Neonatal Mortality Risks Similar in Careful Comparison of the CPM2000 and the 2004 U.S. Neonatal Mortality among Term Births to non-Hispanic White Women

A FOLLOW-UP ON: Johnson, K.C., Daviss, B.A., 2005. Outcomes of planned home births with certified professional midwives: large prospective study in North America. British Medical Journal 330, 1416. June 18th, 2005

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SUMMARY

We compare the CPM2000 neonatal mortality rate among planned homebirths to the U.S. National Institutes of Health (NIH) neonatal mortality rate for births in hospital to U.S. non-Hispanic white women of 37 weeks plus gestation in the year 2004. Adjustments are made to ensure that the comparison is as close as possible to comparing like with like. This includes removal from the CPM2000 death rate of intrapartum mortality, 3 deaths involving lethal birth defects unlikely to have been carried to term in the hospital population, and 1 death and 286 births among African-American and Hispanic women. After making the necessary adjustments that were possible, the neonatal death rate in both datasets was just under 1 death per 1000. Our conclusions remains unchanged from those in the original article—the neonatal mortality rate for low risk women in North America using certified professional midwives is similar to that for low risk women in hospital in the U.S., and the intervention rates are much lower. We note that the premature birth rate for the NIH non-Hispanic white births in hospital was 11.3%, more than double the rate for the women cared for by Certified Professional Midwives (CPMs). Higher prematurity is a serious concern for the U.S. hospital births, because prematurity is associated with much higher perinatal mortality and morbidity.

COMPARING LIKE WITH LIKE

The issue of comparing neonatal mortality between different datasets requires caution—it is important to be comparing like with like. In the BMJ 20005 article we provided the following caution regarding such comparisons as a footnote to Table 3:

"The Table is presented for general comparison only. Direct comparison of relative mortality between individual studies is ill advised, as many rates are unstable because of small numbers of deaths, study designs may differ (retrospective versus prospective, assessment and definition of low risk, etc.), the ability to capture and extract late neonatal mortality differs between studies, and significant differences may exist in populations studied with respect to factors such as socioeconomic status, distribution of parity, and risk screening criteria used. For example, see the study by Schlenzka. Although the crude mortality for low risk babies weighing over 2500 g intended at home was 2.4 per 1000 and intended in hospital was 1.9 per 1000, when standard methods were employed to adjust for differences in risk profiles of the two groups (indirect standardization and logistic regression), both methods showed slightly lower risk for intended home births." (Johnson and Daviss, 2005)

COMPARISON TO US NIH BIRTH DATA

The U.S. NIH provides data on all live births in the US. As with any other comparisons, however, for intrapartum and neonatal mortality we were careful in our article in the BMJ to caution against direct comparisons with the NIH as it was not a comparison of like with like. Since our article was submitted for publication in 2004, the NIH has published analysis more closely comparable than

was available at that time, and some have tried to use it as a comparison. While we still do not offer the comparison as a completely direct one, as it is the closest we have and the comparison is occurring regardless of our cautions, we offer the following adjustments that have to be made to provide the comparison of the CPM2000 analysis in as accurate a manner as is possible with the published NIH analysis.

In doing the analysis, we came to the same conclusion as we did in the original article: the neonatal and intrapartum deaths for low risk women in North America using certified professional midwives are similar to both the NIH and other studies on low risk women in hospital in the U.S., and the intervention rates are lower.

(NOTE: Definitions of mortality categorization are provided at the end of this report.)

Here are some issues:

a) Adjustment needed to the NIH 2004 Neonatal Mortality Rate to more closely approximate the rate for the year 2000

The reported NIH data for the U.S. for 2004 based on linkage of live birth and death certificates was 0.76 neonatal deaths/1000 births for Non-Hispanic white neonates of 37 weeks plus gestation. (Matthews *et al*, 2007) (The NIH only started to publish analysis of gestation – specific neonatal mortality rates by ethnic type for the 2004 data, after the BMJ article was submitted for publication.) Adjustments for the decreasing death rate among term babies documented by NIH would have placed the unpublished NIH 2000 rate slightly higher at about **0.91 neonatal deaths/1000 live births** (based on the changes in death rates for babies of 2500 grams or more between these two time periods). (Matthews *et al*, 2002, Matthews *et al*, 2007)

b) Exclusions required for the CPM2000 data

A crude comparison of the CPM2000 death rate to the neonatal mortality rate among U.S. Non-Hispanic White women with 37 week plus births would also require the following exclusions:

- 5 intrapartum deaths need to be removed as the NIH data report only on live births and thus include only neonatal deaths

- 1 home birth neonatal death that was among the 286 Hispanic and African-American births in the dataset. Both the death and 286 births need to be removed from the comparison as they did not fit the non-Hispanic white women category provided by the NIH.

- 3 neonatal deaths caused by fatal birth defects need to be removed. All three of these deaths would have occurred regardless of whether the birth was planned at initiation of labour to be in hospital or at home. We are only adjusting for "when" the death would likely have happened for better comparison of like with like for the term neonatal death rates. Had these three deaths occurred among the hospital population in the present medical culture, according to studies of abortion prevalence based on severity of anomalies in the U.S. in the 1990s, they would have had a high probability of being both discovered and aborted. Studies in Boston, St. Louis and even rural Missouri indicated that 70-90% of serious defects were aborted, (Schechtman *et al*, 2002, Peller *et al*, 2004). All 3 of these babies had serious anomalies that were both detectable and fraught with multiple anomalies. Therefore they do not fit as a legitimate part of a comparison with the NIH database. The first was a baby who was dwarf with associated lethal anomalies, discovered on ultrasound but declined for hospital birth by the Amish couple, as they did not want to be induced or have the routine autopsy done at their hospital. The second was a baby with Trisomy 13 born to a woman of 42 years. This is picked up with routine genetic testing, recommended especially for women over 35, and if the mother has declined genetic testing, usually picked up on ultrasound. The woman simply chose not to have any testing. The third was a baby with Acrocallosal Syndrome (multiple severe anomalies) that would have been picked up on ultrasound, born to a couple that disagreed

with routine ultrasound and did not have one. These three women chose to let nature take its course, in spite of their awareness of medical knowledge, and this changed the timing of the eventual deaths but not the outcome. Thus the death had nothing to do with the place of birth.

The exclusions are summarized in Table 1.

c) Crude Comparison Yields Similar Risk Estimates

Thus a crude comparison of the comparable rates for non-Hispanic white >37 week babies in hospital in the year 2000 would be about 0.91 neonatal deaths/1000 live births and for the CPM2000 0.97 deaths per 1000 live births, virtually indistinguishable.

d) Further Comparison Refinements Required

Even among low-risk women, risk will vary within subgroups of this population, so a proper comparison requires adjustment for the differences in the populations one is trying to compare. Adjustment for differences in the characteristics of the two populations (distribution of socioeconomic status, parity, risk screening, percentage of older mothers, percent Amish, percent grand multips, etc.) would be required to compare more closely. This adjustment is only possible when both datasets have collected the same information using a similar methodology. The NIH report does not provide rates of neonatal deaths for these subgroups (for example, Amish, grand multips, etc) among the non-Hispanic white 37 weeks gestation plus women, to allow us to make these adjustments for comparison to the CPM2000 dataset.

As an example of how the differences in even low-risk populations can affect the comparison, we provided the example of the study by Schlenzka in California in the section above "Comparing Like with Like".

e) The Larger Issue – Perinatal Mortality and the High Rate of Prematurity in U.S. Hospital Births

Our study focussed on term intrapartum and neonatal death rather than death before labour in order to understand the difference in outcomes between women who choose at the initiation of labour to birth in home or, conversely, to birth in hospital. The larger issue is the perinatal mortality associated with different caregivers, which includes pre-labour as well as intrapartum and neonatal deaths.

It is relevant to note that 11.3% of non-Hispanic white U.S. live births in hospital in the NIH stats happen before 37 weeks. (i.e. 11.3% of non-Hispanic White U.S. births are premature.) This is substantially different from the data among the CPMs in the year 2000 in the BMJ article, where there is an approximate 4% prematurity rate. (This rate is based on the 1.1% of women who delivered at home before 37 weeks, the 58 (1 %) of women risked out because of prematurity; an estimated half of the 205 women who were risked out for complications before labour (2%) and the 3 neonatal deaths that involved fatal birth defects that would have likely been induced or terminated before term in the hospital population.) We draw attention to this difference because the infant mortality is much higher in premature births. Even for women who deliver between 34 and 36 weeks the infant mortality is 3 times as high as it is for 37 weeks plus. Births between 34 and 36 weeks represent 2/3 of the 11.3% premature births in white non-Hispanic births in the U.S.

f) Fewer Premature Babies with CPMs

Thus either there really are fewer premature births with CPMs, or gestation reporting is different in hospital and some of the babies identified as premature in hospital were actually term or would have been included as term births (and deaths) rather than preterm in the CPM2000 project. A possible reason for this from anecdotal evidence is that defining risk can take place subsequent to delivery. Some babies who by date were term might have been re-categorized, after delivery and a newborn exam, as preterm in search for an explanation for their demise.

With either explanation for the difference between the CPM and NIH rates of prematurity, this has a serious impact on neonatal mortality estimates and comparisons that need to be carefully considered

TABLE 1: CPM2000 neonatal death rate adjusted for crude comparison to National Institutes of Health Non-Hispanic White >37 weeks neonatal death rate

Description of Deaths in the CPM2000 Study	Total Deaths	Death Rate
Total Deaths (including 3 birth defect deaths): 5 intrapartum, 9 neonatal deaths	14 deaths among 5418 births	
Deaths removed from CPM2000 to make data comparable to NIH rate:		
· 5 intrapartum		
· 3 birth defect deaths		
· 1 death among 286 Hispanic/ African- American		
"Comparable" Crude Death Rate	5 deaths among 5,132 births	0.97 neonatal deaths per 1,000 live births

and would very likely remove any excess risk in the CPM2000 in comparison to the US non-Hispanic births from the NIH data. This is also why comparison of perinatal mortality rather than neonatal mortality is usually preferred because it includes premature births and gives a better overall risk picture. The nature of our study, however, called for intrapartum and neonatal mortality in order that the risks associated with planned homebirth could be isolated--i.e., mortality that occurred from the time labour began and a homebirth was still planned. Thus we provide the extra information about the antenatal status of clients of CPMs here, to provide the more complete picture.

As we reported in our discussion of the BMJ article, there was no such difference for perinatal mortality between planned out of hospital and hospital births for all of California in 1989-1990 in a study that was able to establish similar low risk profiles because birth and death certificates in California include intended place of birth and these certificates had been linked to hospital discharge abstracts for 1989-1990 for a cesarean section study. This is one of the reasons why we identified the Schlenzka study as an important adjunct to our study.

g) Comparison of the CPM2000 Study to the MacDorman Study

A study by MacDorman *et al* (MacDorman *et al*, 2006) found that for women with no risk factors, the perinatal mortality was 0.7 deaths per thousand. It has been suggested as a comparison group for examining risk in the CPM2000 study. However that would not be a comparison of like with like. It is not appropriate as a comparison group to the BMJ study results because it excluded not only births with risk factors discovered before labour but also those births where a risk factor occurred during labour. The CPM2000 study did not exclude women with risk factors that occurred during labour. Ida Darragh's response to one blogger is succinct and accurate:

"…you continue to claim that the MacDorman study published in Birth indicates a much lower hospital neonatal mortality rate than the BMJ study of CPM births. The MacDorman study looked at only one thing - the difference in mortality rate based on cesarean or vaginal delivery. The BMJ study did screen out high-risk factors that were identified before labor began, but the MacDorman study screened out any risk factor that was identified during labor, including prolonged rupture of membranes, prolonged labor, and meconium. They looked at outcomes related only to method of delivery. The BMJ study looked at all outcomes of births attended by CPMs once labor began, and included any transports for risk factors in labor. The low mortality rate you quote was also only for the vaginal births; the cesarean births in the MacDorman study had a higher neonatal mortality rate. The BMJ mortality rate included both cesarean and vaginal deliveries. It is not unexpected that the mortality rate for vaginal births with no risk factors at any time would be lower than the rate that includes labor transports and cesarean births. As was clearly stated in the BMJ study, it is impossible to compare research outcomes without using the same criteria for evaluation."

DEFINITIONS

Intrapartum Mortality: baby who died during labour (before birth)

Neonatal Mortality: live born baby that died in the first 28 days of life

Early Neonatal Mortality: live born baby that died up to 7 days of life

Late Neonatal Mortality: live born baby that died between 7 and 28 days of life

Infant Death: live born baby that died in the first year of life

Perinatal Mortality: deaths between 20 or 22 or 28 weeks gestation and 7 days of life

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OTHER QUESTIONS ALSO ADDRESSED AT WWW.UNDERSTANDINGBIRTHBETTER.COM:

1. Why did you choose to publish this study in the British Medical Journal?
2. Why did you choose to compute an intrapartum and neonatal mortality rate rather than a perinatal mortality rate (PMR)? How were birth defects, premature births, and stillbirths reported?
3. Why does the Washington home birth study have different conclusions than almost all other articles on home birth?
4. Are you comparing apples to oranges?
5. Why did you choose not to provide confidence intervals for the intrapartum and neonatal death rates?
6. Why did you choose not to provide details about other outcomes?
7. Could the funders have biased the study in any way?