JECH Online First, published on February 17, 2011 as 10.1136/jech.2010.132886

Racial disparities in health-related quality of life in a cohort of very-low-birth-weight 2- and 3-year-olds with and without asthma

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Accepted 19 January 2011

ABSTRACT

Background Children born very low birth weight (VLBW) are at risk for low health-related quality of life (HRQoL), compared with normal-birth-weight peers, and racial disparities may compound the difference. Asthma is the most pervasive health problem among VLBW children and is also more common among black than white children, partly due to unfavourable environmental exposures. This study explores racial disparities in HRQoL among VLBW children and examines whether potential disparities can be explained by asthma and neighbourhood disadvantage.

Methods The study population was the Newborn Lung Project, a cohort of infants (n=660) born VLBW in 2003–2004 in Wisconsin, USA, who were followed up at age 2–3. Multilevel linear regression models were used to examine the contributions of asthma, neighbourhood disadvantage, and other child and family socio-demographic covariates, to racial disparities in HRQoL at age 2–3. A child's HRQoL was measured using the Paediatric Quality of Life Inventory 4.0.

Results VLBW, black, non-Hispanic children, on average, score nearly 4 points lower ($p<0.01$) on HRQoL than do white, non-Hispanic children. Including asthma reduces the difference between black and white children from −3.6 ($p<0.01$) to 0.08 ($p>0.05$). The authors found no evidence that the relationship between asthma and HRQoL differs by race. The interaction between neighbourhood disadvantage and asthma is statistically significant, with further examination suggesting that racial disparities are particularly pronounced in the most advantaged neighbourhoods.

Conclusion The authors found that the black disadvantage in HRQoL among 2–3-year-old VLBW children likely stems from a high prevalence of asthma. Neighbourhood attributes did not further explain the disparity, as the racial difference was particularly pronounced in advantaged neighbourhoods.

INTRODUCTION

There is an inverse relationship between birth weight and neonatal morbidity,[1 2] and an increasing awareness of the long-term health consequences of being born very low birth weight (VLBW, <1500 g). Respiratory disease is the most common morbidity in VLBW children, and asthma risk is threefold higher in the VLBW than in the general population.[3] Not surprisingly, preschoolers born VLBW demonstrate a lower health-related quality of life (HRQoL) than their normal-birth-weight peers,[4 5] and asthma with its symptoms is a likely contributor.[6 7]

In a large cohort of premature births in Northern California, black children were found to have over four times the odds of white children of receiving oral asthma medications in the first year of life.[8] It is therefore likely that racial disparity in HRQoL found in a nationally representative study of US children[7] may be more pronounced among VLBW children, and asthma may be on the causal pathway (ie, effect mediator) explaining these racial disparities. Furthermore, it is unclear if black VLBW children experience asthma differently than do white VLBW children with asthma, and whether this might affect HRQoL (ie, race is an effect modifier between asthma and HRQoL). Black children with asthma have a worse symptom severity, miss more school, have more hospitalisations and have more frequent emergency department visits than their white peers with asthma.[9 10] Black VLBW children might experience more frequent exposure and greater susceptibility to environmental asthma triggers than white VLBW children with asthma, contributing to more severe morbidity and lower HRQoL. Such a hypothesis is also supported by the fact that black VLBW neonates have similar or better respiratory outcomes than white VLBW neonates in the Neonatal Intensive Care Unit,[11 12] yet appear to experience worse asthma in childhood.

The social ecological model[13] suggests that child health, and presumably HRQoL, stem from all ecological niches (individual, family, community and social policy). Our previous work[14] suggests that neighbourhood disadvantage is associated with HRQoL in children born VLBW. Moreover, there is a large literature[15–18] to support the influence of neighbourhood disadvantage on asthma prevalence, especially among minority children. Black, non-Hispanic children are more likely to live in socially disadvantaged neighbourhoods,[19] which may cumulatively increase their risk for lower HRQoL, at least partly due to the high asthma prevalence. To our knowledge, the combined contributions of race, asthma and neighbourhood disadvantage on HRQoL have not been examined in the VLBW population.

Population-health scientists advocate for a research agenda focusing on health-related quality of life (HRQoL) rather than pathophysiology or impairment outcomes.[20 21] HRQoL captures not only health difficulties but problems stemming from children's interactions with their surroundings.[22]

Thus, understanding disparities in HRQoL has important implications for child-health interventions. Despite a lack of definitional consensus, HRQoL encompasses physical, social and emotional well-being and function.[23] HRQoL in young children is collected via parent report. Several measures, including the Paediatric Quality of Life Inventory (PedsQL), are reported to have excellent reliability and to be sensitive to changes pre- to postintervention, and are easily administered.[24]

The aims of this study are to (1) describe racial differences in HRQoL among a cohort of 2- and 3-year-olds born VLBW, and (2) examine whether asthma and neighbourhood disadvantage help explain any racial differences in HRQoL in this cohort. We hypothesise that (1) black VLBW children will experience lower HRQoL than their white counterparts; (2) racial differences in HRQoL will be attributable to greater asthma burden among black VLBW children; (3) black VLBW children with asthma will experience lower HRQoL than their white VLBW peers with asthma; and (4) black children with asthma who live in socially disadvantaged neighbourhoods will be particularly vulnerable to low HRQoL.

METHODS

Study population

The Newborn Lung Project is a regional cohort of infants born very low birth weight in 2003–2004 and hospitalised in one of 16 NICUs in Wisconsin or near the state border. The original study obtained follow-up addresses for 979 infants and also collected extensive clinical data. At age 2–3, 748 were alive and located, and of these, 719 children have complete HRQoL data. Data on 59 children were omitted from analyses due to missing data on smoking during pregnancy (n=20), maternal education (N=2), asthma (N=16), income (N=10) and prenatal care (N=11). The final sample included 660 children. To test for potential bias from missing data, we ran a series of sensitivity analyses including these latter cases with missing data in analyses excluding the covariates with missing data from the model. These model estimates were very similar whether cases with missing data were included or excluded. Thus, we restrict our analyses to children (n=660) with complete data.

Outcome measures

Children's HRQoL was measured using the Paediatric Quality of Life Inventory (PedsQL)[25] reported by parents for toddlers aged 2–4. The PedsQL contains 21 items that obtain parental assessment in four domains: physical, emotional, social and school/daycare. Parents are asked to rate, on a 5-point scale (0=never a problem to 4=almost always a problem), the degree of difficulty their child faced in the last month with regard to each item. A total score (0–100) reflects scaling and reverse scoring. A higher score indicates better HRQoL.

Child and family characteristics (level 1)

Parental education was categorised as less than high school (HS) education, HS degree or equivalent, post-HS schooling including college or technical school, and completion of a college degree, and was treated as an ordinal variable. Severity of neonatal distress was measured by the Score for Neonatal Acute Physiology-II,[26] an index ranging from 0 to 115 that comprises six physiologically based items (eg, blood pressure, temperature, oxygen requirement and seizures). Child's race and ethnicity was grouped as white, non-Hispanic; black, non-Hispanic (hereafter referred to as black); Hispanic; and other (including American Indian and multiracial families). Annual family income was categorised as less than $30 000, between $30 000 and $60 000 and greater than $60 000, and modelled as an ordinal variable. Birth weight was measured in grams and gestational age in weeks. Exposure to environmental tobacco smoke was measured by two variables—whether the mother smoked during pregnancy and whether the child lives with a current smoker. We also included the sex of the child, whether or not the child attends daycare, single-parent household and whether the mother received no prenatal care. Diagnosis of asthma at age 2 was collected by parent report. The distribution of these variables for the full sample and within racial/ethnic subgroups is presented in table 1.

Neighbourhood disadvantage (level 2)

A neighbourhood disadvantage index (table 2) was created using a principal-component analysis of five census tract socio-demographic variables[27]: percentage of families in poverty, percentage of households with an income above the state median, percentage of females with a bachelor's degree or more, percentage of single mothers and percentage of unemployed mothers of young children. Principal-component analysis[28] is a data-reduction technique that determines how to combine variables linearly into a single score that captures as much as possible of the overall variability in the variables and has been previously used in perinatal epidemiological research.[29] Specifically, the five census variables were standardised (after reverse-coding the percentage of females with a bachelor's degree or more and the percentage of households with an income above the state median) following the standard procedure. An overall neighbourhood disadvantage score (mean=0, SD=1, and α=0.86) was created as an average of items weighted by the item loadings (whose elements measure the strength of the relationship between the variable and principal component).

Statistical analyses

Multilevel modelling[30] was used to examine the contributions of asthma, neighbourhood disadvantage, and other child and family socio-demographic covariates to HRQoL. Multilevel models account for the clustering of children (Level 1) in neighbourhoods (Level 2) and explicitly model the fixed effects of each covariate on the outcome (β) and the neighbourhood variability (σ^2) around the predicted value.

Our analyses entailed fitting a series of multilevel linear random intercept models. A previous study[5] suggests that HRQoL in VLBW children has a skewed distribution. To evaluate the appropriateness of a linear model, we also utilised Tobit regression (ie, allows for ceiling effect of data) and obtained results similar to the linear specification. We report the results of the linear models.

First, the null model (ie, contains no covariates) describes the overall neighbourhood census tract (CT) variability in HRQoL. Second, we added the child's race to examine their contribution to explaining HRQoL and neighbourhood variability in HRQoL, conditional on other child and family characteristics. The third model added parent-reported asthma. The fourth model added an interaction between asthma and child race and ethnicity. The final models included, first, neighbourhood disadvantage and, second, interaction terms between child race and ethnicity, asthma and neighbourhood disadvantage.

For each model, we report the estimated fixed effect ($\widehat{\beta}$) for each covariate, the SE of β, estimated neighbourhood variability ($\widehat{\sigma}^2$) and SE of $\widehat{\sigma}^2$. A substantial reduction in ($\widehat{\sigma}^2$) indicates that the variables in the model help explain the observed differences in HRQoL across neighbourhoods.

Multilevel linear regression analyses were conducted in SAS v9.13[31] using PROC MIXED. Tobit models were fitted in

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Table 1 Bivariate analysis of independent and dependent covariates by race and ethnicity* for cohort (N=660) of very-low-birth weight 2- and 3-year-olds in Wisconsin

Characteristic	Percentage (N) Whole cohort (n=660)	Black (n=82)	White (n=517)	Hispanic (n=32)	Other (n=29)	p Value
Total annual income						<0.001
<$30 000	32.1 (212)	78.1 (64)	21.9 (113)	81.3 (26)	24.1 (7)	
$30 000–$60 000	32.4 (214)	17.1 (14)	36.0 (186)	15.6 (5)	31.0 (9)	
>$60 000	35.6 (235)	4.9 (4)	42.2 (218)	3.1 (1)	44.8 (13)	
Parental education						<0.0001
Less than a high-school degree	7.3 (48)	29.3 (24)	3.1 (16)	21.9 (7)	3.5 (1)	
High-school degree or equivalent	21.8 (144)	36.6 (30)	18.2 (94)	40.6 (13)	24.1 (7)	
Some post-high-school	35.9 (237)	22.0 (18)	38.3 (198)	31.3 (10)	31.0 (9)	
Bachelor degree	35.1 (232)	12.2 (10)	40.4 (293)	6.3 (2)	41.4 (12)	
Sex of the child						0.54
Boys	48.3 (319)	48.8 (40)	47.8 (247)	46.9 (15)	48.3 (14)	
Girls	51.7 (342)	51.2 (42)	52.2 (270)	53.1 (17)	51.7 (15)	
Child diagnosed as having asthma†	13.9 (92)	48.8 (40)	7.2 (37)	21.9 (7)	27.6 (8)	<0.001
Mother smoked during pregnancy	16.4 (107)	29.3 (24)	7.2 (37)	6.3 (2)	24.1 (7)	<0.001
Child currently lives with a smoker	5.8 (38)	15.8 (13)	3.9 (20)	6.3 (2)	10.3 (8)	<0.001
Child lives in single parent household	22.8 (151)	70.7 (58)	14.7 (76)	37.5 (12)	13.8 (4)	<0.001
Child attends daycare	41.0 (269)	41.5 (34)	40.8 (211)	31.2 (10)	45.8 (13)	0.009
Mother received no prenatal care	2.6 (17)	7.3 (6)	1.4 (7)	12.5 (4)	0	<0.001
	Mean (SD)					
Score of medical severity at birth‡	15.4 (12.7)	20.0 (13.2)	14.3 (12.7)	16.2 (10.4)	17.3 (12.9)	0.006
Birth weight (g)	1078 (280)	1033 (281)	1096 (280)	1038 (309)	1052 (230)	0.13
Gestational age (weeks)	28 (2.8)	27 (2.9)	28 (2.7)	29 (3.1)	29 (3.0)	0.05
Health-related quality of life§	87.4 (11.9)	83.0 (14.3)	88.2 (11.6)	89.8 (8.8)	86.5 (10.7)	0.005
Neighbourhood Disadvantage Index¶	0 (1)	1.4 (1.2)	0 (0.6)	1.0 (1.1)	0 (1)	<0.001

*Includes black, non-Hispanic, white, non-Hispanic and Hispanic; the 'Other' category was omitted for this table because of the small cell sizes.
†Parent-reported asthma.
‡Neonatal severity is measured using the Score for Neonatal Acute Physiology-II (range 0–115), higher score-associated with greater severity.
§Health-related quality of life, measured using the Paediatric Quality of Life Inventory, scores ranging from 0 to 100; higher scores indicate a higher health-related quality of life.
¶The Neighbourhood Disadvantage Index sums the standardised neighbourhood variables, weighted by their factor loadings.

MPlus.[32] The institutional review board at University of Wisconsin-Madison and all participating institutions approved this study.

RESULTS

Race differences in HRQoL

In table 3, Model 1 indicates that there is a significant between-neighbourhood variability in HRQoL ($\widehat{\sigma}^2$=43.1, SE ($\widehat{\sigma}^2$)=12.5). Model 2 suggests that over 30% of this between-neighbourhood variability is attributable to family sociodemographic and child characteristics (Model 2 reduces the variability to $\widehat{\sigma}^2$=28.8, SE ($\widehat{\sigma}^2$)=13.9).

In Model 2, we test our first hypothesis and find that black race is associated with a statistically significant, nearly four-point deficit in HRQoL relative to being white, non-Hispanic (β =−3.6, SE (β)=1.7), even when controlling for other child and family characteristics. Hispanic children and those whose race is reported as 'other' do not have HRQoL scores that are different from those of non-Hispanic white children. Lower birth weight, higher neonatal severity and prenatal smoking exposure are each associated with a lower level of HRQoL. The detrimental effects on HRQoL of lower birth weight persist across all models.

Table 2 Descriptive statistics and factor loadings of neighbourhood disadvantage characteristics for a cohort of children (n=660), age 2–3, who were born very low birth weight

Characteristic	Percentage (SE %)	Range across CT (%)	Factor loading
Neighbourhood disadvantage			
Percentage of families living in poverty	8 (0.001)	0 to 61	0.95
Percentage of households with incomes above the state median	48 (0.006)	4 to 88	−0.77
Percentage of females with a bachelor's degree	15 (0.003)	0 to 45	−0.57
Percentage with a single female head of household	3 (0.001)	0 to 21	0.84
Percentage of unemployed mothers of young children	4 (0.002)	0 to 44	0.57
	Mean		
Summative Neighbourhood Disadvantage Index*	0	−2 to 4	

*Neighbourhood Disadvantage Index sums the standardised neighbourhood variables, weighted by their factor loadings.

Influence of asthma on the relationship between a child's race and HRQoL

Model 3 adds asthma and demonstrates that asthma has a statistically significant, independent, negative association with HRQoL (β =−3.6, SE (β)=1.2). Moreover, when asthma is included, the parameter estimate for black race is reduced from −3.6 (p<0.05) in Model 2 to 0.08 (p>0.05) in Model 3. This confirms our second hypothesis that the lower HRQoL of black children who were born VLBW is substantially due to their greater likelihood of having asthma. The attenuation of the parameter estimate for black children indicates that an asthma diagnosis is likely a significant explanatory contributor to the relationship between black race and HRQoL. However, including asthma diagnosis substantially increases the between neighbourhood variability in HRQoL, suggesting cross-level confounding[30] (ie, an increase in between-neighbourhood variance associated with an individual-level covariate suggests the

Table 3 Fixed and random effects for a series of linear multilevel random intercept models of quality of life among a cohort (n=660), aged 2–3, who were born very low birth weight

Variable	Model 1: null	Model 2: socio-demographic	Model 3: asthma	Model 4: asthma by race	Model 5: neighbourhood disadvantage	Model 6: neighbourhood disadvantage, race and asthma
	Fixed-effects β se (β)					
Intercept	87.5 (0.48)***	90.9 (6.9)***	88.8 (6.7)***	92.8 (6.7)***	91.1 (7.1)***	90.2 (6.1)***
Parent's education		−0.30 (0.70)	0.28 (0.68)	0.28 (0.68)	0.08 (0.71)	0.36 (0.73)
Neonatal health severity (mean)†		−0.10 (0.05)*	−0.05 (0.04)	−0.05 (0.04)	−0.06 (0.05)	−0.04 (0.05)
Child's race						
White, non-Hispanic		Reference	Reference	Reference	Reference	Reference
Black, non-Hispanic		−3.6 (1.7)*	0.08 (1.7)	−1.37 (3.0)	0.64 (2.4)	1.9 (3.3)
Hispanic		048 (1.9)	2.1 (1.8)	0.98 (21)	0.99 (2.2)	2.2 (2.5)
Other		−0.48 (2.8)	0.09 (2.7)	−1.4 (3.0)	−0.87 (3.4)	−2.0 (3.4)
Family income		0.18 (0.79)	0.09 (0.76)	0.16 (0.78)	0.33 (0.82)	0.18 (0.81)
Birth weight (g)		0.005 (0.003)*	0.005 (0.003)*	0.005 (0.003)*	0.005 (0.003)*	0.005 (0.003)*
Gestational age (weeks)		−0.23 (0.28)	−0.31 (0.27)	−0.31 (0.27)	−0.20 (0.27)	−0.27 (0.27)
Mother smoked during pregnancy						
Yes		−3.32 (1.4)*	−2.4 (1.4)	−2.3 (1.4)	−2.4 (1.5)	−2.1 (1.5)
No		Reference	Reference	Reference	Reference	Reference
Child attends daycare						
Yes		0.32 (0.63)	0.39 (0.60)	0.29 (0.59)	0.15 (0.63)	0.13 (0.63)
No		Reference	Reference	Reference	Reference	Reference
Child's sex						
Male		−1.6 (0.98)	−0.56 (0.93)	−0.55 (0.90)	−0.53 (0.98)	−0.46 (0.97)
Female		Reference	Reference	Reference	Reference	Reference
Child lives in a single-parent household						
Yes		−3.3 (1.4)*	−1.1 (1.4)	−1.19 (1.5)	−1.38 (1.5)	−1.0 (1.5)
No		Reference	Reference	Reference	Reference	Reference
Mother received no prenatal care						
Yes		1.1 (2.9)	1.1 (2.9)	1.0 (2.7)	1.5 (3.0)	1.5 (2.2)
No		Reference	Reference	Reference	Reference	Reference
Child lives with a current smoker						
Yes		3.1 (2.1)	2.6 (2.0)	2.3 (2.0)	2.2 (2.1)	1.9 (2.1)
No		Reference	Reference	Reference	Reference	Reference
Child has asthma‡						
Yes			−6.2 (1.7)***	−5.6 (2.1)**	−4.1 (1.1)**	−5.2 (2.1)*
No			Reference	Reference	Reference	Reference
Child's race×asthma						
WNH×asthma				Reference	Reference	Reference
BNH×asthma				−2.59 (2.0)	−5.8 (2.7)*	−9.5 (5.0)*
Hisp×asthma				3.41 (4.6)	1.5 (4.3)	2.4 (5.4)
Other×asthma				6.44 (2.9)	2.7 (6.7)	4.3 (6.7)
Neighbourhood disadvantage§					0.09 (0.2)	0.39 (0.3)
Child's race×neighbourhood disadvantage						
WNH×disadvantage						Reference
BNH×disadvantage						−0.21 (0.5)
Hisp×disadvantage						−0.28 (0.4)
Other×disadvantage						2.9 (1.2)
Neighbourhood disadvantage×asthma						
Disadvantage×asthma						−2.1 (0.8)*
Disadvantage×no asthma						Reference
Neighbourhood disadvantage×asthma×race						
Disadvantage×WNH×asthma						Reference
Disadvantage×BNH×asthma						1.2 (0.8)
Disadvantage×Hisp×asthma						−0.79 (1.1)
Disadvantage×Other×asthma						−3.4 (1.6)*
	Random-effects σ^2 SE (σ^2)					
Between-neighbourhood variability	43.1 (12.5)**	28.8 (13.9)*	59.1 (14.1)***	57.9 (14.1)***	58.0 (14.7)**	58.0 (14.7)**
−2 res log likelihood	5577.3	4787.7	4628.6	4463.9	4280.9	4251.1

*p<0.05; **p<0.01; ***p<0.001.
†Neonatal severity is measured using the Score for Neonatal Acute Physiology-II (range 0–115), higher score associated with greater severity.
‡Measured by parent report.
§Neighbourhood Disadvantage Index sums the standardised neighbourhood variables, weighted by their factor loadings.
BNH, black, non-Hispanic; Hisp, Hispanic; WNH, white, non-Hispanic.

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individual-level covariate asthma is highly correlated with observed and unobserved contextual factors).

Interaction between child's race and asthma on HRQoL

Model 4 tests our third hypothesis that the association between asthma and HRQoL is stronger for black children. This hypothesis is not supported, as the results demonstrate a non-significant race by asthma interaction ($p>0.05$). When children born with VLBW have asthma, it is associated with worse HRQoL, no matter what the race of the child.

Influence of neighbourhood disadvantage, child's race and asthma on HRQoL

Model 5 suggests that inclusion of neighbourhood disadvantage results in a significant estimate for the interaction between black race and asthma ($\beta=-5.8$, SE (β)=2.7). This suppressor effect of neighbourhood disadvantage, coupled with the previously mentioned increases in between-neighbourhood variance (Models 3–5), suggests confounding (at either level 1 or 2) and provides justification for the inclusion of additional two-way and three-way interactions between race, asthma and neighbourhood disadvantage (Model 6).

Figure 1 depicts the results of a model including a three-way interaction between race, asthma and neighbourhood disadvantage (and all relevant two-way interaction terms) (Model 6). While there are no appreciable differences in HRQoL for children (black and white) without asthma, there is a neighbourhood risk gradient in HRQoL whereby black and white children with asthma fare worst. These results suggest that the interactive effects of having asthma and living in a disadvantaged neighbourhood are detrimental to the HRQoL of both black and white children. The graph indicates that the three-way interaction arose from white children in the most advantaged neighbourhoods not having a decrement in HRQoL with asthma.

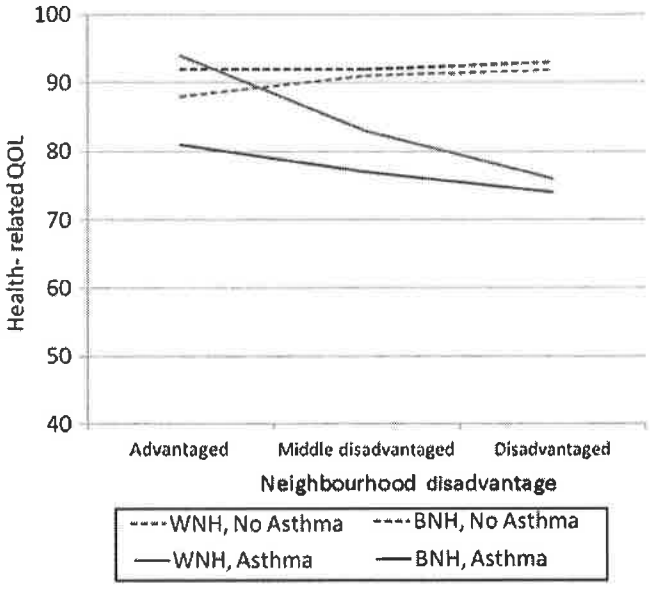

Figure 1 Health-related quality of life (QOL), by neighbourhood disadvantage, for white, non-Hispanic (WNH) and black, non-Hispanic (BNH) children with and without asthma, conditional on child and family characteristics (conditional on birth weight, gestational age and neonatal severity at mean values, living in a single-parent household, and being exposed to prenatal smoking, living with a smoker, no prenatal care and child attending daycare). Neighborhood disadvantage was derived from a principal-component analysis of five socio-economic indicators measured at the census tract level. For interpretation, index scores were categorized as advantaged (>−1 SD below mean), disadvantaged (>1 SD above the mean) and otherwise, middle disadvantage (mean).

DISCUSSION

We examined racial disparities in HRQoL in a regional cohort of 2- and 3-year-olds born VLBW. Our results demonstrate significant black–white disparities in HRQoL that appear to operate, largely, through childhood asthma. Moreover, neighbourhood disadvantage appears to be associated with lower HRQoL for children with asthma. To our knowledge, this is the first study to explore multiple ecological determinants of racial disparities in HRQoL in young children born VLBW.

We find nearly a four-point lower HRQoL among black than white VLBW children, controlling for child and family characteristics. Unpacking explanations for this remaining black–white disparity in HRQoL is challenging, yet our results highlight the potential impact of asthma. Above and beyond race and other child and family socio-economic characteristics, asthma is a statistically significant and clinically meaningful predictor of lower HRQoL. Furthermore, the significant association between race and HRQoL is substantially attenuated (virtually eliminated) when asthma is taken into account. That is, we found evidence that asthma is on the casual pathway (ie, effect mediation) between race and HRQoL. We found no evidence that HRQoL differs between black versus white VLBW children with asthma (ie, effect modification).

We found evidence that, net of child and family characteristics, neighbourhood disadvantage independently influences HRQoL among children with asthma. Children with asthma who live in socially disadvantaged neighbourhoods may not have access to, and the quality of, resources such as health clinics and safe housing. Alternatively, families may have difficulty accessing resources owing to limited transportation, safety or lack of coordinated systems of care. Nonetheless, our findings are consistent with previous research[14 33] suggesting a persistent black–white disparity in asthma morbidity, even when adjusting for individual and neighbourhood socio-economic characteristics. This persistent racial disparity warrants further investigation. For example, there is growing evidence[16 17] to suggest the importance of process (eg, collective efficacy and community psychosocial stress), residential segregation and housing quality. Cagney and Browning[17] demonstrated a higher prevalence of asthma, not explained by family characteristics and neighbourhood poverty, in neighbourhoods with low levels of collective efficacy and social disorder.

We expected that living in a socially disadvantaged neighbourhood would be particularly harmful for HRQoL of black children with asthma, yet we find no evidence of this. Instead, our findings (although not statistically significant) suggest that black VLBW children with asthma living in socially advantaged neighbourhoods fare worse than their white counterparts. Thus, it is plausible that black children with asthma living in more socially advantaged areas experience differential psychosocial (eg, racial discrimination) and environmental stressors than their white peers with asthma, which contribute to a lower HRQoL. Moreover, at the worst levels of neighbourhood social disadvantage, all children fare poorly regardless of their race. The exception to this seems to be children characterised as 'other' race, who seem to fare particularly poorly in socially disadvantaged areas. However, these results should be interpreted with caution owing to sparse sample sizes.

We acknowledge several limitations to this study. First, these data are cross-sectional, which precludes inferring causal

relationships. The relationships we describe are difficult to fully capture through measured characteristics. It is possible that biases are introduced not only by unobserved characteristics of children and families that influence asthma and HRQoL, but also by virtue of the fact that families are not randomly assigned to neighbourhoods, but rather may select or are forced to select their residence based on factors that are also relevant to disease burden and HRQoL.

There is no gold standard for measuring paediatric HRQoL. However, as previously mentioned, the PedsQL not only has strong psychometric properties overall,[24] but also is appropriate for use with paediatric asthmatic populations.[6] Finally, we utilised parent-reported asthma, which may introduce biases if differences in reporting occur across relevant racial and social subgroups. We have no means of quantifying potential reporting bias but rely on previously research[3] suggesting the strength of parent-reported health status of children. In sensitivity analyses, we used a measure of children's use of asthma medication (a marker for at least some access to the medical system) and found similar results. Lastly, the results of the model including the interactions between race, asthma and neighbourhood social disadvantage should be interpreted with caution, as the sample sizes were small (n=34, 18 and 11 for the most, middle and least disadvantaged groups, respectively). While potentially interesting, this finding requires replication with larger sample sizes.

Despite these limitations, the study strengths underscore its population health significance. We utilise a measure of HRQoL rather than measures of impairments or disability. To our knowledge, this is the first study to explore not only overall racial disparities in HRQoL among VLBW children but also the potential mechanisms associated with a common and costly chronic disease in the VLBW population—asthma. We provide evidence for the importance of social context (neighbourhood disadvantage) on understanding the inter-relationships between race, asthma and HRQoL.

A growing number of babies are born VLBW, with consequences for both their own quality of life and society's use of resources to address potential negative outcomes. Racial disparities in VLBW that manifest early in the life course have implications for subsequent disparities in a range of long-term outcomes for children and society. Understanding how we might intervene to reduce the negative outcomes of VLBW, such as asthma, or to buffer their implications for HRQoL, seem appropriate societal concerns. Our study suggests that the neighbourhood context within which asthma arises may be an important ecological niche within which to intervene or focus attention to improve both asthma and its HRQoL consequences.

Funding BMM acknowledges the Robert Wood Johnson Health and Society Scholar Program for funding this research. This research was also funded by the National Heart, Blood, and Lung Institute (HL038149).

Competing interests None.

Ethics approval Ethics approval was provided by the University of Wisconsin-Madison.

Provenance and peer review Not commissioned; not externally peer reviewed.

What is already known on this subject

- Very-low-birth-weight (VLBW; <1500 g) children are at heightened risk for poorer overall physical health.
- VLBW children have a lower health-related quality of life (HRQoL) and risk for asthma than their normal-birth-weight peers. Asthma risk is substantially increased for black, non-Hispanic VLBW children and children living in disadvantaged neighbourhoods.
- To our knowledge, no study has examined racial disparities in HRQoL among VLBW preschoolers, generally, or the additive or interactive effects of child's race, asthma and neighbourhood disadvantage.

What this study adds

- This study utilises multilevel linear regression modelling to describe the contributions of child's race, asthma and neighbourhood disadvantage on HRQoL conditional on a host of child and family health and socio-demographic covariates, in a cohort of VLBW children.
- In a regional cohort of VLBW 2- and 3-year-olds, black, non-Hispanic children demonstrate a statistically significant and clinically meaningful lower HRQoL than their white, non-Hispanic counterparts.
- Asthma explains most of the observed association between black race and HRQoL.
- The HRQoL of children born VLBW is particularly compromised for children with asthma living in more disadvantaged neighbourhoods.

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American Journal of Epidemiology Advance Access published April 17, 2012

American Journal of Epidemiology

DOI: 10.1093/aje/kwr454

Original Contribution

Differential Associations Between the Food Environment Near Schools and Childhood Overweight Across Race/Ethnicity, Gender, and Grade

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Initially submitted July 22, 2011; accepted for publication November 9, 2011.

Epidemiologic studies have observed influences of the food environment near schools on children's overweight status but have not systematically assessed the associations by race, sex, and grade. The authors examined whether the associations between franchised fast food restaurant or convenience store density near schools and overweight varied by these factors using data for 926,018 children (31.3% white, 55.1% Hispanic, 5.7% black, and 8% Asian) in fifth, seventh, or ninth grade, nested in 6,362 schools. Cross-sectional data were from the 2007 California physical fitness test (also known as "Fitnessgram"), InfoUSA, the California Department of Education, and the 2000 US Census. In adjusted models, the overweight prevalence ratio comparing children in schools with 1 or more versus 0 fast food restaurants was 1.02 (95% confidence interval (CI): 1.01, 1.03), with a higher prevalence ratio among girls compared with boys. The association varied by student's race/ethnicity ($P = 0.003$): Among Hispanics, the prevalence ratio = 1.02 (95% CI: 1.01, 1.03); among blacks, the prevalence ratio = 1.03 (95% CI: 1.00, 1.06), but among Asians the prevalence ratio = 0.94 (95% CI: 0.91, 0.97). For each additional convenience store, the prevalence ratio was 1.01 (95% CI: 1.00, 1.01), with a higher prevalence ratio among fifth grade children. Nuanced understanding of the impact of food environments near schools by race/ethnicity, sex, and grade may help to elucidate the etiology of childhood overweight and related race/ethnic disparities.

childhood obesity; food environment; health disparities; log-binomial models; schools

Abbreviation: CI, confidence interval.

In the past decade, concern has been growing over the retail food environment because of its potential influence on children's diet and body weight (1–7) and the need for interventions to reverse the childhood obesity epidemic (8–10). Although influences of the food environment on children's weight have been observed, little research has systematically assessed whether the associations differ across race/ethnic groups (8), by sex or grade level, or whether there are threshold effects.

Because children spend a large proportion of their time in schools (11) and are exposed to the environments surrounding schools (e.g., on their way to school), food types available near or around schools may be particularly important for children's weight. The presence of fast food restaurants and convenience stores near schools may shape children's diet and body weight directly through availability (12–14) and consumption (15) of energy-dense foods and beverages or indirectly via advertising (16–18). One-third of all US public middle and high schools have at least 1 fast food restaurant or convenience store within walking distance, with higher rates around schools attended by Hispanic and black children (5, 12, 19–22).

Using data pertaining to children in the fifth, seventh, and ninth grades attending public California schools, we examined 1) the association of fast food restaurants and convenience store availability within a half-mile (0.81-km) radius of schools with childhood overweight; 2) differences in the association across 4 major race/ethnic groups—Asian, black, Hispanic, and white children, by gender or grade levels; and 3) the possibility of nonlinearity in these associations.

MATERIALS AND METHODS

Data sources

We utilized data for children who participated in the 2007 California physical fitness test (also known as "Fitnessgram"), collected between February and May of 2007 (23). Obtained through a special request from the California Department of Education, Fitnessgram data contain direct measures of children's weight, height, and physical fitness among all children attending fifth, seventh, and ninth grades, as well as their age, sex, race/ethnicity, and a school identifier indicating the school attended by each child at the time of the test.

The locations of fast food restaurants and convenience stores in California were purchased from InfoUSA, a commercial source (24), in January 2009. To select food outlets, Standard Industry Classification codes were used in conjunction with a list of national and local food outlet franchises generated by InfoUSA. Any businesses opened after 2007 were removed. Fast food places were selected if their primary, secondary, or tertiary Standard Industry Classification code was 581208 (restaurant) and the food outlet was a known franchise (refer to Web Table 1 for the list used) (Web Table 1, along with the Web Appendix and Web Figures 1–5, is posted on the *Journal*'s Web site (http://www.aje.oxfordjournals.org/)). Convenience stores were those establishments with a primary, secondary, or tertiary Standard Industry Classification code of 541103 (convenience store) regardless of their franchise status. Geocodes (latitude and longitude) for all locations were provided by InfoUSA, with a reported 90% accuracy in code to the business street address (24).

We obtained school-level data from California Department of Education databases (25) and the 2000 US Census (26). Information on enrollment by grade and race/ethnicity and percentage of students eligible for free or reduced price meals during the 2006–2007 school year, as well as school open and close dates and school addresses, was obtained. Using school addresses, we successfully geocoded 96.7% of schools to latitude and longitude and to census tracts via geographic information system software. Census data from Summary File 3 of the 2000 Census were merged with school-level data from the California Department of Education by using census tract identifiers and subsequently with Fitnessgram data by using school identifiers.

Measures

We used combined overweight or obese status (hereafter "overweight") versus not as a dichotomous outcome. Children were classified as overweight (27) if their age- and sex-specific body mass index (weight (kg)/height $(m)^2$) was at or above the 85th percentile of the 2000 Centers for Disease Control and Prevention reference body mass index distribution (28). Given the evolving definitions of "overweight" (27) and the debated cutoffs to classify Asians as being overweight, we also used continuous body mass index and body mass index z scores (28) and dichotomous obese classification (body mass index: $\geq$95th percentile (27)) in sensitivity analyses.

The primary predictors were the number of fast food restaurants or convenience stores within a half mile buffer around each school, a distance that can be walked by an adult in 10 minutes (29). Using geographic information system software, fast food restaurant and convenience store locations were merged with school locations to obtain the count of food outlets within the school buffer.

We included age, gender, race/ethnicity (Asian, blacks, Hispanics, and whites), as well as physical fitness, in the analyses. Using established criteria (30), we classified each child as unfit, fit, or fit above standard on the basis of the time the child took to run or walk 1 mile (1.6 km). Because Fitnessgram data do not include children's socioeconomic status, we used the proportion of students eligible for free or reduced-price meals within a school as a proxy.

To account for the influences of school-level factors on childhood obesity, we used the proportion of adults with a college degree or higher within the school's census tract as a measure of the school's neighborhood socioeconomic resources (31, 32). School census tract annual median household income was not included in the final models because of high collinearity with the education variable. To adjust the models for racial/ethnic composition of the school, we computed a racial/ethnic heterogeneity measure (33) previously used to quantify residential segregation (34). The measure is computed on the basis of the proportions of the major racial/ethnic groups in the school; it ranges from 0, indicating homogeneity, to 1, indicating that all groups were equally represented.

For descriptive purposes, we categorized schools according to the racial/ethnic majority of the student body. For example, if a school's student body was more than 50% Hispanic, the school was categorized as having an Hispanic majority, and similarly for other race/ethnicities.

Data exclusions

The 2007 Fitnessgram data consisted of 1,419,159 children. Children with race/ethnicity other than the 4 major groups were excluded (5.2%). To protect children's confidentiality, the California Department of Education masked school identifiers for children who attended a school with 10 or less children of the same grade, sex, and race/ethnicity. Thus, these children (8.9%) were excluded because their school environment could not be identified. An additional 0.3% of children with available identifiers were excluded because they could not be matched to a school (e.g., they likely attended one of the 3.3% of schools that could not be geocoded). Children with incomplete data on demographics (3.2%), fitness (13.4%), or overweight status (3.7%) were also excluded, yielding an analytical sample of 926,018 children.

Statistical analysis

We calculated descriptive statistics for children and schools, overall and by either individual race/ethnicity or the racial/ethnic composition of the school. Using individual-level overweight status, we fitted log-binomial regression models (35) for clustered data, treating the school as the clustering

Table 2. Characteristics of California Public Schools Included in This Study According to 2007 Data[a]

	All (n = 6,362, 100%)			Racial/Ethnic Majority in the School[b,c]													
				None (n = 1,493, 23%)			White (n = 1,691, 27%)			Hispanic (n = 2,913, 46%)			Black (n = 95, 1.4%)			Asian (n = 170, 2.6%)		
	%	Median	IQR[d]	%	Median	IQR	%	Median	IQR	%	Median	IQR	%	Median	IQR	%	Median	IQR
No. of fast food restaurants																		
0	55.6			56.1			68.2			49.2			42.1			45.3		
1	16.4			14.9			14.2			18.0			16.8			24.7		
2	11.2			12.2			8.6			11.9			13.7			15.9		
≥3	16.7			16.8			9.0			20.9			27.4			14.1		
No. of convenience stores																		
0	54.8			59.6			71.2			42.9			49.5			57.1		
1	25.7			25.4			19.7			29.2			23.2			28.8		
2	12.8			10.7			6.6			17.5			17.9			7.6		
≥3	6.7			4.3			2.5			10.4			9.5			6.5		
Diversity index		0.60	0.43–0.71		0.79	0.73–0.85		0.57	0.46–0.65		0.49	0.30–0.62		0.53	0.40–0.57		0.61	0.54–0.68
Percent of free or reduced price meals participation		55.4	26.3–79.4		41.6	26.7–58.1		18.7	8.0–35.1		78.3	64.0–89.0		73.6	62.9–82.3		31.4	5.9–67.4
Enrollment		637	462–827		652	484–992		558	441–817		682	502–926		370	245–555		673	520–887
Percent with bachelor's degree or more in the census tract surrounding the school		18.6	9.7–32.5		25.2	16.0–36.6		30.8	19.3–46.0		10.2	5.8–17.7		16.2	10.0–29.5		42.3	24.3–57.5

Abbreviation: IQR, interquartile range.

[a] Data are from databases of InfoUSA on food outlet locations, the 2000 US Census, and the California Department of Education.

[b] Racial/ethnic majority in the school was defined as the group that constituted greater than 50% of the student body, according to enrollment data by race/ethnicity administered by the California Department of Education. When no racial/ethnic group exceeded 50% of the student population, the racial/ethnic majority was coded as "none."

[c] For comparison of the distribution of school-level factors by racial/ethnic majority of the schools, all Ps < 0.001 and were obtained from chi-squared tests for categorical variables and Kruskal-Wallis nonparametric tests for continuous variables.

[d] First and third quartiles of distribution.

unit. Fast food restaurant or convenience store densities were modeled separately, each as a categorical variable (i.e., 0, 1, 2, or ≥3 stores). We tested the hypothesis that children in schools within at least 1 grouping of 0, 1, 2, or ≥3 stores differed in overweight prevalence from the others (denoted as $P_{\geq 1 \text{ differs}}$). We included interaction terms to test if the food store count–overweight association differed by race/ethnicity, sex, or grade (denoted as, e.g., $P_{\geq 1 \text{ differs} \times \text{ethnicity}}$). We used these models to estimate and plot crude and adjusted prevalence. We then assessed whether the associations between food store counts and (log) overweight prevalence deviated from linearity by using orthogonal polynomial contrasts, which enable testing of deviations from linearity without fitting models with parametric trends (36). When the association did not deviate from linearity, models with continuous food store counts were used to succinctly present numerical summaries. When the associations were nonlinear (e.g., ceiling effect) for at least 1 group, we estimated differences in overweight odds comparing specific levels of outlet densities (e.g., more than 1 against 0, denoted as >1 vs. 0).

Models were estimated with and without adjustment for potential confounders and predictors of children being excluded (i.e., missing). Adjusting for predictors of missingness can minimize bias (37). Both individual- and school-level correlates were treated as confounders and predictors of missingness. Because children attending schools with smaller enrollments were more likely to have masked school identifiers, we adjusted for total school enrollment. Quadratic terms of school-level variables were included in the models to account for their nonlinear associations with children's overweight.

All analyses were conducted in R, version 2.13.1, language and environment (38). Additional modeling and coding details are available in the Web Appendix.

RESULTS

Of the 926,018 students included in this study, 31.3% were white, 55.1% Hispanic, 5.7% black, and 8.0% Asian (Table 1). Overall, 22.0% of children were obese, and 41.0% were overweight. Asian children had the lowest overweight prevalence (25.7%), followed by white (29.8%), black (42.4%), and Hispanic (49.5%) children.

Study participants were nested within 6,362 schools (Table 2), of which 16%, 11%, and 17%, respectively, had 1, 2, or ≥3 fast food restaurants within a half-mile radius. There were 25% of schools with 1, 12% with 2, and 6.7% with ≥3 convenience stores nearby. The Spearman correlation between the food store counts was 0.38. The correlations between school-level factors and fast food restaurants (or convenience stores) were as follows—diversity index: $r = 0.0$ ($r = -0.11$); percentage of students in free and reduced-price meals program: $r = 0.15$ ($r = 0.31$); enrollment: $r = 0.07$ ($r = 0.04$); and percentage with bachelor's degree in census tract: $r = -0.07$ ($r = -0.24$).

Compared with schools that had a majority white student population, schools with no racial/ethnic majority had a higher concentration of food outlets: 43.9% and 41.4%, respectively, had at least 1 fast food or convenience store compared with 31.8% and 28.8% of white majority schools. The number of these outlets was even greater around schools with majority Hispanic, black, or Asian students: 50.8%, 57.8%, and 55.7%, respectively, had at least 1 fast food restaurant; 42.9%, 49.5%, and 51.1%, respectively, had at least 1 convenience store.

Overall associations

The crude and adjusted associations between fast food restaurant and convenience stores near schools and overweight are shown in Figure 1, A and B. The unadjusted difference in overweight prevalence comparing ≥3 against 0 fast food restaurants or convenience stores was 4% and 9%, respectively (Figure 1A). After adjustment for individual- and school-level covariates, fast food restaurant density appeared to have a ceiling effect (Figure 1A), with no additional increases in overweight prevalence associated with the availability of 2 versus 1 or ≥3 versus 2 outlets in the school's neighborhood (deviation from linearity test, quadratic contrast $P = 0.021$). Although the confidence interval for the linear dose-response was consistent with a null association with the prevalence ratio = 1.00 (95% confidence interval (CI): 1.00, 1.01) ($P = 0.08$), children attending schools with 1 or more fast food restaurants had 2% higher overweight prevalence with the prevalence ratio = 1.02 (95% CI: 1.01, 1.03) than those in schools with no fast food outlets within the school's half-mile radius (Figure 1B). In adjusted models, the number of convenience stores was linearly associated with higher overweight prevalence (Figure 1A). Each additional convenience store available within a half-mile radius of a school was associated with an estimated 1% higher overweight prevalence with the prevalence ratio = 1.01 (95% CI: 1.00, 1.01) (Figure 1B).

Who is most affected?

There was evidence of a differential effect of the food environment near schools by race/ethnicity but no strong indication of differential effects by grade or sex.

Race/ethnicity. In adjusted models, we found a differential influence of fast food restaurant density near a school on overweight by student race/ethnicity ($P_{\geq 1 \text{ differs} \times \text{ethnicity}} = 0.003$) (Figure 2A). Although the gradients were attenuated after adjustment for individual and school characteristics, greater fast food restaurant density was associated with higher overweight prevalence, except among Asians. Confidence intervals for the fast food restaurant–overweight associations among white children included the null value, but among Hispanics and blacks each fast food restaurant near a school was associated with higher overweight prevalence (prevalence ratio = 1.02, 95% CI: 1.01, 1.03; and prevalence ratio = 1.03, 95% CI: 1.00, 1.06, respectively) (Figure 2B). Among Asian children, fast food restaurants and overweight were inversely associated (prevalence ratio = 0.94, 95% CI: 0.91, 0.97 for ≥1 vs. 0 fast food restaurants).

Although an increasing trend in the association between convenience store density and overweight was found among white, black, and Hispanic but not Asian children, the associations were consistent with homogeneous effects across race/ethnicity in adjusted models (Web Figure 1A;

Table 1. Characteristics of California Children in the Study Sample Overall and by Race/Ethnicity According to 2007 Data[a]

	Overall (n = 926,018, 100%)		Individual-Level Race/Ethnicity[b]							
			White (n = 289,599, 31.3%)		Hispanic (n = 510,089, 55.1%)		Black (n = 52,553, 5.7%)		Asian (n = 73,777, 8.0%)	
	%	Mean	%	Mean	%	Mean	%	Mean	%	Mean
Body mass index[c]		20.8		19.9		21.8		21.3		19.5
Body mass index *z* score		0.78		0.49		1.02		0.84		0.36
Overweight or obese (body mass index *z* score, ≥85th percentile)	41.0		29.8		49.5		42.4		25.7	
Obese (body mass index *z* score, ≥95th percentile)	22.0		13.3		28.5		22.2		10.5	
Female sex	48.8		48.2		49.0		49.6		48.6	
Grade level										
Fifth	30.2		28.6		33.2		20.7		22.5	
Seventh	36.0		35.8		35.3		41.9		37.8	
Ninth	33.8		35.6		31.5		37.4		39.8	
Age group, years										
10	15.9		14.5		17.5		11.2		13.5	
11	13.4		13.6		14.4		8.7		8.9	
12	19.8		18.8		19.7		22.8		22.6	
13	15.9		16.8		15.4		18.2		14.8	
14	18.8		18.9		17.8		21.5		23.7	
≥15	16.0		17.5		15.0		17.4		16.5	
Fitness status[d]										
Fit	44.5		46.3		42.7		39.8		53.5	
Unfit	36.8		28.2		42.3		46.8		25.3	
Fit above standard	18.6		25.4		15.0		13.5		21.3	

[a] Data are from the 2007 California physical fitness test (also known as "Fitnessgram") and the California Department of Education.
[b] For comparison across race/ethnicity, all P's < 0.001 and were derived from chi-squared tests for categorical variables or Kruskal-Wallis nonparametric rank tests for continuous variables.
[c] Body mass index: weight (kg)/height $(m)^2$.
[d] Fitness status indicates whether a child was within, below, or above the Cooper Institute's age- and gender-specific healthy fitness zone based on his/her performance in the 1-mile (1.6-km) run or walk.

$P_{\geq 1 \text{ differs} \times \text{ethnicity}} = 0.35$). Irrespective of race/ethnicity, these associations did not deviate from linearity. Whereas the adjusted convenience store–overweight associations for white and Asian children were consistent with no difference in prevalence, each additional convenience store within a half-mile radius of a school was, respectively, associated with 1% and 2% higher overweight prevalence among Hispanic and black children, with prevalence ratios = 1.01 (95% CI: 1.00, 1.01) and 1.02 (95% CI: 1.00, 1.03) (Web Figure 1B).

For Asian children, adjustment for school- and individual-level correlates had a strong impact on the food environment–overweight associations. Adjustment for school-level correlates impacted the gradient, while individual-level correlates primarily impacted adjusted prevalence. (Refer to the sensitivity analyses in Web Figure 2).

Grade. The estimated fast food restaurant–overweight association appeared to be homogeneous across grade in adjusted models ($P_{\geq 1 \text{ differs} \times \text{grade}} = 0.60$) (Web Figure 3A). Nevertheless, a stronger association between fast food restaurant density and overweight was found among fifth grade children (prevalence ratio = 1.03, 95% CI: 1.01, 1.04) (Web Figure 3B) compared with seventh or ninth grade children. There was weak indication of heterogeneity of the convenience store density–overweight association by grade ($P_{\geq 1 \text{ differs} \times \text{grade}} = 0.12$) (Figure 3A). For each additional convenience store, the prevalence ratio was 1.01 (95% CI: 1.00, 1.02) among fifth graders and 1.01 (95% CI: 1.00, 1.02) among seventh graders. Ninth grade children did not demonstrate higher prevalence with greater convenience store concentration (prevalence ratio = 1.00, 95% CI: 0.99, 1.01) (Figure 3B).

Sex. For both types of food stores, girls had stronger food store–overweight associations. Comparing ≥1 versus 0 fast food restaurants, the prevalence ratio = 1.01 (95% CI: 1.00, 1.02) among boys and 1.02 (95% CI: 1.01, 1.04) among girls. Further, for each additional convenience store,

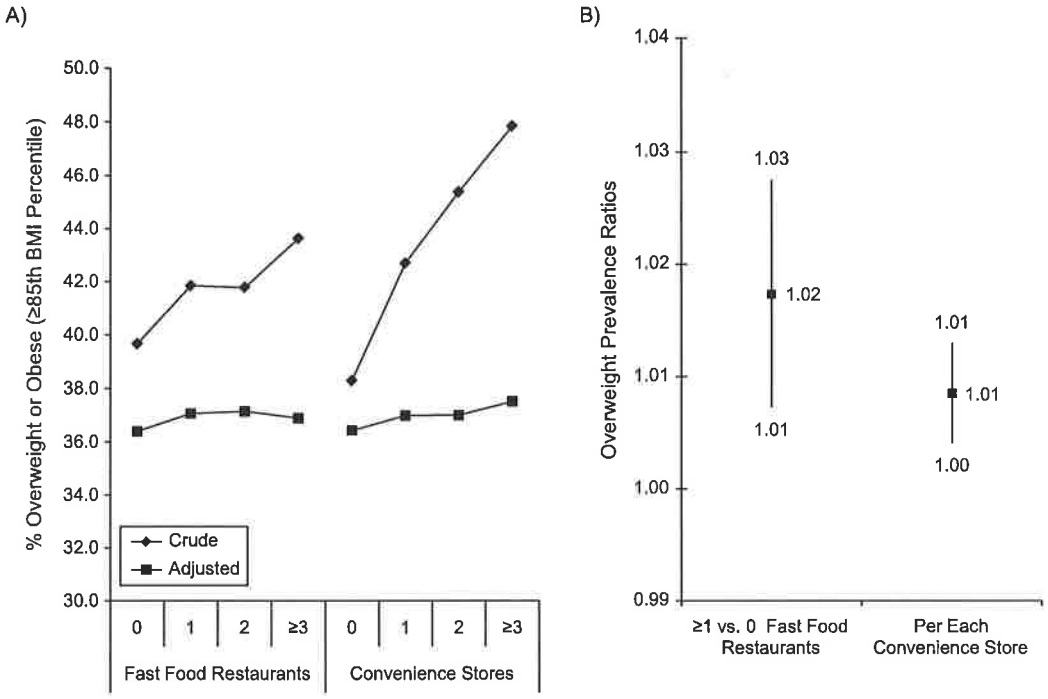

Figure 1. Crude and adjusted model-predicted overweight prevalence by fast food restaurant or convenience store density (A) and adjusted overweight prevalence ratios associated with greater fast food restaurant or convenience store availability (B) among California children tested in 2007. The data sources were the 2007 California physical fitness test (also known as "Fitnessgram"), InfoUSA, the 2000 US Census, and the California Department of Education. Adjusted models include age, sex, physical fitness, and school-level characteristics. In A, the models include categorical food store counts. Because no deviations from linearity were found for the associations with convenience stores, the numerical summaries in B are from models with continuous convenience stores. The point estimate for the fast food restaurant association in B was derived from the model with categorical fast food restaurants by using contrasts. BMI, body mass index.

the prevalence ratio = 1.01 (95% CI: 1.00, 1.01) among boys and 1.01 (95% CI: 1.01, 1.02) among girls. However, overall the evidence of heterogeneity of the food store–overweight associations by sex was weak, particularly for convenience stores (fast food restaurant: $P_{\geq 1 \text{ differs} \times \text{sex}} = 0.06$; convenience store: $P_{\geq 1 \text{ differs} \times \text{sex}} = 0.25$).

The patterns of the association between the nearby food environment and overweight were largely similar regardless of the outcome measures used, including obesity, body mass index, and body mass index z scores (refer to Web Figure 4 and Web Figure 5).

DISCUSSION

Using data involving nearly 1 million racially/ethnically diverse children attending public schools in California, we documented 4 findings in this study concerning the association between the food environment near schools and childhood overweight. First, after control for student and school characteristics, fast food restaurant density around schools was significantly related to overweight prevalence in the overall sample. Second, we found that the influence of fast food restaurant density on overweight was contingent upon student race/ethnicity, with greater density associated with higher overweight prevalence among Hispanics and blacks but lower prevalence among Asians. Third, we documented an association between greater convenience store density and childhood overweight in the overall sample. Fourth, we observed some evidence, although weak, that the food environment may have a stronger influence among younger (fifth grade) children and girls. This study contributes to the limited though growing body of literature concerning the food environment near schools and its potential influence on children's body weight.

Fast food restaurants

Our study provides novel findings concerning the differential influence of fast food restaurant density near schools and overweight prevalence, with greater density associated with higher prevalence among Hispanic and black children but lower prevalence among Asian children. Although the influence of fast food restaurant density on overweight was small among Hispanics and blacks, this may translate into large detrimental effects for the population as a whole (39, 40). In their California study, Davis and Carpenter (1) also found a stronger effect for blacks than for the sample as a whole. Exposure to fast food restaurants near schools may influence susceptibility to overweight especially among

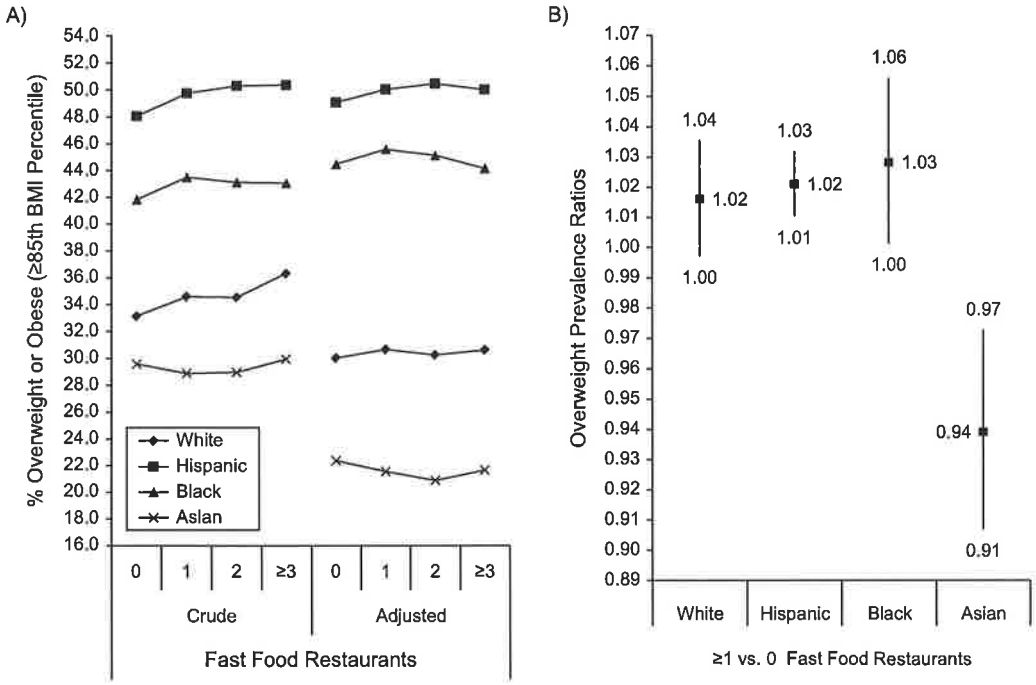

Figure 2. Crude and adjusted model-predicted overweight prevalence by race/ethnicity according to fast food restaurant density (A) and adjusted race/ethnicity-specific associations between fast food restaurant and overweight prevalence ratios (B) among California children tested in 2007. The data sources were the 2007 California physical fitness test (also known as "Fitnessgram"), InfoUSA, the 2000 US Census, and the California Department of Education. The *P* value for race/ethnicity by fast food density interaction = 0.003, derived from a 9-df Wald test, testing if at least 1 interaction term in the adjusted model is significant. Adjusted models include age, sex, fitness, school-level characteristics, and all their interactions with race/ethnicity. The models represented in A include interactions between categorical food store counts and race/ethnicity. Because deviations from linearity were found for the associations with fast food stores, numerical summaries in B were derived by using contrasts from models with interactions of categorical fast food restaurants and race/ethnicity. BMI, body mass index.

black and Hispanic children given this and prior research documenting that schools attended by Hispanic and black youth are disproportionately surrounded by fast food restaurants (20). Moreover, compared with white children, black and Latino youth are more likely to be exposed to fast food advertisements (17) or to recognize fast food logos (16) that may shape their food preferences. Fast food prices are inversely associated with adolescent body mass index, and the effect is greater for teens with lower socioeconomic status (4). The higher obesity risk among black and Hispanic children may be due to increased vulnerability resulting from a combination of greater access, greater exposure to marketing, and affordability of fast foods.

Further research investigating the observed inverse relation between fast food restaurant density and obesity among Asian children is warranted. One plausible explanation is that, although there is diversity in socioeconomic resources across Asian ethnic subgroups (41), in general, Asian children have greater socioeconomic resources than black and Hispanic children (42). Asian children may have increased access to the healthier, but more expensive, food options available in the same fast food restaurants. It is also plausible that other cultural factors such as dietary choices play a role (43), alone or in combination with socioeconomic resources.

Finally, our finding regarding the ceiling effect in the overall association between fast food restaurant density and obesity risk may partly explain the mixed results in this area. Similar to our analyses with linear terms, those of Powell and Bao (4) show null effects of fast food density on adolescent obesity. However, our analyses accounting for ceiling effects are also consistent with those of a study that found that students attending schools with at least 1 fast food restaurant had a 6% increase in the odds for overweight compared with students whose schools were not near fast food outlets (1). Estimated linear dose–response associations will tend to be diluted when associations demonstrate ceiling effects.

Convenience stores

This is one of a handful of studies that examined the impact of convenience store density near schools on student weight outcomes (3, 5, 44). Novel aspects of our study include its systematic analyses regarding the shapes of the associations between convenience store density on obesity across race/ethnic groups and grade levels. The patterns of associations between convenience stores and overweight prevalence were consistent with linear effects among fifth and seventh grade

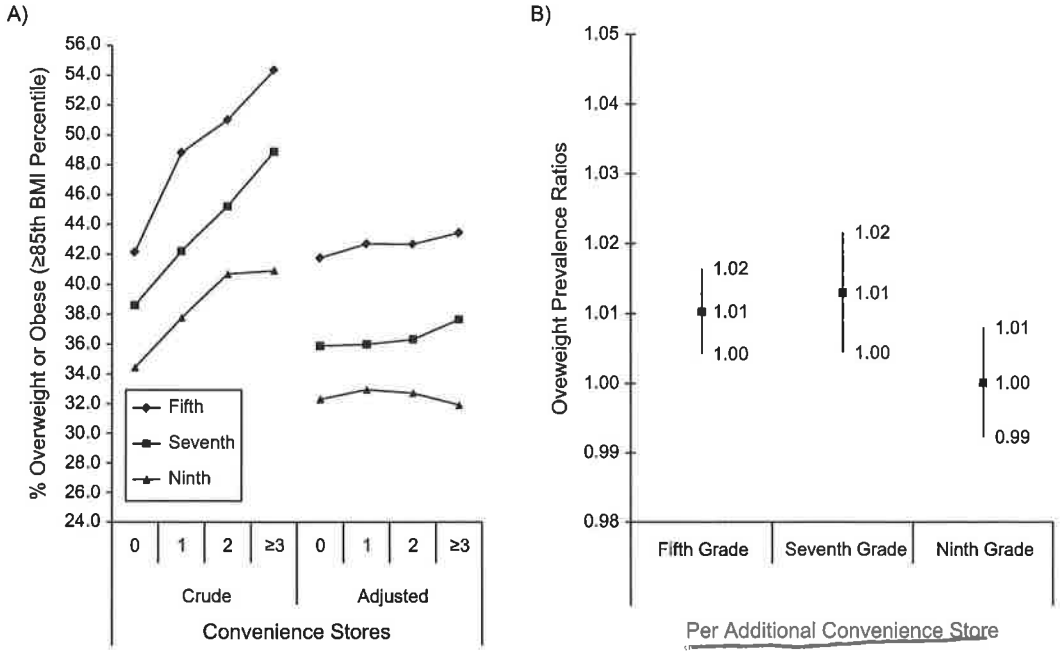

Figure 3. Crude and adjusted model-predicted overweight prevalence by grade according to convenience store density (A) and adjusted grade-specific associations between convenience store density and overweight prevalence (B) among California children tested in 2007. The data sources were the 2007 California physical fitness test (also known as "Fitnessgram"), InfoUSA, the 2000 US Census, and the California Department of Education. The *P* value for the grade by convenience store density interaction = 0.12, derived from a 6-df Wald test, testing if at least 1 interaction term is different from 0. Adjusted models include ethnicity, sex, physical fitness, school-level characteristics, and all their interactions with grade. The models represented in A include interactions between categorical food store counts and grade. Because no deviations from linearity were found for the associations, numerical summaries in B are from models with interactions of continuous convenience stores and grade. BMI, body mass index.

students. Linear dose–response associations between the density of convenience stores and obesity have been reported (3, 7, 17, 45).

Our findings are consistent with literature on both school and residential exposures to convenience stores (45). Powell et al. (3) documented that each additional convenience store per 10,000 capita at the school zip code level was associated with a 0.15% increase in overweight, although their effect is not directly comparable to ours because of their per 10,000 capita adjustment and their use of school-level zip code as a measure of food outlet exposure. School zip codes likely overlap with children's residential environment, which has also been shown to affect childhood overweight. For instance, living on street blocks with 1 or more convenience stores (46) would suggest that the level of aggregation may have a role for capturing effects. It has also been found that a greater concentration of convenience stores at the county level was associated with 30% higher obesity odds among children aged 5–18 years (7).

We documented weak evidence regarding heterogeneity of the food environment's influence on overweight by sex, with point estimates for the associations being higher for girls than boys, as in a prior study (47). However, it is plausible that adjustments for differential effects of other school neighborhood predictors by sex (48) (i.e., models that included sex interactions with all other factors) may have resulted in nearly homogeneous associations between food stores and weight by sex. Further research in this area is warranted.

Limitations

This study is cross-sectional and relied on secondary data, lacking information on potentially important individual- and family-level factors, including socioeconomic status. Although fidelity with test administration protocols may vary, Fitnessgram data included direct measures of height and weight and allowed us to control for physical fitness. Food environment measures were from a commercial source, the accuracy of which is uncertain (20), although these data sources are one of the few available to conduct large-scale studies regarding the availability of food outlets near schools. Because identifying other limited-service restaurants with precision is difficult and the effect of franchised establishments was found previously to be stronger compared with other restaurants (1), we used only franchised fast food restaurants. To our knowledge, the list of convenience stores is comprehensive. Finally, estimating various types of associations (e.g., store-type stores, linear vs. threshold effects, subgroups) may increase false positive findings. However, examination of specific food stores and the shapes of the associations is needed to better understand their potentially distinct influences.

Conclusion

Our study findings suggest a differential influence of fast food restaurant density on overweight, with detrimental effects for Hispanic children and black children. Convenience store density exerted a detrimental influence on children's weight, particularly among fifth and seventh graders. Future programs and policies to reduce race/ethnic disparities and to prevent childhood obesity at the population level stand to benefit from a nuanced understanding of the food environment near schools.

ACKNOWLEDGMENTS

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This research was funded in part by grants from The Robert Wood Johnson Foundation's Healthy Eating Research Initiative, the Salud! America Research Network to Prevent Childhood Obesity among Latino Children, and the Kaiser Permanente Burch Minority Leadership Awards Program. B. N. S. also acknowledges salary support from grant 1-P20-SE018171-01 from the National Institutes of Health.

The authors thank the Center for Advancing Health for providing other support.

Conflict of interest: none declared.

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American Journal of Obstetrics and Gynecology (2004) **191**, 939–44

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The continuum of maternal morbidity and mortality: Factors associated with severity

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Received for publication February 23, 2004; revised May 11, 2004; accepted May 24, 2004

KEY WORDS
Maternal mortality
Maternal morbidity
Preventability
Pregnancy complications

Objective: The goal of this study was to examine whether sociodemographic, clinical, and other service-related factors, as well as preventability issues affect a woman's progression along the continuum of morbidity and mortality.

Study design: This was a case-control study of pregnancy-related deaths, women with near-miss morbidity, and those with other severe, but not life threatening, morbidity. Factors associated with maternal outcome were examined.

Results: Provider factors (related to preventability) and clinical diagnosis were significantly associated with progression along the continuum after controlling for sociodemographic characteristics ($P < .01$ for both associations).

Conclusion: In order to improve mortality rates, we must understand maternal morbidity and how it may lead to death. This study shows that important initiatives include addressing preventability, in particular, provider factors, which may play a role in moving women along the continuum of morbidity and mortality.

More than 25 years ago, the concept of sentinel health events was introduced with maternal deaths cited as an example, yet there has been no improvement in maternal mortality since 1982.[1] The most recent maternal mortality ratio, for 1991 to 1999, was 11.5 deaths per 100,000 live births.[2] One approach to reducing maternal deaths is to examine indicators of maternal health, such as morbidity during pregnancy. Maternal morbidity has been recognized as a public health problem, affecting nearly 1.7 million women annually.[3] Research has shown that the magnitude of maternal morbidity during labor and delivery is greater than generally appreciated. Although the prevalence of any specific morbidity is low, the burden of total morbidity is high, with 43% of women experiencing some form of maternal morbidity, much of which is preventable.[3] Evidence has shown that

Presented at the Twenty-Fourth Annual Meeting of the Society for Maternal Fetal Medicine, February 2-7, 2004, New Orleans, La.

Funding from the Centers for Disease Control and Prevention and the Association of Schools of Public Health "Investigation of Factors Associated with Maternal Mortality" (S1069/19-20).

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0002-9378/$ - see front matter © 2004 Elsevier Inc. All rights reserved.
doi:10.1016/j.ajog.2004.05.099

at least one half of maternal deaths may be preventable through changes in patient, provider, and system factors.[4-6]

Historically, maternal mortality and morbidity have been studied in isolation from one another. By looking only at maternal deaths, developed countries might be in danger of overlooking other major problems in obstetric care.[7] Integrating the study of mortality and morbidity provides the opportunity to gain a clearer picture of how best to improve the care of high-risk women. This approach recognizes that death is the last stop on a continuum of adverse pregnancy events, and focuses research on the nature of how a morbid condition can lead to death.

This continuum can be partitioned into meaningful clinical and epidemiologic ranges that permit an analysis of factors, including preventability factors that may differentiate deaths, near misses, and other severe morbidities. The following set of labels broadly defines major categories along the continuum:

Normal/healthy pregnancy → Morbidity → Severe morbidity → Near miss → Death. While the 2 ends of this continuum are easily identified and labeled, locating intermediate points is far more complex. It has been difficult to define clear thresholds at which a woman can be reliably categorized as a severe morbidity or a near miss, and also difficult to identify the reasons that a woman progresses from one category to the next.

Recently, we developed a scoring system for assigning women to the categories of severe and near-miss morbidity.[8] The research presented here used these categories to examine whether sociodemographic, clinical, and other service-related factors, as well as preventability issues affect a woman's progression along the continuum.

Material and methods

This study was conducted at the University of Illinois Medical Center at Chicago (UIMC), a tertiary care and Regionalized Perinatal Network center that coordinates maternal and neonatal services for 10 hospitals. The hospital serves a predominantly African American and Latina population, and has approximately 2200 births per year. A case control design was used to select pregnancy-related deaths, women with near-miss morbidity, and women with other severe, but not life threatening, morbidity for analysis.

Maternal deaths were identified through state-mandated maternal death reports from the 10 hospitals in the UIMC Perinatal Network from 1992 to 2001. Pregnancy-related death was defined as a death caused by direct or indirect complications of pregnancy occurring during the prenatal period, or within 90 days after delivery or termination of pregnancy. This was the definition used by the state of Illinois during the study time period. Of the 51 deaths initially identified, 14 were deemed unrelated to pregnancy and were excluded from analysis. The remaining 37 mortalities were included in the study sample.

Maternal morbidities from 1995 to 2001 were identified from a variety of data sources at the UIMC, including the hospital discharge data, hospital and departmental quality assurance reports, maternal transport logs, and provider referral. Because UIMC is the tertiary center, many of the women defined as near miss or severe morbidities were transferred to UIMC from the perinatal network hospitals. Because severe maternal morbidity is not always captured through the ICD diagnostic coding system, 3 sets of obstetric indicators were defined to guide the sampling process and reduce the chance of bias from differentially sampling particular diagnoses. The sets of indicators were: (1) diseases/health conditions, (2) morbid events, and (3) procedures or interventions. For example, health conditions such as embolism, morbid events such as hemorrhage, and procedures such as emergency hysterectomy were included.

A team of clinicians, including 2 nurse practioners, 3 maternal and fetal medicine physicians, and 1 PhD researcher with a specialty in maternal health reviewed the medical records in a group setting in order to arrive at consensus on clinical determinations. Of the initial sample of 339 morbidities, 134 women were included in the study based on the criteria described above. More detail regarding the sampling of morbidities can be found in previous publications.[8,9]

The review team also identified factors for inclusion in a scoring system designed to further refine classification of the morbidities along the morbidity continuum. A 5-factor scoring system, including organ system failure, ICU admission, extended intubation, transfusions of >3 units, and surgical intervention was developed to categorize morbidities as either near-miss or other severe morbidity. The total score was a weighted sum of the clinical factors present for each woman. Women with scores of 8 or more out of the possible 15 points were classified as near-miss morbidity (n = 33); women with scores below 8 were classified as severe morbidity (n = 101). Further details of the development of this scoring system have been previously published.[8,9]

A death or morbidity was classified as preventable by consensus of the expert committee. An event was considered preventable if it could have been avoided by any action or inaction on the part of the health care provider (eg, mismanagement of patients, failure or delay in diagnosis), the system (eg, failure in communication), or the patient (eg, noncompliance) that may have caused or contributed to progression to more severe morbidity or death. For this study, therefore,

Table I Proportions of women at different points on the morbidity/mortality continuum with select characteristics

	Death n = 37	Near-miss morbidity n = 33	Other severe morbidity n = 101
Race/ethnicity*			
African American	69.4	45.5	57.4
Asian	8.3	9.1	2.0
Caucasian	22.2	6.1	6.9
Hispanic	0.0	39.4	33.7
Frequency missing = 1			
Age			
10-19	10.8	9.1	12.9
20-34	64.9	66.7	72.3
≥35	24.3	24.2	14.9
Parity*			
Nulliparous	25.0	42.4	34.7
Previous deliveries 1-4	69.4	39.4	59.4
Previous deliveries >4	5.6	18.2	5.9
Frequency missing = 1			
Marital Status*			
Married	54.1	42.4	25.7
Unmarried	46.0	57.6	74.3
Insurance*			
Private	55.9	30.3	37.6
Medicaid	35.3	57.6	60.4
Self-pay/other	8.8	12.1	2.0
Frequency missing = 3			
Prenatal care			
First trimester	68.8	76.9	60.2
Late entry	28.1	15.4	33.3
No prenatal care	3.1	7.7	6.5
Frequency missing = 20			

* Race/ethnicity ($P < .01$), parity ($P = .05$), marital status ($P = .005$), insurance ($P = .02$).

Table II Clinical diagnoses of women at different points on the morbidity/mortality continuum

Diagnosis	Death n = 37	Near-miss morbidity n = 33	Other severe morbidity n = 101
Cardiac (n = 12)	21.6	6.1	7.9
Hemorrhage (n = 44)	18.9	39.4	23.8
Cerebral vascular accident (n = 7)	16.2	3.0	0.0
Embolic (n = 11)	16.2	0.0	5.0
Pregnancy induced hypertension* (n = 58)	10.8	18.2	47.5
Infection (n = 21)	8.1	21.2	10.9
Other medical† (n = 7)	8.1	9.1	1.0
Renal (n = 5)	0.0	3.0	4.0

Chi-square $P < .0001$.

* Preeclampsia, eclampsia, HELLP.

† Other medical: lupus, sickle cell disease, molar pregnancy, pancreatitis, and anesthesia.

preventability does not necessarily mean that the medical condition itself was preventable, but rather that a woman's outcome might have been at a less severe point along the continuum were it not for the preventable event. For example, while preeclampsia itself may not have been preventable, progression from mild to severe preeclampsia or eclampsia might have been avoided.

Medical record abstraction was carried out for the entire study sample of 37 deaths, 33 near misses, and 101 other severe morbidities. Demographic, clinical, and service delivery data were collected and entered into a Microsoft Access database and imported into SAS (Cary, NC) for analysis.

Analysis focused on the association of sociodemographic, clinical, and other service-related factors with progression along the continuum of maternal morbidity. The 3 groups of women—deaths, near misses, and other severe morbidities—were described in terms of sociodemographic characteristics as well as their primary diagnosis. In addition, preventability and components of preventability were described. Chi-square tests were used to compare the 3 groups.

In multivariable analysis, we used multinomial logistic regression to jointly consider the 3 outcome groups. This permitted a unified assessment of the association of factors with the 3 outcome groups acknowledging that they are points on a single continuum. It also permitted separate comparisons of deaths to near misses and near misses to severe morbidities without any assumption that these 2 comparisons would be the same. Using this approach, it is possible to highlight in a single model whether factors associated with death vs survival among the most extremely ill women are different than those associated with severity of illness among the women who survive. For the first comparison, the deaths are the "cases," and the near misses are the "controls." For the second comparison, the near misses are the "cases," and the other severe morbidities are the "controls."

Results

Table I provides a profile of the women in the study according to their location along the morbidity/mortality continuum. The distribution of race/ethnicity, parity, marital status, and insurance status differed within the 3 groups, while there was no difference by age or time of entry into prenatal care. The deaths included higher proportions of African American and Caucasian women compared with the survivors ($P < .01$), and women who died were also more likely to be multiparous, married, and privately insured. These differences may be a reflection of this hospital's population and do not necessarily conform to national data.

The distribution of clinical diagnoses for the deaths, near misses, and other severe morbidities is shown in

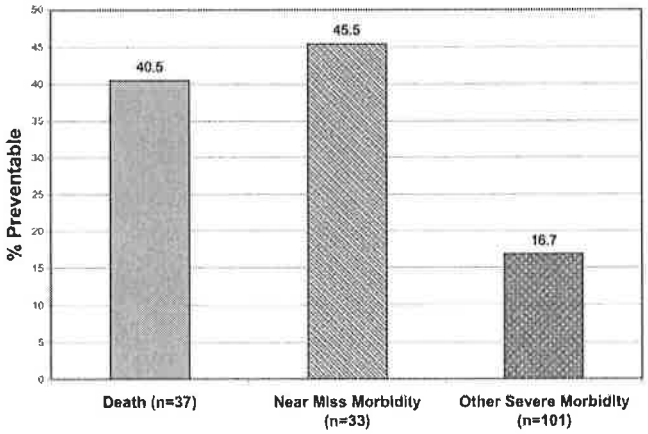

Figure Overall preventability for women at different points on the morbidity/mortality continuum. Overall $P < .01$.

Table II. The pattern of diagnoses differed across the 3 groups ($P < .0001$), with only hemorrhage being a frequently occurring diagnosis in all 3. For example, hemorrhage was the most common diagnosis among the near misses, accounting for 39.4%, while PIH was the most common diagnosis among the severe morbidities, accounting for 47.5%. Among maternal deaths, while cardiac accounted for 21.6% of the deaths, no particular condition was predominant.

The Figure shows the proportion of women whose progression along the morbidity/mortality continuum was deemed preventable. The severe morbidities had a significantly lower rate (17%) of preventable events compared with the deaths (41%) and near-miss morbidities (45%). The proportions of preventable events were significantly different ($P < .01$), with the major difference being between the women who died or were near misses compared with women with severe morbidities

Specific preventability factors are examined in Table III for the women whose place on the continuum was deemed preventable. No significant differences in types of preventability were found across the 3 groups. Patient factors were cited in 13% to 20%, and system factors were cited in 33% to 47% of the women with preventable events. However, provider-related factors were cited for approximately 90% of the preventability in all 3 groups. In particular, incomplete or inappropriate management was the major preventability factor, regardless of the point along the morbidity/mortality continuum.

Table IV provides the results of multivariable modeling. The model included race/ethnicity, maternal age, parity, marital status, insurance status, clinical diagnosis, and types of preventability. The odds ratios and other statistics in Table IV describe the association of these factors with a woman's place on the morbidity/mortality continuum. Clinical diagnosis and provider-related preventability factors were significantly associated with progression along the continuum after controlling for sociodemographic characteristics ($P < .01$ for both associations). System factors and patient factors were not related to progression along the continuum, nor were the sociodemographic characteristics, except for insurance status. Women without health insurance were significantly more likely to progress along the continuum after controlling for other factors in the multivariable model.

Table III Proportion of preventable events by type of preventability among women at different points on the morbidity/mortality continuum

	Potentially preventable outcomes*		
Types of preventability	Death n = 15	Near-miss morbidity n = 15	Other severe morbidity n = 17
Overall system	33.3	46.7	29.4
Communication	13.3	26.7	5.9
Policies/procedures regarding: staff development, care process, equipment, medication, etc	6.7	13.3	11.8
Care process	26.7	20.0	17.7
Overall provider	86.7	93.3	88.2
Failure to identify high-risk status	20.0	13.3	29.4
Incomplete or inappropriate management	86.7	93.3	82.4
No referral to a tertiary care center	0.0	6.7	5.9
Overall patient	20.0	13.3	17.7

* Multiple types of preventability could have been noted for each woman.

Looking at the separate comparison of the deaths to the near misses, none of the adjusted odds ratios for types of preventability were statistically significant, although the odds ratio for provider factors was elevated at 1.71 (95% CI 0.31-9.34). In addition, compared with a diagnosis of hemorrhage, which served as the reference group, women with cardiac disease as well as diagnoses in the Other category (CVA, embolism, renal, and other medical conditions) were more likely to be maternal deaths than near misses.

Looking at the separate comparison of the near misses to severe morbidities, the adjusted odds ratio for preventability due to provider factors was highly significant at 4.22 (95% CI 1.41-12.66), but there was no significant association with either system or patient factors related to preventability. Compared with hemorrhage, women with PIH were more likely to be severe morbidities rather than near misses.

Comment

This study examined the relationship of sociodemographic, clinical, and other service-related factors

Table IV The relationship between types of preventability and clinical diagnosis with different points on the morbidity/mortality continuum*

	Overall comparison	Death vs near miss		Near miss vs other severe morbidity	
	P value	Odds ratio	95% CI	Odds ratio	95% CI
System factors	.96	0.84	0.14-4.87	1.20	0.33-4.33
Provider factors	<.01	1.71	0.31-9.34	4.22	1.41-12.66
Patient factors	.49	0.34	0.05-2.29	1.82	0.51-6.54
Diagnosis	<.01				
Cardiac		11.93	1.00-149.06	0.49	0.07-3.58
Hemorrhage (ref)		—			
Infection		0.89	0.12-6.83	1.24	0.30-5.21
PIH		0.79	0.09-6.95	0.15	0.04-0.58
Other†		9.58	1.46-62.83	1.05	0.23-4.74

* The estimates for types of preventability and diagnosis shown here were adjusted for race/ethnicity (overall $P = .82$), maternal age (overall $P = .76$), parity (overall $P = .18$), marital status (overall $P = .06$), and insurance status (overall $P = .01$).

† CVA, embolism, renal, and other medical conditions.

among women at the 3 most severe points on the morbidity/mortality continuum. Similar to other research, there were different distributions of clinical diagnoses among the 3 groups.[7] The proportion of cardiac, CVA, and embolic diagnoses was higher among the deaths, hemorrhage and infection were higher in the near-miss group, and PIH was higher in the other severe morbidity group. Some diagnoses, such as CVA and embolism, are by definition very severe, and therefore, it is not surprising that the proportion of the deaths with these conditions is greater compared with the proportion of the near misses or other severe morbidities. Conversely, some diagnoses, such as PIH and common infections, are not necessarily life-threatening and afford a better opportunity for intervention. Interestingly, hemorrhage accounted for a high proportion of morbidity and mortality across the 3 groups.

The probability that a woman would progress along the morbidity/mortality continuum was significantly related to whether she had a preventable event. This association between preventable factors, regardless of type, and progression along the continuum remained strong even after controlling for clinical diagnosis and sociodemographic factors. The multivariable results showed that this association was specifically due to provider factors, incomplete or inappropriate management, as opposed to system or patient factors. This is critically important because it means that changes in provider decision-making could reduce the severity of disease experienced by high-risk women.

The association between provider factors and place on the continuum was stronger when comparing the near misses with the severe morbidities than when comparing the deaths with the near misses (OR 4.22 and 1.71, respectively). These observed associations are both strong, even though only the comparison of near misses to severe morbidities reached statistical significance. The odds ratios indicate that a woman with near-miss morbidity was more than 4 times as likely to have had provider-related preventability factors compared with her counterpart with other severe morbidity, while a woman who died was close to 2 times more likely to have had provider-related preventability factors compared with her counterpart with near-miss morbidity. This suggests that changes in provider behavior may have a bigger impact for women at earlier stages along the continuum. It appears that it is much more difficult to impact the outcome of a woman who already has a life-threatening condition.

One limitation of the study was that the mortalities were collected from the entire perinatal network, while the morbidities were collected only from UIMC. However, because UIMC is the tertiary center, many of the near-miss and severe morbidities initiated care at one of the network hospitals, and were transported to UIMC. Additionally, deaths were collected for 3 additional years, which was necessary to gain enough power for analysis because maternal death is a rare event.

It is interesting to speculate how a woman's place on the morbidity/mortality continuum would change if medical decision-making were improved. In our study, if all the women with preventable factors were reassigned to the next less severe point along the continuum, approximately 41% fewer women would have died and would instead be in the near-miss group; 45% of women who were near misses would instead have been in the severe morbidity group; and 17% of women who were other severe morbidities would have been even farther back along the continuum.

Although pregnancy-related mortality is rare, many of these deaths are preventable. Likewise, much pregnancy-related morbidity, which is far more frequent, may also be preventable. This study, similar to other research, suggests that issues of preventability remain a critical concern in decreasing maternal mortality and morbidity.[3,7] Through an increased understanding of preventable events, we can begin to develop the inter-

ventions necessary to improve health care delivery systems and decrease the associated risk for mortality and morbidity. In cases such as hemorrhage and infection, primary prevention may be possible, while for preeclampsia, secondary prevention related to the care and management of these women is paramount.

The results of this study suggest that the farther along the continuum—the more severe the morbidity—the less opportunity there is for clinical control or effective intervention. Once a woman reaches the near-miss level of morbidity, whether she lives or dies becomes difficult to predict. This study highlights the importance of focusing attention on keeping women from progressing along the continuum of morbidity.

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