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COMMENTARY

MATERNAL CIGARETTE SMOKING AND PERINATAL MORTALITY

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A positive association between maternal cigarette smoking and reduced infant birth weight emerges from every study of these two characteristics. Among infants of smoking mothers there is a downward shift of the entire distribution of birth weights by amounts ranging in various studies from 150 to over 300 grams. This difference is not accounted for by a very slight reduction in the average gestation of smokers' babies. The hypothesis that the relationship between smoking and weight reduction is one of cause and effect is supported by several types of evidence. First, it has been consistently observed in a wide variety of populations differing by race, geographic location, and social and economic circumstances. Within heterogeneous populations, when other factors that tend to influence birth weight are held constant, the mean weights of smokers' babies are lower than those of nonsmokers' babies in every category. Furthermore there is an inverse relationship between mean birth weights and the number of cigarettes smoked during pregnancy, an evident dose-response effect (1–12)[2].

On the other hand, whether or not the babies of smokers have higher mortality rates than the babies of nonsmokers is a question not yet answered unequivocally. Some observers have found increased fetal and neonatal loss among smokers' babies (1, 2, 5, 7, 8) and others have not (9–11). The purpose of this commentary is to suggest some possible reasons for the inconsistent findings.

The focus of concern is to learn whether babies who would otherwise be alive and healthy may die, before or after birth, because their mothers smoked. The lack of a clear answer to this important question emphasizes the need for a sharper look at existing data, and for further study. We shall discuss two general sources of inconsistency: the first arises from bias in the selection of study cases, and the second is related to certain practices in data analysis. The need for adequate numbers is self-evident, and requires no further comment.

Bias in selection. Selection of mothers by smoking habit may result in a study population consisting of two groups that are dissimilar in other respects. The basic problem is that some well-known risk factors affecting mortality are also independently related to the frequency of smoking. A few examples will illustrate this point: 1) Mortality

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[2] Reference 3 summarizes 28 previous studies of smoking and human pregnancy.

rates are higher for the first than for the second child, and rise with parity after the second child. Smoking, on the other hand, increases directly with increasing parity. Fewer mothers having their first baby are smokers, and those who do smoke tend to be light smokers (1, 6, 7, 12). A random sample of smokers and nonsmokers would therefore select more primiparous nonsmokers and more smokers having a second or later child. 2) Besides the direct relationship between parity and mortality, there can be an indirect influence of parity on the ascertainment of mortality. Unregistered early abortions would be missed in any case. Early registration, which declines with increasing parity (5), would lead to a relative increase in the ascertainment of abortions and early fetal deaths among the nonsmokers who tend to be younger and lower in parity. Past history of abortion, on the other hand, would be more frequent in older mothers of higher parities and thus more common among smokers. 3) In the United States, mortality rates are higher for black than for white babies, while white mothers smoke more frequently and heavily than black mothers. A division by smoking would tend to select more nonsmokers from the high risk group of blacks and more smokers from the low risk group of whites. 4) In Great Britain, rates of smoking and mortality are both higher in the lower social classes (1). In this instance, the risk for infants of smokers would be exaggerated by social class differences between smokers and nonsmokers.

Fortunately, the types of selective bias described above can be controlled by appropriate analytic methods. A concrete example of the interaction of social class, parity, and smoking both illustrates the problem and offers an approach for separating the smoking effect from other confounding factors. Table 1A shows the wide range of mortality by social class and parity, from a low rate of 17 per 1000 for second babies of social class 1 and 2 mothers to a high of 43 per 1000 for first babies of social class 3–5 mothers. In part B of table 1 we see that the proportion of smokers varies within these same categories, being lowest for social class 1 and 2 primiparous mothers (18 per cent) and highest for mothers of lower social class and high parities (37 per cent). In part C, mortality rates for babies of smoking and nonsmoking mothers are compared within each birth order and social class group. Mortality rates are higher among babies of smokers than of nonsmokers in every subgroup except those of high social class and low parity, where the numbers are small (1).

The analytic approach illustrated in table 1 is one way of controlling for bias in selection, if the bias is identified. This method of separating the smoking effect from other risk factors is valid, but leads rapidly to tables with many cells, small numbers, and complexity of interpretation. A number of multivariate statistical approaches are available as alternatives, setting up models to predict the mortality risk for each of the variables as if it were acting alone, with adjustment made for all the others (1, 13). Table 2 summarizes the factors now appearing to be the most important ones to control, showing their independent relationships to smoking and to mortality.

Birth weight adjustment. A number of workers have compared mortality rates for infants of smokers and nonsmokers within birthweight-specific categories, finding lower rates among the smokers' babies in the low birthweight group (1, 5, 8, 11). This procedure can obscure rather than clarify existing relationships between the two groups. If a factor affects both birth weight and mortality, the process of adjusting for one outcome effect while examining the other can be misleading. The examples in table 3 should clarify this point. Comparisons of neonatal death rates for the United States in 1950 are shown by race, parity, and plurality. Neonatal mortality rates are higher for black than for white babies, higher in multiple than for sin-

TABLE 1

Analysis of data from The British Perinatal Mortality Survey (1)

A) Perinatal mortality rates per 1000 total births by parity and social class*

Birth order	Social classes 1 + 2		Social classes 3–5		All social classes	
	No.	Rate	No.	Rate	No.	Rate
First	30/1039	29	186/4362	43	216/5401	40
Second	16/955	17	110/3771	29	126/4726	27
Third or later	24/707	34	167/4181	40	191/4888	39
All	70/2701	26	463/12314	38	533/15015	35

B) Maternal smoking by parity and social class

Birth order	Social classes 1 + 2		Social classes 3–5		All social classes	
	No.	% smokers	No.	% smokers	No.	% smokers
First	1039	18	4362	28	5401	26
Second	955	22	3771	29	4726	28
Third or later	707	25	4181	37	4888	35
All	2701	21	12314	31	15015	29

C) Perinatal mortality rates per 1000 total births by maternal smoking, by parity and social class

Birth order	Social classes 1 + 2		Social classes 3–5		All social classes	
	Nonsmoker	Smoker	Nonsmoker	Smoker	Nonsmoker	Smoker
First	29	(27)†	39	53	37	50
Second	19	(10)	27	35	25	31
Third or later	30	(45)	34	50	34	49
All	26	26	34	47	32	44

* Social class 1 is the highest.

† Figures in parentheses indicate rates based on fewer than 10 deaths.

gle births, and higher for the first than for the second child. Each of the characteristics in these three examples is also associated with differences in birth weight; black babies, babies in plural sets, and first babies are generally smaller than their counterparts. The last column in the table shows mortality rates, weight specific for babies under 2501 grams. In all instances the relative risks among "low birthweight" babies are the reverse of the situation for infants of all weights. In other words, among "low birthweight" babies, blacks survive better than whites, first babies survive better than later ones, and plural births survive better than single births (14, 15).

A clue to the basis for the apparently paradoxical relative risks for small babies is given by the fact that for each characteristic (race, plurality, parity), the mortality rates for "low birthweight" babies are lower among the group containing a larger proportion of small babies. In each of these examples, as with maternal smoking, the infant population is divided into two groups with similarly shaped distributions of birth weights but with different mean values. Cutting these distributions at a fixed point causes a different proportion of each distribution to be classified as "low birthweight." Figure 1 shows birthweight distributions of live births and of neonatal deaths by plu-

TABLE 2

Important factors affecting both smoking and mortality

Factor	Relationship between factor and:	
	Smoking	Neonatal mortality
Parity	Most studies show increase in smoking with increase in parity (1, 6, 7, 12). Finland: more smoking in young primiparas (10).	J-shaped curve. First child has higher mortality than second child. Mortality rises with parity therafter.
Maternal age	Smoking frequency may vary with age, either in association with or independent of parity (1).	Mortality increases by age within each parity after age 25 (14). High mortality at maternal ages under 20 is associated with low socioeconomic status and primiparity (13).
Calendar time	Complex changes in smoking habits with time, depending on age, race, and location should be evaluated in long term studies.	The general trend in neonatal mortality has been downward, with local variations and exceptions 1935–1971.
Socioeconomic status & race	Great Britain: more smoking in lower social classes (1). Washington Co., Maryland: more smoking in upper socioeconomic levels (2). USA: More smoking in whites (17).	Mortality increases as socioeconomic status decreases. USA: Higher mortality in blacks.

TABLE 3

Comparison of neonatal mortality rates for all live births and for low birthweight infants, by race, plurality, and parity, United States, January 1 to March 31, 1950

	All birth weights			Birth weight under 2501 gm		
	Live births	Neonatal deaths		Live births	Neonatal deaths	
		No.	Rate*		No.	Rate*
Race (14)						
White	717,133	13,521	18.8	49,934	8,779	175.8
Black	120,653	3,220	26.7	11,703	1,927	164.6
Plurality (14) (whites only)						
Single	702,966	12,183	17.3	42,459	7,501	176.7
Plural	14,167	1,338	94.4	7,475	1,278	171.0
Parity (15)						
First birth	261,269	4,980	19.1	20,205	3,116	154.2
Second birth	251,083	4,459	17.8	17,220	2,988	174.1

* Rate per 1,000 live births.

rality. The weight-specific mortality rates of "low birthweight" babies are based on 62 per cent of the deaths and 6 per cent of the births for single births, and on 96 per cent of the deaths and 53 per cent of the births for births in plural sets.

On the basis of information such as that depicted in figure 1, the World Health Organization has designated 2500 grams as a dividing line between normal and low birthweight babies as a practical way to identify a high risk group. Although gestational age is a better criterion of immaturity and risk, weight is chosen because it is more frequently and accurately known. This definition of "low birth weight" has fostered the idea that it is dangerous to be small. Although the risk of mortality is high for babies who are born at very low weights, the risk of premature birth is low, for it is obvious that we have all survived all degrees of smallness from a one cell stage to our present sizes. The vast majority of live babies (93 per cent of U.S. births in 1950) are born at weights over 2500 grams. For the remainder, low birth weight is probably not a risk factor per se, but is more likely to be

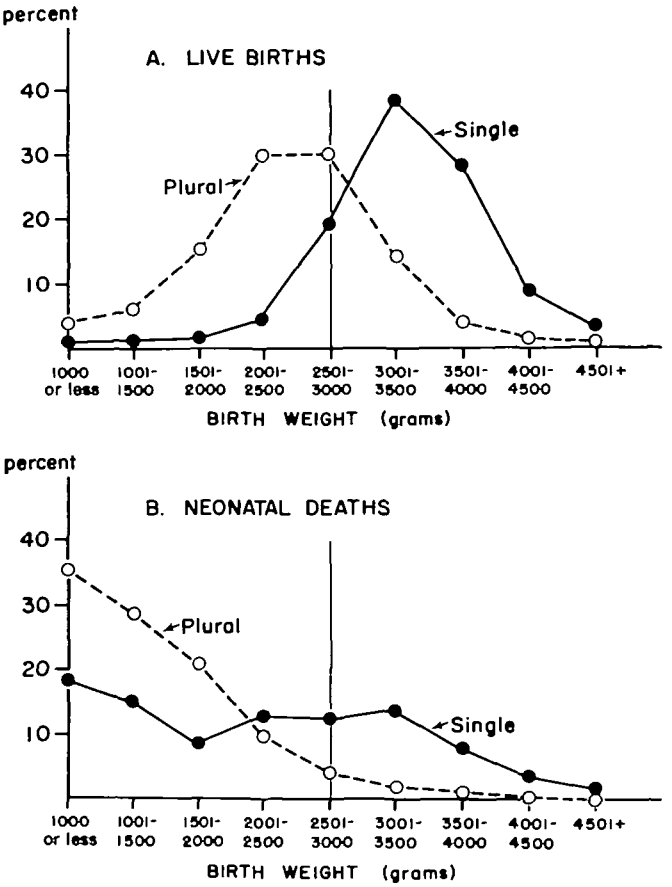

FIGURE 1. Birthweight distributions of live births and of neonatal deaths, United States, January 1 to March 31, 1950, by plurality—per cent in individual weight groups (14).

a *result* of causes that vary greatly in their influence on survival.

Short gestation (less than 37 weeks) is a major cause of small size, and occurs in about half of the babies born at 2500 grams or less. It is also almost certainly a major cause of their high mortality. The important relationship between gestational age and mortality is illustrated in figure 2, which shows that for each birth weight, neonatal mortality rates increase as gestational age decreases. At 2500 grams, for example, the death rate of 28–31-week babies is nine times that of equal-weight babies born at 37 weeks or over (15). The mortality rates of babies who are small from relatively nonpathological causes and born at term would be expected to fall on the lower curve.

Causes of "smallness" may be lethal to a high degree (early delivery, malformations, plurality, toxemia), a moderate threat to survival (small maternal stature, low socioeconomic status, primiparity, high altitude, maternal smoking), or advantageous (female sex). Because of wide disparities in the associations of smallness with mortality, it is apparent that the effect of adjusting for birth weight will also vary according to the cause or causes of low birth weight.

The assessment of a factor's lethality is usually based on the comparison of the mortality rate among persons with the factor with the rate among persons without the factor, the relative risk. It is generally accepted that the two groups so compared should be as similar as possible in all pertinent initial characteristics except the one

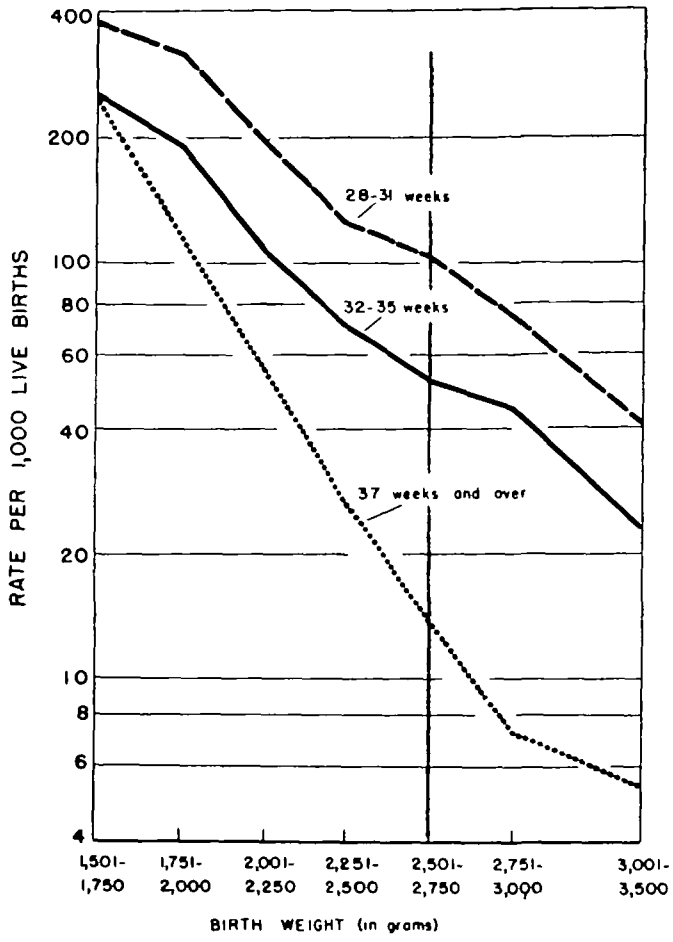

FIGURE 2. Neonatal mortality rates among single white births in hospitals, by detailed birth weight and specified gestation groups, United States, January 1 to March 31, 1950 (15). Note: Weight scale may be viewed as being continuous with the rates plotted at midpoints of the weight intervals.

under study. When similarity is not present, it can be approximated by the use of rates that are specific or adjusted for the dissimilar initial characteristics.

While there would be little disagreement about adjusting for initial or independent variables, studying one outcome by adjusting for another outcome variable is a more dubious procedure, except as an indirect measure of the correlation between the two outcomes. The use of relative risks based on rates adjusted for one outcome variable will give assessments of risks associated with a characteristic that are valid only under very special circumstances. Care must be taken not to generalize about the overall risk associated with a factor from outcome-adjusted rates.

A hypothetical example from another field will illustrate this point. Two populations are selected for study. Both appear to be similar except that one is exposed to factor A and the other is not. All members of both groups are found to have normal electrocardiographic tracings and to have no other evidence of coronary disease. Some time later, all are again examined electrocardiographically. For the sake of arithmetic simplicity, it is assumed that the two study populations are equal in size at this

TABLE 4

Hypothetical example of relative risks and mortality rates per 1000 initial population among persons with and without factor A, subdivided by another outcome variable, electrocardiographic changes

Study population	Total			Electrocardiographic change			No electrocardiographic change		
	No.	Deaths	Rate	No.	Deaths	Rate	No.	Deaths	Rate
With factor A	10,000	80	8.0	2,000	48	24.0	8,000	32	4.0
Without factor A	10,000	40	4.0	1,000	30	30.0	9,000	10	1.1
Relative risk	2.0			0.8			3.6		

time. After another interval, deaths occurring among the two populations since the second examination are recorded, with the results shown in table 4. It is clear that the experience of the total group without factor A is more favorable than that of the total group with factor A, the relative risk being 2.0. Although it is perfectly true that among persons with electrocardiographic changes the death rate for persons with A is lower than for persons without A, one would hesitate to conclude from this truncated experience that A was not harmful. Similar caution should be taken in generalizing about the effects of maternal smoking from relative risks determined among low birthweight babies.

FINDINGS AND UNANSWERED QUESTIONS

Table 5 describes the populations and methods used in major studies of maternal smoking and neonatal or perinatal mortality. Table 6 summarizes the results of these studies.

The British study and the Washington County study were based on all births and deaths in their respective populations. Both found significantly higher mortality in the offspring if the mother smoked during pregnancy. Mortality varied with other risk factors also, but in these two studies adjustment for the other factors had very little effect on the overall difference between the rates for babies of smokers and of nonsmokers. All births in 10 teaching hospitals were included in the Ontario study, and again the mortality (perinatal) was significantly higher if the mother smoked during pregnancy. Frazier's study of a homogeneous group of low socioeconomic status women in Baltimore and Kullander's study of women in Malmo, Sweden also showed increased mortality rates among babies of smoking mothers. On the other hand, Rantakallio (Finland) and Underwood (U.S. Naval installations, worldwide) found no significant differences in infant mortality by maternal smoking habit. Yerushalmy (California) found no mortality difference in white births and only a slight difference in black births to smokers and nonsmokers.

While the weight of the evidence supports the observation that perinatal mortality is significantly higher if the mother has smoked during pregnancy, a number of important questions remain to be answered.

Would analysis of the data along the lines we have suggested reveal differences in the studies where none have been found? Comparisons of mortality rates of smokers' and nonsmokers' babies should be made within subgroups according to parity, socioeconomic status, and other appropriate risk factors and not separated by birth weight. Although Underwood's population included 14 per cent non-Caucasian mothers, his mortality rates were not given by race, despite his observation that Caucasians were more frequent and heavier smokers. No information was given about parity or socioeconomic status. Yerushalmy subdivided his population by race, but not by other risk factors. In addition, he selected only one

TABLE 5

Population characteristics and controls in studies of maternal smoking during pregnancy

Study	Population	Mortality rates controlled for other factors
Butler (1)	Great Britain, 1958. All births registered during 1 week. All fetal and neonatal deaths during 3 months.	Yes; social class, parity, time of death, birth weight, type of death.
Comstock (2)	Washington County, Maryland, 1953–1963. Live births over 2500 gm, 3% sample. Live births 2500 gm or less, 33% sample. Fetal and neonatal deaths, 100%.	Yes; socioeconomic factors, parity, maternal age, sex of child.
Frazier (7)	Baltimore black women, 1959, who received prenatal care from Baltimore City Health Dept. and were scheduled for delivery at Baltimore City Hospitals.	No; homogeneous population with low socioeconomic status. Prospective.
Ontario (5)	Ontario, Canada, 1960–1961. All deliveries in 10 teaching hospitals in Ontario. Single births.	No; birth weight only.
Kullander (8)	Malmo, Sweden. 1963–1964. Repeated questionnaires starting with first visit to gynecologist.	No; homogeneous population. Prospective.
Rantakallio (10)	Oulu and Lapland, Finland, 1966. All liveborn and stillborn children weighing 600 gm or more.	No; prospective.
Underwood (9)	Deliveries at 44 worldwide U.S. Naval Installations. Single live births weighing over 500 gm. Questioned just before delivery. July 1, 1963–June 30, 1965.	No; 14% non-Caucasian. Rates not given by race.
Yerushalmy (11)	Kaiser Foundation Health Plan members. California 1960 to ? Selection of only one pregnancy for each gravida, the first terminating in a single live birth for which smoking information was available.	No; race and birth weight only.

birth per mother for inclusion in the analysis. If smoking increases with parity, as has been frequently observed, this procedure would tend to select more first babies among nonsmokers and more second babies among smokers. The well recognized increased mortality risk for first babies might therefore counterbalance any increased risk associated with smoking.

If a re-analysis along these lines did not indicate an increased risk for the babies of smoking mothers, this finding would raise the possibility that there are populations or population subgroups in which maternal smoking is *not* accompanied by increased perinatal mortality. Whereas the birthweight differences associated with maternal smoking appear to be greatest when all other circumstances of birth are optimal, there is some evidence that mortality differences are greatest in the presence of other risk factors.

Are there special inter-relationships between smoking, mortality and primiparity? Mortality is higher among first births of nonsmokers than of smokers in the Washington County study and in the British Perinatal Mortality Survey for the higher social classes (1, 2). Is there a connection between this apparently paradoxical finding

TABLE 6

Mortality findings in a number of studies of maternal smoking during pregnancy

Study	No. total births		No. stillbirths		No. neonatal deaths		Stillbirths per 1000 total births		Neonatal deaths per 1000 live births	
	Nonsmokers	Smokers	Non-smokers	Smokers	Non-smokers	Smokers	Non-smokers	Smokers	Non-smokers	Smokers
Butler (1)	11,145	4,660	215	129	146	80	19.3	27.6	13.1	17.2
Comstock (2)	7,646*	4,641*			118	100			15.4	21.5
									15.8†	21.0†
Frazier (7)	1,717	1,019	11	16	40	28	6.4	15.5	23.3	27.5
Ontario (5)	28,358	21,909			659§	645§			23.2§	29.4§
Kullander (8)‡	3,282	2,458	32	32	32	42	9.8	13.0	9.8	17.1
Rantakallio (10)	9,221	2,521			214§	59§			23.2§	23.4§
Underwood (9)	24,865	23,629					8.4	8.7	11.3	12.1
Yerushalmy (11)										
White	6,067	3,726			67	42			11.0	11.3
Black	2,219	1,071			38	23			17.1	21.5

* Total number of births represented by sample.

† Adjusted for socioeconomic factors, maternal age, parity, and sex of infant.

‡ Derived from table VI (8).

§ Perinatal.

and the repeated finding that pre-eclampsia rates are lower among smoking than among nonsmoking mothers (1, 6, 8, 12, 17, 18)? Or may it be particularly advantageous for a first-time mother to have a somewhat smaller baby?

Finally, what are the mechanisms involved in these differences between smokers and nonsmokers? The considerable levels of carboxyhemoglobin, found in both maternal and fetal blood when the mother smokes, point to hypoxia as the most probable cause both of lower birth weights and of increased mortality (19–21). There are striking parallels between births to smoking mothers and births at high altitudes. These include reduction in birth weight without a corresponding shortening of gestation, some increase in perinatal mortality, and an increase in the ratio of placental weight to fetal weight (22–24). A single study showed a significant increase in capillary hematocrit and a reduction in bicarbonate levels in babies whose mothers smoked (16), findings similar to those among babies born at high altitudes (25).

Some of these questions may be answered by further analysis of existing data. Experimental and observational studies are also needed to elucidate physiological responses to smoking, to explain the negative association of pre-eclampsia and hypertension with smoking, and to sort out the complex interactions between maternal smoking, reduced birth weight, and increased mortality in the infant.

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