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Parental Age As a Factor in Pregnancy Outcome and Child Development

DOROTHY NORTMAN

ABSTRACT A quantification of age patterns of reproductive risk, based on major research studies, shows that there is a reproductive maternal age of minimum risk. As age departs, either on the younger or the older side, from the optimal age, risk rises continuously. Thus, age patterns for various types of reproductive risk usually take the form of U-, J-, or reverse J-shaped curves. These patterns are illustrated for maternal morbidity and mortality, fetal mortality, stillbirths, perinatal mortality, and infant mortality. Although the pattern of risk persists at all levels of risk, absolute risk varies tremendously over space and time and across income classes. A high relative risk at a given age does not necessarily mean a high absolute rate at that age. Maternal mortality may be as much as ten times higher among women in their forties than among women in their twenties, but the rate among older women in a low mortality country may be lower than the rate among young women in a high mortality country. These observations support the hypotheses that biological processes are the chief determinants of the age pattern of reproductive risk and that social, cultural, and economic factors largely determine the degree of risk, whatever the mother's age.

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In general, ranges were trimmed when the number of studies represented in the table was five or more.

A dash indicates that there were too few cases in the sample to warrant calculation of rates.

A rectangle indicates that the index is based on the unweighted average rate of the age groups enclosed in the rectangle equals 100.

Symbol definitions, lettered footnotes, and sources appear at the end of each table. These must be read in conjunction with each table.

NOTES ON THE FIGURES:

In general, absolute rates were plotted on a logarithmic scale and index numbers of rates on an arithmetic scale. When the range of numbers to be plotted was so large as to render an arithmetic scale impractical, a logarithmic scale was used.

Age groups appearing in parentheses indicate that the index is based on the unweighted average rate of those age groups equals 100.

The question whether there are optimal childbearing ages and birth intervals has long commanded the attention of both the medical profession and the public at large. This is a difficult question to answer because pregnancy and its outcome are influenced by all the circumstances of life—cultural practices, genetic disposition, nutrition, health, hygiene, housing, medical care, maternal competence—in short, by the total configuration of biologic, social, and economic circumstances of parents and children. With so many forces operating, the measurement of the influence of any one factor is obviously a complex substantive and statistical pursuit.

The rationale for trying to isolate the influence of parental age on pregnancy outcome is that age differentials persist even as the level of risk introduced by other variables is reduced, and, in many societies, very early and late childbearing are correlated with reproduction under unfavorable circumstances. Also, age per se offers an easily identifiable, readily available, and ethically acceptable handle for coming to grips with the factors in childbearing and pregnancy that are the chief determinants of higher mortality.

It is not well understood why reproductive capacity declines with advancing age even when a woman continues to have regular menstrual periods and is in seemingly good health. However, biological factors appear to play an important role. Certainly they are all-important in determining the end of the reproductive period. Unlike the female of almost all other species, the human female is unique in that the stock of oocytes (immature eggs, which are produced only during fetal life) is virtually exhausted relatively early in life.[1] In contrast to most animals, the human female thus has a long postreproductive period, amounting in the course of the normal female life span to as many years as are spent in the fertile state. (One is tempted to observe that the long postreproductive period provides the human female with a lengthened opportunity to transmit a cultural rather than genetic heritage to the next generation, a point of view that suggests that rational fertility control is compatible with natural development.) There is also evidence that the stock of oocytes may decrease in quality as well as quantity as the women nears the end of her reproductive span. Early spontaneous abortion, often of a genetically defective fetus, increases with age of woman. Hormonal, morphological, and histological changes with age noted in the reproductive and related organs of many animals, including man, indicate a deterioration in reproductive capacity with age. And, although the human male is capable of producing viable germ cells, that is, spermatogenesis, for a much longer part of his life span, the quantity and quality of spermatozoa deteriorate with advancing age (Bishop, 1970).

As further evidence of the significance of biological factors in reproductive capacity, age schedules of fertility of all human populations have several common features. According to Coale, who has studied these extensively:

> All rise smoothly from zero at an age in the teens to a single peak in the twenties or thirties, and then fall continuously to near zero in the forties and to zero not much above age 50. The lowest mean age of fertility I have found is a little less than 26 years (in recent years in Hungary); the highest a little over 33 years (in nineteenth century Sweden). In all but a few fertility schedules, 75% of total fertility occurs within a span of 16 years, and in every case I have examined within a span of 20 (1972, pp. 5–6).

Parental age patterns of reproductive risk (that is, risk of mortality or morbidity to mother or child) also show striking similarities. Indeed it is common knowledge that risk is high at the very young childbearing years, drops to a minimum usually sometime in the twenties, and then rises continuously to the end of the childbearing years. The inverse relationship between capacity to conceive, which is an inverted U with respect to maternal age, and reproductive risk, which is U (or J) shaped with respect to maternal age, supports the a priori proposition that biological processes largely determine the age pattern of reproductive risk.

On the other hand, the tremendous variation in the rate of risk, in space and time and across different social classes, is strong testimony that social and economic factors have a great deal to do with the absolute levels of risk at any age. Age at marriage, proportions married, pre- and extramarital intercourse, incidence of divorce and widowhood,

[1] This is not to say that oocytes are completely exhausted when the ability to conceive ends. Statistical data on oocyte stock at different ages are scarce, but to indicate order of magnitude, the few ovaries examined showed an average supply of 10,900 at ages 39–45 compared with a stock of 733,000 at birth, with a supply at in-between ages to indicate an increasing attrition rate with increasing age. Thus, of the ¾ million immature eggs with which the human female is born, only about 400, one or so a month for about 30 years, ripen for potential fertilization (Talbert, 1968, p. 462).

practice of contraception and abortion, and in some cultures, proscription of intercourse during religious holidays or for a period following parturition strongly modify fecundity. Because of these exogenous factors, while the relative risk with age seems to persist across different classes, there is a wide variance in the absolute level of risk. Maternal mortality, for example, is often five to ten times as great over age 40 as it is among women in their twenties, but in countries of low mortality, even among older women the rates cannot be thought of as "high" in an absolute sense and may even be lower than among younger women in high mortality countries.

Thus, we are led to hypothesize that (1) biology poses a persistent U- or J-shaped irreducible minimum of reproductive risk with respect to age; and (2) the socioeconomic background of the parents is the chief determinant of the level of risk. To the extent that poorer health, lower income, and inadequate medical care coexist with reproduction at the very young and older parental ages—as they do in most societies—the age differentials of risk are sharpened.

This review evaluates, objectively and quantitatively, the age gradient of *relative* risk.

NATURE, SOURCES, AND ORGANIZATION OF THE DATA

Since vital data and research results are usually aggregated into 5-year age groups, for statistical purposes the 30-year period from age 15 to age 44 is generally accepted as the human female reproductive age span. Individuals vary with respect to both the age of menarche (when the menstrual cycle is first established and with it the capacity to conceive) and the age of menopause (when the capacity to conceive virtually terminates). The average reproductive age span also varies among countries and regions. In the West, age of menarche has been declining in the past century, and in the United States, it is currently at about 13 years (National Academy of Sciences, 1970). At the other end, studies of diverse populations at various times have shown considerable proportions of women who had not at age 50 yet reached menopause.[2] Thus, births can and do occur to women before age 15 and after age 44, but the relatively few such births (and pregnancies) are readily accommodated without significant statistical bias by dealing with them as though they had occurred to women aged 15 and 44, respectively.[3]

The tendency in published data to aggregate teenage pregnancies and births into one composite 15–19-year-old age group is unfortunate because biologically this grouping may be a composite of both the poorest and the best time to start childbearing. That is, at age of menarche, having yet to grow and mature themselves, most girls are biologically unprepared to provide adequate nourishment for a growing fetus. On the other hand, by age 18 or 19, the human female may be at or close to her prime physical condition for reproduction. Thus, rates of risk for the composite 15–19-year-old age group are heavily influenced by the distribution of mothers within the group. The general effect of aggregating teenage pregnancies and births into one 15–19 age group is to transform the age gradient risk pattern from what might be a U- to a J-shaped curve.

Similarly, curves representing parity or social-class aggregates may also be the resultant of rather different shapes for individual parities[4] or classes. Thus, for true age patterns to emerge, the distorting effects of a heavy concentration in particular age-parity classes of either high- or low-risk pregnancies should be removed. To do this, the age gradient of risk must relate to a homogeneous population—that is, a population as much alike as possible in all factors, except age, that have an important bearing on pregnancy outcome. Ideally, therefore, the data should be subdivided into socioeconomic subgroups, but cross-classification requires a large sample size for statistically significant findings.

Of the many studies available on the relation between parental age at birth and reproductive outcome, almost all deal with maternal age because of the obviously critical role of the mother in the offspring's pre- and perinatal period, although genetically both parents contribute equally. Limitations of the data, therefore, necessarily restrict the analysis and quantification of relative risk to maternal age but some information on the paternal age risk to the child is also available.

Because of the long-standing interest in the optimal ages of childbearing, the literature on the subject is rich almost to the point of being inexhaustible. For this review, it has been combed, mostly in secondary sources published in English, for

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[2] In the US health interview survey of 1960–1962, for example, of the women who had not had any operative menopause, 41 percent of those aged 50–51 at the time of the survey reported they were not menopausal, but by age 55–56 the figure was down to 4 percent (US HEW, 1966c, pp. 5, 8).

[3] It should be noted that rates computed with denominators of number of females aged 15–19, 40–44, or 15–44 are not adjusted for the number of women who reproduce outside the 15–44 age span. However, the fact that numerator (pregnancies or births) and denominator (number of females per relevant age group) may not be exactly matched does not affect the rates to any appreciable degree. It should also be noted that in some studies, the upper and lower age groups are open-ended, that is, literally under age 20 and aged 40 and over, while in other studies they are closed-ended, that is, aged 15–19, exclusive of ages under 15, and aged 40–44, exclusive of ages 45 and over. The listing of all studies as though these age classes were identical is not considered to affect the findings significantly.

[4] Parity is a term that relates to the mother, representing number of previous births. Birth order is a term that relates to the child, representing the number that the child is in relation to births to the mother. The practice varies with regard to the inclusion or exclusion of stillbirths and abortion.

studies considered sufficiently well designed and executed, and of large enough sample size, to yield statistically meaningful findings on age patterns of pregnancy outcome, controlled for birth order. This rather rigorous statistical standard in the choice of materials was relaxed in two respects: (1) to include some information on developing countries; and (2) to include information on childhood performance in relation to parental age at birth, a topic on which very few data exist. It was also possible to include some important unpublished data for example, infant mortality in the United States by single year of age among teenagers, as well as a few studies published in other languages. Thus the studies are of large populations, based mainly on national or regional vital statistics records, or else they are medical investigations of more limited scope but considered well designed and often conducted with control groups.

A standard technique was used to quantify the findings for studies relating to a particular risk of pregnancy outcome into a maternal age gradient. The materials were organized by risk, and the hypothesis was posed that the relative risk by age remains much the same regardless of the absolute level of risk. Accordingly, the age-specific rates by birth order for each study were the basic data. For each birth order independently, the age-specific rates were converted into index numbers based on an unweighted average (arithmetic mean of a broad age base) for that birth order equal to 100.[5] (The range of age classes was such as to permit, for each risk, the inclusion of all the studies listed.) For each risk, by birth order, the median age-specific index number and the range in index numbers over the various studies were then determined. A least squares parabola was fitted to the medians to portray the hypothesized basic age gradient for that birth order for the risk under discussion. This technique was applicable to all except the last two of the following categories into which the materials are organized:

1. Effects on mother
   a. Maternal mortality
   b. Maternal complications
   c. Female mortality
2. Fetal loss
3. Stillbirth or late fetal deaths
4. Perinatal mortality (stillbirths plus deaths shortly after a live birth, usually within a week)
5. Infant mortality and its components
6. Congenital malformations
7. Childhood development and performance
   a. Physical handicaps
   b. Mental performance
   c. Psychological or behavior patterns

OVERVIEW

The documentary evidence on the relationship between maternal age and pregnancy outcome establishes that risk of mortality or morbidity, to mother or child, is minimal when the mother is neither too young nor too old and when the child is of moderate birth order, not exceeding, say, four. The few studies on paternal age suggest that results are best if mother and father are not too disparate in age, but as a causal factor, father's age may play a role only in some congenital conditions.

It is not surprising that the relationship between risk and maternal age is not random, considering that aging and reproduction are related biological processes. The empirical data show that there is a reproductive maternal age of minimum risk; as age departs, either on the older or younger side, from the optimal age, or age of minimum risk, risk rises continuously.

This is not to imply that it is dangerous for women outside the optimal age range to bear children. As observed in the introduction, levels of absolute risk vary tremendously over space and time and across income classes, indicating that social, cultural, and economic factors largely determine the degree of absolute risk regardless of the mother's age. However, U- and J-shaped age risk curves persist at all levels of absolute risk, supporting the a priori proposition that biological processes are the chief determinants of the age pattern of reproductive risk.

What, then, is the age of minimum risk—of maternal mortality, infant mortality, and other risks in reproduction—and how rapidly does risk increase as age departs from the optimal, when risk is lowest? The answers to these questions, generalized from findings of major studies covering 20–30 countries, on the relationship of maternal age and pregnancy outcome, are summarized in Table 1. The table gives the mathematical expression of the relationship (a parabola, which was found to yield a good fit to the observations) in a form such that the value of c in the equation, $R = a + b(x - c)^2$ is the age of minimum risk and the value of a is the index of risk at age c compared to an average risk of 100 for reproduction in the age span shown as the index base. For example, for maternal mortality, a woman reproducing at the age of minimum risk runs one-fourth (24.7 percent) the average risk for women aged 15–44 in countries where the maternal mortality level is low, one-third (33.3 percent) in countries of moderate mortality level, and a little less than half (47.3 percent) in countries of high maternal mortality. Table 1 also shows (on the basis of the mathematical expression) how much greater the risk is at 5 years, 10

[5] C. Tietze suggested as an index base the unweighted average instead of the overall average, which is a crude rate influenced by the frequency of occurrence per age class. Although the relative age relationship remains the same, the unweighted average is not affected by the age distribution so that findings from study to study are more comparable.

TABLE 1 **Curve of Index of risk (*R*) by maternal age, and percent by which risk is excess over least risk at 5, 10, and 15 years from age of least risk, for specified risks**

Risk	Age span of index base	$R = a + b(x - c)^2$ (Least squares parabola, x in single years of age, origin at 0)[a]: a	b	c (= age of least risk)	χ^2 of data[b]	Percent of excess risk at ages ± 5 years from age of least risk[c]	± 10 years	± 15 years
Maternal mortality								
Low level	15–44	24.7	0.53	21.9	2.8	54	216	486
Moderate level	15–44	33.3	0.46	21.6	1.9	34	137	308
High level	15–44	47.3	0.40	22.9	1.0	21	85	191
Fetal mortality								
All birth orders	20–34	80.5	0.40	21.9	0.1	12	50	113
Birth order								
1	20–34	63.7	0.38	18.0	4.4	15	59	133
2	20–34	77.7	0.53	22.8	0.5	17	69	155
3	20–34	87.3	0.57	25.0	1.0	16	65	147
4	20–34	92.0	0.48	26.0	1.0	13	52	118
Stillbirths								
All birth orders	20–39	71.3	0.33	22.5	0.3	12	47	105
Birth order								
1	20–39	(46.2)	0.15	(12.4)	2.4	8	33	67
2	25–39	59.5	0.44	23.7	2.0	18	73	165
3	25–39	72.4	0.45	26.3	3.0	16	63	141
4	25–39	72.8	0.43	25.5	1.2[d]	15	60	134
Perinatal mortality								
All birth orders	20–34	89.8	0.41	25.2	0.3	11	45	102
Birth order								
1	20–34	84.0	0.18	18.1	2.1	5	21	24
2	20–39	75.5	0.58	26.9	1.0	19	76	171
3 and 4	20–39	84.7	0.51	29.0	3.2	15	61	136
Neonatal mortality								
All birth orders	20–34	96.8	0.26	27.7	0.5	7	26	59
Birth order								
1	20–39	82.1	0.25	23.6	1.9	7	30	67
2	25–39	87.0	0.44	29.4	0.6	13	50	113
3	25–39	89.3	0.50	31.5	1.1	14	55	125
4	25–39	93.5	0.36	33.3	1.2	9	38	85
Infant mortality								
All birth orders	20–34	97.2	0.23	28.6	0.6	6	23	52
Birth order								
1	20–34	96.0	0.35	26.1	1.5	9	36	81
2	20–39	83.4	0.53	31.6	3.6	16	63	142
3	25–39	83.9	0.65	33.5	6.9	19	77	174
4	25–39	86.1	0.55	34.0	1.2	16	64	144

( ) Data outside the 15–44 age span.

[a] Fitted to the medians of the observations that were age-specific rates in the various studies expressed as index numbers based on the unweighted average of the rates in the age span shown in the table. The equation of the parabola is in a form such that the *c* value gives the age of minimum risk and the *a* value is the index of risk at age *c*, based on the average (simple, unweighted) risk of the age span shown = 100.

[b] The parabola is considered a good fit to the observations if the χ^2 of the data is less than $\chi^2_{.05}$ which, with 3 degrees of freedom (6 observations less 3 for the parameters) equals 7.8.

[c] Computed from the least squares parabola.

[d] Involves 2 degrees of freedom (5 observations less 3 for the parameters) for which $\chi^2_{.05} = 6.0$.

years, and 15 years, plus or minus,[6] from the age when the risk is minimal.

The increase in the optimal childbearing age as birth order goes up is striking. Of course, this was to be expected because it has long been known that too frequent, closely spaced reproduction is correlated with higher risk. What is new is the pinpointing, from the best empirical evidence, of the age when risk is minimal, specific for risk and birth order. As can be seen in Table 1, for fetal mortality, for example, risk was found to be lowest at maternal age 18 among mothers having a first birth, compared with age 26 among mothers having a fourth birth. Corresponding infant mortality figures are 26.1 years as the optimum age for first births,[7] 34.0 years for fourth births.

[6] Since the parabola is symmetrical with respect to its minimum point, equal age distances, plus or minus, from the age of minimum risk give equal excess risk. Of course, distances from the optimal age that fall outside the reproductive age span, herein defined as ages 15–44, have no meaning.

[7] Age 26 as the optimum for minimizing infant mortality among first births deserves some comment. It seems high, but no explanatory clue is to be found in the data. The finding merits further research, a plausible hypothesis being that women having their first child in their twenties are in better socioeconomic circumstances or are better equipped than teenagers to protect the child in its later months of infancy against increasingly important environmental—as opposed to biological—hazards.

The data in Table 1 also show that when the mother is five years older or younger than the age corresponding to the lowest risk, the excess risk ranges, from 5 percent (perinatal mortality, birth order 1) to 19 percent (perinatal mortality, birth order 2 and infant mortality, birth order 3). At ten years away from the optimal age, the excess risk ranges from 21 percent to 77 percent, varying by specific risk and birth order. Finally, reproducing 15 years earlier or later than the age of least risk generally more than doubles the minimum risk, whatever its particular absolute level.

The age pattern of maternal mortality was analyzed by grouping countries into three mortality levels: low, moderate, and high.[a] For all three groups, the age of minimum risk is about the same, 22 or 23 years (all birth orders combined). Of interest is the widening of the age differentials as the level of mortality is reduced. To illustrate, ten years from the optimal age, the excess risk is 85 percent for the high mortality countries, but 216 percent for the low mortality countries. The major explanation probably is that, as maternal mortality comes down, constitutional rather than obstetrical factors become increasingly important as a cause of death. Other explanations include the selecting out of higher order births as a factor in the decline in maternal deaths and the possibility that at high mortality levels, only the hardy survive to reproduce later in life.

That certain congenital malformations are positively associated with parental age has long been known. Except for chromosomal defects, however, the relationship is not usually statistically significant. Down's syndrome, also known as mongolism, a genetic defect involving the (nonsex) chromosome number 21, is definitely known to increase with maternal age, and Penrose, a leading investigator of the subject, also implicated advancing paternal age independent of maternal age. Down's syndrome was not included in Table 1 because its rise with maternal age is exponential rather than parabolic, so that the younger the age, the lower the risk. Thus, there is no sensible age of minimum risk. (This also turns out to be the case for stillbirths of birth order 1.)

Again, it is important to stress that wide age differentials do not necessarily mean high absolute levels of risk at ages of high relative risk. For example, among the studies cited, at ages 40–44, rates of malformations of all types found at birth range from 1.4 to 8.2 percent (for a conglomeration of various places and birth orders, listed in Table 11b, p. 41), and the incidence of Down's syndrome among births to women aged 40–44 is generally less than 1 percent. Clearly, in the absence of a personal history to the contrary, older women run only a small risk of producing a congenitally malformed child, although their risk is much higher than that faced by younger women.

Studies relating parental age at birth with performance or physical condition after infancy are understandably limited in number. A primary difficulty in the research design of such studies is to account for nonsurviving children. Studies of well-selected samples, with properly matched control groups, suggest that children who were born of difficult deliveries or were of low birth weight or premature are more likely than others to show some handicap. Since maternal age is related in U- and J-shaped patterns to complications of pregnancy and delivery, and teenage mothers have a much higher than average proportion of premature and low birth weight babies, age is implicated, if not etiologically, then as an intermediate variable, in the risk of handicap. On the other hand, some evidence suggests that surviving children of older mothers may have higher than average intelligence. Here, however, socioeconomic factors may be operating, since, controlled for birth order, the older the mother the more favorable the parental background may be. In any case, the evidence is mixed and the selecting out of dead children from most child population studies renders tentative the findings on relationship between parental age and intellectual performance.

The best prescription for reducing reproductive mortality and morbidity in the world is to improve living conditions and access to competent medical care. Not only is this a time-consuming process at best, but it is one over which the individual has little control. On the other hand, by voluntarily restricting the ages of childbearing and by spacing births, individual couples can insure for themselves the minimum risk prevalent in their societies during their childbearing years.

A few calculations made along these lines indicate the orders of magnitude by which mortality and morbidity could be reduced by restricted reproduction. If women had births only in the age interval 20–34, maternal mortality would come down by 19 percent in Mexico, Thailand, Venezuela, and the United States; by 23 percent in Colombia and France; and by 25 percent in the Philippines. Wright (1972) found that if women in the United States had their first child not before age 20 or after age 30, a second or third child only between ages 25 and 34, and no further births, infant mortality would be reduced by 29 percent. Halevi (1967) made the point that congenital mal-

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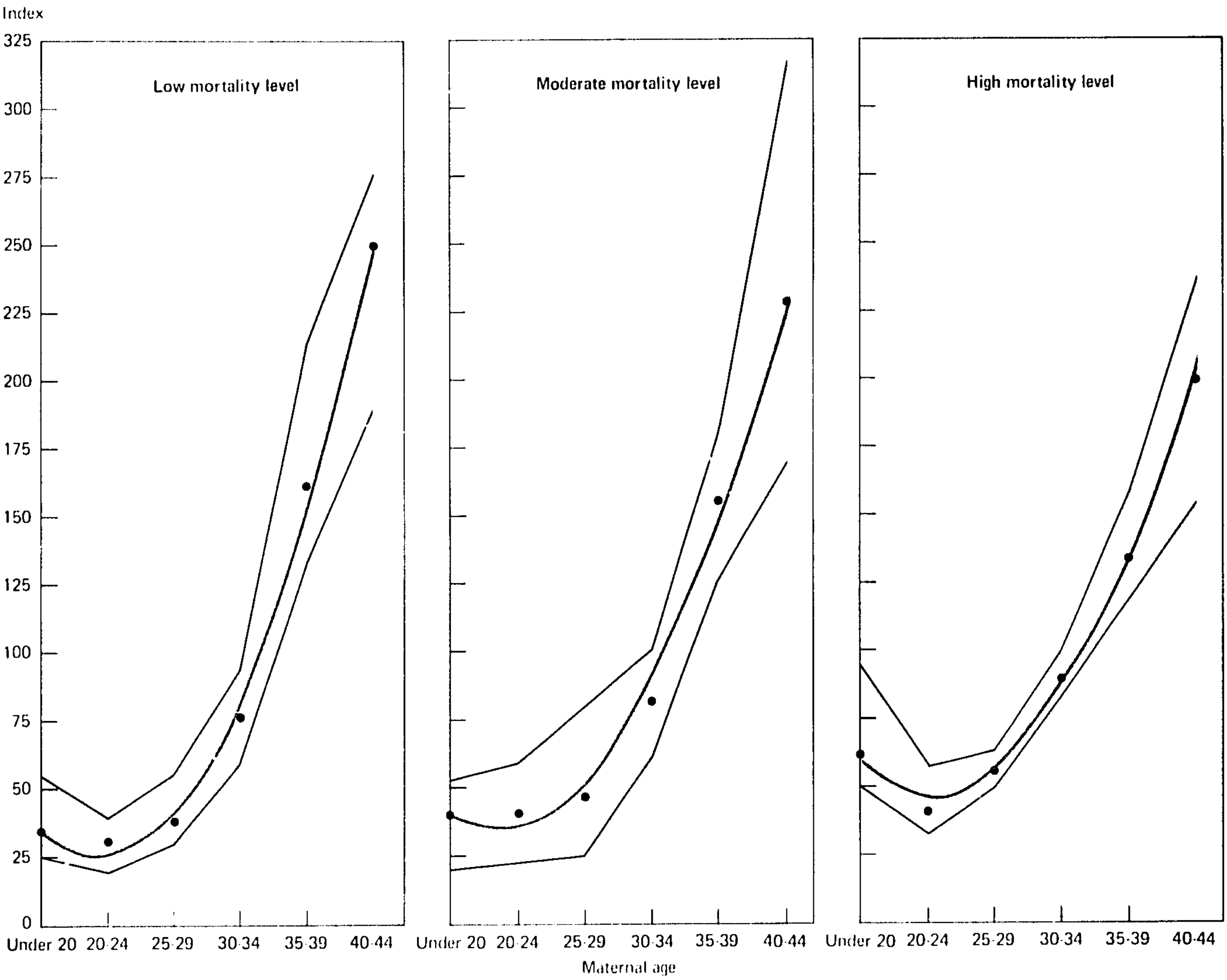
[a] Low mortality was defined as under 35 deaths per 100,000 live births; moderate as a rate between 35 and 100; and high as a rate over 100. Countries in the low mortality group are Australia, Belgium, Canada, Czechoslovakia, Denmark, France, Ireland, Netherlands, New Zealand, Norway, Poland, Sweden, United Kingdom, United States; in the moderate group, Austria, Bulgaria, Ceylon, Germany, Greece, Hungary, Italy, Japan, Mauritius, Portugal, Puerto Rico, Singapore, Spain, Switzerland, Venezuela, Yugoslavia; and in the high group, Chile, Colombia, Costa Rica, Dominican Republic, El Salvador, Guatemala, Mexico, Panama, Philippines, Thailand, Trinidad and Tobago.

formations, largely associated with late childbearing, have an impact not only on mortality, but also on health needs. He noted that in Israel in 1962, congenital malformation cases accounted for 195 per 10,000 hospitalization days. A consideration of the demographic impact of limiting reproduction to maternal ages 20–34 suggests that, for the short run at least, world population growth would decline from its present level of 2 percent per year to about 1.2 percent. The difference is that the population would double in 58 years instead of 35.

Details of the studies are given in their respective sections, organized by risk, but Figures 1–7 depict graphically for each risk the range among the studies in age-specific index numbers, the median index, and the smooth curve.

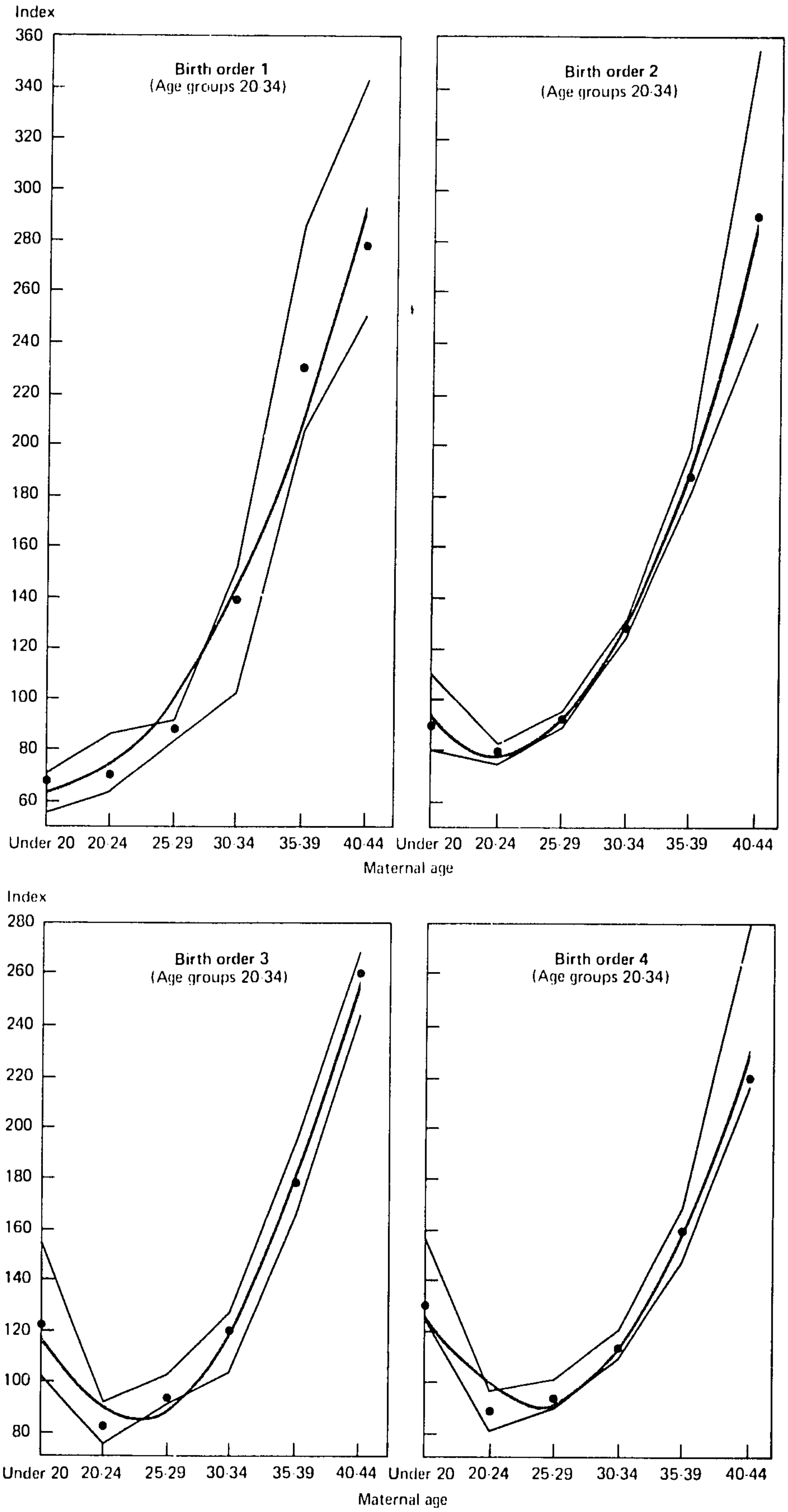
FIGURE 1 **Maternal mortality: Index by maternal age, for selected countries, 1964–66**
(Index based on unweighted average rate = 100)



NOTE The band represents the range, the dots the median, and the line through the band is the smooth pattern fitted to the median. Low mortality level is based on 14 countries with median unweighted average death rate of 40 per 100,000 live births. Moderate mortality level is based on 16 countries with median unweighted average death rate of 87 per 100,000 live births. High mortality level is based on 11 countries with median unweighted average death rate of 216 per 100,000 live births.

SOURCE Table 2.

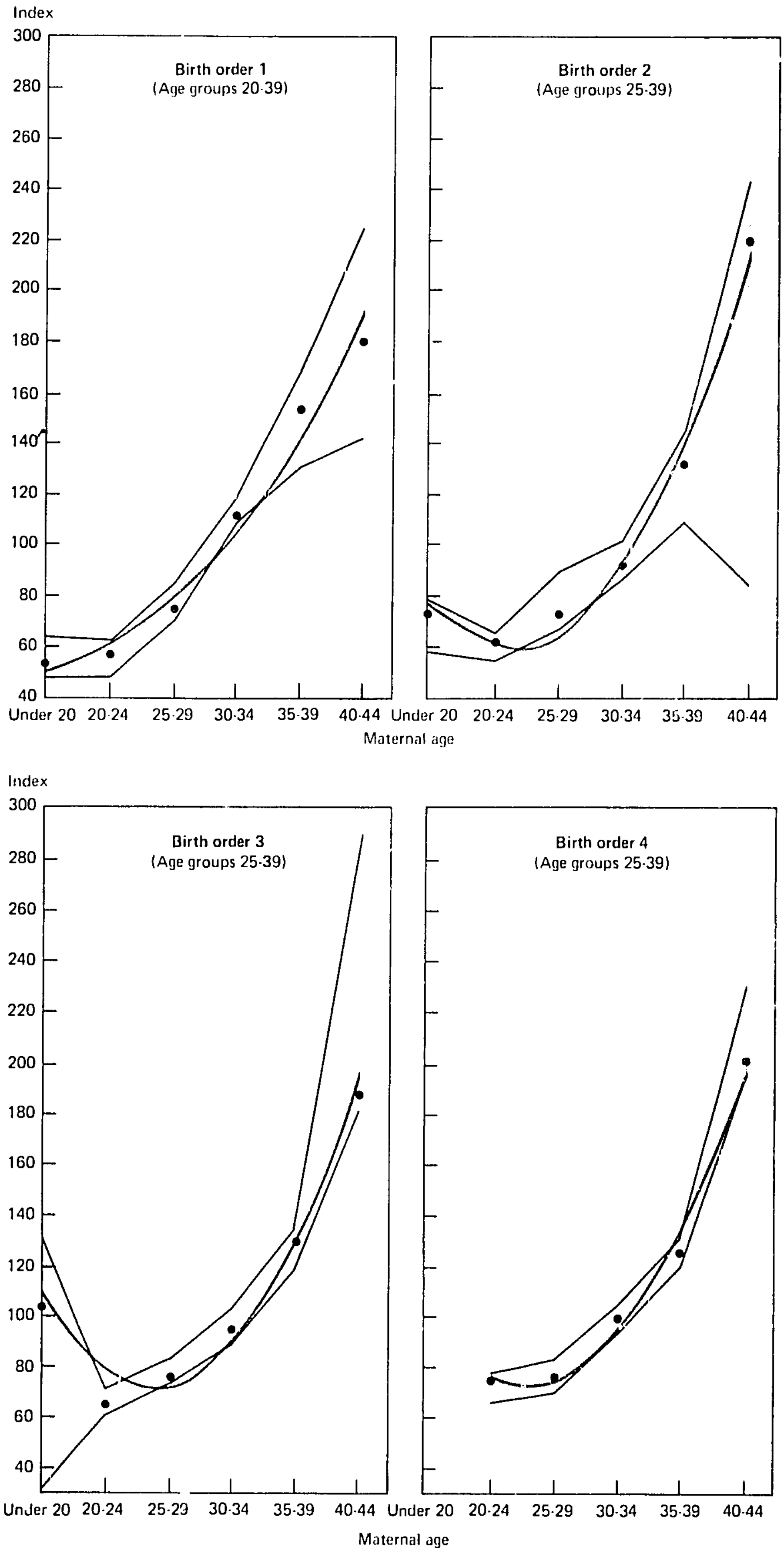
FIGURE 2 **Fetal mortality: Index by maternal age, for selected birth orders, areas, and years**
(Index based on unweighted average rate of age groups in parentheses = 100)



NOTE: The band represents the range, the dots the median, and the line through the band is the smooth pattern fitted to the median.

SOURCE Table 3.

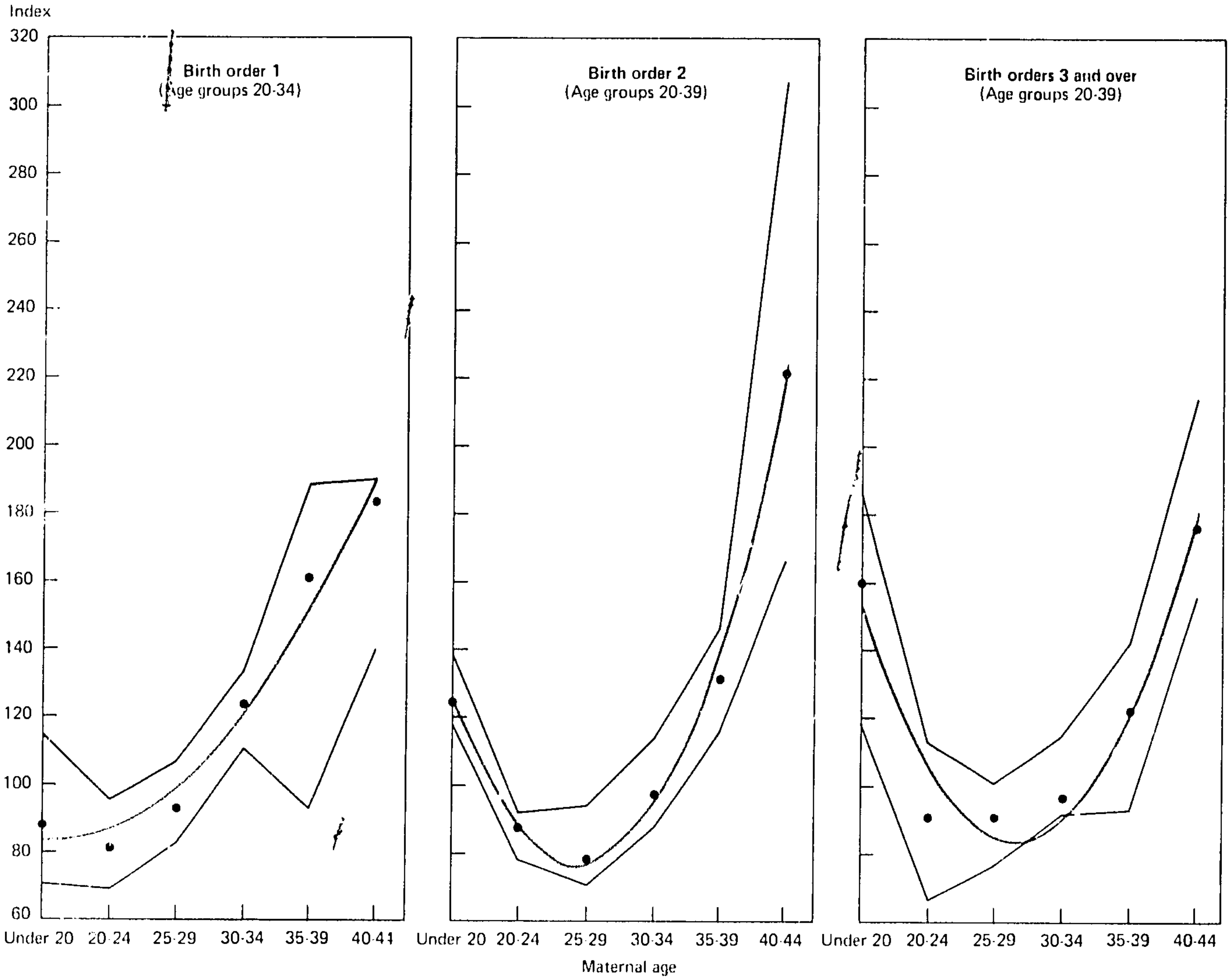
FIGURE 3 **Stillbirth: Index by maternal age, for selected birth orders, areas, and years**
(Index based on unweighted average rate of age groups in parentheses = 100)



NOTE: The band represents the range, the dots the median, and the line through the band is the smooth pattern fitted to the median.

SOURCE: Table 4.

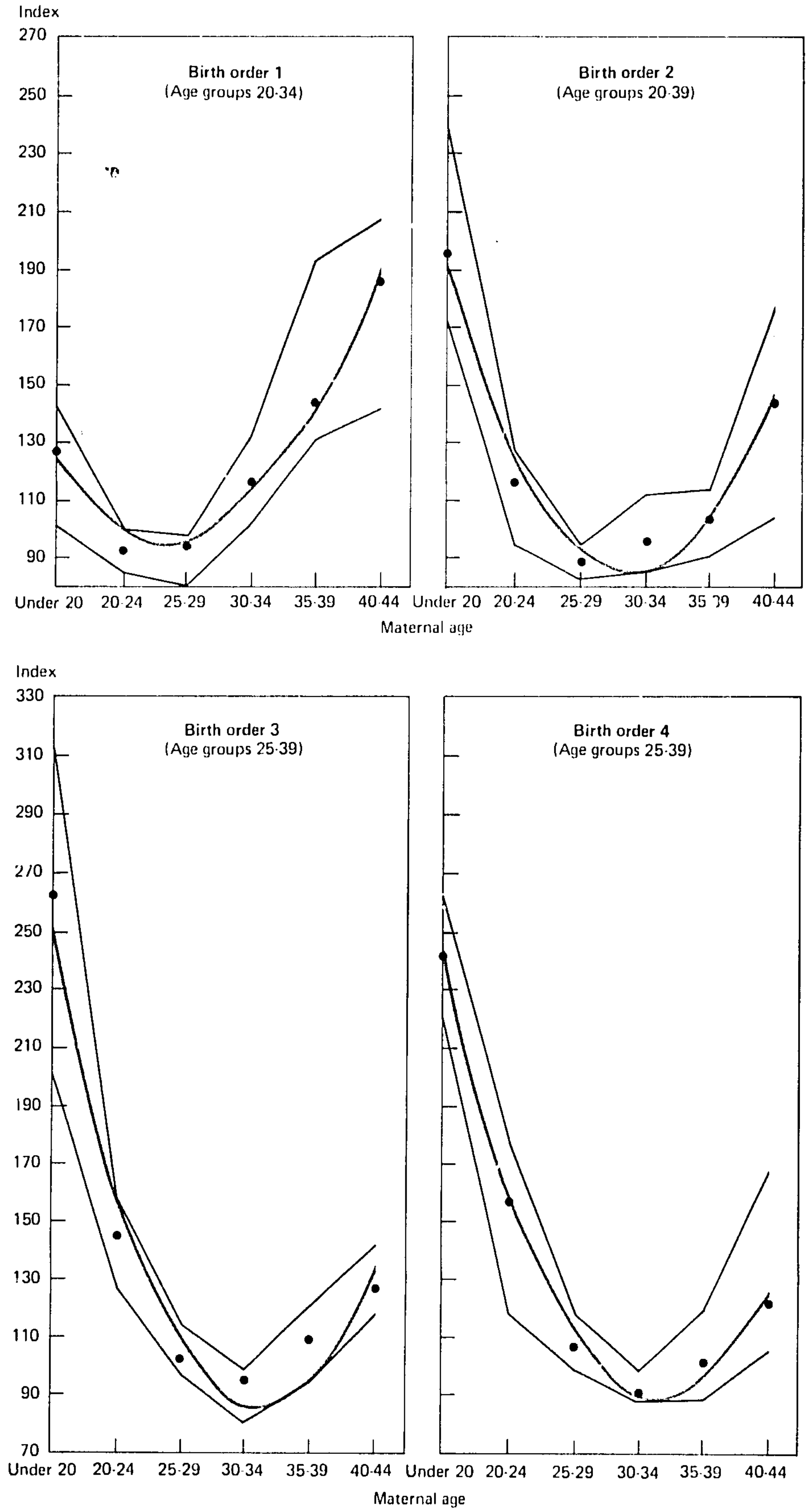
FIGURE 4 **Perinatal mortality: Index by maternal age, for selected birth orders, areas, and years**
(Index based on unweighted average rate of age groups in parentheses = 100)



NOTE The band represents the range, the dots the median, and the line through the band is the smooth pattern fitted to the median.

SOURCE Table 5.

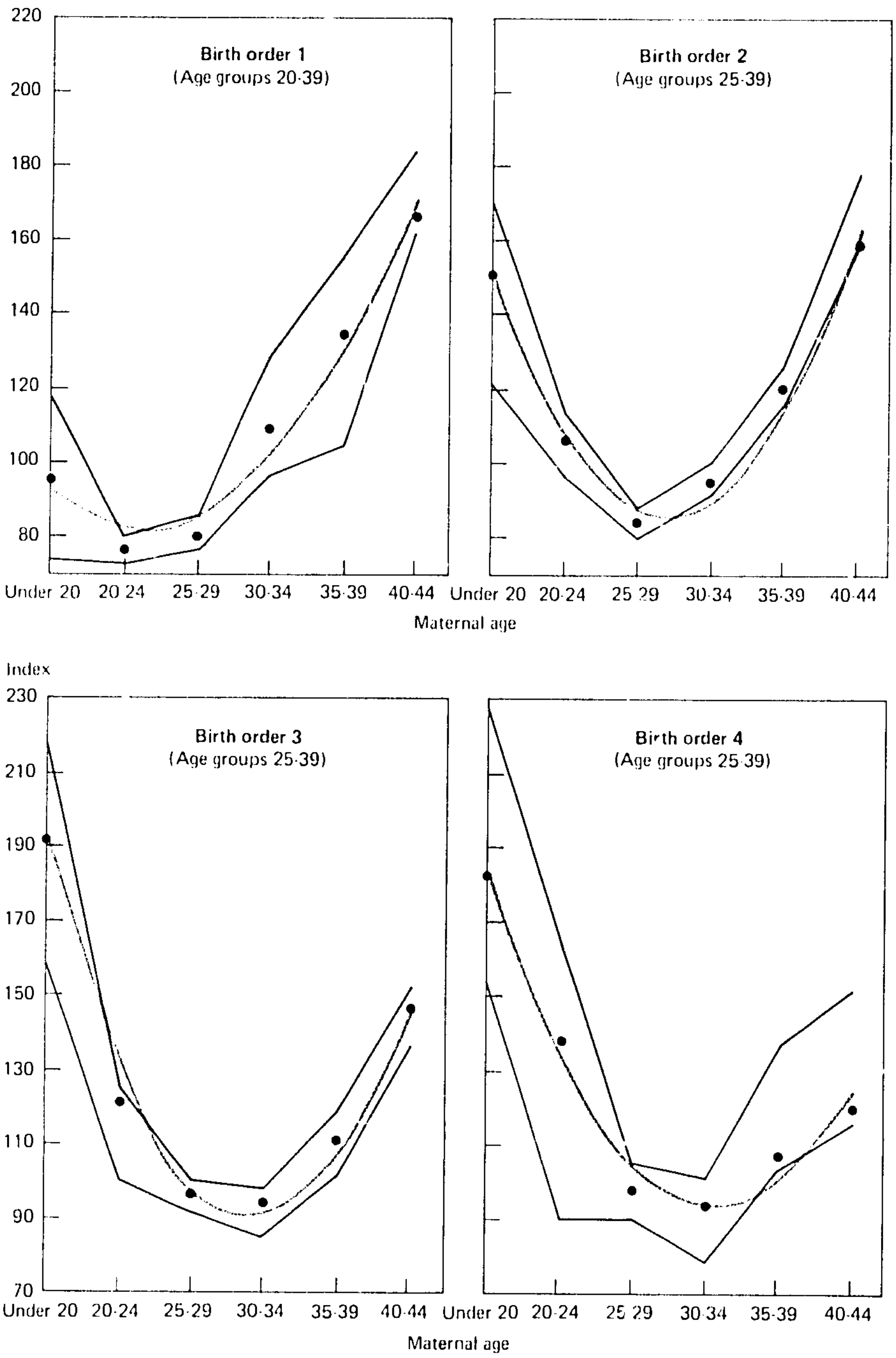
FIGURE 5 **Infant mortality: Index by maternal age, for selected birth orders, areas, and years**
(Index based on unweighted average rate of age groups in parentheses = 100)



NOTE: The band represents the range, the dots the median, and the line through the band is the smooth pattern fitted to the median.

SOURCE: Table 7.

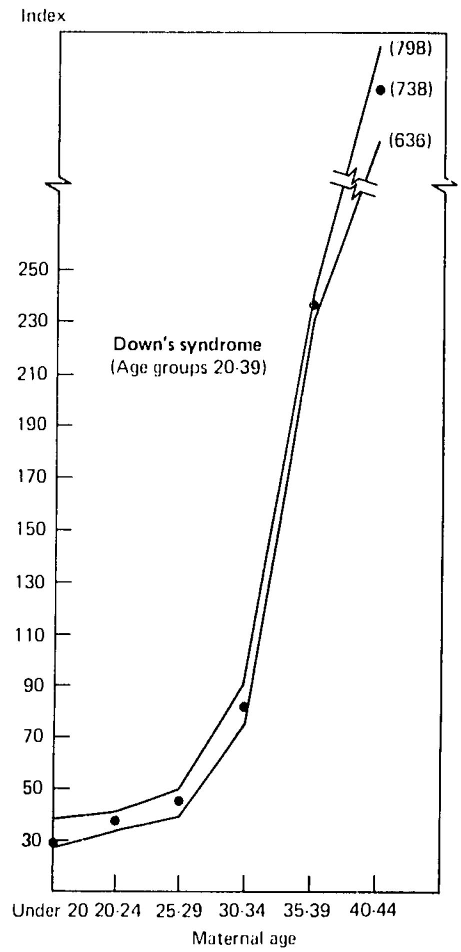
FIGURE 6 **Neonatal mortality: Index by maternal age, for selected birth orders, areas, and years**
(Index based on unweighted average rate of age groups in parentheses = 100)



NOTE The band represents the range, the dots the median, and the line through the band is the smooth pattern fitted to the median.

SOURCE Table 8.

FIGURE 7 **Down's syndrome: Index by maternal age, for selected areas and years**
(Index based on unweighted average rate of age groups 20–39 = 100)



NOTE The band represents the range and the dots the median.

SOURCE Table 11a.

EFFECTS ON MOTHER

Maternal Mortality

Most causes of maternal mortality and morbidity are necessarily related to the changes a woman undergoes during the course of pregnancy, delivery, and the puerperium (the period right after childbirth). According to an obstetrical authority:

> Normal pregnancy is characterized by profound hemodynamic, metabolic, and hormonal changes and a substantial increase in body fluids. Whether preeclampsia represents an exaggeration of changes normally incident to pregnancy or depends upon some wholly new aberration is moot. The causes of preeclampsia, eclampsia, and essential hypertension are unknown, despite decades of intensive research, and they remain among the most important unsolved problems in obstetrics (Hellman and Pritchard, 1971, p. 685).

Major complications are generally divided into two areas: (1) those that are essentially obstetrical in nature; and (2) those due to constitutional illnesses, which, although perhaps exacerbated by the pregnancy, are in any case likely to produce stress and hasten death. Obstetrical complications are broadly classified in three large groups: preeclampsia and eclampsia (toxemia), hemorrhage, and sepsis or infection.

It is still debatable whether the toxemias are a single entity or two or more related but nonidentical entities. An uncomplicated preeclampsia may appear suddenly late in pregnancy and disappear completely after delivery, a type most common in the primigravida. In the other entity, a preeclampsia-like symptom-complex, with an increased level of blood pressure, albuminuria, and edema, is superimposed on a preexisting hypertension. This type, as might be expected, is most common in the older age group, when vascular disease itself is more frequent.

The causes of hemorrhage are miscellaneous. Before delivery, it may result from placenta praevia, a condition characterized by a low situation of the placenta, obstructing passage of the fetus, or from so-called premature separation of the normally situated placenta. After delivery, there may be postpartum hemorrhage from delayed or incomplete expulsion of the placenta or from failure of the uterus to contract firmly enough, conditions probably more frequent in the older and more parous woman. In different categories, but also important as causes of hemorrhage, are abortion and ectopic pregnancy.

Sepsis, the third of the triad, remains an important cause of maternal mortality. The proximate cause is, of course, the introduction of pathogenic organisms, which are more likely under unsanitary surroundings or with untrained or careless personnel. It is, however, predisposed to by any complications that traumatize tissues, the most important being prolonged and obstructed labor and damaging methods of delivery. There is again a special group of cases in which sepsis is associated with abortion. It is difficult to relate age as a predisposing factor to the heterogeneous causes of sepsis.

The constitutional causes of complications in pregnancy consist of the total list of illnesses to which a woman may be subject during the reproductive years of life. With the exception of tuberculosis and one or two others, the majority of such complications are more frequent in the older age group. Of these, the cardiovascular diseases are the most important. As the specific complications of pregnancy are controlled through improved obstetrical care, the constitutional, nonobstetrical disorders may emerge as the principal causes of maternal deaths. Under these circumstances, older women are certainly at greater risk during pregnancy, although with good medical care the absolute level of risk is considerably reduced.[9]

In Figure 8, US 1967 rates illustrate the relation of age and maternal mortality by cause of death, among whites and nonwhites. For all age groups combined, the major triad—sepsis, toxemia, and hemorrhage—accounted for 46 percent of total deaths, with abortion providing an additional 16 percent. (In the under 20 age group, abortion accounted for 21 percent of all deaths.) It may be worth noting that, except for ages under 20 for sepsis, the nonwhite rates exceed the white rates for all three conditions. With sepsis and hemorrhage, the differential may be due to less or poorer care among nonwhites; with toxemia, it is perhaps related to a higher frequency of hypertension among nonwhites.

Of the major triad of causes of death, the risk of hemorrhage rises sharply with age; the toxemias are a greater risk under age 20 than in the twenties, after which decade the risk rises rapidly; and the risk of sepsis is J shaped among whites, zig-zag but upward by age among nonwhites. For all causes combined, the increase of death rates with age is clearly evident, at a very uniform average rate of 12 percent per year among whites from their low point at ages 20–24 and 10 percent per year among nonwhites from their lowest level found in the under 20 age group[10] (see Table 2 for age-specific index numbers).

Control of hemorrhage and sepsis in Western countries in the past generation has contributed to dramatic declines in maternal deaths at all ages. Indeed, the order of magnitude of the decline can give the il-

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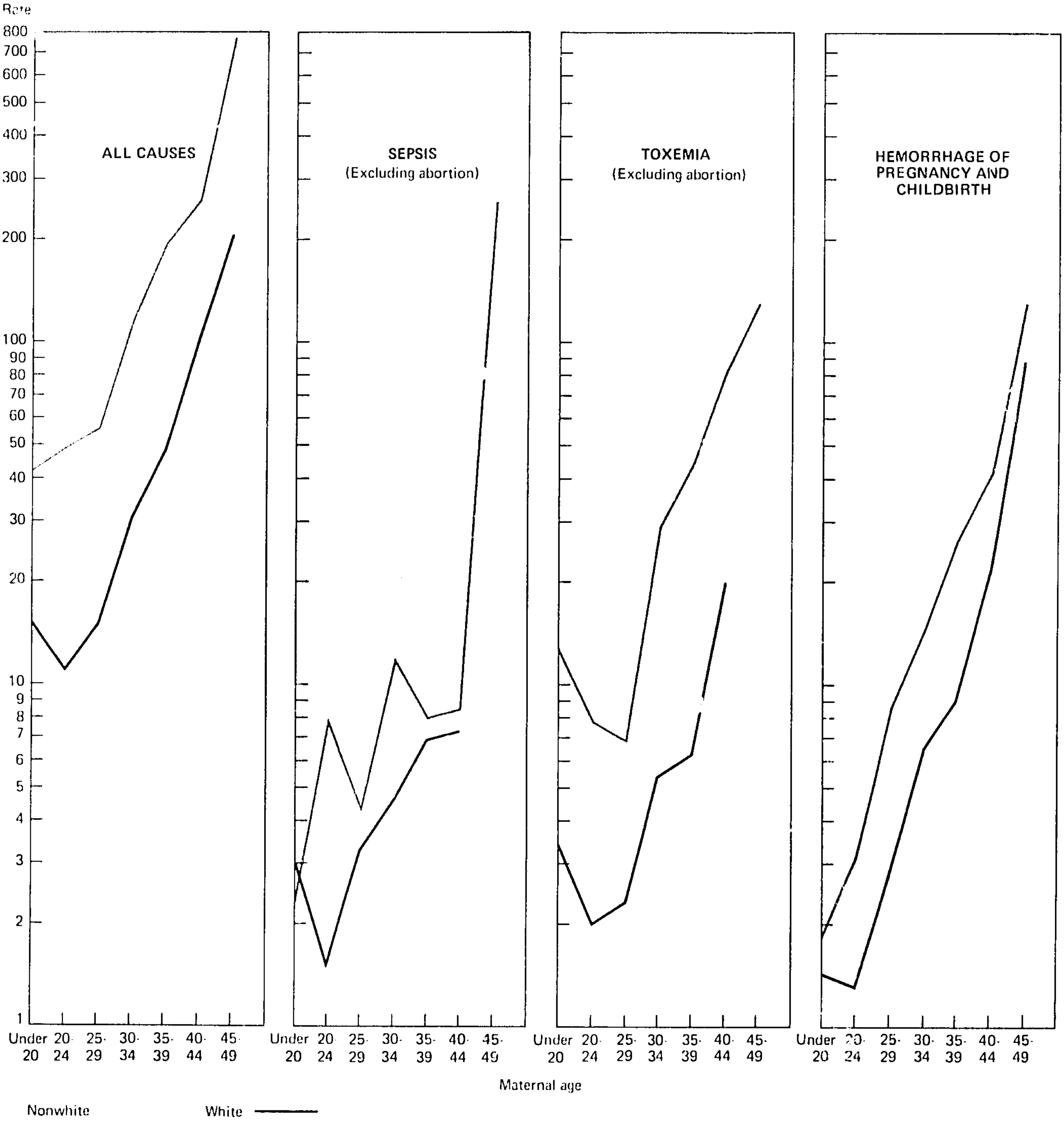
[9] H. C. Taylor, personal memorandum, n.d.

[10] Computed by fitting a linear least squares line to the logarithm of the US rates depicted in Figure 8.

FIGURE 8 **Maternal mortality: Rate per 100,000 live births by maternal age, for specified cause of death, by race, United States, 1967**
(Logarithmic scale)



SOURCE US HEW, 1967d, Vol. 2, Part A.

lusion that maternal mortality has all but been conquered. In the United States, for example, the 1972 rate of 24 maternal deaths per 100,000 live births is a decline of 94 percent from the 1940 rate of 376, of 71 percent from the 1950 rate of 83.3, and of 35 percent from the 1960 rate of 37.1 (US HEW, 1967d, Table 1-16; 1973b, p. 4).

In the underdeveloped countries, on the other hand, where home de-

TABLE 2 Maternal mortality: Rate per 100,000 live births, and index number by maternal age, for selected areas, years, and birth orders

(Index based on unweighted average rate of age groups enclosed in rectangle = 100)

Area, years, and birth order	Average rate: Weighted	Average rate: Un-weighted	Index number for maternal age group: Under 20	20–24	25–29	30–34	35–39	40–44
1964–66[a]								
Low mortality level (14 countries)								
Median	26	40	33	31	39	77	162	248
Range (trimmed)[b]	13-34	22-53	24-55	20-40	27-55	60-95	136-215	192-274
Moderate mortality level (16 countries)								
Median	55	87	39	39	47	81	156	230
Range (trimmed)[b]	38-96	52-150	19-53	23-58	26-79	61-99	127-184	169-319
High mortality level (11 countries)								
Median	156	216	62	42	55	89	134	201
Range (trimmed)[b]	105-219	135-316	48-96	34-59	49-64	84-101	120-157	155-236
United States, 1967								
White	20	37	40	29	40	83	130	278
Other	69	118	35	41	47	96	162	219
Latin American cities, 1962–64								
Cali	138	223	30		51		219	
Caracas	52	71	32		93		175	
Lima	135	194	33		72		195	
Santiago	150	204	34		71		195	
New York State (except NYC), USA, 1936–38	269	354	44	47	66	93	141	211
Birth order								
1	282	598	28	36	45	95	160	292
2	198	292	38	41	69	107	124	107
3	185	229	–	24	102	90	108	195
4 and 5	297	299	–	62	53	75	172	324
6 and 7	379	340	–	–	99	71	130	231
8 and over	551	500	–	–	96	85	119	126

[a] The data exclude abortion for which rates were computed by deflating age-specific rates for all causes (World Health Organization, 1969, Table 3) by the proportion of absolute deaths due to abortion to absolute deaths due to all causes (World Health Organization, 1969, Table 2). Low mortality was defined as under 35 deaths per 100,000 live births; moderate as a rate between 35 and 100; and high as a rate over 100. Countries in the low mortality group are Australia, Belgium, Canada, Czechoslovakia, Denmark, France, Ireland, Netherlands, New Zealand, Norway, Poland, Sweden, United Kingdom, United States; in the moderate group, Austria, Bulgaria, Ceylon, Germany, Greece, Hungary, Italy, Japan, Mauritius, Portugal, Puerto Rico, Singapore, Spain, Switzerland, Venezuela, Yugoslavia; and in the high group, Chile, Colombia, Costa Rica, Dominican Republic, El Salvador, Guatemala, Mexico, Panama, Philippines, Thailand, Trinidad and Tobago.

[b] The range shown excludes the extreme value at each end.

SOURCES:

1964–66: World Health Organization, 1969, Tables 2, 3.

United States: US HEW, 1967d.

Latin America: Puffer and Griffin, 1967, p. 172.

New York State: Yerushalmy, Palmer, and Kramer, 1940.

liveries attended by untrained midwives are common, hemorrhage and sepsis still take a high toll. Worldwide differentials are thus sharp, exceeding a factor of 20, ranging from over 300 maternal deaths per 100,000 live births at the upper extreme[11] down to under 15 in Denmark and Sweden, for example.

Important as it is to reduce death rates everywhere to their biological minimum, our concern here is to investigate the differences and similarities in the age pattern at various mortality levels. Maternal mortality statistics, by age, parity, or cause, are quite readily available for many countries (but rarely cross-classified), probably because the event is of universal interest, highly visible, quite clear regarding immediate cause of death, and, in Western societies, almost always noted by a medical attendant.[12]

A listing by the World Health Organization (1969) of maternal death rates, by age, in 38 countries during 1964–1966 provides suitable data for an age pattern analysis at various mortality levels. The countries were subdivided into three mortality groups of almost equal size by defining low mortality as under 35 deaths (excluding abortion) per 100,000 live births, moderate as a

[11] Rates of 700 deaths per 100,000 live births are not unknown in certain localities.

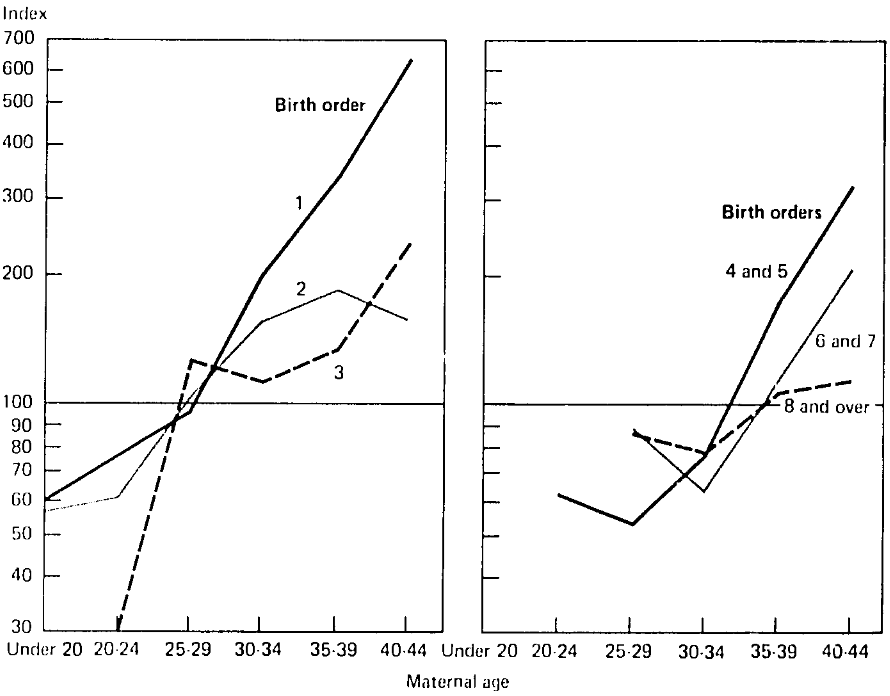
[12] This is not to say that data are of universally high quality. The birth data necessary for denominators in the calculation of maternal mortality rates are often of poor quality in many developing areas.

rate between 35 and 100, and high as a rate in excess of 100. (The latter category consisted mainly of Latin American countries, the other two, mainly Western.)

In accordance with the technique adopted for the analysis, age-specific rates per country were converted into index numbers (based on the unweighted average equals 100). At each mortality level—high, medium, and low—the chain of median index numbers per age class of the countries in the group is hypothesized to represent the age pattern of maternal mortality. These medians and the upper and lower bounds of the range to which they relate are given and depicted in Table 2 and Figure 1.[13] The J-shaped pattern is abundantly clear, the stem rising steeply (exponentially). The similarity in age patterns among countries is indicated by the narrow range (at most ages) within each mortality group and by the shape of the J's among the three mortality groups.

At all three levels, rates tend to be higher before age 20 than in the early twenties, after which age the rate shoots up, more sharply the lower the mortality level. That is, as the mortality level declines, the age differentials become more pronounced. The major explanation for this differential decline by age is probably that, as rates come down, constitutional rather than obstetrical causes of pregnancy complications become increasingly important. Other explanations may lie in the selecting out of higher order births as a factor in the death rate decline and in the possibility that at high mortality levels, poor risks are weeded out earlier in their reproductive careers, leaving a hardier cohort to reproduce later in life. Whatever the reasons, the data suggest that older women face a risk six times greater than that of women in

FIGURE 9 **Maternal mortality: Index by maternal age, for selected birth orders, New York State (excluding NYC), 1936–38**
(Index based on unweighted average rate = 100) (Logarithmic scale)



SOURCE Yerushalmy, Palmer, and Kramer, 1940, pp. 1195-1220.

their early twenties where maternal mortality is low and three times greater, where mortality is high.

Within a country, a cross-sectional examination of maternal mortality by race in the United States in 1967 reveals a shocking disparity between whites and other races, by a factor of three or four, age for age. This is a major social problem, but as far as the age pattern is concerned, as can be seen in Table 2, both groups show the typical pattern of increasing rates with advancing age.

The same pattern emerges in the urban survey of maternal deaths in four Latin American cities in 1962–1964. Although the overall rate in Caracas was about one-third the level in Cali, Lima, and Santiago, all four exhibited similar age patterns, the typical J-shaped curve (see Table 2).

Current data cross-classifying age-specific maternal mortality rates by other potentially important factors, birth order, for example, are not readily available, perhaps because few women now die from the complications of pregnancy and delivery in countries with good vital statistics. If the age pattern is relatively independent of level, however, recency of data is not important. Hence, the study, which controlled for birth order, based on all births in New York State (outside New York City) in 1936–1938 is relevant (Yerushalmy, Palmer, and Kramer, 1940). As can be seen in Figure 9, maternal mortality rises with age for each birth order, and strikingly so for birth order 1. It is to be noted that with increasing birth order, the age group with the lowest maternal mortality shifts to a higher age group [from the under 20 age group[14] for orders 1 and 2 to the

[13] The figure also shows the smooth curve (parabola, the equation of which is given in Table 1) fitted to the medians.

[14] In 1936–1938, the incidence of illegitimacy, with its high adverse effects, was considerably lower than today.

20–24 age group for order 3, the 25–29 age group for orders 4 and 5 (combined), and the 30–34 age group for the higher orders], attesting to the importance of adequate spacing between births. From the lowest rate for each birth order, the increase with increasing age is steep, and yet we have reason to believe from the previous discussion that, since 1936–1938, the age differentials in maternal mortality have widened, not narrowed. From this it can be inferred that the change in pattern of maternal mortality rates by age, with birth order constant, between 1936–1938 and the present was in the direction of an even more startling age differential than is depicted in Figure 9.

A calculation made for several countries by applying their 1964–1966 age-specific mortality rates[15] to the distribution of births by maternal age[16] showed that if births occurred to women only in the age interval 20–34, maternal mortality would be reduced by 19 percent in Mexico, Thailand, Venezuela, and the United States; 23 percent in Colombia and France; and 25 percent in the Philippines. This is not to say that the absolute savings of lives would be large or that the absolute risk of dying from childbirth in the early or late childbearing years is high. With access to competent medical care, under modern medicine, the probability of surviving is good at all ages, especially in the developed countries, but it is best in the age span around 20–34.

Maternal Complications

In contrast to the wealth of material on maternal mortality, few large-scale studies are available on maternal complications by age. The most comprehensive, perhaps, is the medically supervised US Obstetrical Statistical Cooperative study, in which cooperating hospitals in major establishments throughout the United States reported on almost half a million pregnancies and deliveries handled during 1960–1969. The complication rates,[17] standardized within each age and parity category for color, education of the woman, and private versus ward service (as an economic measure), are presented in Figure 10, parts A–E, each part representing a different complication.

Complications, of course, are produced by the same factors that, in dire outcome, lead to death. Based on the US compilation, the increase in the proportion of women with complications as age increases is particularly striking for toxemia (Figure 10A) and, for parities 0 and 1, for both pregnancy complications excluding toxemia and anemia (Figure 10C), and delivery complications (Figure 10D). For toxemia, rates among older women are two to three times greater than among younger women, depending upon parity, with a range in absolute level of 3.1 percent for women aged 20–24, parity 2, to 13.5 percent for women aged 40–44, parity 3, and 14 percent for women aged 35–39, parity 0.[18] Anemia (Figure 10B) is more a hazard for younger mothers, teenagers particularly—a finding perhaps of even greater significance in developing than developed countries. Older women, regardless of parity, are subject to higher risk of complications at delivery (Figure 10D). The relation of age to risk of complications during the puerperium is rather flat except for the increase with age at para 0 (Figure 10E).

Placenta previa rates from the obstetrical cooperative also showed a marked increase with maternal age, from 1.9 to 7.0 per 100 births among primiparous women in age groups 15–24 and 35 or more, respectively, and for multiparous women of the same two age groups, from 2.7 to 10.4, respectively. According to Hellman, "Women over the age of 35 are about three and a half times more likely to have placenta previa than those under 25, regardless of parity" (Hellman and Pritchard, 1971, pp. 611–612).

Data on the incidence of preeclampsia (elevated diastolic blood pressure) by maternal age are available from a remarkable study based on all births in Great Britain during the week of 3–9 March 1958. The original study was concerned with perinatal mortality, but the wealth of information produced in this investigation of a total population of births warranted analysis of other factors. Accordingly, a second report (Butler and Alberman, 1969) followed the first (Butler and Bonham, 1963). The second report found that "the incidence of preeclampsia of all degrees of severity increased with age *despite the exclusion of mothers known to have had hypertension in the first half of pregnancy*" (Butler and Alberman, 1969, p. 38, italics added by author). As shown in Figure 11, 28 percent of multiparous women aged 35 and over developed some degree of preeclampsia compared with 17 percent of women under age 25.

A comprehensive study of complications to the older primigravida was reported on by Kane (1967), based on 36,452 first-time pregnant women of age 25 or more, a sample culled from the over 700,000 births in US hospitals followed over several years. Except for anemia, complications of all types mentioned invariably showed a marked increase in incidence with increasing age. For example, abnormal fetal presentation was 18 percent among women aged 25–29, but 31 percent among

[15] Given in the World Health Organization report (1969) on which the index numbers in Table 2 are based.

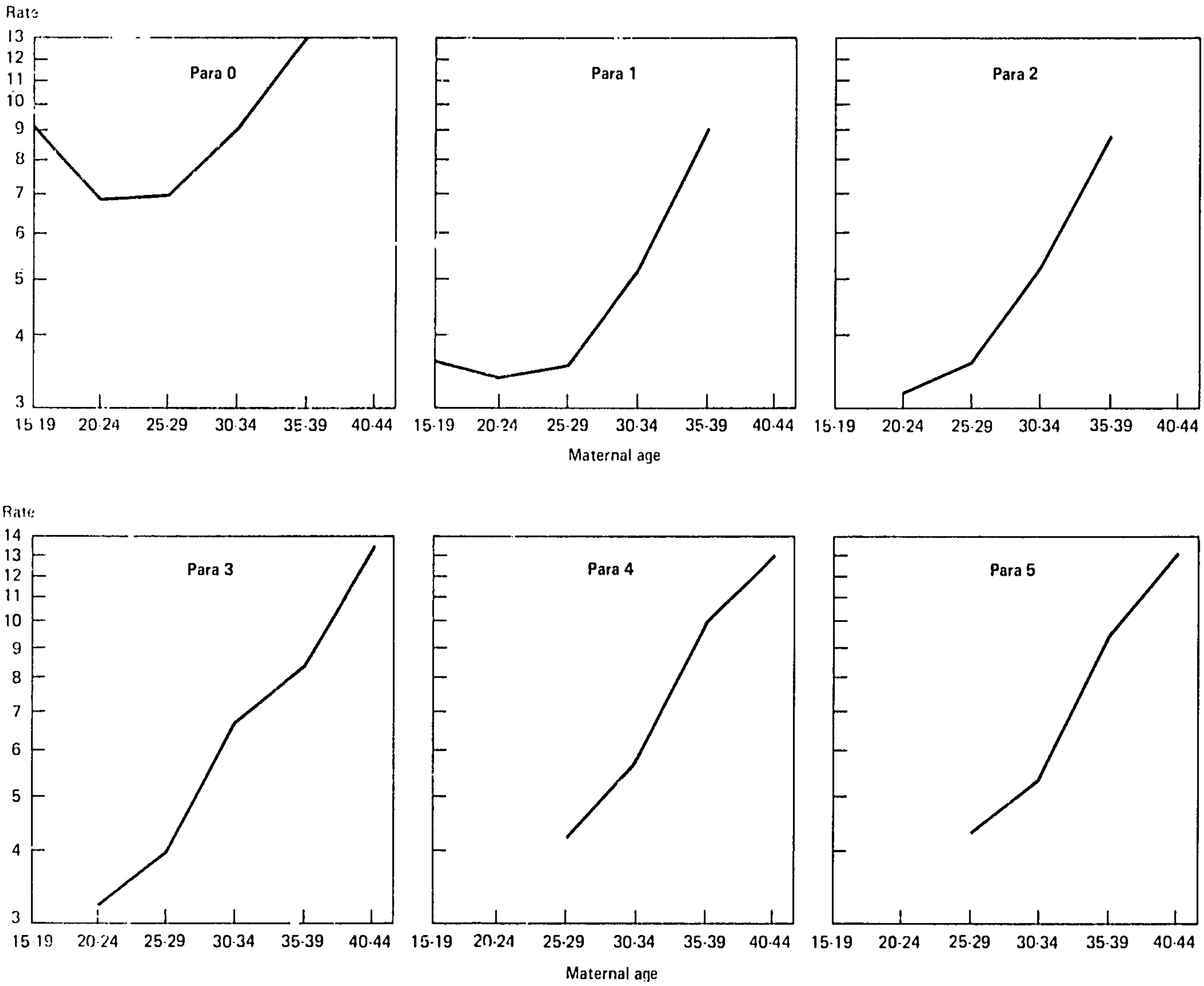
[16] Based on United Nations, 1971, Table 14.

[17] A special tabulation was prepared for the Population Council from the records maintained at the Downstate Medical Center, Brooklyn, New York.

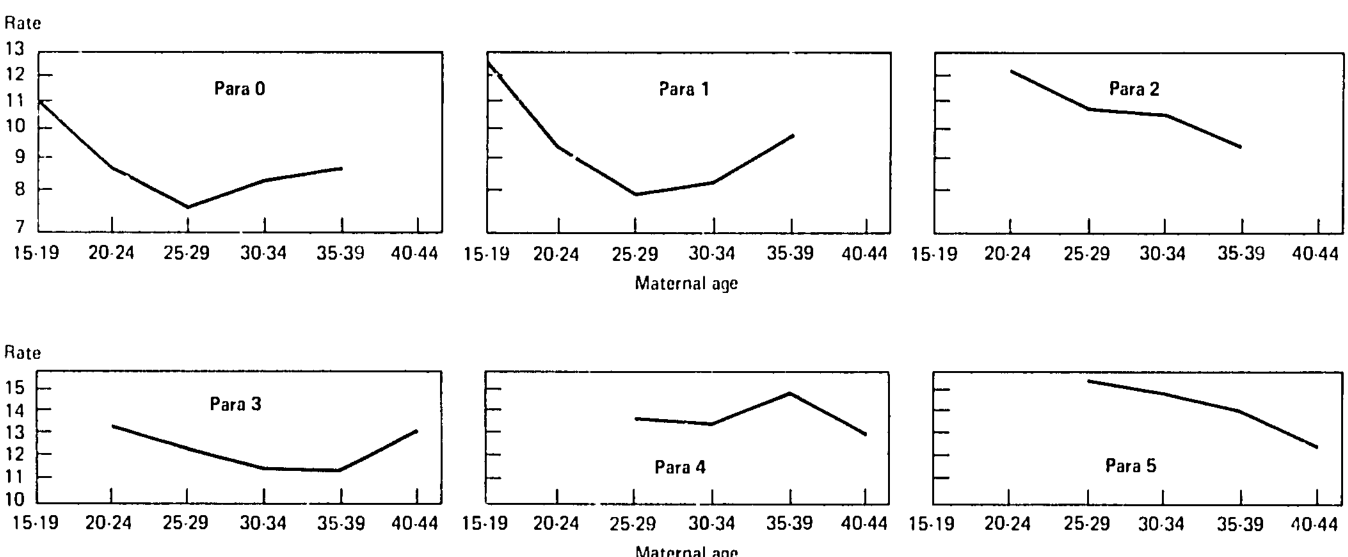
[18] If these rates seem high in an absolute sense, it must be realized that these maternities were medically attended in some of the most sophisticated hospitals in the world, where any complication was likely to be recognized and recorded in the study.

FIGURE 10 **Complications of pregnancy or delivery: Rate per 100 women by maternal age, for selected parities and complications, United States, 1961–69**

A. TOXEMIA



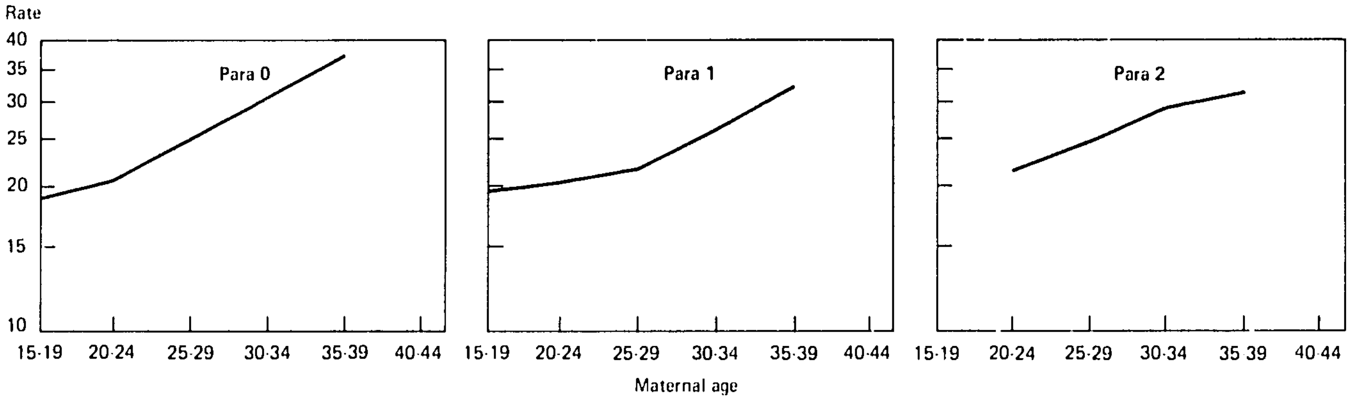
B. ANEMIA



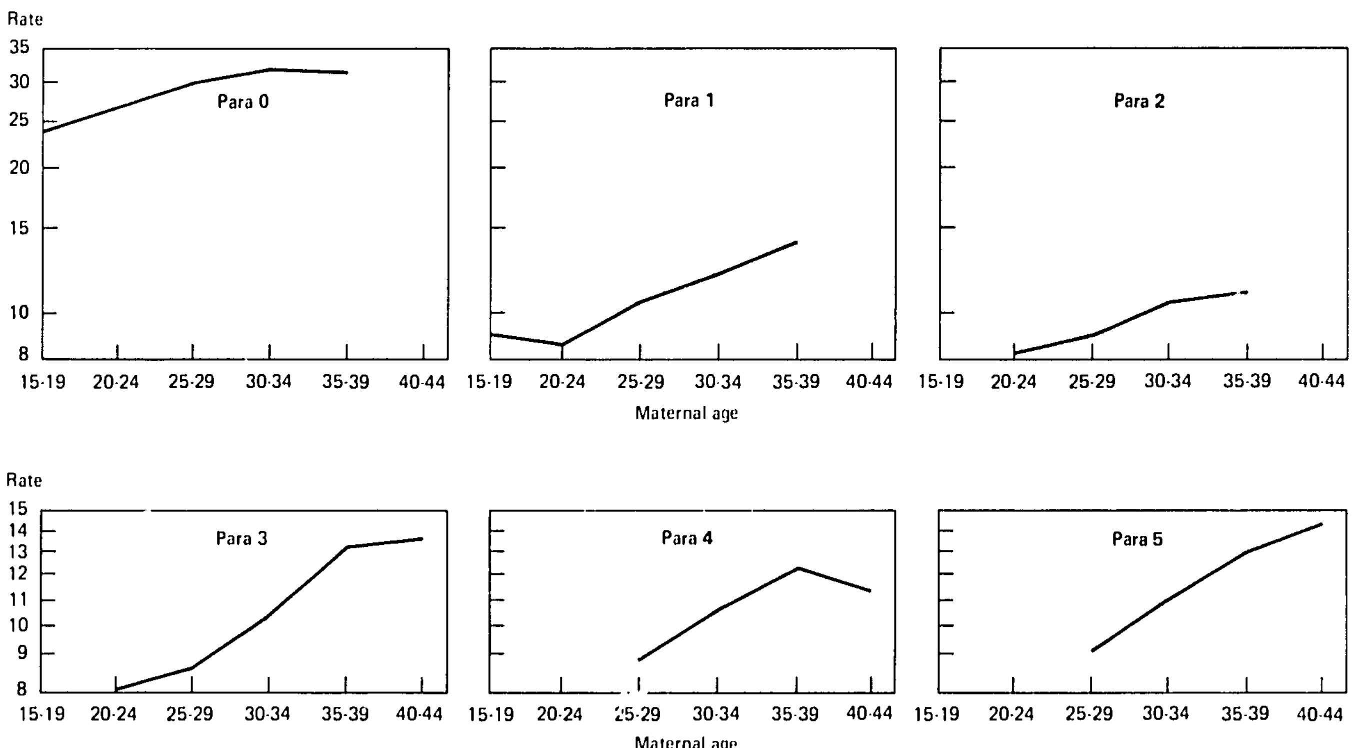
(continued)

FIGURE 10 **Complications of pregnancy or delivery: Rate per 100 women by maternal age, for selected parities and complications, United States, 1961–69** *(continued)*

C. OTHER COMPLICATIONS OF PREGNANCY



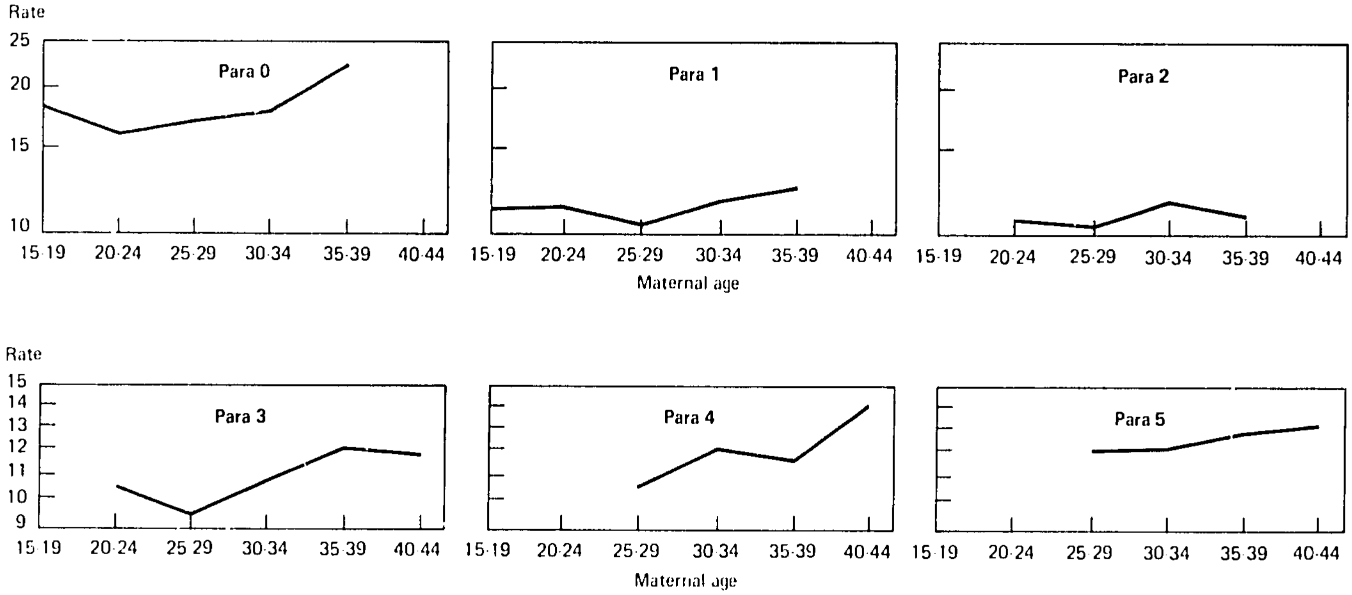
D. COMPLICATIONS OF DELIVERY



(continued)

FIGURE 10 **Complications of pregnancy or delivery: Rate per 100 women by maternal age, for selected parities and complications, United States, 1961–69** *(continued)*

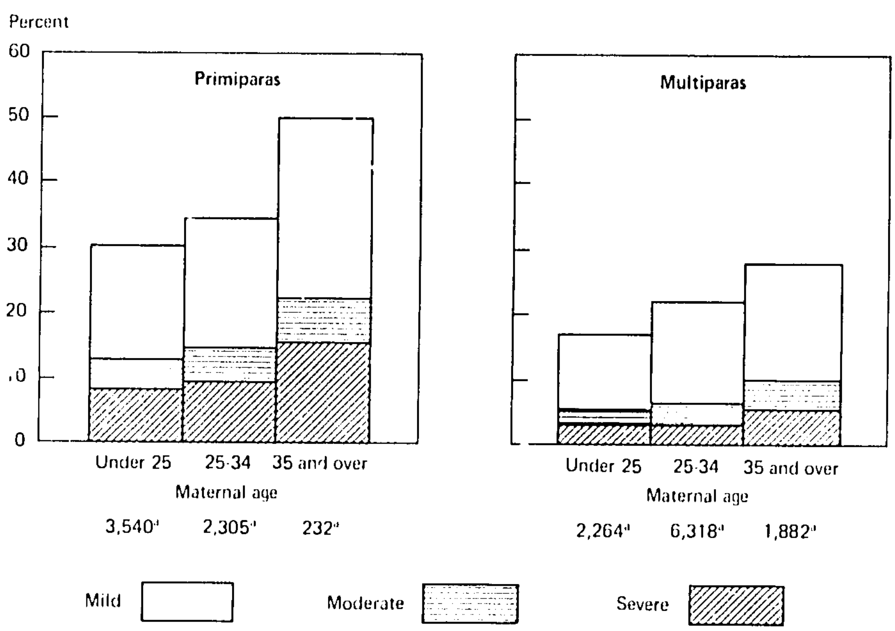
E. COMPLICATIONS OF PUERPERIUM



NOTE The rates have been standardized for age, race, and private and ward service. The influence of these factors is therefore eliminated from the age and parity differentials.

SOURCE Unpublished tabulation of data from over 460,000 pregnancies and deliveries in US hospitals participating in the Obstetrical Statistical Cooperative.

FIGURE 11 **Percent of women giving birth with preeclampsia during pregnancy by maternal age and parity, Great Britain, 3–9 March 1958**



NOTE Preeclampsia was defined as follows:

Degree	Maximum diastolic blood pressure
Severe	110 millimeters or more or 90 or more with proteinuria
Moderate	100-109 millimeters and no record of proteinuria
Mild	90-99 millimeters and no record of the proteinuria

[a]Total number giving birth in each age and parity category, excluding those with known hypertension in first half of pregnancy.

SOURCE Butler and Alberman, 1969, Table 3.3.

women aged 40–44. Postpartum hemorrhage due to uterine inertia rose from 2.7 percent to 6.2 percent for the same two age groups. Since these were presumably first pregnancies, not first live births, a history of difficult pregnancies did not account for the increase in complications with age. No differentials were found by socioeconomic status, based crudely on a four-celled classification by race (white or other) and private or ward service.

Some data are available from developing countries but they must be viewed with caution, first because of international differences in diagnosing, and more important, perhaps, because recorded medically attended cases are not likely to be representative of pregnant women in general in developing countries. Two studies of adequate sample size to warrant citation are Perkin (1969) and Llewellyn-Jones (1965).

Perkin (1969), studying 18,000 deliveries at Women's Hospital in Bangkok, Thailand, in 1964, found the expected J-shaped gradient by age in complicated deliveries, from 13.3 percent among women aged 15–19, dropping to 11.2 percent at ages 20–24, and increasing to 23.4 and 21.3 percent among women aged 40–44 and 45 and over, respectively (p. 60).

Llewellyn-Jones (1965) reported on 10,000 deliveries at the General Hospital in Kuala Lumpur, Malaysia, during the five-year period 1958–1962. An interesting feature of this study is that the hospital had two sections in the obstetrics gynecology department: one for high, the other for low, social class. For both socioeconomic groups, toxemia of pregnancy and prolonged labor (defined as effective contractions for more than 24 hours) increased markedly with increasing maternal age.

Neutra (1973) reported on 220 eclamptic patients in Cali, Colombia, in a well-designed study using a random sample of 345 parturients from the general population as a control group. This latter study found that among women under age 20, primiparas had 6.84 times the eclampsia risk of those aged 25–29 and multiparas had 3.99 times the risk; multiparas over age 35 had 1.75 times the risk of those aged 25–29. (Primiparas over 35 were too few for a statistically significant finding.)

Because of the few studies available and the diversity of maternal conditions reviewed, a generalized median age gradient is not feasible. However, the implication of all the age evidence for the would-be mother is clear. Her chances of a successful, uncomplicated pregnancy are best if she is younger rather than older but not so young that she herself has yet to mature. On the other hand, it cannot be stressed too often that, if other factors are favorable, age alone should not deter a woman who wants a child from bearing one.

Female Mortality

An unusual statistic is an index of mortality, by number of children ever born, among ever-married, white women in the United States who died between May and August 1960 (Kitagawa and Hauser, 1971). It was compiled in a University of Chicago study based on matched death and census records. Although this statistic is unrelated to maternal age at childbirth, it is indirectly relevant to this review. The J-shaped gradient by number of children ever born among women aged 45–64 at death suggests that childbearing may be associated with subsequent female mortality. Standardized for age and education, the index, based on the average for all white, ever-married women equals 100, was 107 for women with no children, 104 for women with one child, and 94, 89, 96, 105, and 113 for women with two, three, four, five or six, and seven plus children, respectively.

EFFECTS ON FETUS AND CHILD

Fetal Loss

During its growth and development, the fetus is subject to the vagaries of its intrauterine environment as well as to its intrinsic genetic endowment. Defective implantation (too deep or too shallow) of the fertilized ovum into the placenta, chromosomal errors, and, to a lesser extent, maternal infections that attack the fetus are the major factors that account for fetal loss (Reid, Ryan, and Benirschke, 1972).

Liberalization of abortion laws and sophisticated medical techniques for early detection of some types of defective fetuses are heightening interest in the question of fetal loss. Pursuit of this subject with improved statistical and medical procedures (to some extent facilitated by earlier prenatal care) has shown that the incidence of spontaneous abortion is considerably greater than had once been thought.

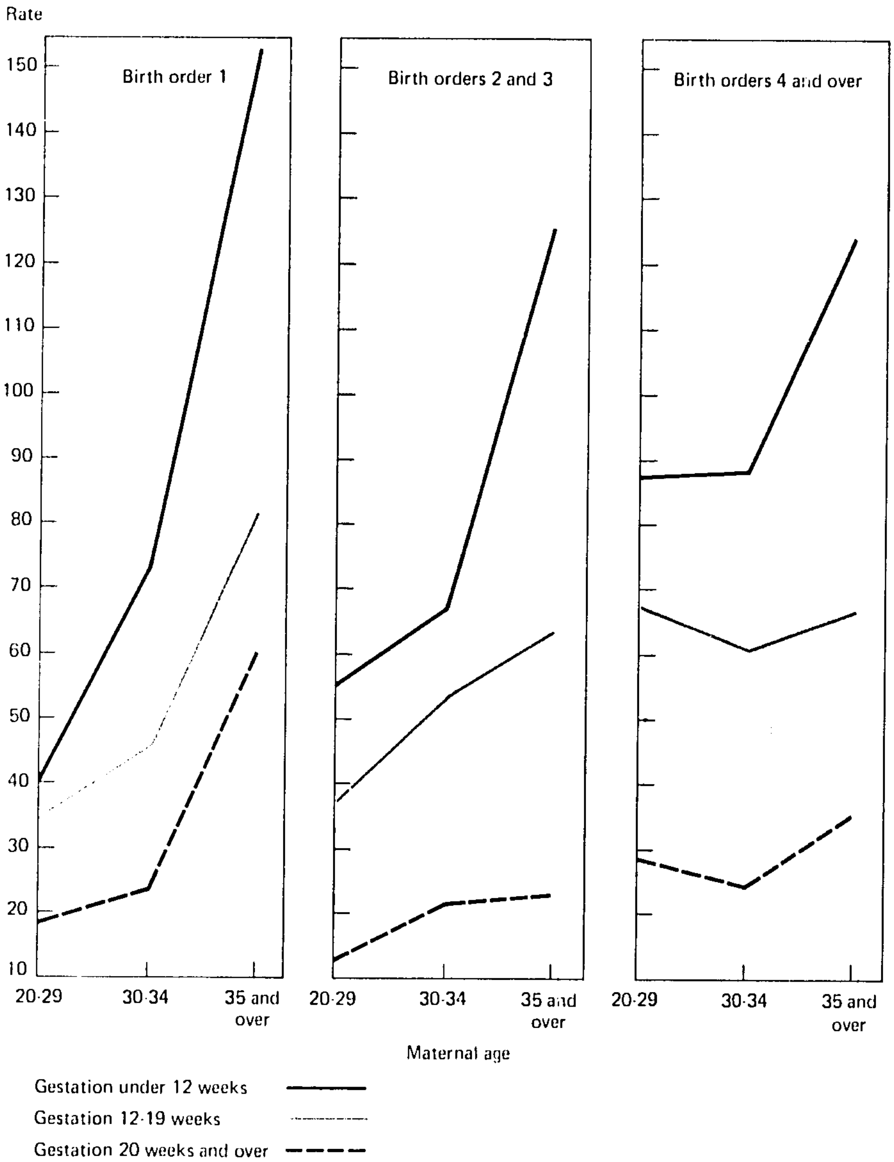
As gestation progresses, the chance of fetal loss diminishes (except, perhaps, for an increase in the very early weeks of pregnancy). Early fetal loss, including the point in time when the rate is probably at a maximum, is difficult to detect because the woman is likely to think that her next menstrual period was merely delayed, not that she had an aborted pregnancy. By extrapolating backward from empirical observations of loss among recognized pregnancies of reliable gestation age, it is possible to estimate the proportion of conceptuses that fail to eventuate into live births. On this basis, human pregnancy wastage is thought to be of the order of 50 percent of all fertilized ova (Kerr, 1971, p. 236). James's analysis suggests that 35 percent of fertilized ova are lost before the first missed period and another 14 percent lost between the first missed period and delivery (1970, p. 245). In their well-known follow-up study of pregnancies on the island of Kauai, Hawaii, in 1953–1956, French and Bierman calculated on a life-table basis that 24 percent of pregnancies reaching four weeks of age terminate in fetal loss.

As gestation progresses, the influence of biological and genetic factors diminishes while that of environmental factors increases. Examination of conceptuses aborted early in pregnancy indicates that losses are due mainly to genetically defective sperm or ova and to errors in fertilization and early cleavage divisions (Kerr, 1971). In later pregnancy, maternal and exogenous factors become dominant, with loss attributable to such reasons as poor maternal nutrition and bodily build, Rh incompatibility of mother and fetus, transmission to the fetus of communicable disease contracted by the mother, and abnormal position at delivery.

In countries where most deliveries occur in hospitals, vital records are kept of fetal deaths, usually limited, for obvious reasons, to gestation of 20 or more weeks. Absolute rates are, therefore, influenced by the proportion of cases of fetal loss that go unrecorded and by the problems of estimating gestation age. To illustrate the effect of more complete recording, efforts in New York City to improve fetal death registration resulted in an increase in the number of recorded fetal deaths of all gestational ages per thousand pregnancies from 69.3 in 1950 to 121.8 in 1960 (Shapiro, Levine, and Abramowicz, 1970). Comparison of rates among different populations, even for the same gestation duration, are therefore hazardous, but on the assumption that the problems of data collection affect all age groups within a population about equally in direction and magnitude, the age patterns of fetal loss are not significantly affected by incomplete recording.

Both for the maternal age pattern and validity of the absolute rates at different gestation ages, an outstanding study on fetal wastage was conducted by the Health Insurance Plan in New York City, a prepaid, comprehensive medical care plan (Shapiro and Abramowicz, 1969). The underlying basis for the validity of the rates, besides technical competence, is the fact that since the medical care had been paid for in advance, it is reasonable to suppose that the women sought prenatal medical care as soon as they thought they were pregnant. The sample comprised 12,000 women whose last menstrual period had occurred between 1 March 1958 and 29 February 1960.

FIGURE 12 **Fetal mortality: Rate per 1,000 pregnancies in a New York City prepaid health plan, 1958–60**



SOURCE Shapiro and Abramowicz, 1969, p. 1633.

The findings, presented in Figure 12, show a clear association between maternal age and fetal deaths (per thousand pregnancies) of specified birth order and gestation duration. The chance of survival improves with duration of gestation, suggesting that nature selects out its own, presumably genetic, mistakes. The point of interest here, however, is that, for any given gestation period or birth order, the fetus of a younger woman has a much better chance of survival than that of the older woman (except that, at gestation period 12–19 weeks for birth orders 4 or more, the age differential is very flat).

The studies for a generalized age pattern, mostly government vital data, are listed in Table 3 with the rates by age given as an index based for each study on the unweighted average of age groups 20–34 equals 100. The absolute average rates (weighted or unweighted) vary widely by a factor of more than 4, which is to be expected, considering the problems of data collection and the fact that the data relate to different population groups, different time periods, and different gestation ages. Nevertheless, the similarity in the age patterns is evident, typically J shaped. This can be seen in Figure 2, where the band represents the range in index numbers per age group, the points surrounding the smooth curve the medians, and the smooth curve the parabola fitted to the medians.

To generalize from the studies listed, fetal loss is lowest at maternal

TABLE 3 **Fetal mortality: Rate per 1,000 deliveries, and index number by maternal age, for selected birth orders, areas, and years**

(Index based on unweighted average rate of age groups enclosed in rectangle = 100)

Birth order, area, and years	Average rate: Weighted	Average rate: Un-weighted	Index number for maternal age group: Under 20	20–24	25–29	30–34	35–39	40–44
All birth orders								
Denmark, 1951–53	18.3	16	101	86	98	116	176	200
Hawaii, USA, 1953								
Gestation								
Under 20 weeks	57.3	59	86	79	94	127	154	211
20 or more weeks	25.9	23	161	93	110	97	136	362
United States, 1960–61								
White	13.9	13	88	80	95	125	176	258
Other	26.2	26	82	77	94	129	178	241
United States, 1967								
Legitimate								
White	12.4	12	94	82	90	128	188	288
Other	23.3	23	76	74	92	133	202	274
Illegitimate								
White	19.7	27	57	70	91	139	173	178
Other	25.7	31	71	74	86	140	178	212
Pennsylvania, USA, 1960–64	19.1	18	98	82	94	124	170	250
North Carolina, USA, 1969								
White	13.1	13	106	84	85	131	177	214
Other	24.6	26	72	82	90	128	156	315
New York City, USA, 1958–60, Health Insurance Plan								
Gestation								
20 or more weeks	20.9	19	–	89	88	123	167	
12–19 weeks	51.2	47	78	83	96	121	144	
Under 12 weeks	78.6	65	90	76	103	121	206	
Median	23.3	23	87	82	94	127	176	254
Range (trimmed)[a]	13–57	13–59	71–106	74–89	86–103	116–139	144–202	211–315
Birth order 1								
Denmark, 1951–53	20.9	25	71	70	91	139	227	252
Hawaii, USA, 1953	14.9	20	71	64	86	150	235	344
United States, 1960–61								
White	13.8	18	66	64	89	147	228	307
Other	26.5	38	55	65	84	150	230	252
New York City, USA, 1958–60, Health Insurance Plan								
Gestation								
20 or more weeks	21.2	21		87		113	286	
12–19 weeks	38.9	40		86		113	206	
Under 12 weeks	52.8	57		71		129	269	
Median	21.2	25	68	70	88	139	230	279
Range	14–53	18–57	55–71	64–87	84–91	113–150	206–286	252–344
Birth order 2								
Denmark, 1951–53	13.4	13	80	80	95	125	199	355
Hawaii, USA, 1953	10.8	11	110	81	90	129	190	250
United States, 1960–61								
White	10.1	11	95	80	94	126	182	281
Other	19.0	21	86	77	92	131	185	298
Median	12.1	12	90	80	93	128	188	290
Range	10–19	11–21	80–110	77–81	90–95	125–131	182–199	250–355
Birth order 3								
Denmark, 1951–53	15.8	13	155	76	103	121	185	246
Hawaii, USA, 1953	13.1	12	135	93	93	114	165	270
United States, 1960–61								
White	12.1	11	111	89	93	118	173	266
Other	20.5	22	103	76	96	128	194	254
Median	14.4	13	123	83	94	120	179	260
Range	12–21	11–22	103–155	76–93	93–103	114–128	165–194	246–270

(continued)

TABLE 3 **Fetal mortality: Rate per 1,000 deliveries, and index number by maternal age, for selected birth orders, areas, and years** *(continued)*

(index based on unweighted average rate of age groups enclosed in rectangle = 100)

	Average rate		*Index number for maternal age group*					
Birth order, area, and years	*Weighted*	*Un-weighted*	*Under 20*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*
Birth order 4								
Denmark, 1951-53	19.2	15	–	82	101	117	167	284
Hawaii, USA, 1953	15.9	15	157	97	91	112	148	218
United States, 1960-61								
White	14.7	13	131	95	95	110	153	221
Other	23.3	23	125	85	94	121	169	217
Median	17.5	15	131	90	94	114	160	220
Range	15-23	13-23	125-157	82-97	91-101	110-121	148-169	217-284

NOTE: Gestation period of 20 weeks or more unless otherwise specified.

[a] The range shown excludes the extreme value at each end.

SOURCES:
Denmark: US HEW, 1967c, p. 13.
Hawaii: Yerushalmy et al., 1956, p. 88.
United States, 1960–61: US HEW, 1966a, p. 79.
United States, 1967: US HEW, 1967d, Table 3.3 (34 reporting states).
Pennsylvania: Department of Health, 1967, p. 3.
North Carolina: State Board of Health, 1969, p. 16.
New York City: Shapiro and Abramowicz, 1969, p. 1633.

age 18 for first order births, rising to age 26 for fourth order births (see Table 1, p. 6). At five years, plus or minus, from the age of minimum risk, the excess risk is 13–17 percent, depending on birth order; ten years earlier or later, excess risk exceeds 50 percent, and 15 years from the optimal age, excess risk is more than 100 percent. Although only a few studies cross-classify maternal age by birth order for fetal loss, the generalized age pattern seems warranted since those studies listed in Table 3 are large-scale.

The conclusions of some of the investigators of fetal loss are worth noting. In connection with their retrospective survey in 1953 of all women under age 50 in Kauai Island, Hawaii (N = 6,039 with 27,528 previous live births and fetal deaths), Yerushalmy et al. stated "The fact that late fetal deaths [gestation 20 or more weeks] are affected by the age of mother much as early fetal deaths [under 20 weeks gestation] are affected by this factor points to possible biological influences of aging on reproductive wastage" (1956, p. 88). Helen Chase noted in her unpublished report based on single, white births in New York State (excluding New York City) in 1950–1952, "with regard to fetal mortality, advancing mother's age is indicated to be more important than declining socio-economic level [based on father's occupation]. From age 25 on, the trend is definitely upward, and the pitch of the trend increases with advancing maternal age" (1962, pp. 33, 47).

Stillbirths or Late Fetal Deaths

Stillbirths are fetal deaths of late or mature gestation, generally defined as 28 weeks or more, at which time a fetus is considered old enough for a live birth. The factors that cause early fetal death continue to operate throughout pregnancy, but stillbirths are caused mainly by failure to make the many necessary physiologic adjustments of birth. The stillbirth concept implies death before birth, but just as it is difficult to pinpoint precisely when a person has died, so it is difficult to pinpoint precisely when a birth has occurred. Because it is thus often a matter of definition or chance whether an infant dies just before or shortly after delivery (Baird et al., 1954), the stillbirth concept has been giving way in recent years to the perinatal mortality measure. The latter includes, in addition to stillbirths, live-born babies who die shortly (often defined as seven days) after birth.

The landmark study of stillbirths is the British analysis of stillbirths among single, legitimate births that occurred in 1949–1950, noteworthy because it provided socioeconomic controls available from the wartime national register still in effect for the period covered. The other studies listed in Table 4, on which the generalized age gradient is based, are the US birth cohorts for 1931–1935 (Yerushalmy, 1939), New York State births in 1936 (Yerushalmy, 1938), Scottish rates for several time periods, and national data for Canada, Denmark, and Norway Recourse to primary vital statistics sources could enlarge this list, but the studies cited are considered sufficiently comprehensive to warrant generalization of the age pattern.

The J-shaped relationship between maternal age and stillbirth rates is evident in Figure 3, which depicts the range, median, and smooth curve fitted to the age-specific median index numbers of the studies listed in Table 4. Again attention is called to the narrow range, suggesting a more or less invariant age pattern at all absolute levels of risk. Except for birth order 1, risk is minimal when mother's age is in the twenties. The excess risk is 8 to 18 percent (depending on birth order) at five years from the age of minimum risk, 33 to 73 percent at ten

TABLE 4 **Stillbirth: Rate per 1,000 deliveries, and index number by maternal age, for selected birth orders, areas, and years**
(Index based on unweighted average rate of age groups enclosed in rectangle = 100)

Birth order, area, and years	*Average rate* Weighted	Un-weighted	*Index number for maternal age group* Under 20	20–24	25–29	30–34	35–39	40–44
All birth orders								
Canada, 1963	12.0	13	76	69	80	99	152	234
Denmark, 1951–53	18.3	20	84	73	82	98	147	218
England and Wales, 1949–50	21.4	22	78	73	81	104	142	210
Socioeconomic class								
1 (high)	15.9	15	–	79	90	112	119	219
2	19.4	19	95	75	81	108	136	207
3	21.0	22	77	73	81	102	144	204
4	22.9	24	71	64	84	109	143	225
5 (low)	25.5	26	67	69	79	107	145	195
New York State (except NYC), USA, 1936	27.6	29	84	67	85	105	143	235
Norway								
1936–40	22.1	21	73	64	84	111	141	198
1946–50	17.1	16	76	72	85	104	139	185
1956–60	13.9	14	74	74	85	104	137	202
Scotland								
1939	42	43	69	71	81	105	143	169
1950	27	28	84	71	84	97	148	213
1960	22	22	103	79	79	105	137	216
1961–63[a]	19.5	21	83	77	81	104	138	204
United States, 1931–35	35.7	35	109	86	85	99	130	171
Median	21	22	78	73	82	104	142	207
Range (trimmed)[b]	14–36	14–35	69–103	64–79	79–85	98–111	130–148	171–234
Birth order 1								
Denmark, 1951–53	20.9	32	55	53	69	106	172	192
England and Wales, 1949–50	23.4	31	57	57	77	112	154	180
Socioeconomic class								
1 (high)	20.4	25	–	52	73	115	160	–
3	23.1	31	57	57	76	113	154	180
5 (low)	27.1	39	48	57	74	118	151	142
Norway								
1936–40	25.7	32	48	48	71	112	169	225
1946–50	21.6	27	48	48	73	112	167	195
1956–60	15.5	20	53	63	76	118	143	175
Scotland, 1961–63[a]	20.3	24	72	74	85	110	131	91
New York State (except NYC), USA, 1936	30.3	39	64	61	87	123	129	252
Median	22	31	55	57	75	112	154	180
Range (trimmed)[b]	20–27	24–39	48–64	48–63	71–85	110–118	131–169	142–225
Birth order 2								
Denmark, 1951–53	13.4	18	58	57	68	89	143	243
England and Wales, 1949–50	15.3	18	76	65	75	95	130	213
Socioeconomic class								
1 (high)	10.7	10	–	–	90	101	109	–
3	15.2	18	66	65	76	92	132	200
5 (low)	17.6	22	–	60	72	92	136	228
Scotland, 1961–63[a]	14.1	17	78	70	73	98	129	229
New York State (except NYC), USA, 1936	19.0	27	73	54	67	87	146	84
Median	15	18	73	62	73	92	132	220
Range	11–19	10–27	58–78	54–65	67–90	87–101	109–146	84–243

(continued)

years, and 67 to 165 percent at 15 years from the age of minimum risk (see Table 1, p. 6).

Another study, that of Scottish births during 1950–1959 controlled for social class, found an exponential increase in stillbirths with respect to mother's age (Baird and Hytten, 1958).[10]

Two studies found in the literature considered paternal as well as maternal ages. Yerushalmy's anal-

[10] These were not included in Table 4 because the published report provided only the graph, not the figures.

TABLE 4 **Stillbirth: Rate per 1,000 deliveries, and index number by maternal age, for selected birth orders, areas, and years** *(continued)*
(Index based on unweighted average rate of age groups enclosed in rectangle = 100)

	Average rate		Index number for maternal age group					
Birth order, area, and years	*Weighted*	*Un-weighted*	*Under 20*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*
Birth order 3								
Denmark, 1951-53	15.8	18	114	55	76	89	135	181
England and Wales, 1949-50	18.8	20	–	67	78	92	130	189
Socioeconomic class								
1 (high)	14.7	13	–	–	84	108	108	309
3	18.9	20	–	72	74	88	138	182
5 (low)	22.7	26	–	65	75	95	130	185
Scotland, 1961-63[a]	17.5	19	32	72	85	96	119	194
New York State (except NYC), USA, 1936	22.4	24	132	61	76	103	121	290
Median	19	20	114	66	76	95	130	189
Range	16-22[b]	18-24[b]	32-132	61-72[b]	75-84[b]	89-103[b]	119-135[b]	182-290[b]
Birth order 4								
Denmark, 1951-53	19.2	19	–	66	79	91	130	223
England and Wales, 1949-50	25.0	23	–	78	78	101	121	203
Socioeconomic class								
2 (high)	24.1	21	–	–	56	113	131	196
3	24.5	23	–	75	83	99	118	202
4	27.7	26	–	75	70	105	125	230
5 (low)	25.3	26	–	77	74	94	132	189
Scotland, 1961-63[a]	22.7	23	–	76	84	95	121	199
New York State (except NYC), USA, 1936	26.7	28	–	56	72	100	128	254
Median	25	23	–	75	76	100	126	202
Range (trimmed)[b]	23-27	21-26	–	66-77	70-83	94-105	121-131	196-230

[a] Figures are for legitimate births only.
[b] The range shown excludes the extreme value at each end.

SOURCES:
Canada: Dominion Bureau of Statistics, 1966, p. 258.
Denmark: US HEW, 1967c, p. 13.
England and Wales: Heady and Heasman, 1959, Table 5a.
New York State: Yerushalmy, 1938, p. 263.
Norway: US HEW, 1967b, p. 15.
Scotland: US HEW, 1966b, pp. 8, 24.
United States: Yerushalmy, 1939, p. 348.

ysis of stillbirths among the 1931–1935 birth cohorts in the US registration area yielded U shapes by father's age with mother's age held constant and J shapes by mother's age with father's age constant, leading him to conclude that "the ages of the two parents are, therefore, shown to be independently correlated with the stillbirth rate" (1939, p. 349).

The second was a study of cause of stillbirths in Arizona during 1958–1961, using live births during 1960 as a control. Cause of death was limited to those ascribable to fetal, that is, nonmaternal origin. Based on a covariance analysis of 742 stillbirths, Woolf concluded, "The results demonstrate clearly that an association exists between stillbirths in certain coded groups (unspecified and ill-defined) and increased maternal and paternal age. However, any biologic or sociologic interpretation . . . is obscured by the etiologic heterogeneity. . . . Of interest is the observation that increased paternal age is of etiologic importance for stillbirths coded as ill-defined cause of death" (1965, p. 7).

To conclude this section, two noted investigations attributed the higher stillbirth rate directly to greater age and not to some other factor. Yerushalmy, in his study of 82,000 legitimate births in New York State in 1936, stated that "birth order has very little effect on the stillbirth rates and almost the entire variation is due to the age of mother" (1938, p. 263). Tabah and Sutter (1948) also concluded that age of mother, not birth order, was the significant factor from their consideration of stillbirth rates in France in 1943. By single year of maternal age, they found J-shaped rates which were minimal at age 23 and which rose more than three-fold by age 45.

Perinatal Mortality

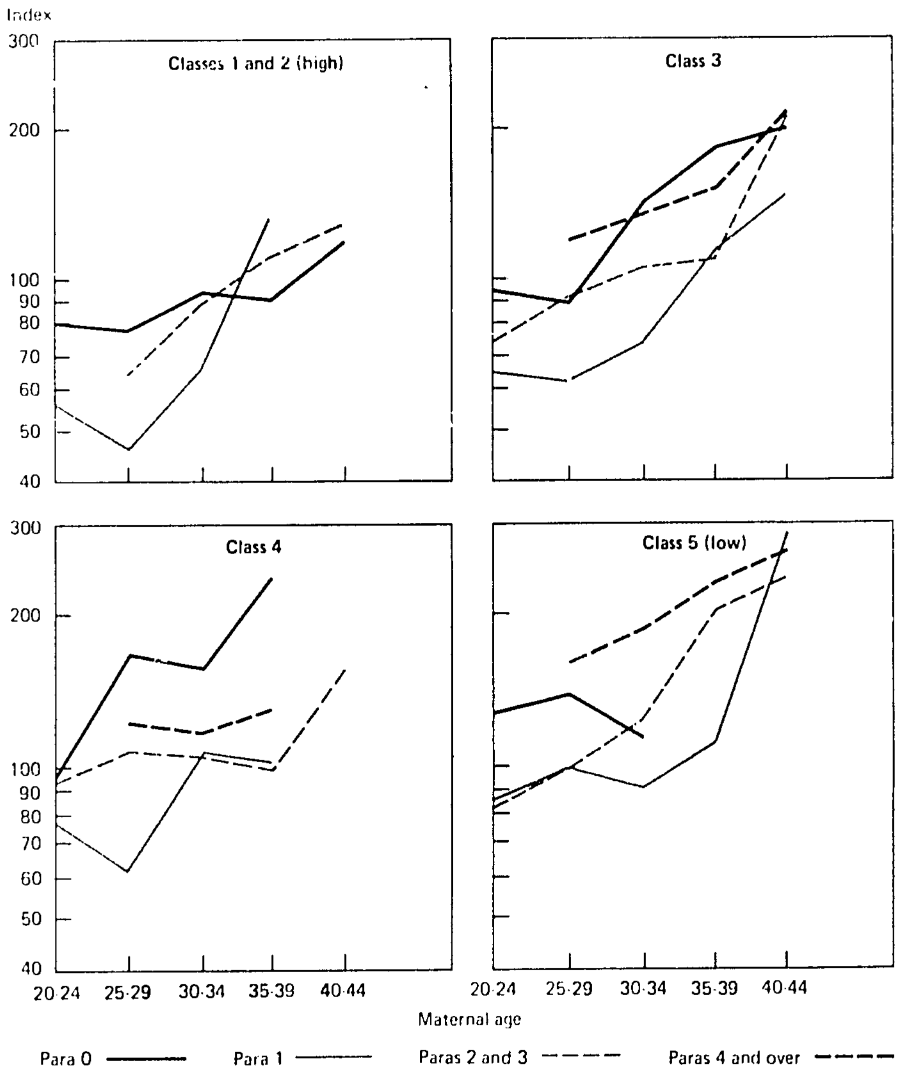
Perinatal mortality relates to the death of a child around the time of its birth. As already mentioned, it is a single measure of two components: (1) babies of mature gestation age, usually at least 28 weeks, who show no sign of life after separation from the mother (commonly called stillbirths); and (2) live-born babies who die shortly after birth, usually within seven days. The former component overlaps with fetal deaths, already discussed, while the latter (also known as early neonatal deaths) overlaps with neonatal (first month) and infant mortality. The perinatal measure is useful because it overcomes the problem of distinguishing between a live and a stillbirth.

A wealth of information exists on perinatal mortality, partly because it is easier to compile than measures of fetal or child mortality since death occurs around the time of birth, and partly because the decline in perinatal mortality in the modern era, although not inconsequential, has not kept pace with the decline in maternal and later than first week infant mortality. To a large extent, the lag in the decline can be attributed to the fact that perinatal mortality, like fetal mortality, is heavily weighted with biological mistakes not very amenable to sociological and medical control. To illustrate, an analysis of the subject, based on medical diagnosis of cause of perinatal mortality among the infants of 26,116 patients who had single deliveries at the Aberdeen Maternity Hospital, Scotland, in the 15-year period 1938–1952, found that in a death rate of 38.6 per thousand deliveries, 20 percent of the deaths were due to prematurity, cause unknown; another 14 percent were of mature infants, also cause unknown; and 16 percent were attributable to fetal deformity (Baird et al., 1954, p. 436). The medical details, cause of death by parity per maternal age group, are too specific for this review, but with regard to age, the authors concluded that ". . . the ageing process, especially in primiparae, is associated with a decline in reproductive efficiency, and this causes a tendency for the obstetric (perinatal) death rate to rise with age. . . . The lowest possible obstetric death rate cannot be achieved by good obstetric care alone. For this, a higher level of health among mothers and reproduction at more favorable ages would be required" (Baird et al., 1954, pp. 447, 448).

The most analytic statistical study on the subject is the *British Perinatal Mortality Survey* based on 7,117 deaths among single, legitimate births in Great Britain in the three-month period March–May 1958. To get denominators for the calculation of rates, by maternal age per parity per social class (measured by husband's occupation), these characteristics were recorded on all single, legitimate births that occurred during a control week 3–9 March 1958, a total of 16,994. The rates were then computed as the number of deaths in the three-month period divided by 13 times the number of births in the control week, based on the reasonable supposition that the population of births in the three-month period was the same as during the control week.

As previously mentioned, two reports emanated from this survey, Butler and Bonham (1963) and Butler and Alberman (1969). Figure 13, prepared from the first report, shows that social class affects the level of the rate (which is about twice as high in the lowest compared with the highest class, even when controlled for parity) but not the age

FIGURE 13 **Perinatal mortality: Index by maternal age, parity, and social class, for single, legitimate births, Great Britain, 1958**
(Index based on all ages, parities and classes = 100) (Logarithmic scale)



SOURCE Butler and Bonham, 1963, pp. 28, 30.

pattern. Within each social class and parity group, the increase with mother's age is unmistakable. In social class 3, for example, women in their thirties encountered about 75 percent greater perinatal mortality at parity 0 than women in their twenties. At parity 1, the corresponding excess was about 50 percent, and at parities 2 and 3, about 25 percent. The increase with maternal age for the composite parities 4 and over is also evident, although disaggregation of the parities could yield more of a U shape or a reverse J shape because higher risk is usually associated with short birth intervals.

The Butler and Alberman report (1969) provided data on the maternal age gradient of perinatal mortality by cause, parity, and social class. Cause was dichotomized into "obstetrical" and "environmental," the latter not in the sense of being unrelated to the biologic but in the sense of being unrelated to the delivery. For obstetrical causes, rates by age tended to be J shaped, but they were somewhat mixed for environmental causes. Prematurity was U shaped for all parities and social classes; malformations decreased with age for parity 0 but increased for other parities. That risk was also U or J shaped with respect to age for the professional class is meaningful because among them presumably, with sociological factors optimal, biological influences are least diluted.

Another point of interest in the Butler and Alberman report (1969) was the differential in rates between smoking and nonsmoking mothers, by two maternal age groups, social class, and parity. The findings showed a differential in favor of the nonsmoking mother, but the point of interest is that the mortality risk was greater for the nonsmoking mother aged 35 or over than for the smoking mother under age 35 of the same parity.

Because of its uniqueness, the British study has been presented in some detail, but several additional studies of large scope are available for a generalized age pattern. These are the Aberdeen data already mentioned; a study by Fischler, Peritz, and Wingerd (1971) of NYC births, by race, in 1957–1959; vital data for two US states, New York for four time periods and North Carolina for 1969; Scottish national data, 1961–1962, by social class; data on births in Vancouver, Canada, 1963–1967; and finally, to include an underdeveloped area, a prospective study in a Hyderabad, India, maternity hospital of 3,792 deliveries in a 12-month period 1969–1970 (Karan et al., 1972). Although the uncharacteristically low rates of the latter study make them suspect, we are assuming that the age pattern is not unduly affected.

The J-shaped relationship between maternal age and perinatal death rates is evident in Figure 4 showing the age-specific index numbers of the studies listed in Table 5. Again, the generally narrow range at most ages is noteworthy, considering the wide differential in the absolute level of risk among the various studies and social classes. Based on the smooth curves fitted to the median index numbers of the studies, risk is minimal at age 18 for birth order 1, at age 27 for birth order 2, and at age 29 for birth orders 3 and 4. For birth order 1, excess risk rises slowly with departure from age 18 when risk is minimal, being only 24 percent greater at age 33, 15 years later. Birth orders 2, 3, and 4, however, show substantial increases in risk as the childbearing age departs from the optimum, as is shown in Table 1 (p. 6).

Some important studies not included in the data for the generalized picture because of differences in category or emphasis also deserve mention. A major study is the ongoing US National Institute of Health project on cerebral palsy. From this study, on the basis of 22,201 unselected gravidas of a single fetus each, Israel and Deutschberger (1964) found higher perinatal mortality rates, by race, among offspring of women over age 30 than among offspring of younger women, particularly for nulliparous mothers. Donnelly et al., reporting on 22,126 university hospital white births in North Carolina, United States, over a period of time starting in 1954, with data available by father's occupation, mother's education, legitimacy, and hospital financial classification, stated, "The mother's age was found to be a highly significant factor in perinatal mortality even when adjusted for race, age, and parity (and subjected to covariant analysis)" (1957, p. 1253).

Two other studies in India are noteworthy. In one, based on 6,101 single live births among two social classes in a Gujarati community, the investigator concluded, "There is a significant difference in the mean maternal age of survivors (lived more than 7 days after birth) and non-survivors (born dead or died within 7 days), the non-survivors having [mothers of] a higher mean maternal age. It would seem that maternal age represents an independent influence on survivorship . . . [the other factors being birth weight and sex of child]" (Jayant, 1964, pp. 265, 266). The other, a study of 125 pregnant women attending the clinics in Indore, showed 3 perinatal deaths among 11 births to women under age 20 and 4 deaths out of 12 births among women aged 30–34 compared with 4 deaths in 67 births to women in their twenties (Mittal and Ketker, 1970). Although of small sample size and uncertain sampling quality, these studies are consistent with the generalized findings of sophisticated surveys.

Infant Mortality and Its Components

Infant mortality, long accepted as a sensitive barometer of a country's progress in controlling disease and warding off death, has always commanded great interest. A few countries, such as Denmark and Sweden,

TABLE 5 **Perinatal mortality: Rate per 1,000 deliveries, and index number by maternal age, for selected birth orders, areas, and years**
(Index based on unweighted average rate of age groups enclosed in rectangle = 100)

	Average rate		*Index number for maternal age group*					
Birth order, area, and years	*Weighted*	*Un-weighted*	*Under 20*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*
All birth orders								
Aberdeen, Scotland, 1938–52	38.6	37	94	89	97	114	——179——	
England and Wales, 1958	38.2	36	115	93	92	115	146	218
Socioeconomic class								
1 and 2 (high)	29	23	119	119	102	79	193	208
3	37	35	107	92	90	118	144	221
4	41	39	108	86	103	111	127	226
5 (low)	49	45	122	90	97	113	156	228
India, 1969–70	8.1	8	113	94	91	115	131	–
New York City, USA, 1957–59								
White	32.9	32	–	92	95	113	149	–
Black	60.5	60	–	86	96	118	137	–
New York State (except NYC), USA								
No previous child loss								
1936	–	38	123	91	92	117	154	224
1951	–	27	148	102	92	106	143	231
1958–59	–	24	164	100	95	105	139	191
1965–66	–	23	173	102	94	104	135	181
North Carolina, USA, 1969								
White	27.6	25	145	97	90	113	152	222
Other	46.6	46	97	90	96	114	143	254
Scotland, 1961–62	34.4	32	114	92	94	114	156	190
Socioeconomic class								
1 and 2 (high)	25.1	23	149	97	92	111	110	198
3	33.9	32	114	92	95	113	177	192
4 and 5 (low)	40.1	38	96	88	93	119	147	189
Vancouver, British Columbia, Canada, 1963–67	23.1	23	93	105	90	105	149	134
Median	36	32	114	92	94	113	146	213
Range (trimmed)[a]	23–49	23–46	94–164	86–105	90–102	104–118	127–179	181–231
Birth order 1								
Aberdeen, Scotland, 1938–52	42.5	49	70	70	96	134	——183——	
England and Wales, 1958	41	42	97	89	91	120	152	190
Socioeconomic class								
1 and 2 (high)	32	33	90	96	93	111	106	140
3	38	41	88	86	83	131	169	187
4	46	52	77	70	118	112	174	174
5 (low)	50	48	115	101	107	92	63	251
Malaysia, 1958–62								
Low status	21.4	30	71	65	72	163	——153——	
New York City, 1957–59								
White	27.5	29	–	82	94	124	188	–
Black	54.4	60	–	81	86	133	197	–
Scotland, 1961–62	35.8	38	95	83	93	124	149	122
Socioeconomic class								
1 and 2 (high)	26.0	27	128	86	92	122	94	–
3	36.3	39	93	82	95	123	176	–
4 and 5 (low)	40.6	47	75	75	94	131	135	–
Vancouver, British Columbia, Canada, 1963–67	23.6	26	75	84	90	126	184	183
Median	37	40	89	82	93	124	161	183
Range (trimmed)[a]	24–50	27–52	71–115	70–96	83–107	111–134	94–188	140–190

(continued)

with present levels of under 15 deaths per thousand live births, may be close to the irreducible biological minimum.

In her comparison of the US rates of infant mortality and its components with those of six West European countries, Chase examined a number of demographic factors to assess their possible statistical effects. She found that, adjusted for maternal age and birth order, the US neonatal (first month) mortality rate was 36 percent above that of the Netherlands compared with a 47 percent excess in the unadjusted rates. "It may be concluded, therefore, that differences in the maternal age and birth order distributions account for part but not all of the difference . . ." (US HEW, 1967a, p. 59). Pursuing this same line of inquiry, Wright (1972) recently found that if women had their first child

TABLE 5 **Perinatal mortality: Rate per 1,000 deliveries, and index number by maternal age, for selected birth orders, areas, and years** *(continued)*
(Index based on unweighted average rate of age groups enclosed in rectangle = 100)

Birth order, area, and years	*Average rate*		*Index number for maternal age group*					
	Weighted	*Un-weighted*	*Under 20*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*
Birth order 2								
England and Wales, 1958	28	31	123	84	76	94	146	221
Socioeconomic class								
1 and 2 (high)	24	29	–	74	60	86	180	226
3	27	30	120	82	78	95	145	180
4	31	33	139	88	71	124	117	418
5 (low)	37	37	118	90	101	93	116	307
New York City, 1957–59								
White	27.3	30	–	92	82	102	124	–
Black	48.5	57	–	81	76	97	146	–
Scotland, 1961–62	26.1	27	126	92	87	104	117	180
Socioeconomic class								
1 and 2 (high)	21.5	23	183	94	87	88	131	166
3	24.6	25	129	92	94	109	105	241
4 and 5 (low)	32.1	37	96	78	75	114	133	109
Median	27	30	124	88	78	97	131	221
Range (trimmed)[a]	24–37	25–37	118–139	78–92	71–94	88–114	116–146	166–307
Birth orders 3 and 4								
England and Wales, 1958[b]	40	38	387	80	94	108	118	197
Socioeconomic class								
1 and 2 (high)	33	30	–	57	85	115	143	159
3	39	36	186	81	95	110	114	214
4	40	38	118	93	105	103	99	157
5 (low)	45	48	–	67	77	96	160	187
New York City, 1957–59[c]								
White	33.2	35	–	111	85	91	113	–
Black	56.4	62	–	85	81	110	124	–
New York City, 1957–59[d]								
White	40.2	41	–	113	95	94	98	–
Black	62.4	64	–	93	92	105	110	–
Scotland, 1961–62[c]	30.8	31	147	92	92	92	124	177
Socioeconomic class								
1 and 2 (high)	21.6	18	–	97	101	122	80	301
3	31.1	32	173	93	91	92	124	164
4 and 5 (low)	35.0	36	113	78	91	92	139	176
Scotland, 1961–62[d]	37.7	37	–	91	91	96	122	158
Socioeconomic class								
1 and 2 (high)	30.4	32	–	136	76	95	93	156
3	36.9	35	–	77	99	96	128	195
4 and 5 (low)	41.3	43	–	91	81	97	131	112
Median	38	36	160	91	91	96	122	176
Range (trimmed)[a]	30–56	30–62	118–186	67–113	77–101	92–115	93–143	156–214

[a] The range shown excludes the extreme value at each end.
[b] Birth orders 3 and 4 combined.
[c] Birth order 3.
[d] Birth order 4.

SOURCES:
Aberdeen: Baird et al., 1954, p. 433.
England and Wales: Butler and Bonham, 1963, pp. 3, 29.
India: Karan et al., 1972, p. 101.
New York City: Fischler, Peritz, and Wingerd, 1971, p. 406.
North Carolina: State Board of Health, 1969, p. 16
Scotland: US HEW, 1966b, p. 30.
Malaysia: Llewellyn-Jones, 1965.
New York State: Schlesinger, Mazumdar, and Logrillo, 1972, unpublished data corresponding to Figure 3, p. 261.
Vancouver, British Columbia, Canada: Tysoe, 1971.

not before age 20 or after age 30, and a second or third child only between ages 25 and 34, and no further births, US infant mortality would be reduced by 29 percent.

The positive correlation between low infant mortality and a preponderance of births in young age and low birth order categories is also suggested by the data in Table 6, which show a striking difference between the United States and four low infant mortality countries in the proportion of births within and outside low birth orders confined to ages 20–34.

The inference from these data is that despite its low level, infant mortality in Japan should be still lower, considering the concentration of births in the optimal age and birth order categories. On the other hand, in the United States, where over half the births in 1968 were outside the optimal age and birth order categories, reduction in infant mortality from the 1968 level may involve more restricted and better timing of births, as well as improvement in medical care.

TABLE 6 **Percent of total live births, and infant mortality per 1,000 live births, by maternal age and birth order, for selected countries and years**

Maternal age and birth order	*United States 1968*	*Denmark 1966*	*Sweden 1966*	*Netherlands 1968*	*Japan 1967*
	Percent of total live births				
Ages 20–29, orders 1 and 2	42.9	51.0	58.1	55.3	68.6
Ages 30–34, orders 2 and 3	4.6	7.9	11.4	10.8	14.8
Ages 20–34, orders 1, 2, and 3	47.5	58.9	69.5	66.1	83.4
Balance of ages and orders	52.5	41.1	30.5	33.9	16.6
	Infant mortality per 1,000 live births				
All ages and orders	21.8	16.9	12.6	13.6	14.9

SOURCE:
United Nations, 1970b, Table 17; 1971, Table 16.

In the United States, infant mortality declined substantially until about 1950, largely as a result of prevention and cure of infectious diseases. Between 1950 and 1965, however, the rate seemed to stagnate, declining by an average of only 1 percent per year, from a level of 29.2 deaths per thousand live births in 1950 to 24.7 in 1965. Since this was a period of great economic expansion and prosperity, failure of the rate to drop more sharply, especially when rates were falling faster and to lower levels in some West European countries, provoked widespread criticism that medical maternity care was inadequate in the United States, particularly among its low-income population. Recently, infant mortality has again been falling rapidly, by an annual average rate of 4 percent to a level of 18.5 deaths per thousand live births in 1972 (US HEW, 1973b, p. 3). It has yet to be determined to what extent improved obstetrical services and other factors such as liberalized grounds for induced abortion and a changing age and birth order mix are responsible for the new decline, but there seems no question that better obstetrical care provides only a partial explanation.

In spite of the wealth of information on infant mortality and its components, relatively few studies provide rates by maternal age and birth order for the good reason that to do so involves the laborious process of matching death with birth certificates. Nevertheless, several birth cohort populations have been so analyzed. For control by socioeconomic status, in addition to the classic analysis of single, legitimate births in England and Wales in 1949–1950 (already discussed under stillbirths), a recent publication gives US infant mortality rates by race, income, and mother's and father's education, derived from a national probability sample of 10,395 legitimate births and 2,160 deaths of legitimate infants in the three-year period 1964–1966. The socioeconomic characteristics were obtained from a mail questionnaire usually completed by the mother (US HEW, 1972c). The New York City study listed in Table 7 also provides some measure of socioeconomic control since the rates are available by race. This study investigated 7,308 infant deaths among 299,136 live births during 1966–1967. Rates for the Indian population of Arizona, obtained from a primary source, can also be considered controlled for socioeconomic status because of the homogeneity of the population.

The Israel data shown in Table 7 were based on a longitudinal study of all births in Jerusalem during 1964–1966 (Legg et al., 1969). Computer linkages of birth and death certificates provided the data for the 305,091 children born in British Columbia, Canada, during calendar years 1952–1960 (Newcombe, 1965). The Taiwan rates are from the prospective Vital Demographic Survey sponsored jointly by the Taiwan Provincial Committee on Family Planning and the Academia Sinica in which 40,000 households totaling over 200,000 persons were visited eight times each over the period May 1966–February 1969 (Sullivan, 1972).

Three other sets of rates complete the data in Table 7 on which the generalized maternal age pattern of infant mortality is based: Danish rates for 1954–1955; rates for the US 1960 birth cohort, by race; and, giving representation to an underdeveloped country, rates obtained in a continuing prospective study conducted by the Cholera Research Laboratory in Matlas Thana, Bangladesh (Stoeckel and Chowdhury, 1972). The latter, considered an unusually reliable study of births and deaths, covered a population of 117,000 in 132 villages between May 1968 and April 1969.

As can be seen in Figure 5 and Table 7, on the basis of the above studies, infant mortality rates are typically J shaped with respect to maternal age. For all birth orders combined, the rate of risk among infants of teenage mothers is 28 percent above the average for offspring of mothers aged 20–34. Mothers in their twenties run the lowest risk of losing their children in infancy; the rate of risk rises to 20 and 38 percent above the 20–34 age base for mothers in their late thirties and forties, respectively.

Disaggregated by birth order, the pattern shifts in the expected direction, from J to reverse J. Also, as birth order increases, the minimum risk shifts to a higher age group, from 26.1 years for birth order 1 to 31.6 years for birth order 2, 33.5 years for birth order 3, and 34.0 years for birth order 4 (see Table 1, p. 6). If age 26 seems high for minimizing the risk of infant mor-

TABLE 7 Infant mortality: Rate per 1,000 live births, and index number by maternal age, for selected birth orders, areas, and years

(Index based on unweighted average rate of age groups enclosed in rectangle = 100)

Birth order, area, and years	*Average rate*		*Index number for maternal age group*					
	Weighted	*Un-weighted*	*Under 20*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*
All birth orders								
Arizona, USA, 1967								
Indian population	35.5	32	49	83	82	135	160	259
Bangladesh, 1968–69	123.8	114	121	97	100	103	122	105
British Columbia, Canada, 1952–60	22.4	21	135	107	93	100	103	133
Denmark, 1954–55	25.7	24	99	104	94	102	132	
Birth weight 2,500 grams and over	(14.5)	(15)	(65)	(96)	(94)	(110)	149	
Birth weight under 2,500 grams	(215.2)	(213)	(92)	(97)	(107)	(96)	(118)	
England and Wales, 1949–50								
Socioeconomic class								
1 (high)	16.9	16	–	101	101	98	138	163
2	19.9	18	128	102	95	103	137	173
3	26.2	25	143	106	96	98	110	149
4	31.7	30	141	105	97	98	109	139
5 (low)	36.0	34	122	111	97	92	111	128
Israel, 1964–66	20.2	19	149	94	98	108	140	149
New York City, USA, 1966–67								
White	16.0	15	156	94	93	113	132	138
Puerto Rican	23.9	24	103	101	97	102	100	79
Other	34.0	32	124	107	90	103	102	91
Taiwan, 1966–69	40.4	40	102	90	90	120	155	169
United States, 1960								
White	22.2	21	134	102	96	102	108	134
Other	41.2	39	128	104	97	99	106	116
United States, 1964–66								
White, by income (US$)	(20.8)	(20)	(127)	(96)	(96)	(108)	(126)	
Under 3,000	27.3	30	83	91	80	129	147	
3,000–4,999	22.1	21	101	107	105	88	147	
5,000–6,999	17.8	17	127	85	111	104	120	
7,000–9,999	19.2	19	–	82	95	123	127	
10,000 and over	19.4	18	–	95	93	112	111	
Black, by income (US$)	39.5	36	153	95	116	89	105	
Under 3,000	42.5	38	148	91	124	85	–	–
3,000–4,999	46.8	–	–	–	–	–	–	–
5,000–6,999	22.0	–	–	–	–	–	–	–
7,000–9,999	37.6	–	–	–	–	–	–	–
10,000 and over	–	–	–	–	–	–	–	–
Median	26	24	128	101	96	102	120	138
Range (trimmed)[a]	17–47	16–40	83–153	83–107	82–116	88–129	102–155	91–173
Birth order 1								
British Columbia, Canada, 1952–60	19.8	19	131	94	89	117	153	–
England and Wales, 1949–50								
Socioeconomic class								
1 (high)	17.3	16	–	99	98	103	144	–
2	19.5	18	112	91	94	115	170	201
3	24.8	24	143	96	92	112	132	185
4	28.4	28	142	94	95	111	131	142
5 (low)	34.1	31	122	105	96	99	195	208
Israel, 1964–66	18.2	21	127	78	76	146	124	–
New York City, USA, 1966–67								
White	13.9	14	143	85	98	117	193	239
Puerto Rican	20.5	22	88	100	99	101	–	–
Other	31.0	32	102	88	80	132	147	–
United States, 1960								
White	19.1	18	127	88	90	122	140	179
Other	36.4	34	115	87	89	124	140	114
Median	20	22	127	92	94	116	144	185
Range (trimmed)[a]	17–34	16–32	102–143	85–100	80–98	101–132	131–193	142–208

(continued)

TABLE 7 **Infant mortality: Rate per 1,000 live births, and index number by maternal age, for selected birth orders, areas, and years** *(continued)*
(Index based on unweighted average rate of age groups enclosed in rectangle = 100)

Birth order, area, and years	*Average rate: Weighted*	*Average rate: Un-weighted*	*Index number for maternal age group: Under 20*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*
Birth order 2								
British Columbia, Canada, 1952–60	21.1	19	208	123	86	90	101	176
England and Wales, 1949–50								
Socioeconomic class								
1 (high)	14.9	15	–	94	94	99	113	–
2	17.2	18	–	120	85	92	103	155
3	23.4	23	179	126	88	84	102	142
4	29.2	27	240	134	88	79	99	103
5 (low)	32.8	31	201	124	91	85	100	97
Israel, 1964–66[b]	20.4	19	187	94	106	110	90	192
New York City, USA, 1966–67								
White	15.8	15	281	110	82	111	97	141
Puerto Rican	24.1	22	170	101	90	80	129	–
Other	34.4	27	197	127	76	111	86	–
United States, 1960								
White	21.1	19	194	107	85	95	113	158
Other	41.3	34	172	102	89	96	113	131
Median	22	20	196	115	88	94	102	142
Range (trimmed)[a]	16–34	15–31	172–240	94–127	82–94	84–111	90–113	103–176
Birth order 3								
British Columbia, Canada, 1952–60	21.7	19	315	144	96	98	106	124
England and Wales, 1949–50								
Socioeconomic class								
1 (high)	17.8	19	–	–	100	70	130	–
2	21.5	20	–	144	101	87	112	145
3	28.9	26	–	157	113	94	93	117
4	33.9	30	–	154	113	90	97	121
5 (low)	38.8	34	–	168	105	90	105	127
New York City, USA, 1966–67								
White	15.9	15	453	129	91	98	111	96
Puerto Rican	26.2	22	138	126	108	113	79	–
Other	34.1	26	264	157	101	79	120	–
United States, 1960								
White	21.2	18	260	137	98	94	108	141
Other	40.4	33	200	118	95	95	110	128
Median	26	22	262	144	101	94	108	126
Range (trimmed)[a]	18–39	18–33	200–315	126–157	95–113	79–98	93–120	117–141
Birth order 4								
British Columbia, Canada, 1952–60	23.1	20	–	164	114	98	88	125
England and Wales, 1949–50								
Socioeconomic class								
1 (high)	19.1	–	–	–	–	–	–	–
2	26.9	25	–	–	109	72	119	127
3	33.0	31	–	197	117	93	90	123
4	38.8	35	–	174	115	88	97	119
5 (low)	38.6	35	–	177	127	87	86	105
Israel, 1964–66[c]	17.7	18	–	129	72	90	138	168
New York City, USA, 1966–67								
White	22.0	21	–	152	105	90	105	95
Puerto Rican	25.0	26	–	97	103	90	107	–
Other	37.8	35	–	117	102	92	106	203
United States, 1960								
White	22.7	20	262	161	105	96	99	116
Other	40.4	34	220	132	98	102	100	110
Median	26	26	241	156	105	90	100	121
Range (trimmed)[a]	19–39	20–35	220–262	117–177	98–117	87–98	88–119	105–168

( ) = Figures not included in the median or the range.

[a] The range shown excludes the extreme value at each end.

[b] Includes birth order 3.

[c] Includes birth orders 5 and 6.

SOURCES:
Arizona: Department of Health, 1967.
Bangladesh: Stoeckel and Chowdhury, 1972, p. 118.
Denmark: US HEW, 1967c, pp. 11, 14.
England and Wales: Heady and Heasman, 1959, Tables 2 Ciii, 5 Biii.
Israel: Legg et al., 1969, p. 1110.
New York City: Erhardt et al., 1970 (supplemented by unpublished data provided by F. Nelson, New York City Department of Health).
Taiwan: Sullivan, 1972.
United States, 1960: US HEW, 1972a, p. 58.
United States, 1964–66: US HEW, 1972c, Table 7.
British Columbia: Newcombe, 1965, p. 93.

TABLE 8 Neonatal mortality: Rate per 1,000 live births, and index number by maternal age, for selected birth orders, areas, and years

(Index based on unweighted average rate of age groups enclosed in rectangle = 100)

Birth order, area, and years	*Average rate*		*Index number for maternal age group*					
	Weighted	*Un-weighted*	*Under 20*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*
All birth orders								
Baltimore, USA, 1960-64	20.4	19	128	100	92	108	——124——	
England and Wales, 1949-50	(16.2)	(16)	(143)	(104)	(94)	(102)	(127)	(162)
Socioeconomic class								
1 (high)	12.2	11	–	99	98	103	155	150
2	14.2	13	120	101	94	105	146	184
3	15.9	15	137	101	95	104	122	168
4	17.9	16	151	102	96	102	120	161
5 (low)	19.1	18	130	111	92	97	133	139
Hawaii, USA, 1953	17.1	17	135	96	89	115	135	110
New York State (except NYC), USA, 1936[a]	30.5	29	114	99	90	111	124	162
North Carolina, USA, 1969								
White	14.8	13	197	110	96	94	126	235
Other	22.6	20	126	99	103	98	126	181
Taiwan, 1966-68	26.7	26	113	92	88	120	——153——	
United States, 1950	(12.0)	(19)	(128)	(101)	(93)	(106)	(125)	(144)
White	18.9	18	124	101	94	105	126	145
Other	26.7	26	108	95	94	111	115	134
United States, 1960	(18.4)	(18)	(131)	(99)	(96)	(105)	(113)	(133)
White	16.9	16	126	99	96	105	114	136
Other	26.7	25	122	100	96	104	107	116
Median	19	18	126	100	94	105	126	150
Range (trimmed)[b]	14–27	13–26	113–151	95–110	89–98	97–115	114–153	116–184
Birth order 1								
England and Wales, 1949-50								
Socioeconomic class								
1 (high)	13.4	15	–	80	76	96	148	–
2	15.2	17	71	70	78	99	153	188
3	17.1	19	117	76	84	107	133	183
4	18.6	22	172	73	85	110	132	–
5 (low)	20.8	25	88	77	75	81	167	166
New York State (except NYC), USA, 1936[a]	30.7	33	99	86	83	128	104	–
United States, 1950								
White	17.8	21	96	76	80	108	136	144
Other	28.9	36	73	76	87	135	102	–
United States, 1960								
White	15.0	17	102	75	78	114	133	161
Other	25.7	31	83	72	79	115	134	–
Median	18	22	96	76	80	109	134	166
Range (trimmed)[b]	15–29	17–33	73–117	72–80	76–85	96–128	104–153	161–183
Birth order 2								
England and Wales, 1949-50								
Socioeconomic class								
1 (high)	9.8	10	–	–	87	94	119	–
2	11.7	12	–	111	83	100	117	178
3	13.4	14	138	108	85	94	121	188
4	15.5	15	233	118	89	89	122	156
5 (low)	16.4	16	170	113	80	92	128	–
New York State (except NYC), USA, 1936[a]	25.5	27	122	96	80	94	126	–
United States, 1950								
White	16.9	16	164	106	86	92	122	159
Other	25.4	26	119	93	81	129	90	–
United States, 1960								
White	16.1	16	169	96	84	98	118	159
Other	27.2	26	137	87	86	98	116	–
Median	16	16	151	106	84	94	120	159
Range (trimmed)[b]	12–26	12–26	122–170	96–113	80–87	92–100	116–126	159–178

(continued)

TABLE 8 **Neonatal mortality: Rate per 1,000 live births, and index number by maternal age, for selected birth orders, areas, and years** *(continued)*
(Index based on unweighted average rate of age groups enclosed in rectangle = 100)

Birth order, area, and years	*Average rate*		*Index number for maternal age group*					
	Weighted	*Un-weighted*	*Under 20*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*
Birth order 3								
England and Wales, 1949–50								
Socioeconomic class								
1 (high)	11.7	13	–	–	96	73	131	–
2	14.7	14	–	138	97	85	118	152
3	16.0	15	–	115	99	99	102	146
4	17.8	17	–	124	100	90	110	149
5 (low)	18.4	18	–	124	83	98	119	166
New York State (except NYC), USA, 1936[a]	26.6	26	191	104	97	96	107	108
United States, 1950								
White	19.3	18	230	125	96	94	110	142
Other	22.3	21	138	100	116	92	92	–
United States, 1960								
White	15.9	15	217	121	94	94	112	145
Other	25.8	24	158	98	91	95	114	136
Median	18	18	191	121	96	94	111	146
Range (trimmed)[b]	15–26	14–24	158–217	100–125	91–100	85–98	102–119	136–152
Birth order 4								
England and Wales, 1949–50								
Socioeconomic class								
1 (high)	14.1	–	–	–	–	–	–	–
2	17.6	16	–	–	90	61	149	151
3	17.8	17	–	164	105	93	102	157
4	20.1	18	–	178	94	103	103	120
5 (low)	18.9	18	–	149	110	83	107	–
New York State (except NYC), USA, 1936[a]	29.1	29	–	90	98	94	108	144
United States, 1950								
White	20.3	19	–	135	98	97	105	116
Other	25.0	28	154	81	85	78	137	–
United States, 1960								
White	17.2	16	229	140	100	97	103	117
Other	25.4	24	182	109	92	101	107	110
Median	19	18	182	138	98	94	107	120
Range	17–25[b]	16–28[b]	154–229	90–164[b]	90–105[b]	78–101[b]	103–137[b]	116–151[b]

( ) = Figures not included in median or range.
[a] Figures are for legitimate births only.
[b] The range shown excludes the extreme value at each end.

SOURCES:
Baltimore: Shah and Abbey, 1971, p. 44.
England and Wales: Heady and Heasman, 1959, Tables 5 Bi, 2 Ci.
Hawaii, USA: Yerushalmy et al., 1956, p. 88.
New York State: Yerushalmy, 1938, p. 261.
North Carolina: State Board of Health, 1969, p. 16.
Taiwan: Sullivan, 1972.
United States: US HEW, 1972b, Tables M and P, supplemented by absolute data on births and rates in US HEW *Special Reports* 47, no. 3, Tables 1 and 3.

tality for first births, it may be speculated that women bearing their first child at an older age are in better socioeconomic circumstances than younger women and that women in their twenties are better equipped than teenagers to protect the child against increasingly important environmental—as opposed to biological—hazards.

A study by Spiers (1972) investigated father's age and infant mortality based on North Carolina vital statistics data, with cause of death dichotomized into congenital malformations and other causes. For both groups of causes, the lowest death rates occurred when mother and father were in about the same age group. With mother's age constant, death rates were J shaped with respect to father's age; but further analysis, using father's education from an independent source as a socioeconomic measure, suggested that, except for congenital malformations, variations in infant mortality rates with father's age were attributable to irregular social circumstances.

At least half, and in developed countries about three-quarters, of infant deaths occur in the first month of life, the so-called neonatal period, from causes largely attributable to complications of delivery, prematurity, and abnormal birth weight. In Chase's analysis of several Western countries (US HEW, 1967a), unqualified prematurity (International Standard Classification Code 776)[20] accounted for an average (unweighted mean) of 22 percent of neonatal and 15 percent of infant deaths. With the addition of postnatal asphyxia and atelectasis

[20] The International Standard Classification Code is an internationally agreed upon method of classifying diseases, under the auspices of the World Health Organization.

(Code 762, lack of air in the lungs), which are associated with prematurity, these percentages were 43 and 27, respectively. If congenital malformations, some highly correlated with maternal age, are also considered (Code 750–759), more than half the causes of neonatal and infant mortality are accounted for (US HEW, 1967a, pp. 37, 42).

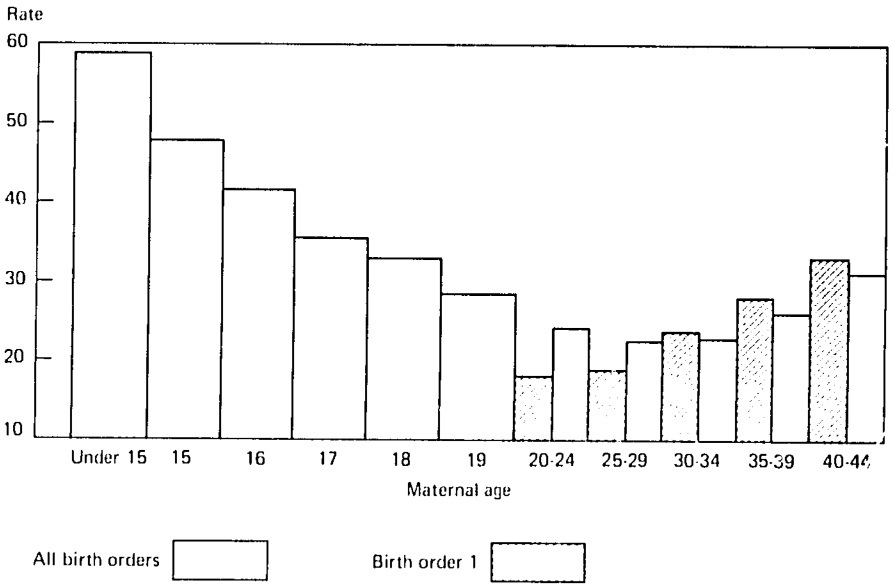
The several studies of neonatal death cross-classified by maternal age are listed in Table 8, while Figure 6 depicts the age gradient suggested by the data. Again the U- and J-shaped patterns are evident, the smoothed curves (parabolas) giving a minimum risk of neonatal death when mothers are age 23.6 for birth order 1, age 29.4 for birth order 2, age 31.5 for birth order 3, and age 33.3 for birth order 4 (see Table 1, p. 6).

A rather striking finding is the degree of excess risk of neonatal and infant mortality among offspring of teenage mothers compared with that among offspring of mothers in their twenties. At higher birth orders, a large differential is expected (because of the importance of birth spacing), but even at birth order 1, neonatal and infant deaths are over 25 percent higher for babies of teenagers than for babies of mothers in their twenties.

An unpublished tabulation of infant mortality in the US 1960 birth cohort, by single year of age for mothers under age 20, sheds further light on this subject. As can be seen in Figure 14, not only the very young teenage mothers, but even the 18- and 19-year-old mothers were disadvantaged compared with mothers in their twenties or thirties. Moreover, this finding appears even more marked when the comparison is with first order births among the older women (a valid comparison on the plausible assumption that most births to teenagers are first order births).

Because abnormal birth weight is highly correlated with high risk of infant and even childhood mortality and morbidity, it would be useful to document the well-known association between abnormal birth weight and maternal age. To control for birth order, a special tabulation was requested from the US Vital Statistics Division. The findings, based on a one per thousand sample of legitimate births in the 1964–1966 national fertility survey, show clear U- and J-shaped age patterns by birth order. These are statistically significant at the 2 percent level for the 30 cells in Table 9 constituting birth orders 1–5 for each of the six quinquennial age groups (χ^2 = 36.23, degrees of freedom = 20). For all birth orders combined (including 6 and over), the relationship was found to be statistically significant at the 1 percent level (χ^2 = 48.45, degrees of freedom = 5). With this documentation of the relationship between abnormal birth weight and maternal age, it is clear that over half the causes of infant and neonatal deaths are correlated in U- and J-shaped patterns with maternal age.

FIGURE 14 **Infant mortality: Rate per 1,000 live births, United States, 1960**



NOTE Rates are for deaths occurring within first year of life.
SOURCES For single year of age, unpublished data obtained from US HEW, courtesy of Frank Godley, Health Services and Mental Health Administration. Other data from US HEW, 1973a, Table C.

Congenital Malformations

It has long been known that certain congenital malformations are positively associated with parental age. Some are more directly related to operative deliveries, anesthetic effects, or maternal infection, such as rubella, in the early gestation period; but a few, notably chromosomal aberrations, are related unequivocally to age. According to the World Health Organization Scientific Group on Genetic Disorders, "very little is known about the factors that cause chromosomal disorders in man. The most important finding is the association between increasing maternal age and Down's and Klinefelter's syndromes in particular. A decrease in the frequency of childbirth in older mothers could lead to a significant reduction in the incidence of nondisjunctional aneuploidy" (World Health Organization, 1972, p. 16). One investigator has estimated that a reduction (presumably in the United States) of five years in the childbearing pattern after age 30 could decrease the incidence of trisomic children by 40 percent (Lenz, cited in Polani, 1967, p. 15). The extent to which such a reduction in chromosomal disorders would decrease human suffering and the burden of medical care is

TABLE 9 **Percent of live, legitimate births having abnormal birth weight by maternal age, by birth order and weight, United States, 1964–66**

Birth order and weight	*All ages*	*Maternal age group* *Under 20*	*20–24*	*25–29*	*30–34*	*35–39*	*40 and over*
All orders	16.4	15.1	14.1	16.2	19.0	22.7	24.0
Under 2,500 grams	7.8	9.0	7.1	6.9	7.7	11.2	10.3
Over 4,000 grams	8.6	6.1	7.0	9.3	11.3	11.5	13.7
Total number abnormal	1,703	215	530	430	289	182	57
Birth order 1	13.4	13.2	13.0	12.4	17.1	26.0	30.7
Under 2,500 grams	7.2	7.7	6.2	7.4	7.7	23.3	30.7
Over 4,000 grams	6.2	5.5	6.8	5.0	9.4	2.7	0
Total number abnormal	404	134	186	50	20	11	3
Birth order 2	14.4	17.9	13.6	12.9	17.1	22.3	5.7
Under 2,500 grams	7.1	10.4	6.9	5.3	7.6	14.5	0
Over 4,000 grams	7.3	7.5	6.7	7.6	9.5	7.8	5.7
Total number abnormal	378	59	176	87	38	17	1
Birth order 3	16.3	25.2	14.8	16.9	15.4	16.5	29.3
Under 2,500 grams	7.6	16.4	7.9	6.2	6.0	11.8	11.0
Over 4,000 grams	8.7	8.8	6.9	10.7	9.4	4.7	18.3
Total number abnormal	303	17	97	111	49	21	8
Birth order 4	18.5	27.1	17.8	16.2	20.0	18.8	13.9
Under 2,500 grams	7.6	27.1	9.5	4.6	7.2	8.6	3.5
Over 4,000 grams	10.9	0	8.3	11.6	12.8	10.2	10.4
Total number abnormal	221	4	43	77	62	31	4
Birth order 5	25.9	100.0	24.2	24.3	23.4	30.4	38.8
Under 2,500 grams	11.4	100.0	13.8	13.4	9.2	7.4	18.8
Over 4,000 grams	14.5	0	10.4	10.9	14.2	23.0	20.0
Total number abnormal	184	1	21	58	53	40	11
Birth order 6 and over	21.2	0	19.0	19.6	19.6	23.8	23.7
Under 2,500 grams	9.3	0	10.8	8.1	8.1	11.5	9.5
Over 4,000 grams	11.9	0	8.2	11.5	11.5	12.3	14.2
Total number abnormal	213	0	7	47	67	62	30

SOURCE:
Special tabulation prepared at the request of the Population Council by the US Department of HEW, Division of Vital Statistics, Statistical Resources Section, Rockville, Maryland (request #549), based on 10,395 live, legitimate births representing a 1 per 1,000 sample in the 1964–66 National Fertility Survey.

suggested by the finding among patients admitted to a pediatric hospital in Montreal, Canada, during 1969–1970 that about one-third were suffering from genetic disorders and congenital malformations (World Health Organization, 1972, p. 40).

A brief explanation of the technical terms and conditions may be helpful. In the normal individual, chromosomes exist in 23 pairs, each parent having contributed one of each pair. Trisomy means the presence of a third chromosome in one of the pairs. This results when, instead of receiving 23 chromosomes from each parent, the embryo receives 24 from one parent because one pair of chromosomes in the affecting parent fails to disjunct. Trisomy of autosomal (nonsex) chromosomes produces such severe abnormalities that it generally results in prenatal death. The only trisomic conditions observed in life, all of which are associated with severe abnormalities, involve (a) three autosomal (nonsex) chromosomes: number 18 (Edward's syndrome), D_1 (probably number 13, yielding Patau's syndrome), and number 21 (Down' syndrome or mongolism); and (b) various combinations of the sex chromosomes (*X* for female, Y for male).

Trisomy of the sex chromosomes, although nonfatal, produces serious deviant behavior. It includes the well-known nonsterile genotypes *XXX* and *XYY* and the sterile genotype *XXY* (Klinefelter's syndrome). Monosomy of the female sex chromosome, *XO* (Turner's syndrome), also exists, the affected females usually being severely malformed and almost always sterile. That no *YO* individuals exist suggests that the *X* chromosome is indispensable to life. The normal female is *XX*, the normal male *XY*.

Average life expectancy at birth ($^0e_0$) with trisomic conditions is as follows (Polani, 1967, pp. 3, 4):

Trisomy	$^0e_0$
D_1	3½ months
#18	2½ months
#21	10 years
Other autosomal	Prenatally Lethal
Sex chromosomes	little if any influence

Polani estimates the frequency of chromosomal abnormalities to be 1 in 250 pregnancies, with 88 percent of the abnormalities terminated prenatally by spontaneous abortion. Theoretically, a great part of the remaining 12 percent could be terminated by induced abortion because it is now possible, by cytological and biochemical analysis of the cells in the amniotic fluid (obtained by transabdominal amniocentesis), to diagnose genetic defects in the fetus. Although the method is considered practical and foolproof, it is still recognized as potentially dangerous and should be used only when there is a substantial risk of severe fetal abnormality (Ferguson-Smith, 1971).

It is generally agreed that the association between maternal age and most chromosomal aberrations, Down's syndrome (mongolism) in particular, is unequivocal. The frequency of such aberrations is, therefore, a function of the proportion of births occurring to older mothers. Penrose (1967, pp. 54–57), a pioneer in the study of the incidence of Down's syndrome, noted that changes in the maternal age distribution in England and Wales between 1939 and 1964 produced a 24 percent decline in its relative incidence during this period. Recent frequencies and average (mean) parental ages of infants with chromosomal anomalies are presented in Table 10.

It is clear from the data that, except for Turner's syndrome (XO), mean parental age of chromosomally abnormal infants is higher than average. In the case of Down's syndrome, the association between maternal age and the condition is strong enough for age to be considered the causal factor (although the mechanism of action remains speculative). The Michigan data in Table 11a show that birth order makes no difference—that for any order, at ages 35–39, the risk of Down's syndrome is three to four times average, and beyond age 40, over eight times average. For the Israel data, the relationship was statistically significant at the 1 percent level. On the average (medians of the studies reviewed), compared with the risk when women are in their early twenties, the risk of Down's syndrome among offspring is twice as great when women are in their early thirties, 6 times as great when women are in their late thirties, and 19 times as great when women are over age 40 (see Table 11a and Figure 7).

Whether or not increasing maternal age also increases the risk of the autosomal chromosomal abnormality (trisomy 13–15) that gives rise to cleft palate and harelip seems controversial. Because the condition is visible at birth, it is generally recorded on the birth certificate and so is readily matched with maternal age. In a study of 5,838,855 birth records from 17 US state departments of vital statistics, among whom 6,070 infants were identified with cleft lip and/or palate, Donahue (1965, 1967) found that 29.7 percent of mothers of babies with clefts were age 30 or over compared with 25.7 percent for a control group selected by arbitrarily taking the birth record of every fifth case after the one on which a cleft was reported. Of fathers of babies with clefts, 25.4 percent were age 35 or more compared with 24.6 percent among controls. Donahue (1965) considered these data inconclusive. Although they are statistically significant, it may be that he considered the differences trivial.

Similarly in their study of clefts among live births in New York City during 1952–1962, Conway and Wagner (1966) found rates per thousand of 1.0, 0.8, 0.7, 0.8, 1.0, and 1.4 per maternal quinquennial age group from under age 20 to age 40 and over, respectively, a pattern that is J shaped; but the findings lacked statistical significance.

As shown in Table 10, not all chromosomal aberrations are noticeable at birth, and furthermore, defective chromosomes are only one of many causes of congenital malformations. Since statistics on the incidence of congenital malformations are usually limited to those noted at birth, one study reported on here, the New York City study included in Tables 11a and 11b, tried to get more complete coverage by looking into infant mortality attributed on the death certificate to a congenital cause that had not been noted on the infant's birth certificate. The interesting statistic that turned up was that conditions missed at birth that were serious enough to cause death in the first year of life amounted to 28 percent of the malformations noted on the death certificates.[21]

Thus, the absolute rates of congenital malformations are greatly influenced by the completeness of recognition and recording on birth certificates and subsequent follow-up; but as in previous sections, it is reasonable to suppose that deficiencies in reporting and any selectivity of maternities under scrutiny have no appreciable effect on age patterns. In addition to the study of New York City births in 1958–1959

TABLE 10 Chromosomal anomalies: Rate per 1,000 births and institutional population, and mean parental age, by specified syndrome

			Rate per 1,000		*Mean age (years)*	
Syndrome	*Chromosome complement*	*Signs at birth*	*Births*	*Institutional population*[a]	*Maternal*	*Paternal*
Sex chromosomal anomalies						
Turner	45/XO	Lymph angiectatic edema of hands and feet	0.4	—	27.5	30.3
Klinefelter	47/XXY	None	2.0	10–30	33.6	37.7
Triple X	47/XXX	None	0.6	4–7	32.5	35.8
Double Y	47/XYY	None	1.4–4	10–30[b]	u	u
Autosomal chromosomal anomalies						
Down's trisomy, 21	47	Mongoloid facies, simian line	1.6	100	36.7	u
Trisomy, 16–18	47	Finger flexion, low set ears, digital arches	Rare[c]		32.8	35.2
Trisomy, 13–15	47	Cleft palate, harelip, eye defects, polydactyly	Rare[c]		u	u

u – unavailable.

[a] Data refer to people with chromosomal anomalies found in institutions such as mental hospitals; they exclude people living at home.

[b] Refers to penal institutions.

[c] McKusick (1969, p. 31) gives a figure of 1 in 4,500 live births for trisomy 16–18, 1 in 14,500 for trisomy 13–15.

SOURCE:
Hellman and Pritchard, 1971, p. 1068.

[21] Figures were as follows: Among 344,542 births in New York City in 1958–1959, 3,558 malformations were recorded on live-birth certificates, 450 on fetal-death certificates, and another 1,144 on infant-death certificates without mention on the corresponding infant's live-birth certificate (Erhardt and Nelson, 1964, p. 1490).

TABLE 11a **Down's syndrome: Rate and index number per 1,000 births (coded B) or live births (coded LB) by maternal age, for specified population groups and years**

Population group and years	Average rate	Under 20	20–24	25–29	30–34	35–39	40–44	Code base
		Age group						
	Weighted	Rate						
Great Britain, 1940s	1.51	0	0.28	0.29	1.72	3.52	14.2	B
Israel, 1959–60[a]	1.01	0.37	0.45	0.46	1.07	2.74	7.53	B
Michigan, USA, 1950–64								
Crude	(0.891)	0.432	0.428	0.512	0.866	2.64	8.59	LB
Adjusted[b]	(0.891)	0.313	0.399	0.516	0.924	2.72	8.40	LB
Birth order								
1	0.567	0.469	0.426	0.533	1.03	2.71	8.51	LB
2	0.685	0.360	0.471	0.513	1.01	2.99	7.54	LB
3	0.814	–	0.402	0.517	0.841	2.42	8.37	LB
4	1.144	–	0.393	0.486	0.877	3.00	9.40	LB
New York City, USA, 1958–59[c]	0.438	0.1	0.2	0.3	0.3	0.8	2.9	LB
Victoria, Australia, 1942–57	1.45	0.43	0.62	0.83	1.15	3.50	9.93	B
	Unweighted	Index number (unweighted average rate of age groups enclosed in rectangle = 100)						
Great Britain, 1940s	1	0	21	21	119	239	971	B
Israel, 1959–60[a]	1	30	39	39	91	231	636	B
Michigan, USA, 1950–64								
Crude	1	38	38	46	77	238	774	LB
Adjusted[b]	1	27	35	45	81	239	738	LB
Birth order								
1	1	41	37	45	87	231	724	LB
2	1	29	38	41	81	240	605	LB
3	1	–	41	50	79	230	798	LB
4	1	–	34	40	74	252	790	LB
Victoria, Australia, 1942–57	2	29	42	54	75	229	646	B
Median	.91	29	38	45	81	238	738	
Range (trimmed)[d]	0.57–1.45	27–38	34–41	39–50	75–91	230–240	636–798	

( ) – Figures not included in the median or the range.

[a] Significant at 1 percent level according to the investigator.

[b] Adjusted for birth order differences within age group.

[c] Rates shown above by five-year age groups are not carried to adequate number of significant digits for calculation of index. The index numbers shown for the two age groups, under 30 and 30 and over, are based on Table 7.3 in "The influence of season of conception on obstetric problems and casualties," Health Services Administration, New York City Department of Health and Medical and Health Research Association of New York City, Inc., June 1971 (mimeo.).

[d] The range shown excludes the extreme value at each end.

SOURCES:

Great Britain: Carter and MacCarthy, 1951, p. 84. Retrospective survey by mail questionnaire sent in 1940 to 12 maternity hospitals in Great Britain. Data are based on returns relating to 66,366 maternities.

Israel: Halevi, 1967, p. 67. Data based on congenital malformations noted during maternal postnatal hospitalization averaging 4.5 days, among 90,792 births in Israel during 1959–1960.

Michigan: Stark and Mantel, 1966, p. 692. Down's syndrome incidence rates based on birth certificates of live births in lower Michigan during 1950–1964.

New York City: Erhardt and Nelson, 1964. Supplemented by unpublished tables supplied by Nelson from the New York City Health Department Bureau of Records and Statistics dated 20 November 1962. An unusual feature of this study was the inclusion of infant deaths attributed to congenital anomalies that were not recorded on live birth certificates.

Victoria, Australia: Collman and Stoller, 1962, p. 824. Birth and death certificates were examined prospectively for all mongoloid cases during 1942–1957 in Victoria, a small southern state of Australia with population of 2 million in 1942 and 2.7 million in 1957. Among 780,168 births, 1,134 incidences of Down's syndrome occurred.

mentioned above, three other studies are presented in Table 11b on maternal age-specific rates and index numbers for all types of malformations combined: an analysis of 90,792 births in Israel during 1959–1960, in which the investigator (Halevi, 1967) paid careful attention to details on levels of statistical significance; rates based on 19,529 live births in hospitals in Chile during November 1968–October 1969 (a study that turned up as a reward in the search through the medical literature for a nonwestern, developing society); and unpublished (to date) data from the almost 500,000 births reviewed in the US Obstetrical Cooperative study (discussed in the maternal effects section).

Median index numbers are not warranted on the basis of four studies, particularly since they relate to different birth orders. Each study shows an increase in total congenital malformations with increasing maternal age. The United States, the white New York City, and the Israel data suggest, as a rough order of magnitude, twice the average congenital malformation frequency at the upper extreme of the reproductive age span. The Chilean differential is much sharper.

As for congenital malformations of nonchromosomal origin, the findings in relation to maternal age are not usually statistically significant. For example, in the Israel data, only a residual category of bone and joint malformations bore a statistically significant relation to maternal age (at the 5 percent level) (Halevi, 1967, p. 69). Likewise, an analysis of developmental malformations of

TABLE 11b Congenital malformations: Rate and index number per 1,000 births (coded B) or live births (coded LB) by maternal age, for specified population groups and years

Population group and years	Average rate	Maternal age group: Under 20	20–24	25–29	30–34	35–39	40–44	Code base
	Weighted	Rate						
Chile, 1968–69								
Birth order								
1	10.4	12.6	8.6	7.7	7.2	0	81.6	LB
2 and over	8.68	9.1	6.5	6.7	5.1	9.6	47.5	LB
Israel, 1959–60	13.2	11.2	11.6	11.7	14.8	16.0	22.1	B
New York City, USA, 1958–59								
White	14.6	13.8	13.9	13.7	15.0	16.4	26.2	LB
Puerto Rican	16.6	15.1	16.9	15.9	16.6	19.6	21.5	LB
Other	16.9	16.6	16.0	18.2	16.1	19.6	13.6	LB
United States, 1961–69								
Birth order								
1	u	44.2	46.0	48.7	53.3	62.0	–	B
2	u	38.7	40.3	42.0	44.7	50.6	–	B
3	u	–	41.8	37.0	39.3	49.2	–	B
4	u	–	37.9	39.8	42.8	47.3	65.8	B
	Unweighted	Index number (unweighted average rate of age groups enclosed in rectangle = 100)						
Chile, 1968–69								
Birth order								
1	6	213	147	130	123	0	1,385	LB
2 and over	7	131	93	97	73	137	681	LB
Israel, 1959–60	14	83	86	86	110	118	162	B
New York City, USA, 1958–59								
White	15	94	94	93	102	111	178	LB
Puerto Rican	17	88	98	92	96	114	125	LB
Other	17	95	92	104	93	111	78	LB
United States, 1961–69								
Birth order								
1	52	84	87	93	102	118	–	B
2	44	87	91	94	100	115	–	B
3	42	–	100	88	94	118	–	B
4	42	–	90	95	102	113	157	B

u = unavailable.

SOURCES:

Chile: Coke and Navarro, 1970, p. 711. Data based on infant anomalies noted at birth among 19,529 live births in Chilean hospitals during November 1968–October 1969.

Israel: Halevi, 1967, p. 67. See source, Table 11a for nature of survey.

New York City: Erhardt and Nelson, 1964. See source, Table 11a for nature of survey.

United States: Unpublished tabulation from the US Obstetrical Cooperative, Downstate Medical Center, Brooklyn, New York, prepared at the request of the Population Council. Data are based on almost half a million births during 1961–1969 occurring in participating US hospitals, which include almost all the major maternity centers in the country.

fetuses and newborn infants in Poland with particular reference to maternal age, based on six and a half years of observation of 31,857 deliveries to the tenth day after birth, found a strict correlation between age and fetal malformation only for mongolism (Baron, Kuczynski, and Pydzik, 1966).

Despite the inconclusive results (except for Down's syndrome), a higher incidence of most types of congenital malformations seems to go hand in hand with higher maternal age. The data in Table 12 on specific congenital malformations among infants of mothers under and over age 30 who gave birth in New York City during 1960–1967 indicate that for most conditions rates tend to be higher among the infants of older mothers.

Because of its high correlation with maternal age, it is difficult to establish whether paternal age is implicated with congenital abnormalities. Mongolism and clefts have received the most attention in this regard. The same inconclusiveness in the relation between clefts and maternal age also applies to paternal age. As for Down's syndrome, Sigler et al. found "no positive statistical association of paternal age" in their study of 288 mongoloid children among Baltimore births during 1946–1962, compared with a matched control group (1965, p. 641). Penrose, however, perhaps the foremost investigator of mongolism and parental age, concluded that "mongols with somatic fusion of the 21:22 type form a special group in which advancing paternal age, and not maternal age, is a highly significant aetiological factor" (1962, p. 1101).

To estimate the proportion by which congenital malformations would be reduced anywhere or everywhere in the world if childbearing were restricted to the maternal ages of 20–34 is too speculative an exercise to consider. This section, therefore, concludes by noting the point made by Halevi (1967), the investigator in the Israel study, that the impact of congenital malformations is not alone on mortality, but also on health needs. To illustrate this point, he reported that, in 1962, cases of congenital malformation in Israel comprised 159 per 10,000 hospitalizations and 195 per 10,000 hospitalization days.

Childhood Development and Performance

The older the child, the less the association between its physical or mental health and the circumstances surrounding its birth. This is attributable partly to the selecting out by early death (or removal from the population at large by institutionalization) of those seriously damaged by pre- and postnatal factors and partly to the increasing importance of environmental versus biological factors as a child matures. Nevertheless, early influences, good or bad, leave their mark throughout life.

The number of studies relating parental age at birth with performance or physical condition of child after infancy is understandably limited. In addition to the usual difficulty of linking records of subjects and controls to their birth certificates, a difficulty compounded with increasing age of the child, are problems of definition and measurement of the variable under scrutiny and of proper selection of subjects and controls. Three major techniques characterize the research design and sample selection of the studies listed in Tables 13 and 14 relating parental age (or a surrogate, notably birth weight and/or prematurity)[22] to child condition or performance.

1. Pursuit of a birth cohort, retrospectively or prospectively, to ascertain the incidence of a condition (proportion manifesting it) by parental age;

2. Identification of a sample population through a register of persons with a prescribed condition (good or bad), and comparison of the parental age at birth distribution of these subjects with a set of controls matched on other than parental age variables; and

3. Testing of a large population (school children, for example) for the variable, and comparing the distribution by parental age at birth of normal (average) versus poor or high performers.

It is clear that only the first procedure takes account of institutionalized or dead children. The second may or may not include children who failed to survive to the survey date. In the third procedure, any relationship of the variable with parental age is likely to be attenuated by the exclusion of children who, for whatever reason, are not part of the testing population.

For purposes of discussion, the performance variables are considered in three categories: (1) physical handicaps; (2) mental functioning, usually measured by reading, verbal, and IQ scores; and (3) abnormal psychological or behavior patterns. To facilitate presentation of the findings of the 13 studies relating to these variables, it was unfortunately necessary to cut across these categories in the design of Tables 13 and 14, but the reader should have no difficulty in identifying the study under discussion.

Physical Handicaps

Three of the 13 studies listed in Tables 13 and 14 deal with physical handicaps. A fourth, of childhood

TABLE 12 **Congenital malformations: Rate per 100,000 deliveries of 17 or more weeks of gestation, and index number by maternal age, by type of malformation, New York City, 1960–67**
(Index based on unweighted average rate = 100)

Type	Average rate		Index number for maternal age group	
	Weighted	Unweighted	Under 30	30 and over
Neoplasms	37.8	37.1	104	96
Hemolytic disease of newborn	61.1	73.4	65	135
Other diseases of blood and blood-forming organs	15.3	15.8	92	108
Down's syndrome	43.8	61.5	40	160
Anencephalus	57.0	57.8	97	103
Spina bifida	70.1	73.8	89	111
Hydrocephalus	63.3	65.0	95	105
Congenital eye anomalies	8.8	9.8	80	120
Other nervous system anomalies	12.8	12.8	101	99
Respiratory anomalies	11.5	11.4	103	97
Ear, face, and neck anomalies	39.2	40.9	92	108
Heart anomalies	138.1	147.0	87	113
Other circulatory anomalies	28.9	30.4	90	110
Cleft lip and/or palate	79.9	85.0	87	113
Other digestive anomalies	57.8	57.0	103	97
Hernia of abdominal cavity	76.9	76.4	101	99
Rectal and anal canal anomalies	18.1	18.2	98	102
Genital organ anomalies	54.8	55.0	99	101
Hypospadias	61.4	64.5	90	110
Urinary system anomalies	12.2	11.9	106	94
Limb anomalies	235.7	233.2	102	98
Polydactyly	150.5	143.8	110	90
Musculoskeletal anomalies	71.4	73.3	94	106
Congenital hip dislocation	4.5	4.5	106	94
Skin, hair, nails, pigmented nevus	48.1	49.5	94	106
Monstrosity	5.9	6.2	89	111
Other types	131.5	139.0	88	112
All types	1572.6	1654.0	93	107

SOURCE: New York City Department of Health and Medical and Health Research Association of New York City, Inc., 1971, Table 7.3.

[22] Birth weight is a justifiable surrogate for maternal age on the basis of the high U-shaped correlation between the two shown in Table 9.

mortality at ages 1–4 in Hawaii (see Table 13), yielded a rate of 41 percent above average among children of teenage mothers, with a rapid decline as age of mother increased. Since accidents are an important cause of childhood deaths, the implication is that teenagers are too immature to make responsible mothers.

A New York State study (see Table 13) compared the distribution by maternal age at delivery of 553 children registered as blind among the live births of 1948–1959 with the distribution of a 2 per thousand sample of all births during the same period. "While there was little difference between the observed [subjects] and expected distribution for New York City mothers, the difference for upstate New York was statistically significant ($P < 0.05$) . . . . For upstate, the ratio of observed to expected number of mothers in the combined under 20 and 35 or over age groups was 1.38" (Goldberg et al., 1967, pp. 521, 522). The pattern for New York City whites, although not statistically significant at the 0.05 level, was similar to the pattern for upstate New York, with an observed to expected ratio for mothers under age 20 and aged 35 or over combined of 1.20 (Goldberg et al., 1967).

The study of birth weight among children born in Baltimore diagnosed as having strabismus (a squint that inhibits binocular fixation) (see Table 14) found that "case infants were lighter [than controls] even when gravidity was controlled, suggesting a direct association of strabismus with birth weight" (Goldstein et al., 1967, p. 227).

Newcombe's analysis of the 213,353 live births in British Columbia, Canada, during 1953–1958 (see Table 13) is the most comprehensive study found of the relation between maternal age at birth and child handicaps. The study is unique not only for the variety of conditions investigated but also for its research design, which added to the registered handicapped children, the children, live-born during 1953–1958, who died by the end of 1961. The maintenance in British Columbia of a register of handicapped persons and the routine tabulation of all births in the province by maternal age enabled the investigator to link the record of dead and handicapped children to their birth certificates to obtain rates by maternal age. A possible criticism is that children dead or handicapped through accident were not excluded, but their inclusion presumably would have little effect on the findings and would, moreover, detract from rather than enhance any relation with maternal age.

Newcombe's study found a J-shaped relation between parental age and handicapped children, with or without the addition of children who had died. The relationship was statistically significant at the .001 level between maternal age and handicapped children (Newcombe, 1964) and between paternal age (standardized for maternal age) and handicaps plus deaths (Newcombe, 1965). For all birth orders

TABLE 13 **Index number by parental age, for specified survey populations, years, and childhood conditions (or performance)**

		Parental age group						
Survey population, years and condition	*All ages*	*Under 20*	*20–24*	*25–29*	*30–34*	*35–39*	*40–44*	*45 and over*
Baltimore, USA, institutionalized, white mental defectives (N = 404) linked to birth certificates for births during 1941–48. Expected equals age and birth order distribution of all single, white births in Baltimore in 1941–48.								
	Ratio of observed to expected × 100 by maternal age							
All birth orders	100	121	95	88	95	146		
Birth order								
1	100	133	102	75	73	210		
2	100	330	135	52	67	133		
3	100	741	105	120	58	58		
4	100	–	236	97	57	66		
5	100	–	316	128	39	55		
Baltimore, USA, prospective study of children from time their mothers registered for prenatal care to IQ testing (Stanford-Binet) at ages between 3 years 9 months and 4 years 3 months (N = 1,333 black male, 1,354 black female, 576 white children).								
Children		Index number of IQ scores by maternal age (unweighted average rate = 100)						
Black								
Male		96	97	99	102	101	105	
Female		94	98	99	101	104	104	
White		99	95	101	101	101	103	

(continued)

TABLE 13 **Index number by parental age, for specified survey populations, years, and childhood conditions (or performance)** *(continued)*

Survey population, years, and condition		All ages	Under 20	20–24	25–29	30–34	35–39	40–44	45 and over
			Parental age group						
Birmingham, England, school children representing single births during 1950–54 (N = 48,913) tested for verbal reasoning in the standard school examination for children aged 11 and older.	Average rate								
	Weighted	Un-weighted	Index number of verbal scores by maternal age (unweighted average rate = 100)						
All birth orders	100	–	–	–	–	–	–	–	
Birth order									
1	103.0	104.0	93	97	101	103	104	102	
2	101.6	101.2	93	96	101	103	104	103	
3	99.0	99.4	–	95	98	101	102	104	
4	96.8	96.9	–	95	98	100	102	105	
5	94.0	94.2	–	–	97	100	102	101	
New York State, USA, children registered as blind before 1961, born between 1 January 1948 and 31 December 1959. Expected based on a stratified sample of 2 of every 1,000 live births during study period (N = 231 upstate, 322 New York City).			Ratio of observed to expected × 100 by maternal age						
Upstate		100	146	95	78	107	118	195	
New York City		100	94	92	106	97	119	90	
Hawaii, USA, 1953, childhood mortality at ages 1–4 from retrospective survey of all women with at least one pregnancy (N = 6,039).	Rate per 1,000 live births		Index number of mortality by maternal age (all ages = 100)						
	10.3		141	102	103	77	0		
British Columbia, Canada, registered handicapped children (N = 4,711) plus child deaths (N = 6,404) through 1958 among children born alive during 1953–58 (N = 213,353).	Average rate								
	Weighted	Un-weighted	Index number of child handicap by parental age (unweighted average rate = 100)						
Handicaps plus deaths per 1,000 live births			Maternal age						
All birth orders	5.21	6.34	98	80	75	81	88	113	165
Birth order									
1	4.63	5.21	105	81	79	100	110	125	–
2	4.63	5.63	135	84	69	75	94	143	–
3	5.10	7.83	168	76	62	54	62	79	199
4	5.57	8.26	196	99	67	61	54	63	160
			Paternal age						
All birth orders[a]	5.21	5.54	110	88	91	89	97	101	124[b]
Handicapped children per 1,000 live births			Maternal age						
All disease groups[c]	2.21	2.75	82	73	78	84	92	126	171
Infective/parasitic (001–138)[d]	u	u	94	115	92	108	79	73	–
Neoplasms (140–239)[d]	u	u	96	88	96	109	132	99	–
Allergic/metabolic (240–289)[d]	u	u	79	93	123	102	76	70	–
Mental/personality (300–326)[c, d]	u	u	78	83	88	91	131	376	625
Nervous system (330–398)[d]	u	u	107	86	102	102	121	102	70
Bones/organs of movement (720–749)[d]	u	u	122	109	98	93	77	103	101
Congenital malformations (750–759)[d, e]	u	u	102	91	97	107	105	140	209
Early infancy (760–776)[d]	u	u	88	92	99	130	85	78	–
Other	u	u	136	68	101	141	73	96	302

u = unavailable.

[a] Standardized within each paternal age group for maternal age distribution.

[b] Age group 45–49. The index for age group 50 and over is 123.

[c] Age distribution significant at 0.1 percent level.

[d] Numbers in parentheses are the International Standard Classification Code.

[e] Age distribution significant at 1 percent level.

SOURCES:

Baltimore: Institutionalized subjects, Lilienfeld and Pasamanick, 1956, Table II.

Baltimore: IQ index, Lobl, Welcher, and Mellits, 1971, p. 355.

Birmingham: Record et al., 1969, p. 64.

New York State: Goldberg et al., 1967, pp. 519–531.

Hawaii: Yerushalmy et al., 1956, p. 88.

Canada: Maternal age, Newcombe, 1965, p. 97; paternal age, Newcombe and Tarendale, 1965; handicapped children, Newcombe, 1964, pp. 367–382.

TABLE 14 **Studies on relationship between specified childhood disorder and maternal age at birth (or a surrogate)**

<table>
<tr><th>Survey population and years</th><th>Survey type and sample size</th><th>Sample selection and characteristics</th><th>Relationship investigated and findings</th></tr>
<tr>
<td>Baltimore, USA, ward service live births after 1 January 1950 diagnosed with strabismus (squint that renders binocular fixation impossible) up to 1 October 1964.</td>
<td>Birth certificate linkage
SUBJECTS: 406
CONTROLS: 406</td>
<td>SUBJECTS: Single-born children registered as having strabismus (but no other diagnosed neurological disorder) in Baltimore eye clinics that serve essentially similar Negro population groups.
CONTROLS: Single-born children selected from birth certificates and matched for date and place of birth, sex, and maternal age.</td>
<td>(1) Birth weight and strabismus;
(2) Perinatal disorders and strabismus.
FINDINGS: (1) "Case infants were lighter even when gravidity was controlled, suggesting a direct association of strabismus with birth weight" (Goldstein et al., 1967, p. 227).
<table>
<tr><th>Birth weight (grams)</th><th>Percent
Subjects</th><th>
Controls</th></tr>
<tr><td>1,500 or less</td><td>3.2</td><td>0.5</td></tr>
<tr><td>2,000 or less</td><td>11.1</td><td>2.7</td></tr>
<tr><td>2,500 or less</td><td>20.2</td><td>12.1</td></tr>
</table>
(2) Mothers of strabismic children had no more complications of delivery than control mothers.</td>
</tr>
<tr>
<td>Baltimore, USA, births between 1935 and 1945.</td>
<td>Retrospective
SUBJECTS: 205
CONTROLS: 205</td>
<td>SUBJECTS: Boys with reading problems selected from hospital records with complete information on complications of pregnancy and delivery.
CONTROLS: Children without reading problems.</td>
<td>Reading disorders and maternal complications during pregnancy and delivery.
FINDINGS: Among subjects, mothers of 16.6 percent had been exposed to two or more complications compared with 1.5 percent among controls. "Children with reading disorders had a significantly larger proportion of premature births and abnormalities of the prenatal and paranatal periods than their control subjects" (Kawi and Pasamanick, 1958, p. 1423)</td>
</tr>
<tr>
<td>Baltimore, USA, children at ages 6–7 years.</td>
<td>Longitudinal (ongoing large-scale study of prematurity)
SUBJECTS: 107
CONTROLS: 350</td>
<td>SUBJECTS: Children with birth weight under 2,001 grams.
CONTROLS: Children with birth weight over 2,500 grams and matched by race, season of birth, parity, hospital of birth, and social class based on census tract data.</td>
<td>Psychological score (composite based on six measures including IQ, motor development, draw-a-person, and visual and motor coordination).
FINDINGS:
<table>
<tr><th>Composite score (Group 1-high)</th><th>Percent
Subjects</th><th>
Controls</th></tr>
<tr><td>Group 1</td><td>5</td><td>27</td></tr>
<tr><td>2</td><td>15</td><td>24</td></tr>
<tr><td>3</td><td>21</td><td>22</td></tr>
<tr><td>4</td><td>35</td><td>18</td></tr>
<tr><td>5</td><td>24</td><td>9</td></tr>
</table>
"Premature children are psychologically impaired. . . . Such impairment is not secondary to personality trait disturbances" (Wiener et al., 1965, p. 443).</td>
</tr>
<tr>
<td>Baltimore, USA, children at ages 8 and 10 years.</td>
<td>Longitudinal (ongoing large-scale study of prematurity)
SUBJECTS: Unavailable
CONTROLS: Unavailable
(Presumably pursuit of birth cohorts)</td>
<td>SUBJECTS: Children with birth weight over 2,500 grams but gestation under 38 weeks.
CONTROLS: Children with birth weight over 2,500 grams but gestation full term.</td>
<td>Premature birth of adequate weight and IQ.
FINDINGS: Subjects had significantly lower IQs than controls. Thus, prematurity makes a difference even if birth weight is adequate.</td>
</tr>
<tr>
<td>British Columbia, Canada, live births, September 1958–May 1965.</td>
<td>Longitudinal
SUBJECTS: 502
CONTROLS: 207</td>
<td>SUBJECTS: Low birth weight children (under 2,041 grams) born in Vancouver General Hospital.
CONTROLS: Children of birth weight over 2,500 grams born in same hospital and matched by social class of subjects.</td>
<td>Low birth weight and composite developmental and IQ scores at child ages 3, 6, 12, 18, 30, 48, and 78 months.
FINDINGS: Low birth weight children perform less well than matched controls, at least through infancy. At age 48 months, only high social class children had caught up to controls.</td>
</tr>
<tr>
<td>London, England, 1947–48, psychiatric patients,</td>
<td>Cross-sectional
SUBJECTS: 500
CONTROLS: 500</td>
<td>SUBJECTS: Psychiatric patients aged 15 and over selected randomly from 2,500 such patients in London Hospital.
CONTROLS: Physically ill in-patients in same hospital selected randomly and matched for age and sex (and, less reliably, for social class).</td>
<td>Maternal age at birth of subjects compared with controls.
FINDINGS:
<table>
<tr><th></th><th>Maternal age
Subjects</th><th>
Controls</th></tr>
<tr><td>Median</td><td>30.74 (±0.36)</td><td>29.53 (±0.36)</td></tr>
<tr><td>Mean</td><td>31.14 (±0.28)</td><td>30.10 (±0.29)</td></tr>
<tr><td>Standard deviation</td><td>6.35 (±0.20)</td><td>6.42 (±0.20)</td></tr>
</table>
Difference between means = 1.04 ± 0.404
$t = 2.5$, $p < .01$</td>
</tr>
</table>

(continued)

TABLE 14 **Studies on relationship between specified childhood disorder and maternal age at birth (or a surrogate)** *(continued)*

Survey population and years	*Survey type and sample size*	*Sample selection and characteristics*	*Relationship investigated and findings*
United States, around 1930.	Cross-sectional SUBJECTS: 1,045 CONTROLS: None	SUBJECTS: "Problem" cases of Institute of Juvenile Research. CONTROLS: None.	IQ (Stanford-Binet) and maternal age at birth of socially maladjusted juveniles. FINDINGS: "There is no noticeable difference in the intelligence of [children of] young mothers and of old mothers (Thurstone and Jenkins p 45).

SOURCES:
Baltimore: Goldstein et al., 1967, pp. 217–228.
Baltimore: Kawi and Pasamanick, 1958.
Baltimore: Wiener et al., 1965.
Baltimore: Wiener, 1970.
Canada: Eaves et al., 1970.
England: Gregory, 1958.
United States: Thurstone and Jenkins, 1931.

combined, the lowest handicap plus death rate was for the maternal age group 25–29. Controlled for birth order, the shift with increasing order from a J to a reverse J attests to the importance of birth spacing.

By disease category, handicaps that proved to be statistically significant in relation to maternal age were mental, psychoneurotic, and personality disorders (International Classification Categories 300–325), with $P < .001$, and congenital malformations (categories 750–759), with $P < .01$.

MENTAL PERFORMANCE

Just as British Columbia children seem to be the most thoroughly analyzed for the relation between parental age at birth and physical handicaps, the ongoing longitudinal studies of Baltimore births seem to provide most of the data linking maternal age and mental performance.

Of the seven studies in Tables 13 and 14 on the subject, five are based on Baltimore data. In the study comparing the maternal age distribution of 404 institutionalized, white mental defectives among 1941–1948 Baltimore births with the distribution of all white births in the same period (see Table 13), mothers of defectives were overrepresented in the under 20 and over 35 age groups (by 21 and 46 percent, respectively) and underrepresented in the age groups 20–34. Controlled by birth order, overrepresentation of young mothers of defectives increased with increasing birth order (Lilienfeld and Pasamanick, 1956).

Another Baltimore study of 205 boys with reading problems (see Table 14) revealed that 16.6 percent of their mothers had been exposed to two or more complications during pregnancy and delivery compared with 1.5 percent of the mothers of a control group[23] (Kawi and Pasamanick, 1958). A third Baltimore study (see Table 14) tested low birth weight children at ages 6–7 years and a control group of adequate birth weight, matched by age, race, season of birth, parity, and social class, for a composite psychological score (including IQ, motor development, draw-a-person test, and visual motor coordination), with the finding that "premature children are psychologically impaired . . . . Such impairment is not secondary to personality trait disturbances" (Wiener et al., 1965, p. 443). In another Baltimore study (see Table 14), children of adequate birth weight but immature gestation age, tested for IQ at ages 8 and 10, were found to have significantly lower IQs than matched controls. Thus, prematurity makes a difference even if weight is adequate (Wiener, 1970).

A longitudinal study of children born in Vancouver General Hospital, British Columbia (see Table 14), compared the psychological and neuropediatric responses at ages 3, 6, 12, 18, 30, 48, and 78 months of 502 low birth weight children with those of a control group of adequate weight, matched for socioeconomic class based on father's occupation and education. The findings demonstrated that low birth weight children "perform less well than control children of full birth weight, at least throughout infancy," regardless of social class. Since the differentials were greater in the lower than in the upper classes, "the effect of low social class may compound the difficulty which children of low birthweight experience" (Eaves et al., 1970, p. 17).

Two studies looked at IQ of presumably normal children in relation to maternal age (see Table 13). One was based on the verbal score achieved in the standard school examination for children aged 11 by 48,913 single-birth children out of a total of 86,630 born in Birmingham, England, during 1950–1954 (Record et al., 1969). Although the data imply a positive relation between score (the higher the score, the higher the intelligence) and mother's age, for at least two reasons the implication is unwarranted. First, the investigators concluded that "the striking association with maternal age and birth order . . . is determined mainly by differences between rather than within families" (Record et al., 1969, p. 65). Second, the research design, by omitting children incapable of going to school, can conceivably produce findings contrary to expectation.

The second study was better designed in that the children, again in Baltimore, were followed from the time their mothers registered for prenatal care. Tested for IQ (Stan-

[23] The section on maternal complications showed a direct relation between maternal age and maternal complications.

ford-Binet) at ages between 3 years 9 months and 4 years 3 months, among both blacks and whites "intellectual functioning and birth weight increased with maternal age" (Lobl, Welcher, and Mellits, 1971, p. 355). In this study, the investigators recognized that the findings related to the surviving children in the study population, but again, the selecting out of dead children from the sample necessarily renders the findings tentative or even suspect as far as the relationship between child's IQ and maternal age is concerned.

Psychological or Behavior Patterns

In this area, very little is available in relation to parental age. The London Hospital study (see Table 14), which seems well designed and executed, found a statistically significant difference in the average maternal age between psychiatric patients and a control group of physically ill patients from the same hospital, matched for age and sex and, to some extent, for socioeconomic class. According to the investigator, the mean maternal age of the psychiatric cases (31.14) exceeded that of the controls by 1.04 years. The difference, 2.58 times the standard error, implied a probability of only one in a hundred that so large a difference could have occurred by chance (Gregory, 1958).

The Thurstone and Jenkins study (see Table 14) found "no noticeable difference in the intelligence (Stanford-Binet IQ) of [children of] young and of old mothers," but cases were described merely as "problem" children, and there were no controls (1931, p. 45).

An unpublished study (Broverman and Klaiber, 1968) found that "older and younger mothers give rise to offspring who are . . . weak automatizers" (automatization being a measure of the rapidity with which a subject can reduce a routine task to a habit level). The hypothesis was offered that maternal hormonal age differentials subtly influence offsprings' psychological processes. (This study, like the Thurstone and Jenkins one above, is referred to because of the dearth of data on the relation between performance and parental age.)

To conclude this section, a study of birth order, family size, and intelligence among a total population of 19-year-old men born in the Netherlands (Belmont and Marolla, 1973) is worthy of note. Parental age was not considered, but the correlation between child's age, birth order, and family size has implications for parental age. The study is especially noteworthy because variables seemed reliably measured and the data dealt with a total birth cohort at an age when family size could be assumed to be complete. It was found that within each family size group, intelligence decreased with increasing birth order. Moreover, for the same birth order, intelligence was lower the larger the family size. Since the findings were controlled for social class, the inference can be drawn that for a given social class, the child's intelligence is maximized the fewer its siblings, or the earlier its mother stops reproducing.

DEMOGRAPHIC IMPLICATIONS OF ELIMINATING BIRTHS AT AGES OF REPRODUCTIVE INEFFICIENCY

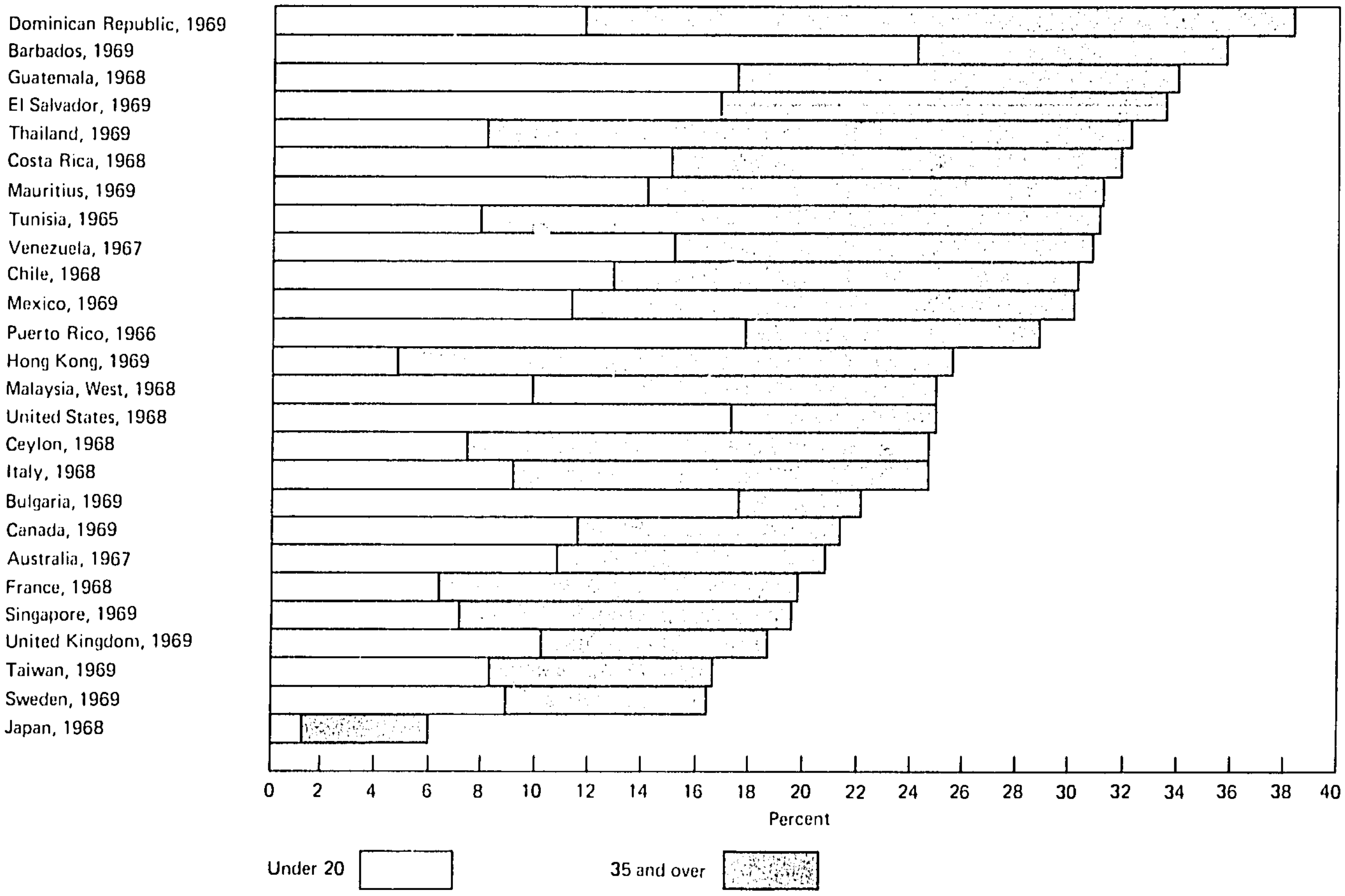
According to Coale (1972), 75 percent of fertility is produced within a 16-year period. Suppose, then, that women were to reproduce only during the 15-year period from age 20 through age 34. What would the demographic impact be? Even for the short run this is a complex and speculative question, outside the main scope of this paper. However, given the concern over the current high rate of world population growth and the attendant efforts to reduce high fertility, the question deserves some consideration here.

An upsurge in births at age 20 is one likely consequence of forgoing births at an earlier age, and, in anticipation of zero fertility after age 34, women might attempt before that age to compensate for "lost" births.[24] Nevertheless, two considerations suggest that, in the short run, little alteration in the present 20–34 fertility schedule would result from limiting reproduction to this age interval. One consideration concerns the developing world where, because much of the differential between the biological capacity to reproduce and actual fertility stems from cultural practices and taboos that are unrelated to conscious fertility control, *ceteris paribus*, the capacity to increase fertility can be considered minimal. As for the developed countries, much of the childbearing at the upper and lower ends of the reproductive age span represents unwanted or out-of-wedlock fertility, which, if averted, is not likely to be regarded as "lost." Of course some premarital fertility may substitute for postmarital fertility, but the direct relationship found between total fertility and the timing of first births (Presser, 1971) suggests that the substitution effect is small. On this line of reasoning, a gross measure of the short-run reduction in birth rates of averting births during the ages of reproductive inefficiency approximates the proportion that these births represent of total births.[25]

[24] For a discussion of the relation between fertility schedules and population growth, see in addition to Coale (1972), R. Lesthaeghe, 1971, "Nuptiality and population growth," *Population Studies* 25, no. 3 (November); also, K. G. Basavarajappa and M. Belvalgidid, 1967, "Changes in age at marriage of females and their effect on the birth rate in India," *Eugenics Quarterly* 14, no. 1 (March).

[25] The proportion of births in a given year occurring to mothers of different ages is not to be confused with the proportion of women of specified age who become mothers in a given year. The former depends upon the latter but also upon the relative number of women at different ages. The latter is the fertility schedule of a particular year, but it is hypothetical to suppose that any woman follows it throughout her childbearing years.

FIGURE 15 **Percent of live births born to mothers under age 20 and age 35 and over, for selected countries and years**



NOTE: Developing countries were selected on basis of availability of data; developed countries, to represent different geographies, economies, and fertility levels.

SOURCE United Nations, 1971, Table 14.

Available data on births by age of mother suggest that about 25 percent of births are to women who have not yet reached or are already beyond the favorable childbearing years. As shown in Figure 15, the range in the proportion of live births to women below age 20 and aged 35 or more is from 17 percent (Taiwan) to 38 percent (Dominican Republic) among 17 developing countries selected for availability of recent data on annual births by age of mother. In 11 of the 17 cases, the figure exceeds 30 percent.

Among nine developed countries, selected to represent different geographies, types of economy, and levels of fertility, the proportion ranges from 6 percent in Japan to 25 percent in the United States. Although usually classified as developing, Taiwan and Singapore typify the developed pattern, while the United States and Bulgaria are more typical of less developed societies in the proportion of births to teenage mothers. Whether it is the older or the younger mother who contributes the greater part of the births at unfavorable ages is quite variable among both developed and developing societies. The most striking case is Japan, which, with 94 percent of its births concentrated in the 20–34 age group, offers convincing evidence of the capacity of a human population to limit its reproduction to the maternal ages of optimum efficiency.

A woman reproducing in accordance with the recent world average age-specific fertility schedule (Frejka, 1973, p. 221) would produce a total of 4.74 children, 3.37 in the age interval 20–34 and 1.37, or 29 percent, in her other childbearing years. Suppose the latter were eliminated with no change in the present schedule of fertility for the age group 20–34. The effect on the average (mean) age of childbearing would be trivial—a one-year reduction, from age 28.6 to 27.6 for a woman pursuing the schedule, or, for all women now of reproductive age, from 27.6 to 27.2 years. This is an important consideration because a lowered average childbearing age acts to increase the intrinsic birth rate, and thus to offset, in part at least, the effect of reduced fertility.

Although limiting births to the age group 20–34 would have a trivial effect on the mean childbearing age, in the short run at least, it would produce a far from trivial decline in the world birth rate from about 33 or 34 (United Nations, 1970a, p. 19) to perhaps 25 per thousand population, on the assumption of little alteration in the fertility schedule in the 20–34 age group. Of course the crude death rate would also decline because of the reduction in maternal, infant, and child mortality, let us say from about 13 to 11. The effect on the growth rate would be a drop from an estimated 20 (United Nations, 1970a) to about 13 per thousand population per year. The impact of this can be seen in the fact that, at a growth rate of 20 per thousand per year, the population doubles in 35 years compared with 53 years at a rate of 13 per thousand per year.

Policy questions concerning the means by which to restrict fertility to ages 20–34 are beyond the scope of this paper, but the orders of magnitude of the probable short-term demographic changes suggest that the immediate effect of a 15-year instead of a 30-year reproductive period would be to bring relief to a world coping with growth rates that retard economic development and threaten nature's ecological balance.

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