Feto-infant mortality in Southern Nevada: A Perinatal Periods of Risk (PPOR) assessment

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Abstract

Objective
This report presents recent trends relating to perinatal mortality in Southern Nevada for available data years from 2000 through 2012.

Data and methods
Data from the live birth/fetal death (preliminary from 2011 onwards) and death (preliminary from 2012 onwards) registries were used to develop rate estimates. The Perinatal Periods of Risk approach was adopted to provide an action-oriented perspective on the relative contributions of Maternal Health/Prematurity, Maternal Care, Newborn Care and Infant Health to the perinatal mortality burden, and to identify areas with the highest potential for mortality reduction.

Main results
The feto-infant death (FID) rate showed a slight uptick between 2009-10 and 2011-12, despite a long-term decreasing pattern. The vast majority of FIDs occurred in the Maternal Health/Prematurity and Maternal Care periods. NHB had FID rates well in excess of the NHW reference for all risk periods. Importantly, the birthweight pathway (high rates of VLBW), rather than that of mortality (poor survival rates), was the predominant cause of excess Maternal Health/Prematurity deaths among NHBs, underscoring the need to address preconception health gaps.

Abbreviations
NHB: non-Hispanic black
NHW: non-Hispanic white
LBW: low birth weight
VLBW: very low birth weight
LBFD: live births and fetal deaths
CI: Confidence interval

Infant mortality is generally considered one of the best barometers of a community's socio-economic wellbeing, as well as its perinatal health. It is related to a range of factors including socio-economic and behavioral characteristics, environmental and biomedical factors, genetics, as well as the quality and accessibility of health care services. Many of these risk or protective factors also impact fetal mortality (stillbirths and miscarriages), a reproductive loss which is less understood in comparison with infant deaths. This report examines both fetal and infant mortality in order to provide a more complete picture of perinatal death in Clark County, and to help focus action where it is most needed.

There were 8.3 feto-infant deaths (FID) per 1,000 live births or fetal deaths (≥24 weeks of gestation) weighing 500 grams or greater, corresponding to 435 perinatal deaths in 2011-12. This was an upturn from the rate in 2009-10 (7.8 per 1,000; 427 deaths), albeit a slight decrease from that in 2003-04 (8.4 per 1,000; 430 deaths). Over the period 2003-04 to 2011-12, the proportion of perinatal deaths that were fetal deaths rose from 40% to 51.5%, and the number of fetal deaths from 172 to 224. To help identify vulnerable neighborhoods or subpopulations with regard to perinatal mortality, spatial variations in FIDs were depicted using zip code as the enumeration area for the 2010-12 period (Appendix A).

Methods

Data sources

Using a previously developed linkage methodology (Appendix B), the live birth cohort for the period 2000-2012 was successfully linked with infant deaths during 2000-2013 (there were 1,962 infant deaths with linked birth certificates issued in 2000-2012, compared to a period infant death total of 1,966). The linked birth/infant death registrations provided the basis for analyzing infant deaths of various maternal as well as birth weight and gestational age characteristics. Analyses using the PPOR approach were based on live births/linked infant deaths between 2003 and 2012, merged with fetal deaths of the same period (a gestational age at death requirement was added to the fetal death registry in 2003).

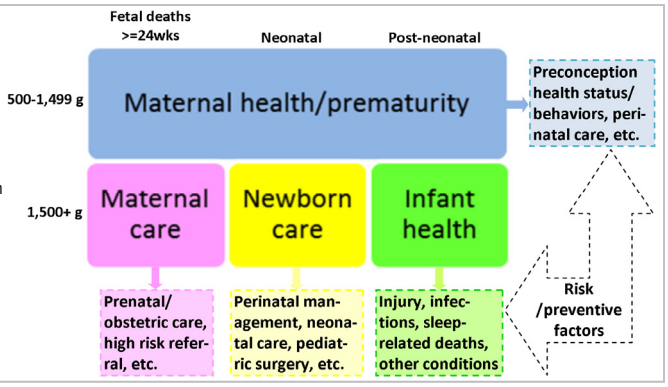
The PPOR framework

The PPOR methodology was first developed by the World Health Organization to guide infant death prevention activities in developing countries. It has recently gained much attention in the U.S. as a framework for targeting investigations and preventions. As an action-oriented community process, the PPOR consists of three main stages: 1) engaging stakeholders to establish task force and community readiness; 2) data assessment to identify important periods of risk and causal pathways contributing to excess feto-infant mortality; and 3) targeting resources and actions to address disparate risks and opportunity gaps. Collectively, the resulting integrated approach offers both a measurement tool and a stimulus to action, bringing stakeholders together to build consensus and partnerships around focus areas likely to have a large impact on maternal and child health (MCH). At the core of the PPOR structure are Phase I and II analyses, comprising the data assessment stage.

Phase I analysis

The Phase I analysis sorts fetal and infant deaths into four periods of risk based on birth/fetal weight and age at death, with each period defined by different risk and preventive characteristics as well as intervening opportunities (Box 1).[1] Maternal Health/Prematurity deaths include fetal (24 or more weeks of gestation) and infant deaths weighing 500-1,499g. The very low feto-infant weight characterizing this period reflects important antecedents such as poor preconception health and/or inadequate access to quality health care across the life span, including before and between pregnancies. Regarding deaths with a birth/fetal weight of 1,500g or more, mortality risks are further divided into the Maternal Care (fetal deaths at 24+ weeks of gestation), Newborn Care (neonatal [0-27 days] deaths), and Infant Health (postneonatal [28-364 days] deaths) periods. Maternal Care deaths point to gaps in antenatal care, particularly management of obstetric risks (e.g. diabetes, seizures, postmaturity) or other medical problems. Newborn Care and Infant Health deaths highlight challenges in neonatal care (e.g. medical/surgical treatment of congenital anomalies) and postneonatal risk management (e.g. Sudden Infant Death Syndrome [SIDS] and injury prevention) respectively. Of note is that fetal deaths less than 24 weeks of gestation and feto-infant deaths of less than 500g were excluded from the PPOR examination to limit pregnancy events to those physically viable and to improve data comparability across time. The intent of the PPOR partitioning is to identify areas with the greatest potential for mortality reduction, and to help guide planning and implementation of interventions aimed at addressing these different windows of opportunities. With further analysis by race/ethnicity, the approach also provides a means to examine excess mortality risks attending population subgroups (relative to a reference race/ethnicity) across the risk periods. This helps paint a broader picture of gaps between population groups with regards to MCH for targeted actions.

Box 1. Schematic depiction of the PPOR



Source: Adapted from *Perinatal Periods of Risk Approach: The U.S. Urban Experience. CityMatCH, 2002.* Available from http://www.citymatch.org/sidewide-keywords/ppor

Phase II analysis

Phase II investigations consist of multiple approaches for evaluating the relative contributions of potential causes of excess feto-infant mortality. The Kitagawa analysis determines whether excess Maternal Health/Prematurity deaths are due to a higher frequency of very low birth weight (VLBW) births (both live births and fetal deaths) or higher mortality rates at given birth weights. A large overall difference in feto-infant mortality (e.g. 2 or more deaths per 1,000 live births and fetal deaths) between the target and reference groups and large numbers of deaths in each group (e.g. 60 or more) are required for the analysis to give stable results.[1,2] Specifically, mortality rate differences between the target and reference groups were calculated for each of the seven birth weight categories (500-749g, 750-999, 1,000-1,249, 1,250-1,499, 1,500-1,999, 2,000-2,499 and 2,500+) , weighted by the averaged birth weight frequencies (as proportions of births) of the target and reference groups; similarly, birth weight frequency differences were calculated and then weighted by the averaged mortality rates between the target and reference for each of the birth weight categories. When summed across birth weight categories, the former indicates the contribution of birth weight-specific mortality to the excess (relative to the reference) feto-infant mortality in the target group, and the latter that of birth weight frequencies. Thus: $MR_1 - MR_2 = \sum_n \left(\left(\frac{(P_{1n}+P_{2n})}{2} \times (M_{1n}-M_{2n}) \right) + \left(\frac{(M_{1n}+M_{2n})}{2} \times (P_{1n}-P_{2n}) \right) \right)$ (n=1,2,...7) where MR_1 and MR_2 denote the overall feto-infant mortality rates, P_{1n} and P_{2n} proportions of births for a specific birth weight category, and M_{1n} and M_{2n} birth weight-specific mortality rates for the target and reference groups respectively. By partitioning excess mortality into birth weight frequency and mortality components, the analysis estimates the relative importance of these two causal pathways, which are associated with different sets of risk and protective factors. The birth weight frequency pathway underlines socio-behavioral, health and economic disparities at play throughout the mother's life span, whereas the mortality pathway generally relates to deficits in perinatal care. To explore reasons for VLBW in the target population, standard mixed-effects logistic regression based on the multilevel pseudo-maximum likelihood method was used.[3] The probability for VLBW was defined in terms of binomial logit. This modeling process allows simultaneous consideration of various individual health/behavioral risks (e.g. smoking, obesity, diabetes, prenatal care behavior, maternal/obstetric risks) with socio-environmental (e.g. neighborhood income) characteristics. Although cross-sectional in nature (i.e. causal claims cannot be made), the multilevel study enables a formal assessment of available evidence for potential VLBW mechanisms. In addition, underlying cause of death from linked live birth/death records was examined to determine predominant causes of excess Infant Health deaths.

The PPOR Phase I analysis (see Methods), which sorted these fetal or infant deaths into distinctive risk periods, reinforced the impact of Maternal Health/Prematurity on fetal well-being. In 2011-12, nearly 40% of FIDs were attributed to the Maternal Health/Prematurity risk period, at a rate of 3.3 deaths per 1,000 live births and fetal deaths (LBFD). Another 32% of FIDs were attributed to Maternal Care (2.7 per 1,000), whereas Newborn Care and Infant Health represented around 13% (1.1 per 1,000) and 16% (1.3 per 1,000) respectively. Given that the vast majority of FIDs occurred in the Maternal Health/Prematurity and Maternal Care periods, increases in mortality associated with these two periods have been responsible for the uptick in overall feto-infant mortality between 2009-10 and 2011-12 (Figure 1).

Feto-infant mortality rates (FIMR) were substantially higher among younger (under 20) and older (35 and over) women; the two age groups registered 51 and 84 FIDs at rates of 11.6 and 10.3 per 1,000 LBFDs in 2011-12 respectively (compared with a FIMR of 8.3 per 1,000 for women overall). In both age groups Maternal Health/Prematurity accounted for about 45% of the FIDs in 2011-12, at rates of 5.2 and 4.6 per 1,000 LBFDs respectively (compared with 3.3 per 1,000 overall). Among women aged less than 20 years, Maternal Care deaths comprised about 25% of the FIDs at a rate of 3 per 1,000 LBFDs; whereas Infant Health deaths represented nearly 20% at a rate of 2.3 per 1000 LBFDs, notably above the rate for all women (1.3 per 1,000). Among women aged 35 and over, on the other hand, a higher proportion (one-third) of the

Definitions

PPOR feto-infant mortality rate= Number of feto-infant deaths × 1,000 / Number of live births and fetal deaths*

Maternal health/prematurity death rate
=Number of feto-infant deaths with a birth weight of 500-1,499 g × 1,000 / Number of live births and fetal deaths*

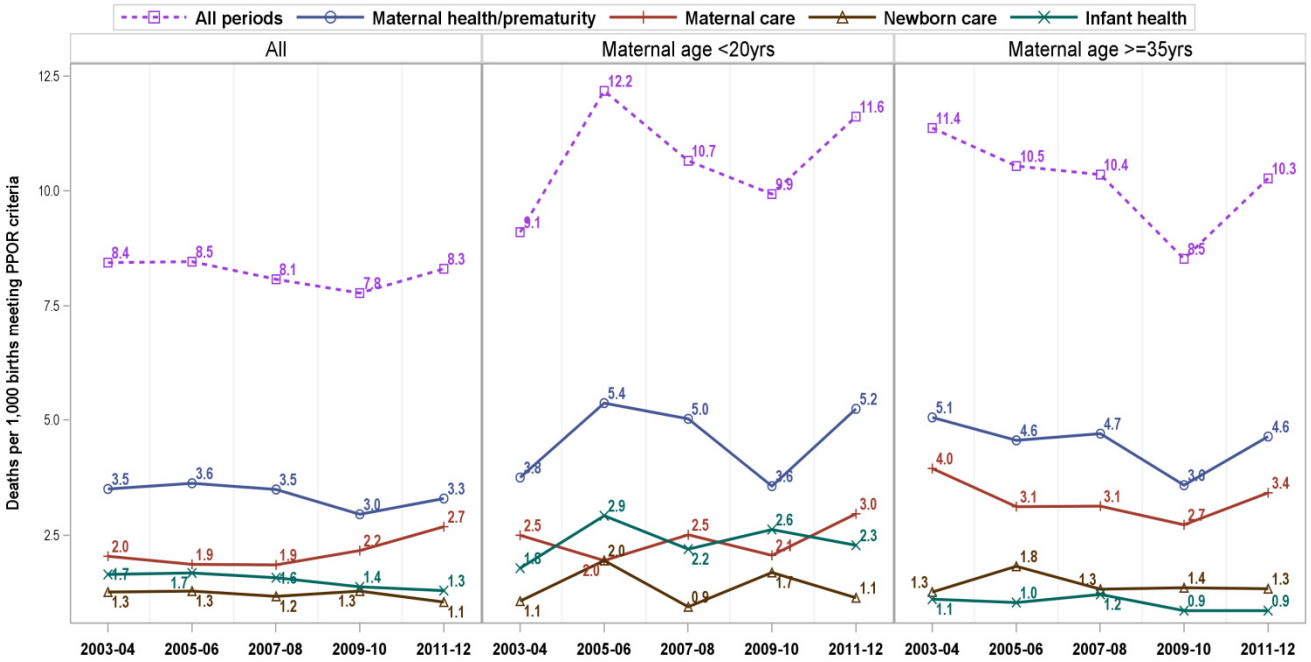
Maternal care death rate
=Number of fetal deaths with a birth weight of 1,500+ g × 1,000 / Number of live births and fetal deaths*

Newborn care death rate
=Number of neonatal deaths with a birth weight of 1,500+ g × 1,000 / Number of live births and fetal deaths*

Infant health death rate
=Number of postneonatal deaths with a birth weight of 1,500+ g × 1,000 / Number of live births and fetal deaths*

*Excludes fetal deaths <24 weeks of gestation and births (live or fetal death) with a birth weight <500 g.

Figure 1. Perinatal Periods of Risk (PPOR) trend by maternal age, Clark County-NV, 2003-12



Source: Birth certificate files/fetal death registry files (preliminary for 2011 onwards) and death certificate files (preliminary for 2012 onwards); excluding fetal deaths<24 wks or births<500g and restricted to mothers residing in Clark County.

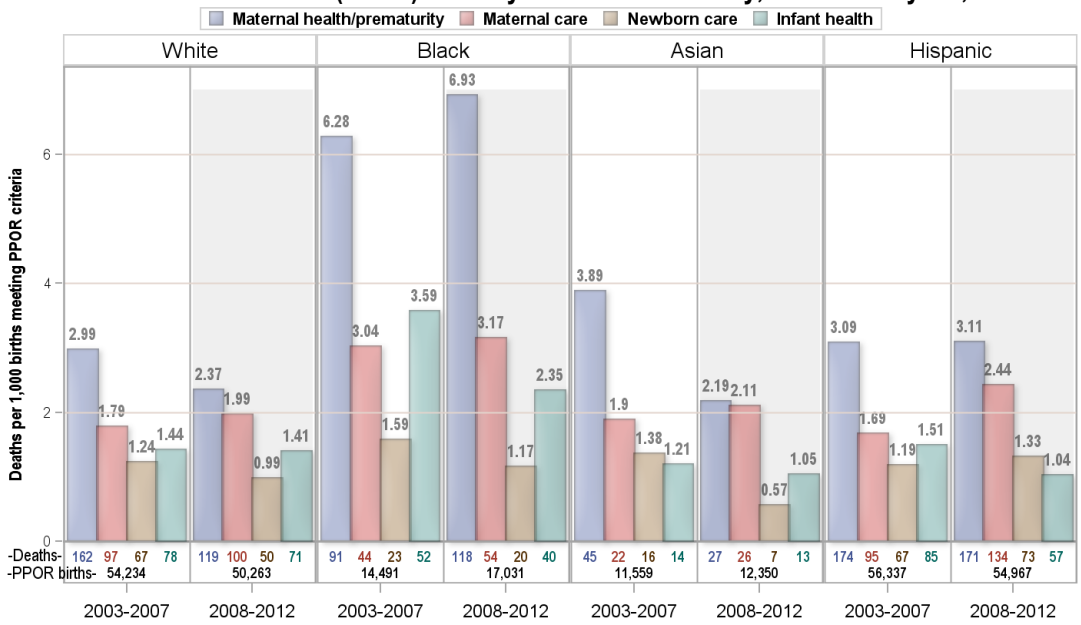
FIDs occurred in association with Maternal Care, at a rate of 3.4 per 1,000 LBFDs (compared with 2.7 per 1,000 overall); to a smaller extent Newborn Care death rates were elevated in the older age ranges as well (1.3 among ages 35 and over compared with 1.1 per 1,000 overall). It is worth noting that the recent increases in Maternal Health/Prematurity and Maternal Care mortality were more pronounced in teenagers and women aged 35 and over (Figure 1).

The higher perinatal mortality from Maternal Health/Prematurity and Maternal Care associated with teenage childbearing may relate to less favorable socio-economic circumstances and behavioral or biological factors (e.g. inadequate perinatal care, biological immaturity), whereas those associated with delayed childbearing often reflect the higher risks older women face during pregnancy and in labor, as well as the higher prevalence of chronic conditions at older ages.

Racial/ethnic comparisons

Most races experienced some reductions in FIMRs between 2003-07 and 2008-12. In particular, the FIMR declined from 7.5 to 6.8 FIDs per 1,000 LBFDs in non-Hispanic whites (NHW), from 14.5 to 13.6 in non-Hispanic blacks (NHB), and from 8.4 to 5.9 in Asians, whereas the rate in Hispanics was slightly up from 7.5 to 7.9. Between 2003-07 and 2008-12, the proportions of FIDs that were NHB and Hispanic rose from 18.4% (210 deaths) and 37% (421) to 21.4% (232) and 40.2% (435) respectively, while the NHW and Asian contributions decreased from 35.5% (404) and 8.5% (97) to 31.4% (340) and 6.7% (73) respectively. Of particular concern, Maternal Health/Prematurity was responsible for over one-half of the FIDs among NHBs in 2008-12, compared to 35% among NHWs and 39.3% among Hispanics (Figure 2). Further, Maternal Health/Prematurity death rates increased 10% for NHBs over the period 2003-07 to 2008-12, from 6.3 to 6.9 deaths per 1,000 LBFDs; whereas for other racial/ethnic groups, mortality due to Maternal Health/Prematurity generally declined or remained stable during this period. On the other hand, Maternal Care mortality showed an increasing trend for most race/ethnicities, with the largest percent increase observed among Hispanics. In 2008-12, Maternal Health/Prematurity, Maternal Care and Infant Health death rates were higher in NHBs than in other racial/ethnic groups, whereas Hispanics had the highest FIMR due to Newborn Care across race/ethnicities.

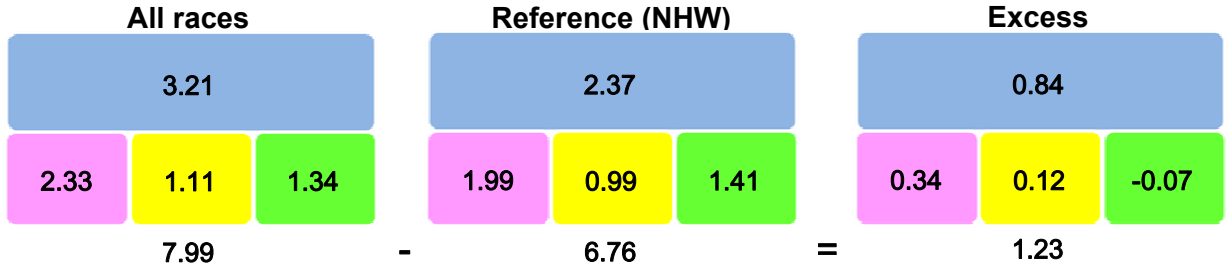
Figure 2. Perinatal Periods of Risk (PPOR) trend by select race/ethnicity, Clark County-NV, 2003-12



Source: Birth certificate files/fetal death registry files (preliminary for 2011 onwards) and death certificate files (preliminary for 2012 onwards); excluding fetal deaths<24 wks or births<500g and restricted to mothers residing in Clark County.

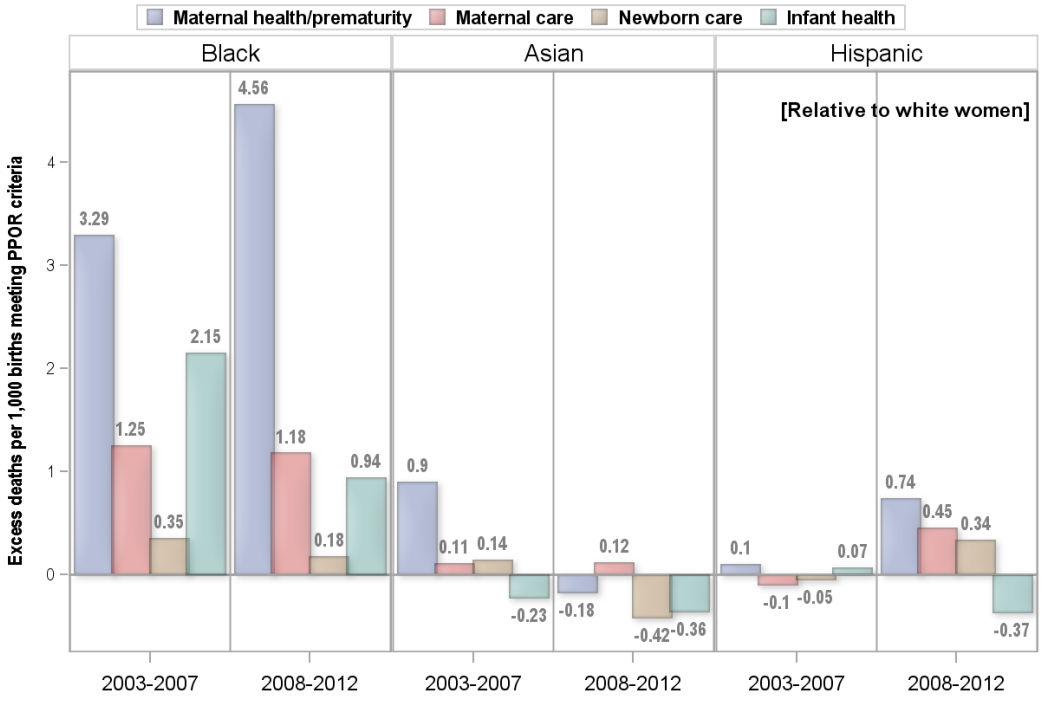
To estimate the potential for feto-infant mortality reduction, NHWs were chosen as the reference group given their comparatively low perinatal death rates and large group size. Perinatal mortality in excess of the NHW baseline generally reflected the PPOR patterns in Clark County: Maternal Health/Prematurity had the highest excess mortality and hence the greatest potential for perinatal mortality reduction, followed by Maternal Care (Figure 3). While the overall FIMR decreased from 8.3 (corresponding to 1,139 deaths) to 8 (1,082) per 1,000 LBFDs between 2003-07 and 2008-12, the excess or preventable mortality portion actually increased, from 10% (0.8 of 8.3) to 15% (1.2 of 8), due to increases in excess perinatal deaths among Hispanics (stemming from Maternal Health/Prematurity, Maternal Care and Newborn Care), and an appreciable rise in excess Maternal Health/Prematurity deaths among NHBs. In 2008-12, Maternal Health/Prematurity comprised over two-thirds (4.6 of 6.9) of the excess FIDs among NHBs, compared with 46.7% in 2003-07 (Figure 4).

Figure 3. Excess feto-infant mortality per 1,000 live births and fetal deaths, Clark County-NV, 2008-12



Source: Birth certificate files/fetal death registry files (preliminary for 2011 onwards) and death certificate files (preliminary for 2012 onwards); excluding fetal deaths<24 wks or births<500g and restricted to mothers residing in Clark County.

Figure 4. Excess feto-infant mortality by select race/ethnicity, Clark County-NV, 2003-12



Source: Birth certificate files/fetal death registry files (preliminary for 2011 onwards) and death certificate files (preliminary for 2012 onwards); excluding fetal deaths<24 wks or births<500g and restricted to mothers residing in Clark County.

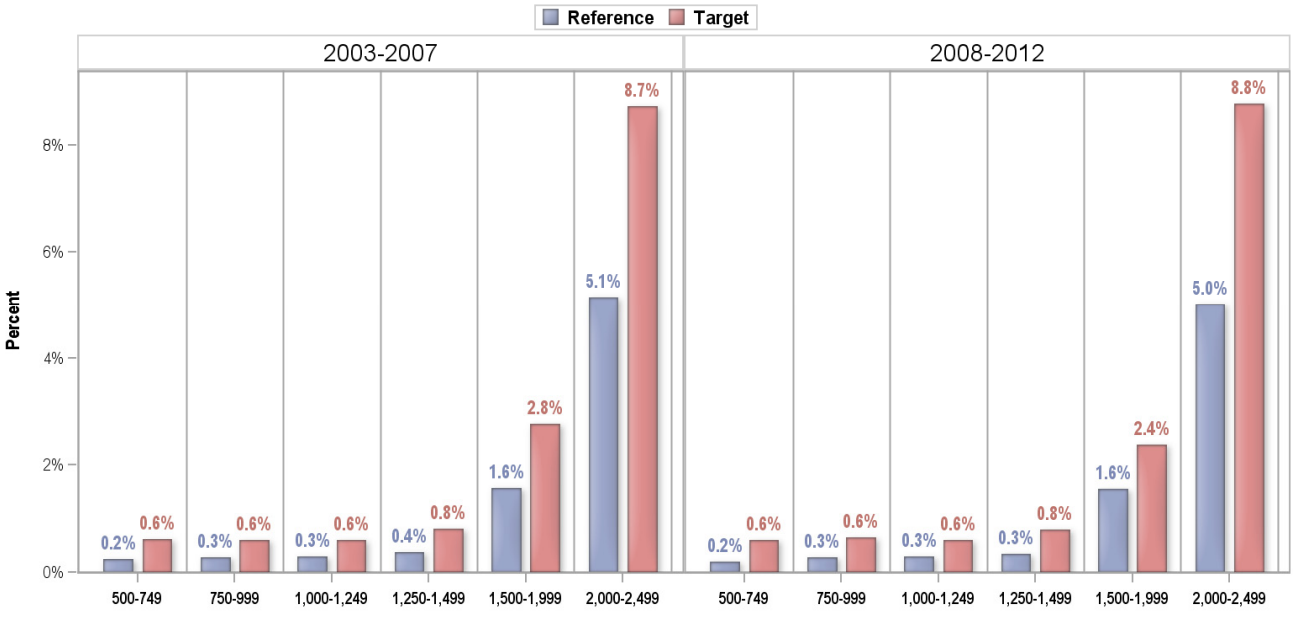
Primary causes for excess Maternal Health/Prematurity mortality

Roughly one-half (6.9 of 13.6) of the NHB feto-infant mortality may be preventable based on the NHW reference rate in 2008-12 (compared with an excess mortality of about 15% [1.2 of 7.9] in Hispanics). Given the high excess perinatal mortality burden experienced by NHBs, and a preponderance of Maternal Health/Prematurity deaths underlying these excesses, Kitagawa analysis was conducted to determine whether the excess Maternal Health/Prematurity mortality was attributable to high rates of extreme prematurity or VLBW (labeled as birthweight), or to poor survival rates at given birth weights (labeled as mortality). The two pathways are different from both an etiologic and action standpoint. The birthweight pathway generally relates to socio-economic disadvantages, behavioral and health disparities of the mother prior to and during pregnancy, and it highlights the need to examine and manage risk factors associated with VLBW in the community. The mortality pathway on the other hand, generally relates to the efficacy and accessibility of perinatal health care services, and underscores the importance of effective interventions to improve the survival of high-risk infants.

When compared across birth weight categories, the weight disparity between NHB and NHW feto-infants (weighing 500+g; fetal deaths at 24+ weeks of gestation) widened at the severe end of the LBW spectrum: LBW (less than 2,500g) rates among NHB births (live or fetal death) in 2003-12 were 1.8 times as high as for NHWs, whereas the NHB VLBW (less than 1,500g) rate was 2.2 times as high as the NHW rate in 2003-07, and 2.4 times as high in 2008-12 (Figure 5). Birth weight-specific mortality rates also tended to be elevated in NHBs, and showed greater NHB excesses among VLBW births in recent years; mortality rates were about 10-30% higher in NHB births weighing 1,500g or more, and about 10-50% higher in those weighing less than 1,250g (and at least 500g), when compared with respective NHW cohorts in 2008-12 (data not shown).

Results from the Kitagawa partitioning of excess perinatal mortality show that the birthweight pathway (i.e. high rates of VLBW), rather than that of mortality (i.e. poor survival rates), was the predominant cause of excess Maternal Health/Prematurity deaths among

Figure 5. Low birth weight distributions of target (NHB) and reference (NHW) groups, Clark County-NV, 2003-12



Source: Birth certificate files/fetal death registry files (preliminary for 2011 onwards) and death certificate files (preliminary for 2012 onwards); excluding fetal deaths<24 wks or births<500g and restricted to mothers residing in Clark County.

NHBs. Of the 4.6 (per 1,000 LBFDs) excess VLBW deaths (corresponding to the excess rate for Maternal Health/Prematurity) among NHBs in 2008-12, 3.9 deaths or 85.6% was attributed to the birthweight pathway (compared with a birthweight contribution of 71.5% to the overall NHB excess mortality) (Figure 6). Subanalyses (results not shown) by maternal age reveal that teen births were not the reason for excess NHB mortality, as excess rates for overall and Maternal Health/Prematurity mortality in teen births were generally lower than or similar to those in all births, while those at older maternal ages (35 and over) were substantially higher. Nonetheless, the excess Maternal Health/Prematurity deaths among NHB teen births were attributed entirely to the birthweight pathway, whereas the attributable proportion in births to older mothers was about 72% (compared with the much attenuated birthweight contributions to excess VLBW deaths among Hispanics, at 56% and 22% for teen and older mothers respectively). These findings emphasize the importance of preventing VLBW births in reducing perinatal deaths among NHBs.

Figure 6 a. Excess NHB feto-infant mortality by pathway, Clark County-NV, 2003-12

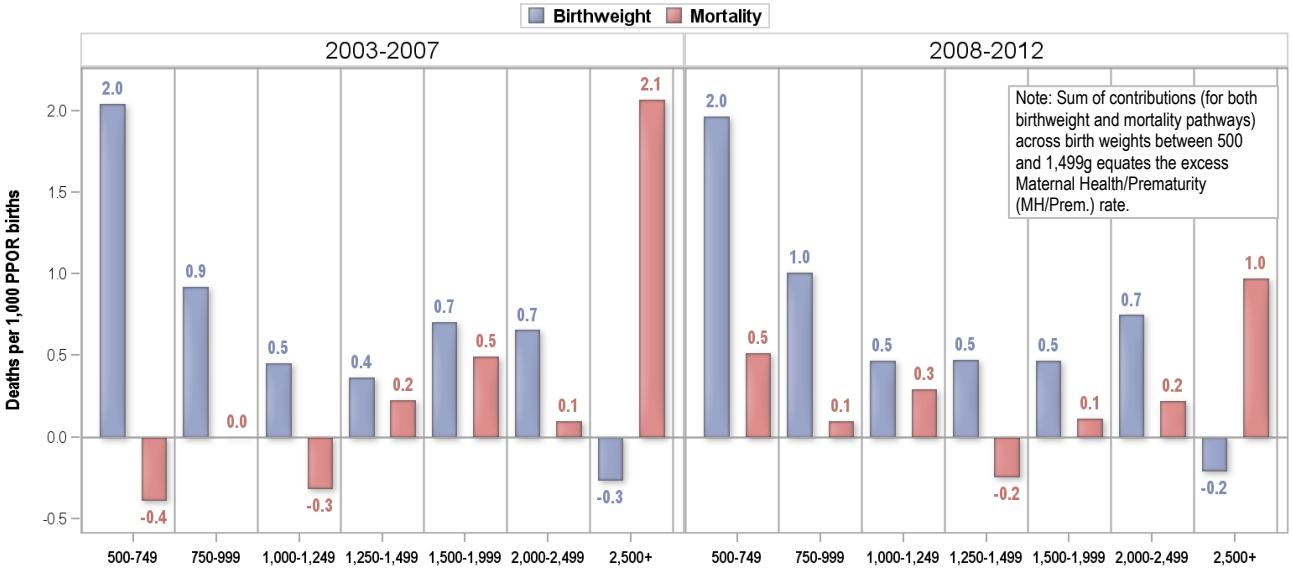
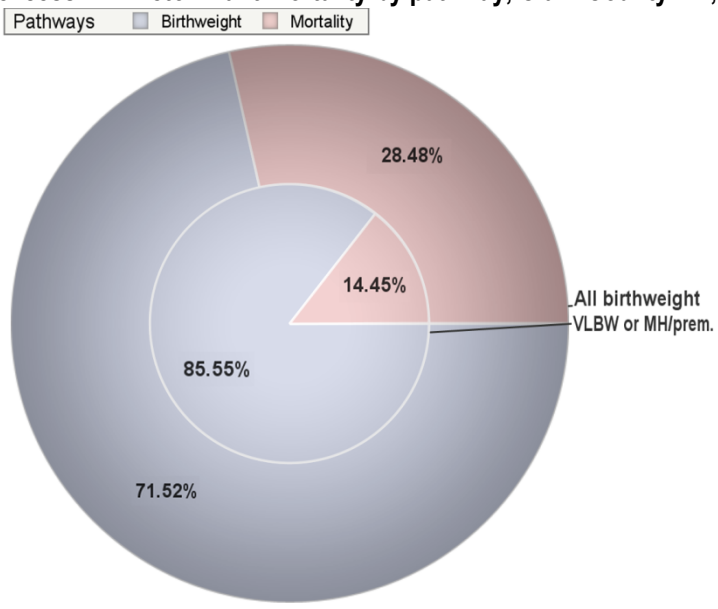


Figure 6 b. Percentage of excess NHB feto-infant mortality by pathway, Clark County-NV, 2008-12



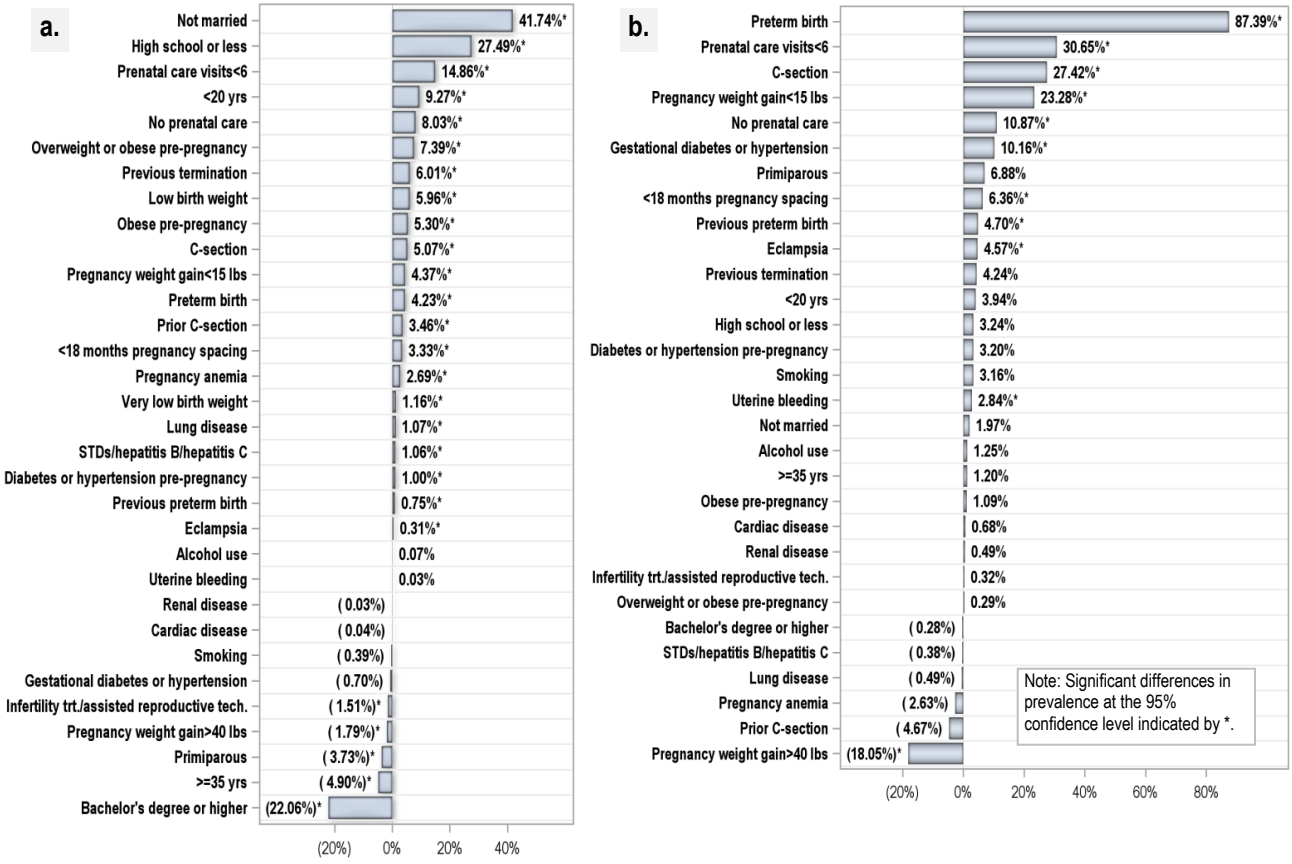
Source: Birth certificate files/fetal death registry files (preliminary for 2011 onwards) and death certificate files (preliminary for 2012 onwards); excluding fetal deaths<24 wks or births<500g and restricted to mothers residing in Clark County.

Risk factors for VLBW

Although vital records do not provide comprehensive health or behavior-related information for an in-depth investigation of the medical and social determinants of VLBW, they are the only ongoing surveillance system that systematically records data covering essentially the whole county-dwelling population, and it is able to estimate population-based prevalence of relevant maternal and infant characteristics that are potential predisposing factors for VLBW. Using vital records of live singleton births in 2010-12, prevalence of various socio-demographic and perinatal health indicators for NHBs were examined, and compared with the NHW reference group for all birth weights in Figure 7a. The extent to which characteristics of NHB VLBW newborns differed from those of their NHB counterparts weighing 1,500g or more was also illustrated in Figure 7b.

Among NHB singleton live births in 2010-12, teenage birth conferred odds of VLBW that were 1.5 (95% CI: 0.9-2.6) times as high as in childbearing at ages 20 years and older, after adjusting for the effects of other co-risk factors at both individual and group levels (see Methods). For older NHB women (35 and over) relative to those younger, the VLBW odds ratio was 0.8 (95% CI: 0.4-1.6) after differences in other variables, including maternal, obstetric and socio-behavioral risk factors (as reported on birth certificates), were taken into account, a finding that perhaps reflects the protective effects of favorable socio-economic status and widespread uptake of prenatal and obstetric care in this age group (above and beyond those captured by the neighborhood income and prenatal care usage variables). Low prenatal care utilization showed significantly positive association with VLBW in NHBs—the odds of VLBW for those reporting less than 6 prenatal

Figure 7 a. NHB excess (relative to NHW) prevalence of selected characteristics (singleton; all birth weights); b. Prevalence excesses (relative to births ≥1,500g) for VLBW births among NHBs (singleton), Clark County-NV, 2010-12



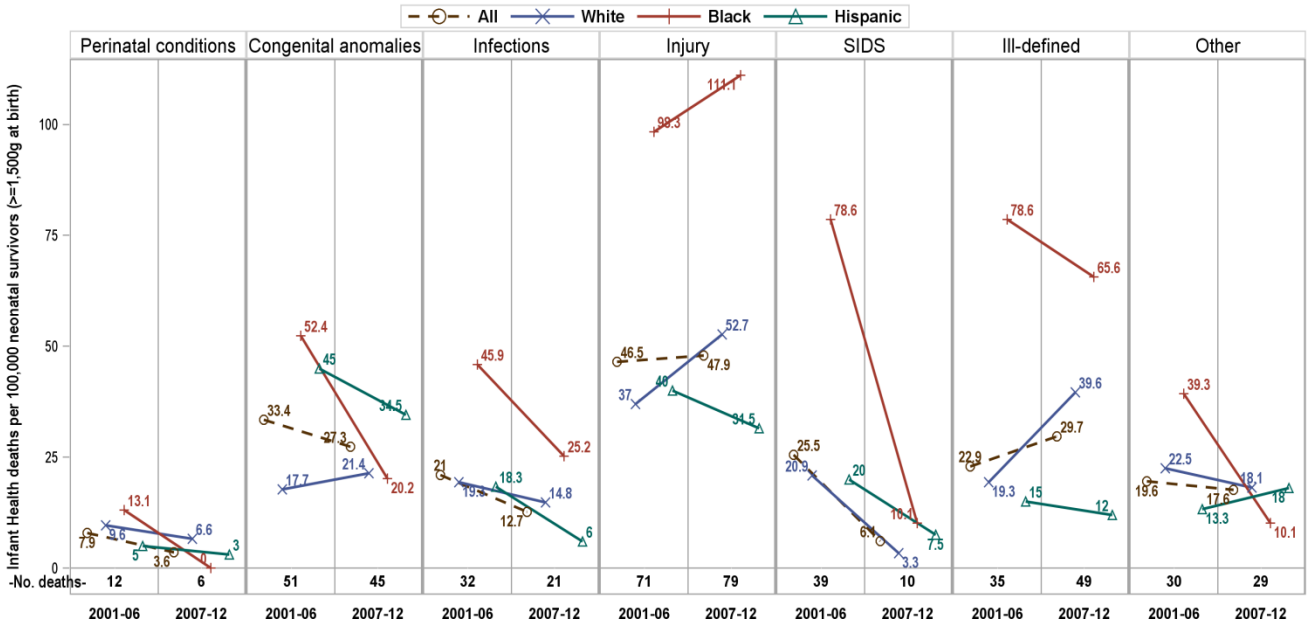
Source: Birth certificate files (preliminary for 2011 onwards); restricted to mothers residing in Clark County.

care visits were 5.1 (95% CI: 3.1-8.3) times the odds for those reporting greater usage. As well, primiparity was positively related to VLBW, with an adjusted odds ratio (AOR) of 1.3 (95% CI: 0.8-2.1) for primiparous relative to multiparous NHB women. On the other hand, the odds of VLBW were nearly three (2.9 [95% CI: 1.7-5.1]) times as high among NHB pregnancies spaced less than 18 months apart, compared with first-parity or higher parity births spaced further apart, while controlling for other factors. Moreover, previous preterm birth was a predisposing factor for VLBW with an elevated AOR of 1.6 (95% CI: 0.7-3.6) relative to NHB births with no such risk. For other risk factors, including smoking during pregnancy, pre-existing health problems (e.g. diabetes, hypertension), and pregnancy complications (e.g. gestational diabetes or hypertension, eclampsia, uterine bleeding), their associations with VLBW among NHBs were largely as expected. The AOR of VLBW for smoking during pregnancy, in comparison with not smoking, was 1.2 (95% CI: 0.7-2.1). The AORs for NHB women with pre-pregnancy diabetes/hypertension and for those with gestational diabetes/hypertension, relative to their counterparts without the conditions, were 3.3 (95% CI: 1.6-7.1) and 2.9 (95% CI: 1.7-5) respectively. Among the complicating factors examined, premature rupture of membranes showed a highly significant association with VLBW (AOR of 9.6 [95% CI: 6.4-14.3]). This association may reflect labor induction practices and other obstetric efforts undertaken to prevent perinatal mortality or disease in the mother and the neonate, which paradoxically have the effects of increasing rates of low-weight or preterm live births with precarious survival chances.

Infant Health mortality

Infant Health deaths, namely infant deaths with births weights of 1,500g or more that occur after 27 days and before one year from birth, reflect different risk factors and intervention opportunities than those attributed to Maternal Health/Prematurity. While the potential for reductions in Maternal Health/Prematurity mortality lies primarily in improving preconception health and early perinatal care, interventions that seek to reduce Infant Health deaths should focus on underlying cause-of-death and associated risk factors. Between 2003-07 and 2008-12, the proportion of NHB excess FIDs attributed to Infant Health decreased from around

Figure 8. Birth cohort-based cause-specific Infant Health mortality by select race/ethnicity, Clark County-NV, 2001-12



Source: Birth certificate files (preliminary for 2011 onwards) and death certificate files (preliminary for 2012 onwards); restricted to mothers residing in Clark County.

31% to 14%; however, racial disparities remained, with Infant Health death rates considerably higher in NHBs than in other race/ethnicities (Figures 2 and 4). To identify causes of excess Infant Health mortality and prevention opportunities, linked live birth/death certificates were examined by race/ethnicity and main causes of postneonatal mortality.[2,4]

The potential for more immediate prevention and thus reduction of Infant Health mortality is greater for injury than other postneonatal causes of death. Injury mortality rates among neonatal survivors (weighing 1,500+g at birth) were substantially higher in NHBs than in other racial groups, and increased from 98.3 to 111.1 per 100,000 neonatal survivors between 2001-06 and 2007-12 (Figure 8). Accordingly, Infant Health mortality attributable to injury rose from 24.2% to 45.8% among NHBs, while less marked increases were observed in NHWs (from 25.3% to 33.7%) and Hispanics (25.5% to 28%). Of note is that part of the observed injury mortality increase may be explained by changes in coding practices since the mid-1990s (e.g. decreased SIDS reporting and increased reporting of suffocation and asphyxia).[5] Infection was another primary cause of excess NHB Infant Health mortality, accounting for around 10% of Infant Health deaths among NHBs in 2007-12, or 25.2 deaths per 100,000 neonatal survivors. As well, ill-defined conditions were responsible for substantial proportions of Infant Health mortality, due mainly to differential coding practices and/or post-mortem examination rates, a potential limiting factor for the utility and comparability of the causes of death information contained in vital records.

With regard to injury mechanisms, suffocation was listed in over three-quarters of the injury-attributable Infant Heath mortality among NHBs in 2001-12, compared to 69% in NHWs and 58% in Hispanics. This finding highlights the urgent need to further investigate sleep-related deaths (e.g. due to bedding, sleep position and environment) and to direct prevention efforts towards related risk factors.

Concluding remarks

The burden of feto-infant mortality was mainly attributed to factors associated with Maternal Health/Prematurity, and was disproportionate for NHB mothers. These findings underline the necessity of further analyses and community engagement efforts to target preconception health disparities and extreme prematurity. The analysis also supports the fundamental importance of data capacity building, community mobilization and alignment to develop evidence-based interventions using the PPOR approach.

Acknowledgements

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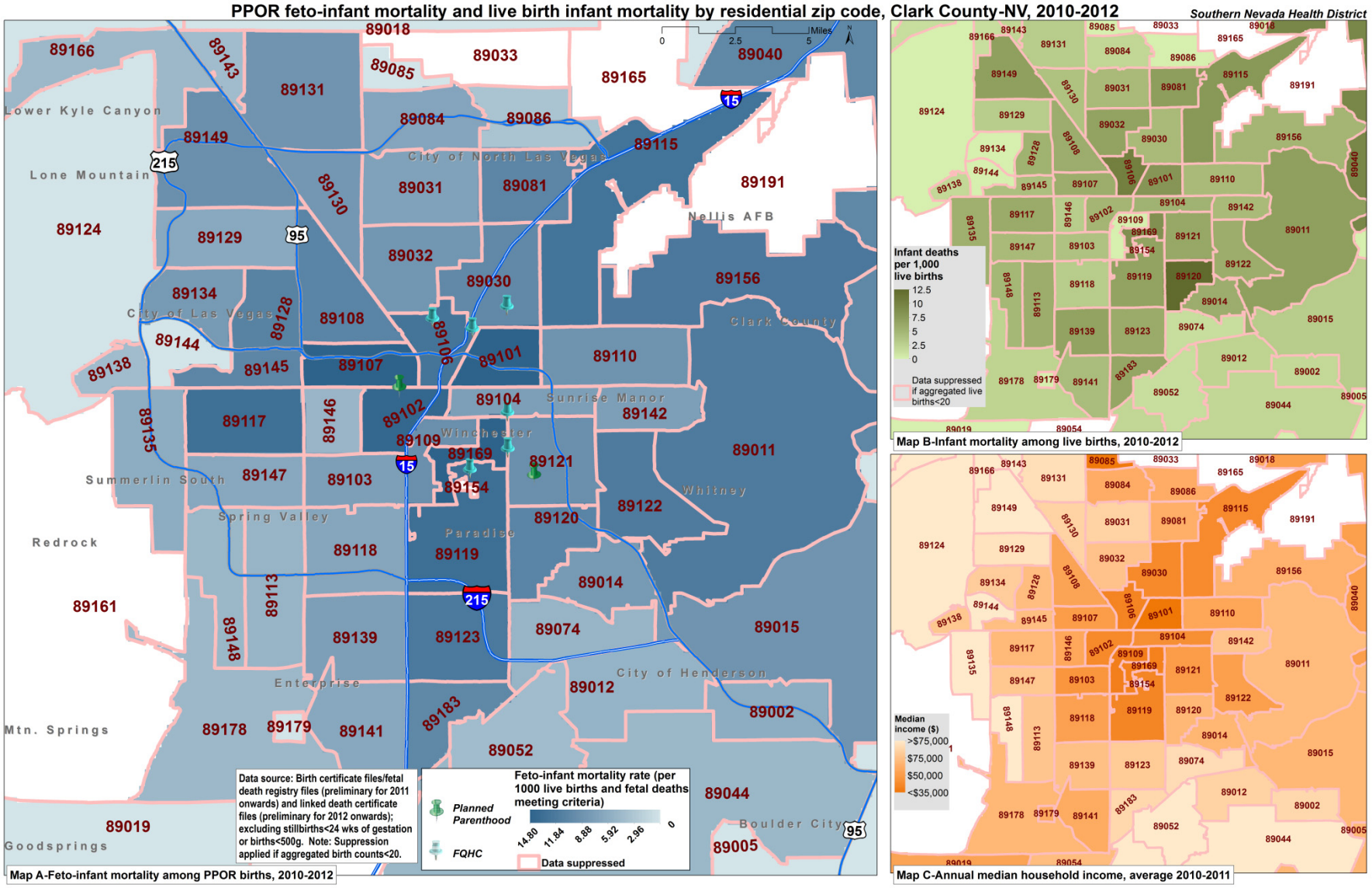
Appendix A.


PPOR feto-infant mortality and live birth infant mortality by residential zip code, Clark County-NV, 2010-2012
Southern Nevada Health District
Lower Kyle Canyon
Lone Mountain
City of North Las Vegas
City of Las Vegas
Nellis AFB
Clark County
Sunrise Manor
Winchester
Summerlin South
Spring Valley
Paradise
Whitney
Redrock
Enterprise
City of Henderson
Mtn. Springs
Boulder City
Goodsprings
Data source: Birth certificate files/fetal death registry files (preliminary for 2011 onwards) and linked death certificate files (preliminary for 2012 onwards); excluding stillbirths<24 wks of gestation or births<500g. Note: Suppression applied if aggregated birth counts<20.
Feto-infant mortality rate (per 1000 live births and fetal deaths meeting criteria)
14.80
11.84
8.88
5.92
2.96
0
Planned Parenthood
FQHC
Data suppressed
Map A-Feto-infant mortality among PPOR births, 2010-2012
Infant deaths per 1,000 live births
12.5
10
7.5
5
2.5
0
Data suppressed if aggregated live births<20
Map B-Infant mortality among live births, 2010-2012
Median income ($)
>$75,000
$75,000
$50,000
<$35,000
Map C-Annual median household income, average 2010-2011

Appendix B.

Cross referencing child/infant deaths with birth certificates that reside in a different vital records database was automated via several rounds of text parsing algorithms. Birth and death records for the select period were first cleaned and edited to improve data quality. Cleaned identifiers including child's and mom's names, child's DOB, and birth certificate number/year were used to identify potential birth-infant death matches. While exact matching can be done on DOB and birth certificate number identifiers, comparisons have to be 'fuzzy' to allow for spelling variations, misspelling, and truncation or omission where names are involved. The major steps involved in the fuzzy matching process are outlined below.

1. Child's last name and first name fields in both death and birth databases were standardized to remove non-informative characters (e.g. * ' - . ,) as well as digits;
2. The same standardization was performed on mom's name fields in both death and birth databases;
3. Standardized first name (SFN) and last name (SLN) were concatenated as a new name field (NNF) for both child and mom; if valid text entries were not available for the name fields, random character strings were assigned to SFN and SLN as unique name identifiers;
4. Soundex codes (for phonetic matching) of SFN and SLN were created for both child and mom;
5. While the SAS version of the soundex algorithm is fairly robust, generalized phonetic matching was supplemented with a spelling distance algorithm ('spelldex') to improve sensitivity to keying errors or nicknames. The spelldex algorithm assumed the first letter in the name fields was correct, and from the rest of the fields took the first three consonants. The first letter was then concatenated with the three selected consonants to form the spelldex codes for SFN and SLN;
6. Linkage criteria included matches on:
    1) NNF of child plus child's DOB
    2) Soundex of child's SFN and of SLN plus child's DOB
    3) Spelldex of child's SFN and of SLN plus child's DOB
    4) NNF of mom plus child's DOB
    5) Soundex of mom's SFN and of SLN plus child's DOB
    6) Spelldex of mom's SFN and of SLN plus child's DOB
    7) Birth certificate number and year