Excess Hispanic Fetal-Infant Mortality in a Midwestern Community

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SYNOPSIS

Objective. We assessed excess fetal-infant mortality for Hispanic, non-Hispanic white, and non-Hispanic black populations in five contiguous counties of Missouri and Kansas.

Methods. We conducted a perinatal periods of risk (PPOR) assessment of fetal-infant mortality using electronic linked birth-death record files from 2001 through 2005. We generated an internal reference group in accordance with established PPOR protocol. We used Kitagawa analysis to determine whether excess deaths were due to birthweight distribution (a higher frequency of prematurity or growth retardation) or to higher mortality rates once born at that birthweight (birthweight-specific mortality).

Results. We found the excess fetal-infant death rates for Hispanic and non-Hispanic white populations to be similar and considerably lower than that for non-Hispanic black populations. Among Hispanic children, we judged 21.6% of fetal-infant mortality to be excess in relation to the reference population. Within the PPOR matrix, Hispanic excess mortality rates were distributed differently from those of non-Hispanic white and non-Hispanic black populations. Among Hispanic children, 93.6% of the excess mortality could be explained by low birthweight and birthweight-specific mortality, with the greatest contribution attributable to low birthweight.

Conclusion. The excess fetal-infant mortality experience of Hispanic people in the five-county region was similar to that of the non-Hispanic white population, but was distributed differently in the PPOR model, which has significance regarding interventions targeting reductions in fetal-infant mortality.

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The infant mortality rate health indicator statistic, while useful for comparisons and tracking trends, has limitations in its utility, in that it lacks enlightenment of the factors that contribute to infant mortality. Follow-up traditional epidemiologic analyses provide a better definition of the factors associated with infant mortality, but often lack the ability to convert knowledge into community actions that can help reduce infant mortality.

As a result, the Centers for Disease Control and Prevention, through CityMatCH—a national organization of city and county health departments' maternal and child health programs—created the National Perinatal Periods of Risk (PPOR) Collaborative to promote the integration of the PPOR assessment in local maternal and child health practice.¹ It is designed to assist communities in better understanding the contributory causes of infant mortality and identification of opportunities for intervention, and is intended to be used in conjunction with other strategies.

From an operational perspective, PPOR does have a major limitation: the required number of fetal-infant deaths for inclusion of any racial/ethnic group in the analysis. The procedural criteria restrict the number of live births and fetal-infant deaths from the overall numbers of such events and then require that each racial/ethnic group have at a minimum 60 of the restricted fetal-infant deaths. This usually is not an issue for statewide data or for data for very large cities or counties, but it is an issue for smaller communities.

The Kansas City Health Department (KCHD) was an early adopter of PPOR and promotes its use by the public health community. Despite Kansas City being the largest city in Missouri and the 40th largest city in the country, it has been unable to include any racial/ethnic groups other than non-Hispanic white and non-Hispanic black populations in its PPOR analyses,^{2,3} even when combining data with Jackson County, the second most populous county in Missouri,⁴ or using multiple years' worth of data.

The PPOR literature is limited and, with the exception of a report from New York City,⁵ is silent with regard to Hispanic populations.^{6,7} Consequently, to examine fetal-infant deaths in the Hispanic population in the Kansas City area, we conducted a PPOR analysis for five core counties of the 17-county bistate Kansas City-Overland Park-Kansas City, MO-KS, combined statistical area (CSA). These five counties—three in Missouri and two in Kansas—represent 81.5% of the population of the CSA and 92.4% of the Hispanic population. In this article, we analyze the results of that bistate PPOR analysis.

METHODS

Upon receipt of authorization from each of the seven local health departments in the five core counties, the Missouri Department of Health and Senior Services and the Kansas Department of Health and Environment provided KCHD with electronically linked birth-infant death and fetal death files for the years 2001 through 2005. These files contained information for Clay, Jackson, and Platte counties in Missouri, and Johnson and Wyandotte counties in Kansas. KCHD merged the data files and conducted data extraction using SPSS® 15.0.⁸ We only used information on residents of the five-county area.

We calculated fetal mortality rates (FMRs) and infant mortality rates (IMRs) for Hispanic, non-Hispanic white, and non-Hispanic black populations in the five-county area. We calculated the percentage of deaths attributable to prematurity using the International Classification of Diseases, Tenth Revision codes for deaths associated with prematurity.⁹

We conducted the PPOR analysis in accordance with the CityMatCH PPOR protocol.¹ We used only fetal deaths at ≥ 24 weeks gestation with birthweights ≥ 500 grams (g) and live births with birthweights ≥ 500 g. We based gestational age on clinical estimates. To be included in the analysis, each racial/ethnic group had to have a minimum of 60 qualifying fetal-infant deaths; this requirement excluded all groups except Hispanic, non-Hispanic white, and non-Hispanic black. PPOR analysis uses a reference group of non-Hispanic white women aged ≥ 20 years, with ≥ 13 years of education. The reference group may be derived internally from the study group provided that there are at least 60 fetal-infant deaths for the internal group; otherwise, an external reference population, such as the one suggested by CityMatCH, must be employed. We used an internal reference group for the current study.

PPOR includes two analytical phases. The first phase estimates excess fetal and infant mortality from the birth and death certificate data. The second phase is a combination of analyses of data and information obtained from community-based organizations. Although second-stage analyses are not included in this article, protocols for the second phase are available on the CityMatCH website.^{10,11}

We distributed the fetal-infant deaths into a two-by-three table with time of death (fetal, neonatal, and postneonatal) represented by the columns and birthweight (500–1,499 g, $\geq 1,500$ g) as the rows. We combined the three cells for birthweights $< 1,500$ g and designated it as maternal health/prematurity

(MHP). We designated the three cells for birthweights $\geq 1,500$ g as maternal care (MC), newborn care (NC), and infant health (IH).

We completed additional tables for the internal reference, non-Hispanic white, non-Hispanic black, and Hispanic groups and calculated the death rate for each category. We subtracted the reference group death rates from these values to obtain the excess death rates. We calculated the number of excess deaths per category by multiplying the excess death rates by the respective total number of fetal deaths and live births divided by 1,000.

We employed the Kitagawa formula¹² to assess whether the excess deaths were due to birthweight distribution (a higher frequency of prematurity or growth retardation) or to higher mortality rates once born at that birthweight (birthweight-specific mortality). The formula uses seven birthweight ranges, with those between 500 g and 2,499 g being divided into 250 g increments, while the final birthweight range includes all births with weights $\geq 2,500$ g. The actual numbers of fetal deaths and live births, as well as fetal and infant deaths, were distributed across the birthweight ranges. This was done for the total population, the three subpopulations, and the reference group. These distributions provided the starting point for the multiple steps of the formula. An online calculator can be found at <http://www.citymatch.org>, and a description of the calculations is presented in the appendix of Cai et al.⁴

We used Chi-square tests to measure statistical significance, with values of $p < 0.05$ being considered significant.

RESULTS

Standard mortality measures

Between 2001 and 2005, the five-county area experienced 115,879 live births, 599 fetal deaths, and 805

infant deaths among residents. The overall FMR was 5.1 per 1,000 fetal deaths plus live births. The FMR for the non-Hispanic black group (9.3) was 2.2 times higher than the FMR for the non-Hispanic white (4.2) and Hispanic (4.1) groups. The overall IMR was 6.9 per 1,000 live births, with non-Hispanic black people having the highest rate (13.8), followed by Hispanic (6.4) and non-Hispanic white (5.4) populations.

Slightly more than 37.0% of infant deaths in the five-county area could be attributed to prematurity. Prematurity accounted for 47.0% of the deaths among non-Hispanic black infants, 44.8% of the deaths among Hispanic infants, and 29.6% of the deaths among non-Hispanic white infants. The percentage of deaths attributable to prematurity was not statistically different for non-Hispanic black and Hispanic populations ($p > 0.05$), but both percentages were significantly higher than that for the non-Hispanic white population ($p < 0.001$ and $p < 0.01$, respectively).

Perinatal periods of risk

The PPOR analysis used 992 fetal and infant deaths (70.6% of the total number of fetal-infant deaths) and 115,044 live births (99.3% of the total live births). The fetal-infant mortality rate for the five-county area was 8.5 per 1,000 fetal deaths and live births (Table 1). The rates for Hispanic and non-Hispanic white infants were similar (7.5 and 7.2, respectively), while the rate for non-Hispanic black infants was twice as high (14.8).

Table 2 summarizes the determination of the excess fetal-death information. Of the 992 fetal-infant deaths, we determined 302 (30.4%) to be excess deaths relative to the experience of the internal reference group. The percentage of excess deaths among the three racial/ethnic groups were 59.8% for non-Hispanic black, 21.6% for Hispanic, and 17.3% for non-Hispanic white populations. The difference in the percentage of excess deaths for Hispanic and non-Hispanic white infants was not statistically significant ($p > 0.05$), while

Table 1. Fetal-infant mortality rates per 1,000 fetal deaths plus live births^a in a five-county area in Missouri-Kansas, 2001–2005

Weight	Age at death, overall			Fetal-infant death rate: 8.5/1,000 fetal deaths + live births
	Fetal	Neonatal	Postneonatal	
500–1,499 grams	Maternal health/prematurity 374 deaths Rate: 3.2/1,000			
$\geq 1,500$ grams	Maternal care 227 deaths Rate: 2.0/1,000	Newborn care 162 deaths Rate: 1.4/1,000	Infant health 229 deaths Rate: 2.0/1,000	

^aAll rates were calculated separately and rounded to the nearest 10th.

Table 2. Perinatal periods of risk analysis of fetal and infant deaths in a five-county area, Missouri and Kansas, 2001–2005

Characteristic	Perinatal periods of risk categories				
	Maternal health/ prematurity	Maternal care	Newborn care	Infant health	Fetal-infant deaths
Internal reference group ^a					
Number of deaths	102	83	58	63	306
Death rate per 1,000 fetal deaths and live births ^b	2.0	1.6	1.1	1.2	5.9
Five-county area					
Number of deaths	374	227	162	229	992
Death rate per 1,000 fetal deaths and live births	3.2	2.0	1.4	2.0	8.5
Excess death rate ^c	1.2	0.3	0.3	0.7	2.6
Excess number of deaths ^d	144	40	31	87	302
Contribution of excess deaths (percent) ^e	48	13	10	29	100
White, non-Hispanic					
Number of deaths	196	128	104	132	560
Death rate per 1,000 fetal deaths and live births	2.5	1.6	1.3	1.7	7.2
Excess death rate	0.6	0.0	0.2	0.5	1.2
Excess number of deaths	42	2	16	37	97
Contribution of excess deaths (percent)	43	2	17	38	100
Black, non-Hispanic					
Number of deaths	121	59	35	71	286
Death rate per 1,000 fetal deaths and live births	6.2	3.0	1.8	3.7	14.8
Excess death rate	4.2	1.4	0.7	2.5	8.9
Excess number of deaths	82	28	13	48	171
Contribution of excess deaths (percent)	48	16	8	28	100
Hispanic					
Number of deaths	46	24	16	16	102
Death rate per 1,000 fetal deaths and live births	3.4	1.8	1.2	1.2	7.5
Excess death rate	1.4	0.2	0.1	0.0	1.6
Excess number of deaths	20	2	1	–1	22
Contribution of excess deaths (percent)	91	9	5	–5	100

^aFive-county area, non-Hispanic white women aged ≥ 20 years with ≥ 13 years of education

^bRates were calculated separately and rounded to the nearest 10th.

^cExcess death rate = death rate for category–death rate for reference group category

^dExcess number of deaths = excess death rate multiplied by total number of fetal deaths and live births divided by 1,000

^ePercent contribution of excess deaths = excess deaths per category divided by total excess deaths

the percentage of excess deaths for non-Hispanic black infants was highly significant from those two groups ($p < 0.001$).

The excess fetal-infant death rates for Hispanic and non-Hispanic white groups were similar (1.6 and 1.2, respectively) and considerably lower than the rate for the non-Hispanic black group (8.9). Yet, the excess mortality rates were distributed differently among Hispanic people. Overall and for non-Hispanic white and non-Hispanic black groups, excess mortality was concentrated in the MHP and IH categories, being 1.3

times higher in the former than in the latter. These two PPOR categories contributed to between 76.0% and 81.0% of the excess deaths for these three groups. Among Hispanic people, however, excess mortality was concentrated in the MHP category (91.0%) and did not contribute to excess mortality in the IH category.

The results of the Kitagawa analysis are shown in Table 3. Overall, 62.9% of the excess fetal-infant deaths were attributable to low birthweight and 37.1% were attributable to birthweight-specific mortality. Within the MHP category, low birthweight and birthweight-specific

mortality contributed to less than half of the excess fetal-infant mortality for all groups except Hispanic. Among Hispanic people, 93.6% of the excess mortality could be explained by these two factors, with the greatest contribution attributable to low birthweight.

DISCUSSION

Demographically, Hispanic people are the fastest-growing ethnic group in the Kansas City-Overland Park-Kansas City, MO-KS, CSA. The July 2007 U.S. Census Bureau population estimate was 2,027,682, with 6.9% being Hispanic—a 38.0% increase from Census 2000. More than 92.0% of the Hispanic population was white. In the five-county core area of the CSA, Hispanic people constituted 7.8% of the population—a 37.0% increase from Census 2000, with more than 92.0% being white and more than 77.0% being of Mexican heritage. The age structure of the Hispanic population is decidedly younger, and the Hispanic general fertility rate has been two to three times higher than that of non-Hispanic white and non-Hispanic black groups.¹³ While births have been declining for the non-Hispanic white population and remaining relatively stable for the non-Hispanic black population, the number of Hispanic births has been on an upward trajectory.

The literature suggests that native- and foreign-born Hispanic people in the United States have perinatal outcomes and infant mortality rates similar to that of non-Hispanic white populations.^{14–16} The FMRs and

IMRs of the five-county area were consistent with those observations. For each of these indicators, the two groups were very different from the non-Hispanic black population. However, the two groups differed very significantly in the percentage of infant deaths attributable to prematurity. Further, the 44.8% prematurity rate for Hispanic infant deaths in the five-county area was significantly higher ($p < 0.05$) than that reported nationally for Hispanic people (34.0%) in 2005.¹⁷ A tabulation of specific causes of infant deaths in the five-county area attributed 55.0% of the deaths among Hispanic infants to disorders related to length of gestation/fetal growth and to congenital malformations/chromosomal anomalies. This percentage was significantly higher ($p < 0.05$) than for non-Hispanic white (42.2%) and non-Hispanic black (42.1%) groups.

The current PPOR analysis found that Hispanic and non-Hispanic white populations have similar fetal-infant mortality rates (7.5 and 7.2, respectively) that were half that for non-Hispanic black people (14.8). This observation differed from the PPOR results published for New York City, in which the rate for Hispanic people (10.0) was intermediate between that for non-Hispanic white (7.3) and non-Hispanic black (18.2) people.⁵ In New York City, it was the Asian/Pacific Islander population whose rate (7.2) was similar to that of non-Hispanic white people.

It is well-recognized that there are ethnic variations in Hispanic health, with Mexican people having health advantages and Puerto Rican people having health

Table 3. Percentage contribution to the differences in excess fetal and infant mortality rates by Kitagawa analysis in a five-county area, Missouri and Kansas, 2001–2005

<i>Perinatal periods of risk category</i>	<i>Percent attributable to low birthweight</i>	<i>Percent attributable to birthweight-specific mortality rates</i>	<i>Total (percent)</i>
Maternal health/prematurity (birthweight 500–1,499 grams)			
Five-county area	53.4	–5.1	48.3
White, non-Hispanic	41.5	2.8	44.3
Black, non-Hispanic	57.2	–8.9	48.3
Hispanic	85.0	8.6	93.6
Maternal care, newborn care, and infant health (birthweight $\geq 1,500$ grams)			
Five-county area	9.5	42.2	51.7
White, non-Hispanic	9.5	46.1	55.6
Black, non-Hispanic	10.7	41.0	51.7
Hispanic	–13.5	19.6	6.1
Total			
Five-county area	62.9	37.1	100.0
White, non-Hispanic	51.0	49.0	100.0
Black, non-Hispanic	67.9	32.1	100.0
Hispanic	71.6	28.4	100.0

disparities.¹⁷ Neither the present PPOR analysis nor the New York City study addresses Hispanic subgroups, yet they are the only published reports known to the authors in which excess fetal-infant mortality information for Hispanic people is presented (the New York City analysis did not use the Kitagawa analysis). In the five-county region, the excess fetal-infant mortality among Hispanic people was concentrated in the MHP category (87.5%). Of the MHP excess mortality, 93.8% was attributable to low birthweight and birthweight-specific mortality. This was very different from non-Hispanic white and non-Hispanic black populations, among which excess fetal-infant mortality and mortality attributable to low birthweight and birthweight-specific causes were nearly evenly divided between the MHP and a combination of the other categories.

The literature shows that low birthweight rates are similar for Hispanic and non-Hispanic white populations, with the exception of Puerto Rican people, who have a higher rate.^{18–20} Also, foreign-born Hispanic women are less likely to have low-birthweight infants than their U.S.-born counterparts.²¹ In the five-county area, women who were U.S.-born had a low-birthweight rate of 7.3% compared with 5.3% for those who were foreign-born ($p < 0.01$).

While the current PPOR examination of fetal-infant mortality focuses on an emergent Hispanic population, Asian populations also are increasing in communities across the nation,²² through immigration and relocation. Therefore, analyses of fetal-infant mortality, whether employing traditional approaches and/or PPOR, should, if feasible, include emerging minority populations.

In general, PPOR analyses report the highest excess fetal-infant mortality rates in the MHP and IH categories. The relative contribution of these categories can vary by geography and race/ethnicity. The inclusion of the Kitagawa analysis, as was done in this article, can further refine the data and assist in the selection of intervention strategies. Because excess fetal-infant mortality among Hispanic people was so concentrated in the MHP category in the five-county region, intervention strategies would target those social, behavioral, and medical factors that contribute to very low birthweight and premature infants, and could place less emphasis on issues such as sudden infant death syndrome. Examples of such interventions would be those that target health-compromising behaviors,^{23,24} low rate of weight gain during pregnancy,²⁵ inadequate prenatal care,²⁶ and maternal age.²⁷

Studies based on birth and death certificates must contend with missing data elements that can influence the reliability and validity of the findings.²⁸ Missing birth certificate data, for example, have been reported

to be associated with those groups at highest risk for perinatal problems.²⁹ In our study, 1.3% of the birth and death certificates lacked information regarding race, while 3.8% had no responses recorded with regard to Hispanic ethnicity. When there are more than 10.0% missing data for both birthweight and gestational age in fetal, infant, or combined deaths, or when more than 10.0% are imputed, the PPOR technique cannot be used.

In the current study, 0.6% of the certificates lacked birthweight information, while 1.0% lacked any gestational age information. We used the physicians' clinical estimate of gestation because a high number of fetal death records in the Kansas City metropolitan area lacked gestational age based on the date of last normal menstrual period.³⁰ The clinical estimate is typically less than the last normal menstrual period estimate,³¹ and there are arguments that the last normal menstrual period misclassifies gestational duration and that clinical estimates should be used.³² There are historic concerns with underreporting of fetal deaths, particularly at earlier gestational ages and often for minority racial/ethnic groups, although the concerns could not be addressed by the current study.^{33,34}

CONCLUSION

The information presented in this article on the fetal-infant mortality experience of the Hispanic population in five core counties of the bistate Kansas City-Overland Park-Kansas City, MO-KS, CSA represents the first phase of the PPOR process. It illustrates the value of PPOR to assist communities in parsing their fetal-infant mortality along racial/ethnic groupings. It also shows that while two groups may have similar overall fetal-infant mortality rates, there may be significant differences in how those deaths are distributed, which, in turn, can influence what interventions are appropriate for each group. First-phase PPOR findings should be used to inform policy regarding appropriate interventions in the second phase of PPOR as well as for program planning and assessment. The key element in decreasing fetal-infant mortality must focus on developing a community response to the information gleaned from the first PPOR phase.

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