RE: "INCREASED POSTPERINATAL CHILD MORTALITY AMONG CHILDREN OF MOTHERS EXPOSED TO MEASLES DURING PREGNANCY"

Aaby et al. (1) have created an imaginative natural experiment from two measles epidemics to explore the prenatal effects of measles on postnatal mortality. They advance what is, at first sight, a less-than-compelling hypothesis about effects that follow, not directly from prenatal measles, but merely from prenatal or postnatal exposure to measles. Yet it is not quite true that "no other study has examined a possible association between exposure during pregnancy and increased postperinatal mortality" (1, pp. 538-9). Their exploration of the effects of exposure to measles on perinatal and postperinatal mortality (1-3) is especially intriguing to us for the light it throws on mortality following the Dutch Famine of 1944/1945 (4).

In our studies of the effects of prenatal famine exposure, we found it necessary to invoke an additional factor to explain infant and child mortality patterns after the Dutch Famine. This factor, we concluded, was most likely to be an unspecified infection, either prenatal or postnatal. The pattern of severe child mortality after the famine led us to postulate an epidemic spreading across the country and interacting with prenatal nutritional deprivation. This conclusion conforms to the results of Aaby et al. (1). Moreover, the suggestion of Aaby et al. of different effects at different stages of gestation is a striking feature of the Dutch Famine results. Our paper on mortality after the Dutch Famine (5, p. 141) summarizes our findings:

Among major conclusions, the most general is that prenatal experience produced a discernible impact on the infant mortality of an entire population. The fatal experiences were of more than one kind. Some were nutritional in origin, some were not, and some experiences required the combination of nutritional and other factors. Some occurred early in gestation and affected survival in the perinatal and neonatal period; some occurred late in gestation and affected survival beyond the neonatal period.

Famine exposure in the first trimester related somewhat equivocally to prenatal mortality. There was a sharp rise in stillbirths and first week deaths among cohorts conceived at the height of the famine and exposed only during the first trimester of gestation (i.e., the D2 cohort). The rise appeared in all areas, and must be attributed to interaction of prenatal nutritional deprivation with an unknown factor, most likely a spreading infection affecting pregnant women. Age-specific cohort mortality curves further support the notion that interaction between severe nutritional deprivation and non-famine factors raised mortality in the D2 cohort.

In relation to famine exposure in the third trimester, the outstanding result was increase in death rates up to 90 days, but not thereafter. The increase in rates appeared in a gradient rising with each successive age group; third trimester effects were absent among stillbirths, slight below the age of 7 days (an age group with pattern transitional between first and third trimester effects), marked from 7 to 29 days, and at a maximum between 30 and 90 days of age. At 90 to 364 days, effects specific to famine were again absent. In the affected age groups, the pattern of mortality in cohort and periodic death rates was in much better accord with the effects of prenatal than of postnatal famine exposure. In the third trimester, as in the first, the effects of famine exposure are not pure and isolated from other factors.

We were unable to attribute the epidemic to a specific organism, and at the time, we did not seek out any particular organism (in later unpublished investigations, we found that scarlet fever and diphtheria related to congenital heart disease). The analysis, without forerunners as far as we know, proved to be a difficult one. Aaby et al. (1) have shown how modern analytical techniques well used can ease the difficulties.

REFERENCES

Mervyn Susser
Zena Stein
Gertrude H. Sergievsky Center
Columbia University
New York, NY 10032

In accordance with Journal policy, Dr. Aaby and his colleagues were asked if they wished to respond to Susser and Stein's letter but chose not to do so.