Trends in Maternal Morbidity Before and During Pregnancy in California

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Objectives. We examined trends in maternal comorbidities in California.

Methods. We conducted a retrospective cohort study of 1 551 017 California births using state-linked vital statistics and hospital discharge cohort data for 1999, 2002, and 2005. We used *International Classification of Diseases, Ninth Revision, Clinical Modification* codes to identify the following conditions, some of which were preexisting: maternal hypertension, diabetes, asthma, thyroid disorders, obesity, mental health conditions, substance abuse, and tobacco use. We estimated prevalence rates with hierarchical logistic regression models, adjusting for demographic shifts, and also examined racial/ethnic disparities.

Results. The prevalence of these comorbidities increased over time for hospital admissions associated with childbirth, suggesting that pregnant women are getting sicker. Racial/ethnic disparities were also significant. In 2005, maternal hypertension affected more than 10% of all births to non-Hispanic Black mothers; maternal diabetes affected nearly 10% of births to Asian/Pacific Islander mothers (10% and 43% increases, respectively, since 1999). Chronic hypertension, diabetes, obesity, mental health conditions, and tobacco use among Native American women showed the largest increases.

Conclusions. The prevalence of maternal comorbidities before and during pregnancy has risen substantially in California and demonstrates racial/ethnic disparity independent of demographic shifts. (*Am J Public Health.* 2014;104: S49–S57. doi:10.2105/AJPH.2013.301583)

Approximately 4 million women give birth each year in the United States, and 1 in 8 of these births is in California.[1] However, after many years of improvement, maternal mortality may be on the rise in both the United States and California.[2,3] Although changes in surveillance may have resulted in better ascertainment of maternal deaths, no discernible reduction in US maternal mortality has been observed in 3 decades.[4]

Increases in maternal mortality likely reflect worsening trends in underlying maternal health. Studies of maternal morbidity associated with labor and delivery have varied widely in methodology and have been especially lacking in consistency regarding the composite measures used to aggregate disparate conditions.[5–9] Depending on the definitions used and the country studied, estimates of "any" maternal morbidity range from 0.6%[10] to 28.6%.[11] Many of the more common conditions included in some of the composite measures, such as lacerations and infections, have been shown to decrease over time,[7,12] and more serious conditions, that is, conditions classified as severe maternal morbidity, have increased.[6,7,13]

Conceptually, severe maternal morbidity exists along a health continuum that extends from normal pregnancy and delivery to organ failure and maternal death and is estimated to affect 52 000 women annually in the United States.[5] Severe maternal morbidity can have lifelong sequelae for both mother and child, with high societal costs. According to Kuklina et al.,[6] conditions classified as severe maternal morbidity, for example, renal failure, pulmonary embolism, adult respiratory distress syndrome, shock, blood transfusion, and ventilation, are all on the rise nationally. In spite of varying reports of overall rates of maternal morbidity, multiple investigations in the United States have confirmed an increase in severe maternal morbidity, showing substantial racial disparity with elevations among non-Hispanic Black women and Hispanic women.[14,15]

Reasons behind these increases remain speculative but include the observation that, for childbirth, women are presenting with an increasing number of comorbidities, both pregnancy related, such as preeclampsia and gestational diabetes mellitus (GDM), and preexisting, such as chronic hypertension, pregestational diabetes, and heart disease.[5,12,13,16–18] The percentage of births to women aged 35 years and older increased from 10.4% in 1993 to 14.5% in 2010,[19,20] and rates of obesity have also risen.[21] According to Wen et al.,[13] the presence of 1 or more preexisting comorbidity appeared related to a 6-fold increase in the risk of severe obstetrical complications. If priorities are to be established and effective interventions designed to improve maternal health, the burden of morbidity among women giving birth must be identified and monitored.

We were commissioned by the California Department of Public Health's Maternal, Child, and Adolescent Health Program to study the trends in maternal morbidity in California. In this article, we report our findings on 6 preexisting or pregnancy-related medical comorbidities: hypertension, diabetes, asthma, thyroid disorders, obesity, and mental health conditions. We also report on 2 risk behaviors: substance abuse (e.g., dependence on opioids or cocaine) and tobacco use. We chose these conditions because of their known association with increased risk of maternal and neonatal pregnancy complications.[7,22–24] Moreover, they can be derived from administrative data and therefore are amenable to tracking and public reporting.

METHODS

We used California Office of Statewide Health Planning and Development linked vital

statistics and hospital discharge cohort data for 1999, 2002, and 2005 for this analysis, which included a total of 1 551 295 deliveries during these 3 nonconsecutive years. The database, which links birth certificates to maternal and infant hospital discharge records, has been shown to match more than 97% of all California deliveries.[25] These records include data on patient characteristics, medical diagnoses, and procedures; the latter are coded according to the *International Classification of Diseases, Ninth Revision, Clinical Modification* (*ICD-9-CM*).[26] Data for 2005 were the latest available at the time of the project's initiation; data are routinely released after several years of delay necessary for data collection and compilation. We selected 3 time points with a 3-year gap between consecutive points to allow for robust trend estimation. To better ensure comparability in hospital service capacity, we excluded 278 deliveries from 26 hospitals with fewer than 50 annual deliveries. After these exclusions, 1 551 017 deliveries in 310 hospitals remained.

We included in the analyses only birth discharge records with diagnosis-related groups related to childbirth hospitalizations (diagnosis-related groups 370, 371, 372, 373, 374, and 375, which include cesarean and vaginal deliveries with or without complications and cesarean and vaginal deliveries with or without associated procedures). For multiple gestations, we kept for analysis only 1 of the multiple-gestation maternal–neonatal record combinations because we used only maternal data, which are identical for all multiple-gestation records for a delivery. We assumed that if *ICD-9-CM* codes for a condition or a procedure were lacking, then the condition or procedure was not present. We did not distinguish between primary and secondary codes; instead, we scanned all codes to determine the presence or absence of a specific code. Details on the codes used to define conditions are provided in Table 1. When possible, we used results from a systematic review of validation studies for perinatal administrative data to report the estimated sensitivity and specificity of the *ICD-9-CM* codes designating the specific conditions of interest.[27] We used the following definitions for covariates: previous cesarean delivery was defined as *ICD-9-CM* code 654.2, and current cesarean delivery was defined as diagnosis-related group codes 370 and 371.

TABLE 1—Definition of Maternal Morbidities and Risk Behaviors Using *ICD-9-CM* Codes: California; 1999, 2002, and 2005

Morbidity or Risk Behavior	*ICD-9-CM* Discharge Codes	Sensitivity, %	Specificity, %
Hypertension	401–405, 642		
Chronic hypertension	401–405, 642.0, 642.1, 642.2	44–86	97–100
Pregnancy-related hypertension	642.3, 642.4, 642.5, 642.6, 642.7	49–88	100
Diabetes	250, 648.0, 648.8		
Pregestational diabetes	250, 648.0	75–100	99–100
Gestational diabetes	648.8	69–96	100
Asthma	493	12–42	98
Thyroid disorders[a]	240–246, 648.1	10–97	100
Obesity[b]	278	11	
Mental health conditions[c]	290–302, 317–319, 648.4, V11		
Substance abuse[d]	304, 305.2–305.9, 648.3, 655.5		
Tobacco use[e]	305.1, V15.82	15–66	99

Note. *ICD-9-CM* = *International Classification of Diseases, Ninth Revision, Clinical Modification.*[26]
[a]Includes hyperthyroid disorder (244.9) and hypothyroid disorder (240.9).
[b]Obesity was defined as a body mass index (weight in kilograms divided by the square of height in meters) of ≥ 30.
[c]Mental health conditions include dementias, alcohol-induced mental disorders, drug-induced mental disorders, transient and persistent mental disorders resulting from conditions classified elsewhere, schizophrenic disorders, episodic mood disorders, delusional disorders, psychoses, pervasive developmental disorders, anxiety, dissociative and somatoform disorders, personality disorders, sexual and gender identity disorders, mild intellectual disabilities, other specified intellectual disabilities, mental disorders complicating pregnancy and childbirth or the puerperium, and personal history of mental disorder.
[d]Substance abuse includes drug abuse; nondependent cannabis abuse; nondependent hallucinogen abuse; nondependent sedative, hypnotic, or anxiolytic abuse; nondependent opioid abuse; nondependent cocaine abuse; nondependent amphetamine or related acting symphathomimetic abuse; nondependent antidepressant type abuse; nondependent other mixed or unspecified drug abuse; drug dependence complicating pregnancy and childbirth or the puerperium; and suspected damage to fetus from drugs affecting management of mother.
[e]Tobacco use includes tobacco use disorder and personal history of tobacco use.

We conducted both unadjusted and adjusted analyses of trends in maternal comorbidities and risk behaviors. Variables used for adjusted analyses included maternal age, parity, education, race/ethnicity, insurance status, prenatal care adequacy, and route of delivery. We categorized race/ethnicity on the basis of the levels reported to the Office of Statewide Health Planning and Development: Asian/Pacific Islander (API), Native American, Hispanic, non-Hispanic Black, non-Hispanic White, and unknown. Maternal education was categorized as elementary, secondary, college, and graduate. Insurance status was reported as private, Medi-Cal (California's Medicaid program), and other. We characterized route of delivery with 2 indicator variables that capture previous cesarean delivery and current cesarean delivery.

We first tested the significance of the unadjusted trends overall and stratified by race/ethnicity with the Cochran–Armitage linear trend test. Differences over the 3 time points were tested with the regression *t* test for the 2002 and 2005 indicator covariates (relative to 1999, the reference year). Additionally, we examined and tested racial/ethnic differences in the maternal comorbidities over the combined study period by using hierarchical logistic regression models, which were implemented using generalized mixed models (SAS GLIMMIX procedure) with random intercepts for counties.[28] Differences in the prevalence of comorbidities such as preeclampsia, GDM, and obesity across racial/ethnic groups have been well documented.[29–31] California data are well suited to test such differences because of California's diverse population, and the persistence of racial/ethnic disparities after adjustment is of particular interest because education level, insurance status, and prenatal care adequacy are often considered potential confounders.

TABLE 2—Unadjusted Trends in Maternal Morbidities and Risk Behaviors Overall and by Race/Ethnicity: California; 1999, 2002, and 2005

Variable	1999 (n = 503 141), %	2002 (n = 514 681), %	2005 (n = 533 195), %	All[a] (n = 1 551 017), %	% Change[b]	P[c]
Hypertension[d]						
All	5.56	5.81	6.35	5.92	14.2	≤ .001
Native American	5.40	6.14	8.04	6.53	48.9	< .001
API	4.16	4.26	4.64	4.36	11.5	≤ .001
Hispanic	5.18	5.35	5.81	5.46	12.2	≤ .001
White, non-Hispanic	5.94	6.33	6.96	6.40	17.2	≤ .001
Black, non-Hispanic	8.46	9.36	10.40	9.38	22.9	≤ .001
Missing	5.54	5.88	6.76	6.07	22.0	
Chronic hypertension						
All	0.63	0.71	0.93	0.80	47.5	≤ .001
Native American	0.26	0.72	1.19	0.78	355.9	< .001
API	0.56	0.75	0.94	0.79	69.5	≤ .001
Hispanic	0.44	0.48	0.68	0.57	54.5	≤ .001
White, non-Hispanic	0.70	0.84	1.05	0.92	50.0	≤ .001
Black, non-Hispanic	1.52	1.86	2.29	2.03	50.7	≤ .001
Missing	0.71	0.67	0.95	0.82	35.0	
Pregnancy-related hypertension						
All	3.84	4.13	4.33	4.10	12.8	≤ .001
Native American	4.22	4.24	5.58	4.69	32.2	.03
API	2.87	2.92	3.02	2.94	5.2	.149
Hispanic	3.69	3.93	4.11	3.91	11.4	≤ .001
White, non-Hispanic	4.06	4.46	4.72	4.42	16.3	≤ .001
Black, non-Hispanic	5.31	6.06	6.25	5.88	17.7	≤ .001
Missing	3.74	4.07	4.48	4.10	19.8	
Maternal diabetes[e]						
All	4.56	5.77	6.50	5.63	42.5	≤ .001
Native American	5.05	6.55	7.43	6.34	47.1	< .001
API	6.70	8.52	9.55	8.33	42.5	≤ .001
Hispanic	4.97	6.26	7.02	6.12	41.2	≤ .001
White, non-Hispanic	3.66	4.44	4.95	4.34	35.2	≤ .001
Black, non-Hispanic	3.84	5.01	5.25	4.67	36.7	≤ .001
Missing	4.19	5.32	6.44	5.33	53.7	
Pregestational diabetes						
All	0.65	0.82	0.83	0.77	27.7	≤ .001
Native American	0.83	1.13	1.67	1.21	101.9	.009
API	0.61	0.81	0.78	0.74	27.3	.003
Hispanic	0.77	0.93	0.94	0.88	22.5	≤ .001
White, non-Hispanic	0.50	0.64	0.63	0.59	25.8	≤ .001
Black, non-Hispanic	0.82	1.04	1.17	1.00	42.9	≤ .001
Missing	0.54	0.69	0.67	0.64	23.9	

Continued

RESULTS

Unadjusted rates of maternal comorbidities and risk behaviors for the combined population and by race/ethnicity are presented in Table 2 by year of study. For the overall sample, trends increased for all conditions ($P \leq .001$). The prevalence of overall hypertension increased from 5.6% in 1999 to 6.4% in 2005, a 14.2% increase in maternal hypertension over the study period. Between 1999 and 2005, pregestational hypertension increased by 47.5%, and pregnancy-related hypertension increased by 12.8%. The prevalence of maternal diabetes also showed a significant rise, from 4.6% to 6.5%, a 42.5% increase over the study period; pregestational diabetes increased by 27.7%, and GDM increased by 44.2%. Notably, asthma rates increased by 74.5%, from 1.02% in 1999 to 1.78% in 2005. The rate of thyroid disorders similarly increased from 0.81% in 1999 to 1.31% in 2005, a 61.7% increase. The prevalence of obesity increased from 0.84% to 1.30% in 2005, a 54.8% increase, and prevalence of mental health conditions increased by 65.0%.

Examination of the prevalence of risk behaviors demonstrated that substance abuse increased from 1.10% in 1999 to 1.31% in 2005, a 19.1% increase, and tobacco use trended upward from 1.26% to 1.62%, a 28.6% increase. All results were significant at a P value of less than or equal to .001.

Unadjusted Trends by Race/Ethnicity

The race/ethnicity distribution of patients was as follows: Native American, 6787 (0.4%); API, 152 099 (9.8%); non-Hispanic White, 518 113 (33.4%); non-Hispanic Black, 85510 (5.5%); Hispanic, 718 003 (46.3%); and missing, 70 505 (4.6%). All race/ethnicity–specific tests were statistically significant, except for asthma and substance abuse among Native Americans and pregnancy-related hypertension and substance abuse among APIs (Table 2). All trends increased over the study period except for a decreasing trend in substance abuse for non-Hispanic Blacks.

Native Americans had the highest increases in hypertension (overall, 48.9%; chronic hypertension, 356%), diabetes (overall, 47.1%; pregestational diabetes, 102%), thyroid

TABLE 2—*Continued*

Gestational diabetes mellitus						
All	3.96	5.01	5.71	4.91	44.2	≤ .001
Native American	4.40	5.51	5.89	5.26	33.9	.024
API	6.15	7.84	8.84	7.68	43.7	≤ .001
Hispanic	4.27	5.38	6.11	5.29	43.1	≤ .001
White, non-Hispanic	3.18	3.84	4.34	3.78	36.5	≤ .001
Black, non-Hispanic	3.06	4.00	4.12	3.71	34.6	≤ .001
Missing	3.69	4.71	5.79	4.75	56.9	
Asthma						
All	1.02	1.12	1.78	1.31	74.5	≤ .001
Native American	1.70	1.44	2.46	1.87	44.7	.057
API	0.68	0.75	1.21	0.89	78.5	≤ .001
Hispanic	0.59	0.67	1.13	0.81	92.2	≤ .001
White, non-Hispanic	1.42	1.60	2.49	1.83	75.4	≤ .001
Black, non-Hispanic	2.43	2.72	4.46	3.18	83.5	≤ .001
Missing	0.96	1.06	1.72	1.26	78.6	
Thyroid disorders						
All	0.81	1.02	1.31	1.05	61.7	≤ .001
Native American	1.04	1.22	1.93	1.40	85.6	.011
API	0.87	1.19	1.60	1.24	83.3	≤ .001
Hispanic	0.44	0.57	0.80	0.61	80.2	≤ .001
White, non-Hispanic	1.31	1.62	2.03	1.65	55.0	≤ .001
Black, non-Hispanic	0.54	0.64	0.84	0.67	55.8	≤ .001
Missing	0.89	1.10	1.53	1.18	72.9	
Obesity						
All	0.84	0.96	1.30	1.04	54.8	≤ .001
Native American	1.18	1.94	2.42	1.84	105.1	.002
API	0.26	0.29	0.44	0.33	68.3	≤ .001
Hispanic	0.68	0.82	1.22	0.92	79.9	≤ .001
White, non-Hispanic	1.06	1.17	1.47	1.23	38.7	≤ .001
Black, non-Hispanic	1.73	2.14	2.90	2.24	67.6	≤ .001
Missing	0.76	0.80	1.00	0.85	31.7	
Mental health conditions						
All	1.77	2.12	2.92	2.28	65.0	≤ .001
Native American	3.09	4.06	8.00	5.05	158.9	≤ .001
API	0.68	0.83	1.05	0.86	54.4	≤ .001
Hispanic	0.81	1.08	1.60	1.18	97.3	≤ .001
White, non-Hispanic	2.81	3.48	4.90	3.72	74.4	≤ .001
Black, non-Hispanic	4.51	4.85	6.49	5.25	43.9	≤ .001
Missing	1.71	2.04	2.67	2.15	56.1	
Substance abuse						
All	1.10	1.04	1.31	1.15	19.1	≤ .001
Native American	2.79	2.48	3.78	3.02	35.5	.05
API	0.29	0.25	0.30	0.28	1.0	.862
Hispanic	0.62	0.64	0.86	0.71	37.8	≤ .001
White, non-Hispanic	1.37	1.39	1.89	1.55	38.0	≤ .001
Black, non-Hispanic	4.19	3.48	3.86	3.85	−7.9	.031
Missing	1.06	1.08	1.17	1.10	10.4	

Continued

disorders (85.6%), and obesity, mental health conditions, and tobacco use (all > 100%).

Notable trends in other racial/ethnic groups included the following:

1. Hypertension increased most among non-Hispanic Blacks (22.9%) and least among Hispanics (12.2%) and APIs (11.5%).
2. Increases in diabetes were similar across non–Native American racial/ethnic groups, ranging from 35.2% to 42.5%.
3. Asthma increased by more than 75% among all racial/ethnic groups except for Native Americans.
4. Thyroid disorders increased by 83.3% and 80.2% among APIs and Hispanics, respectively, and by 55.8% and 55.0% among non-Hispanic Blacks and Whites, respectively.
5. Obesity increased most among Hispanics (79.9%) and least among non-Hispanic Whites (38.7%).
6. Mental health conditions increased most among Hispanics (97.3%) and least among non-Hispanic Blacks (43.9%).
7. Substance abuse increased by 38.0% and 37.8% among non-Hispanic Whites and Hispanics, respectively, compared with a 7.9% decrease among non-Hispanic Blacks and a 1.0% increase in APIs.
8. Tobacco use increased most among non-Hispanic Blacks (57.7%) and least among non-Hispanic Whites (33.6%) and APIs (33.3%).

Rates for deliveries with missing race/ethnicity were comparable with the overall rates with the exception of tobacco use, for which they decreased slightly.

Racial/Ethnic Prevalence Disparities

When aggregating across all study years, we observed significant racial/ethnic disparities (Table 2). Non-Hispanic Black women had the highest prevalence of hypertension, asthma, obesity, mental health conditions, and substance abuse. API women had the highest prevalence of GDM but the lowest prevalence of hypertension, obesity, mental health conditions, and substance abuse.

Non-Hispanic White women had the highest prevalence of thyroid disorders and the lowest prevalence of diabetes. Hispanic women had

TABLE 2—*Continued*

Tobacco use						
All	1.26	1.27	1.62	1.39	28.6	≤ .001
Native American	1.78	2.03	4.62	2.81	159.6	≤ .001
API	0.50	0.53	0.67	0.57	33.3	< .001
Hispanic	0.39	0.41	0.58	0.47	51.4	≤ .001
White, non-Hispanic	2.41	2.53	3.22	2.71	33.6	≤ .001
Black, non-Hispanic	2.15	2.32	3.39	2.60	57.7	≤ .001
Missing	1.25	1.08	1.22	1.18	–2.4	

Note. API = Asian/Pacific Islander; *ICD-9-CM = International Classification of Diseases, Ninth Revision, Clinical Modification*.[26] The race/ethnicity distribution of patients was as follows (n [%]): Native American, 6787 (0.44%); Asian/Pacific Islander, 152 099 (9.81%); White, non-Hispanic, 518 113 (33.40%); Black, non-Hispanic, 83 310 (5.51%); Hispanic, 718 003 (46.29%); missing, 70 505 (4.55%). The sample size was n = 1 551 017.
[a]For all conditions, χ^2 test *P* values using all years of data were < .001 for comparisons of race/ethnicity prevalence.
[b]Percentage of change between 2005 and 1999.
[c]Two-sided Cochran-Armitage linear trend test *P* values.
[d]Chronic and pregnancy-related hypertension coding are not mutually exclusive; The *ICD-9-CM* code 642.9 was not assigned to a category because it is used for unspecified hypertension.
[e]Chronic and gestational diabetes coding are not mutually exclusive; therefore, the total percentage in any given level can be lower than the sum of the chronic and gestational percentages.

the lowest prevalence of thyroid disorders. API and Hispanic women had the lowest prevalence of asthma and tobacco use.

Adjusted Analyses

As with the crude rates, adjusted analyses demonstrated statistically significant increases in the rates of all 12 maternal conditions from 1999 to 2005 ($P < .01$; Table 3). Adjusted rates were minimally different (within 4% change) from the crude rates.

Table 4 presents adjusted odds ratios for maternal morbidities and risk behaviors by race/ethnicity relative to non-Hispanic White women. Odds ratios in Table 4 that are smaller than 1 indicate decreased odds of the outcome relative to the reference group, and we use their reciprocal in the following paragraph for better interpretability. Non-Hispanic Black women had significantly increased adjusted odds of the following conditions relative to non-Hispanic Whites: hypertension (adjusted odds ratio [AOR] = 1.60), asthma (AOR = 1.39), obesity (AOR = 1.58), mental health conditions (AOR = 1.23), and substance abuse (AOR = 1.28). API women had significantly decreased adjusted odds of hypertension (AOR = 1.54), asthma (AOR = 2.17), obesity (AOR = 4.26), mental health conditions (AOR = 4.35), substance abuse (AOR = 5.56), and tobacco use (AOR = 4.24). Hispanics had significantly decreased adjusted odds of mental health conditions (AOR = 3.13), substance abuse (AOR = 3.45), and tobacco use (AOR = 6.10), and non-Hispanic Blacks had decreased adjusted odds of thyroid disorder (AOR = 1.81). Native Americans had significantly higher adjusted odds of obesity (AOR = 1.46) than non-Hispanic Whites.

TABLE 3—Adjusted Rates in Maternal Morbidities and Risk Behaviors: California; 1999, 2002, and 2005

Variable	1999, %	2002, %	2005, %	% Change[a]	*P*[b]
Hypertension	5.56	5.81	6.35	14.2	≤ .001
Chronic	0.63	0.71	0.93	47.5	≤ .001
Pregnancy-related	4.36	4.70	4.91	12.7	.007
Diabetes	4.55	5.80	6.50	42.9	≤ .001
Pregestational	0.65	0.81	0.83	27.7	≤ .001
Gestational	3.96	5.01	5.71	44.2	≤ .001
Asthma	1.02	1.12	1.78	75.5	≤ .001
Thyroid disorder[c]	0.82	1.02	1.32	61.6	≤ .001
Obesity	0.85	0.98	1.33	56.9	≤ .001
Mental health conditions	1.72	2.06	2.85	66.1	≤ .001
Substance abuse	1.04	0.96	1.24	18.9	≤ .001
Tobacco use	1.23	1.27	1.62	31.2	≤ .001

Note. Rates adjusted for maternal age, parity, education, race/ethnicity, insurance status, prenatal care adequacy, and route of delivery.
[a]Percentage of change calculated between 2005 and 1999.
[b]Two-sided *t*-test *P* values for difference in estimated slopes (2005 vs 1999) in hierarchical logistic regression model.

DISCUSSION

We found increasing rates of hypertension, diabetes, asthma, thyroid disorders, obesity, mental health conditions, substance abuse, and tobacco use among women giving birth in California hospitals between 1999 and 2005. These trends were independent of demographic shifts in maternal age, race/ethnicity, education, and other maternal characteristics in California during the same time period. Our findings are consistent with previous reports of the increasing prevalence of maternal comorbidities in the United States.[6,7]

Our analyses not only confirm but also improve on previous studies,[6,7] adjusting for a number of maternal demographic characteristics. Some researchers have attributed this rise in maternal comorbidity to the changing demographics of childbearing.[7] However, even after controlling for demographic shifts, we continued to find significant increases in the prevalence of these maternal conditions, suggesting that not only are more women in California becoming pregnant at an advanced maternal age, but also, irrespective of age, they have an increased prevalence of comorbidity during pregnancy.

Pregnancy-related hypertension increased by 12.7%. Using data from the National Hospital Discharge Survey for 1987 to 2004, Wallis et al.[17] reported that age-adjusted rates

TABLE 4—Racial/Ethnic Disparities Relative to White, Non-Hispanic Women in Maternal Morbidities and Risk Behaviors: California; 1999, 2002, and 2005

Morbidity and Risk Behavior and Race/Ethnicity	AOR (95% CI)
Hypertension	
Native American	1.130 (1.021, 1.251)
API	0.645 (0.627, 0.664)
Hispanic	1.001 (0.982, 1.020)
Black, non-Hispanic	1.596 (1.551, 1.643)
Chronic hypertension	
Native American	1.001 (0.999, 1.003)
API	0.998 (0.997, 0.998)
Hispanic	0.999 (0.999, 1.000)
Black, non-Hispanic	1.013 (1.012, 1.014)
Pregnancy-related hypertension	
Native American	1.005 (1.000, 1.010)
API	0.983 (0.982, 0.984)
Hispanic	1.001 (1.001, 1.002)
Black, non-Hispanic	1.014 (1.013, 1.016)
Diabetes	
Native American	1.029 (1.024, 1.035)
API	1.036 (1.035, 1.038)
Hispanic	1.028 (1.027, 1.029)
Black, non-Hispanic	1.015 (1.013, 1.017)
Pregestational diabetes	
Native American	1.007 (1.005, 1.009)
API	1.001 (1.000, 1.001)
Hispanic	1.003 (1.003, 1.004)
Black, non-Hispanic	1.004 (1.004, 1.005)
Gestational diabetes	
Native American	1.024 (1.018, 1.029)
API	1.036 (1.035, 1.037)
Hispanic	1.024 (1.023, 1.025)
Black, non-Hispanic	1.011 (1.009, 1.012)
Asthma	
Native American	0.934 (0.777, 1.123)
API	0.460 (0.433, 0.489)
Hispanic	0.545 (0.523, 0.567)
Black, non-Hispanic	1.385 (1.317, 1.457)
Thyroid disorders	
Native American	1.242 (1.006, 1.534)
API	0.702 (0.664, 0.742)
Hispanic	0.702 (0.670, 0.735)
Black, non-Hispanic	0.554 (0.505, 0.608)

Continued

of preeclampsia (*ICD-9-CM* codes 642.4 and 642.5) and gestational hypertension (*ICD-9-CM* code 642.3) increased by 25% and 184%, respectively. In 2003–2004, the US age-adjusted rate (per 1000 deliveries) of preeclampsia was 29.4; for gestational hypertension, it was 30.6. Most recently, Berg et al.[7] reported an 11% increase in preeclampsia and eclampsia, from 30 cases per 1000 in 1993–1997 to 34 per 1000 in 2001–2005. Our findings reflect a similar increasing trend in pregnancy-related hypertension in California, although the rate in our study was higher (approximately 49/1000 deliveries in 2005).

Baraban et al.[18] also observed an increase in hypertension associated with childbirth (*ICD-9-CM* code 642) between 1991 and 2003 in Los Angeles County; the age-adjusted prevalence of hypertension increased from 40.5 per 1000 in 1991 to 54.4 per 1000 in 2003. Our findings for all cases of hypertension in childbirth were 63.5 per 1000 in 2005.

Despite differences in the data sources and case definitions used in these studies, they are consistent in their demonstration of an increase in hypertensive disorders in pregnancy. The cause of these increasing trends is not known; plausible contributors include population-level increases in known risk factors for preeclampsia (e.g., prepregnancy overweight and obesity, pregestational hypertension and diabetes), increasing numbers of multiple gestations, and improved coding and case ascertainment.

The increasing prevalence of GDM in California noted here is also consistent with previous reports. A study of women living in Colorado reported that the prevalence of GDM doubled between 1994 and 2002.[32] In a cohort of 267 051 pregnancies screened for GDM in Northern California,[33] the prevalence increased from 5.1% in 1991 to 7.4% in 1997 (a relative increase of 45%). Using National Hospital Discharge Survey data from 5.9 million births in the United States between 1989 and 2004, Getahun et al.[34] found that the prevalence of GDM increased from 1.9% in 1989–1990 to 4.2% in 2003–2004, a relative increase of 122%. Berg et al.[7] also found a 43% increase in GDM, from 2.8% in 1993–1997 to 3.9% in 2001–2005. In our study, the prevalence of GDM increased by 42.5%, from 4.0% of all deliveries in 1999 to 5.0% in 2002 and 5.7% in 2005.

The reason for the increasing rates of GDM is not well understood; we speculate that the recent increases in the prevalence of overweight and obesity among women of childbearing age may have contributed to this temporal increase in GDM. Using survey data from the Pregnancy Risk Assessment Monitoring System in 9 states, Kim et al.[31] found prepregnancy obesity increased 69.3% in a decade, from 13.0% in 1993–1994 to 22.0% in 2002–2003. Other factors, such as demographic shifts in maternal age and race/ethnicity, could also have contributed to the increase in maternal diabetes; however, we adjusted for these demographic factors in our model, and the substantial rise in GDM in California appears to be independent of demographic shifts.

Our analysis demonstrated substantial disparities in maternal morbidity across racial/ethnic groups in California. Notably, API women had the highest rates of diabetes. In 2005, nearly 1 in 10 (9.55%) API women who gave birth had diabetes, a rate that has nearly doubled since 1999. Our finding confirmed 2 recent reports of higher rates of GDM among API women. In a retrospective cohort study of 139 848 women who delivered within a managed care network in California, Caughey et al.[35] found that Asians had the highest rate ($P<.001$) of GDM (6.8%) compared with non-Hispanic Whites (3.4%), non-Hispanic Blacks (3.2%), and Hispanics (4.9%). Using Oregon Pregnancy Risk Assessment Monitoring System data from 3883 women who delivered in Oregon in 2004 and 2005, Hunsberger et al.[36] found that API women had the highest prevalence of GDM (14.8%); this was true for women with both normal and high body mass index (defined as weight in kilograms divided by height in meters squared). The high rate of diabetes among API women who gave birth in California, as well as the high rate of increase, warrants more research and closer public health surveillance. We also found a near 50% increase in maternal hypertension and diabetes among Native Americans. To our knowledge, we are the first to report the rising trends in maternal morbidity among Native Americans, which is even more

TABLE 4—*Continued*

Obesity	
Native American	1.462 (1.210, 1.767)
API	0.235 (0.213, 0.259)
Hispanic	0.845 (0.809, 0.882)
Black, non-Hispanic	1.575 (1.484, 1.672)
Mental health conditions	
Native American	1.041 (0.926, 1.171)
API	0.229 (0.216, 0.243)
Hispanic	0.315 (0.306, 0.325)
Black, non-Hispanic	1.228 (1.182, 1.277)
Substance abuse	
Native American	1.046 (0.895, 1.223)
API	0.182 (0.164, 0.203)
Hispanic	0.291 (0.279, 0.304)
Black, non-Hispanic	1.280 (1.214, 1.348)
Tobacco use	
Native American	0.603 (0.516, 0.706)
API	0.236 (0.219, 0.255)
Hispanic	0.164 (0.157, 0.171)
Black, non-Hispanic	0.695 (0.659, 0.734)

Note. AOR = adjusted odds ratio; API = Asian or Pacific Islander; CI = confidence interval. Rates adjusted for maternal age, parity, education, insurance status, prenatal care adequacy, and route of delivery.

concerning in light of the growing epidemic of overweight and obesity among Native American women of childbearing age.[37]

Strengths and Limitations

The strengths of our study include a large sample size of more than 1.5 million births, differentiation of pregestational and gestational conditions, and adjustment made to account for demographic shifts in California. Our study also has several limitations. First, our findings reflect the lack of sensitivity of administrative data for the coding of maternal comorbidities, tobacco use, and substance abuse. For hospital discharge data, coding standards only require the coding of conditions that affect the current admission, so preexisting conditions and risk factors may not be fully documented.[38] This diminished sensitivity, together with specificities approaching 100%, suggests that our reported rates for many of the conditions studied are likely to be conservative estimates of the true prevalence.

Second, our findings may be limited by potential ascertainment and reporting biases; that is, the observed increases may be the result of improved screening or reporting in later years compared with earlier years rather than real increases in disease prevalence over time. New guidelines for screening and diagnosis of GDM and preeclampsia were issued in 2001[39] and 2002,[40] which may have had an impact on screening and reporting. Likewise, increased emphasis on weight gain during pregnancy and obesity during the study period may have resulted in increased reporting. However, the publication of the Institute of Medicine[41] report on weight gain in pregnancy, the additional requirements for reporting of prepregnancy weight and pregnancy weight gain on California birth certificates in 2007, and recent *ICD-9-CM* codes for documenting overweight and obesity in hospital discharge data took place after the study period and will likely be more relevant to future analyses. Given the low prevalence of obesity noted in the Office of Statewide Health Planning and Development data, the condition was likely underreported. Data from the 2004 Pregnancy Risk Assessment Monitoring System survey of recently pregnant women in the United States document a prepregnancy obesity rate of 21.9% (body mass index ≥ 30).[42] However, asthma, thyroid disease, obesity, mental health conditions, substance abuse, and tobacco use all increased in prevalence without any clear reason for ascertainment or reporting bias. Thus, we believe that the observed rise in maternal hypertension and diabetes in California is unlikely to be entirely attributable to improved screening and reporting over time.

Third, these cross-sectional data do not allow us to make causal inferences regarding increases in these comorbidities and in severe maternal morbidity and mortality. Furthermore, we have an imperfect understanding of how race/ethnicity may be related to both the prevalence of and increasing trend in these comorbidities and risk behaviors, although as we have described, racial disparities have been associated with maternal comorbidities, and maternal comorbidities have in turn been associated with severe maternal morbidity and mortality.

Last, we found substantial racial/ethnic demographic differences when comparing the 2005 maternal race/ethnicity distribution in California with that of the United States as a whole[43] (both excluding missing values): Native American, 0.5% versus 1.1%; API, 10.3% versus 5.6%; non-Hispanic White, 35.0% versus 55.1%; non-Hispanic Black, 5.6% versus 14.1%; and Hispanic, 48.6% versus 24.1%. Therefore, although we hypothesize that the trends and racial/ethnic differences found in this report may be generalizable to the United States, the specific overall rates may not be so.

Conclusions

In this study, we demonstrated that administrative data linked to birth records can be used to monitor the burden of maternal morbidity at the state level. Between 1999 and 2005 in California, we observed increases in asthma, thyroid disorders, obesity, mental health conditions, substance abuse, and tobacco use documented during the childbirth admission and increases in the prevalence of maternal hypertension and diabetes both before and during childbirth. Significant racial/ethnic disparities exist, although maternal morbidity appears to be increasing across all racial/ethnic groups in California. Increased surveillance of these trends is warranted because the rates of maternal morbidity appear to be continuing on an upward trend, which has both a human and an economic toll. These findings support the need to develop and refine case-mix methods using administrative data for monitoring and reporting of childbirth quality indicators. For example, women could be stratified into high- versus low-risk groups on the basis of preexisting or pregnancy-related conditions, and rates of severe morbidity, near-miss morbidity, and mortality could be determined. Such information would support the development of health care facility guidelines or standards regarding the provision of risk-appropriate maternal care,[44,45] as is currently integrated into neonatal intensive care units[46] and trauma centers.[47]

Evolving research regarding fetal programming and adult disease has suggested that maternal health directly affects fetal and child health, which ultimately affect population health. Although a major application of public health practice and policy in the preconception and interconception periods is to focus on prevention of conditions known to contribute to maternal morbidity (obesity, diabetes, and hypertension in particular) through improved

nutrition and physical activity, stress reduction, promotion of safe and healthy neighborhoods, and general efforts to improve other social determinants of health,[48] improved data quality, data collection, database linkages, and ongoing surveillance are also key in that they allow trends to be readily identified and public policy interventions to be realized.[49,50] ■

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This article was accepted July 22, 2013.

Contributors

M. Fridman planned the study and participated in the study design, data analysis, interpretation, and article preparation. L. M. Korst assisted with planning the study, study design, interpretation of the data, and article preparation. J. Chow contributed to interpretation of the data and article editing. E. Lawton assisted with interpretation of the data and article editing. C. Mitchell assisted with the planning of the study, study design, and article editing. K. D. Gregory assisted with planning the study, study design, data interpretation, and article preparation.

Acknowledgments

Financial support for this work was provided by federal Title V block grant funding from the California Department of Public Health, Maternal, Child, and Adolescent Health Division (contract no. 01-15166).

We thank Michael C. Lu for his valuable insight regarding an early version of this article and Samia El Haj Ibrahim for her assistance with drafts of the article.

Human Participant Protection

Institutional review board approval was obtained through Cedars-Sinai Medical Center, David Geffen School of Medicine at UCLA, and California committee for the protection of human subjects.

References

1. Martin JA, Hamilton BE, Ventura SJ, Osterman MJK, Wilson EC, Mathews TJ. Births: final data for 2010. *Natl Vital Stat Rep.* 2012;61(1):1–100.

2. Xu J, Kochanek KD, Murphy SL, Tejada-Vera B. Deaths: Final data for 2007. *Natl Vital Stat Rep.* 2010; 58(19):1–136.

3. California Department of Public Health, Center for Family Health, Maternal, Child and Adolescent Health Division. Maternal mortality rates. 2012. Available at: http://www.cdph.ca.gov/data/statistics/Documents/MO-CAPAMR-MaternalMortalityRates-1999-2010.pdf. Accessed July 8, 2013.

4. Hoyert DL. Maternal mortality and related concepts. *Vital Health Stat 3.* 2007;3(33):1–13.

5. Callaghan WM, Creanga AA, Kuklina EV. Severe maternal morbidity among delivery and postpartum hospitalizations in the United States. *Obstet Gynecol.* 2012;120(5):1029–1036.

6. Kuklina EV, Meikle SF, Jamieson DJ, et al. Severe obstetric morbidity in the United States: 1998-2005. *Obstet Gynecol.* 2009;113 (2 part 1): 293–299.

7. Berg CJ, MacKay AP, Cheng Q, Callaghan WM. Overview of maternal morbidity during hospitalization for labor and delivery in the United States: 1993-1997 and 2001-2005. *Obstet Gynecol.* 2009;113(5):1075–1081.

8. Mann S, Pratt S, Gluck P, et al. Assessing quality in obstetrical care: development of standardized measures. *Jt Comm J Qual Patient Saf.* 2006;32(9):497–505.

9. Geller SE, Rosenberg D, Cox SM, Kilpatrick S. Defining a conceptual framework for near-miss maternal morbidity. *J Am Med Womens Assoc.* 2002;57(3):135–139.

10. Callaghan WM, MacKay AP, Berg CJ. Identification of severe maternal morbidity during delivery hospitalizations, United States, 1991-2003. *Am J Obstet Gynecol.* 2008;199(2):133.e1–8.

11. Brace V, Penney G, Hall M. Quantifying severe maternal morbidity: a Scottish population study. *BJOG.* 2004;111(5):481–484.

12. Srinivas SK, Epstein AJ, Nicholson S, Herrin J, Asch DA. Improvements in US maternal obstetrical outcomes from 1992-2006. *Med Care.* 2010;48(5): 487–493.

13. Wen SW, Huang L, Liston R, et al. Maternal Health Study Group, Canadian Perinatal Surveillance System. Severe maternal morbidity in Canada, 1991-2001. *CMAJ.* 2005;173(7):759–764.

14. Goffman D, Madden RC, Harrison EA, Merkatz IR, Chazotte C. Predictors of maternal mortality and near-miss maternal morbidity. *J Perinatol.* 2007;27(10): 597–601.

15. Berg CJ, Harper MA, Atkinson SM, et al. Preventability of pregnancy-related deaths: results of a state-wide review. *Obstet Gynecol.* 2005;106(6):1228–1234.

16. Getahun D, Nath C, Ananth CV, Chavez MR, Smulian JC. Gestational diabetes in the United States: temporal trends 1989 through 2004. *Am J Obstet Gynecol.* 2008;198(5):525.e1–5.

17. Wallis AB, Saftlas AF, Hsia J, Atrash HK. Secular trends in the rates of preeclampsia, eclampsia, and gestational hypertension, United States, 1987-2004. *Am J Hypertens.* 2008;21(5):521–526.

18. Baraban E, McCoy L, Simon P. Increasing prevalence of gestational diabetes and gestational hypertension in Los Angeles County, California, 1991-2003. *Prev Chronic Dis.* 2008;5(3):A77. Available at: http://www.cdc.gov/pcd/issues/2008/jul/07_0138.htm. Accessed July 8, 2013.

19. National Center for Health Statistics. *Vital Statistics of the United States 1993. Vol 1: Natality.* Hyattsville, MD: National Center for Health Statistics; 1999.

20. Martin JA, Hamilton BE, Sutton PD, et al. Births: final data for 2005. *Natl Vital Stat Rep.* 2007;56(6): 1–103.

21. Chu SY, Bachman DJ, Callaghan WM, et al. Association between obesity during pregnancy and increased use of health care. *N Engl J Med.* 2008;358(14):1444–1453.

22. Pan IJ, Yi HY. Prevalence of hospitalized live births affected by alcohol and drugs and parturient women diagnosed with substance abuse at liveborn delivery: United States, 1999-2008. *Matern Child Health J.* 2013;17(4):667–676.

23. Bansil P, Kuklina E, Jamieson D, et al. Maternal and fetal outcomes among women with depression. *J Womens Health (Larchmt).* 2010;19(2):329–334.

24. Grote NK, Bridge JA, Gavin AR, Melville JL, Iyengar S, Katon WJ. A meta-analysis of depression during pregnancy and the risk of preterm birth, low birth weight, and intrauterine growth restriction. *Arch Gen Psychiatry.* 2010;67(10):1012–1024.

25. Gilbert WM, Nesbitt TS, Danielsen B. The cost of prematurity: quantification by gestational age and birthweight. *Obstet Gynecol.* 2003;102(3):488–492.

26. *International Classification of Diseases, Ninth Revision, Clinical Modification.* Hyattsville, MD: National Center for Health Statistics; 1980. DHHS publication PHS 80–1260.

27. Lain SJ, Hadfield RM, Raynes-Greenow CH, et al. Quality of data in perinatal population health databases: a systematic review. *Med Care.* 2012;50(4):e7–e20.

28. Gelman A, Hill J. *Data Analysis Using Regression and Multilevel/Hierarchical Models.* New York, NY: Cambridge University Press; 2009.

29. Mulla ZD, Gonzalez-Sanchez JL, Nuwayhid BS. Descriptive and clinical epidemiology of preeclampsia and eclampsia in Florida. *Ethn Dis.* 2007;17(4):736–741.

30. Harper M, Dugan E, Espeland M, Martinez-Borges A, McQuellon C. Why African-American women are at greater risk for pregnancy-related death. *Ann Epidemiol.* 2007;17(3):180–185.

31. Kim SY, Dietz PM, England L, Morrow B, Callaghan WM. Trends in pre-pregnancy obesity in nine states, 1993-2003. *Obesity (Silver Spring).* 2007;15(4):986–993.

32. Dabelea D, Snell-Bergeon JK, Hartsfield CL, Bischoff KJ, Hamman RF, McDuffie RS. Increasing prevalence of gestational diabetes mellitus (GDM) over time and by birth cohort: Kaiser Permanente of Colorado GDM Screening Program. *Diabetes Care.* 2005;28(3):579–584.

33. Ferrara A, Kahn HS, Quesenberry CP, Riley C, Hedderson HH. An increase in the incidence of gestational diabetes mellitus: Northern California, 1991-2000. *Obstet Gynecol.* 2004;103(3):526–533.

34. Getahun D, Nath C, Ananth CV, Chavez MR, Smulian JC. Gestational diabetes in the United States: temporal trends 1989 through 2004. *Am J Obstet Gynecol.* 2008;198(5):525.e1–5.

35. Caughey AB, Cheng YW, Stotland NE, Washington AE, Escobar GJ. Maternal and paternal race/ethnicity are both associated with gestational diabetes. *Am J Obstet Gynecol.* 2010;202(6):616.e1–5.

36. Hunsberger M, Rosenberg KD, Donatelle RJ. Racial/ethnic disparities in gestational diabetes mellitus: findings from a population-based survey. *Womens Health Issues*. 2010;20(5):323–328.

37. Brennand EA, Dannenbaum D, Willows ND. Pregnancy outcomes of First Nations women in relation to pregravid weight and pregnancy weight gain. *J Obstet Gynaecol Can*. 2005;27(10):936–944.

38. Hart AC, Stegman MS, Ford B, eds. *ICD-9-CM Expert for Hospitals, Vol 1, 2, & 3 2011*. 6th ed. Salt Lake City, UT: Ingenix; 2010.

39. American College of Obstetricians and Gynecologists (ACOG) Committee on Practice Bulletins–Obstetrics. ACOG Practice Bulletin. Clinical management guidelines for obstetrician-gynecologists. Number 30, September 2001. Gestational diabetes. *Obstet Gynecol*. 2001; 98(3):525–538.

40. ACOG Committee on Practice Bulletins–Obstetrics. ACOG practice bulletin. Diagnosis and management of preeclampsia and eclampsia. Number 33, January 2002. *Obstet Gynecol*. 2002;99(1):159–167.

41. Institute of Medicine and National Research Council. *Weight Gain During Pregnancy: Reexamining the Guidelines*. Washington, DC: National Academies Press; 2009.

42. D'Angelo D, Williams L, Morrow B, et al. Preconception and interconception health status of women who recently gave birth to a live-born infant–Pregnancy Risk Assessment Monitoring System (PRAMS), United States, 26 reporting areas, 2004. *MMWR Surveill Summ*. 2007;56(10):1–35.

43. Martin JA, Hamilton B, Sutton P, et al. Births: final data for 2005. *Natl Vital Stat Rep*. 2007;56(6):1–103.

44. March of Dimes. Toward improving the outcome of pregnancy III. Available at: http://www.marchofdimes.com/professionals/medicalresources_tiop.html. Accessed July 8, 2013.

45. Staebler S. Regionalized systems of perinatal care: health policy considerations. *Adv Neonatal Care*. 2011;11(1):37–42.

46. Lorch SA, Myers S, Carr B. The regionalization of pediatric health care. *Pediatrics*. 2010;126(6):1182–1190.

47. Nathens AB, Jurkovich GJ, Rivara FP, Maier RV. Effectiveness of state trauma systems in reducing injury-related mortality: a national evaluation. *J Trauma*. 2000;48(1):25–30, discussion 30–31.

48. Johnson K, Posner SF, Biermann J, et al. Recommendations to improve preconception health and health care–United States. A report of the CDC/ATSDR Preconception Care Work Group and the Select Panel on Preconception Care. *MMWR Recomm Rep*. 2006; 55(RR-6):1–23.

49. ACOG. Making Obstetrics and Maternity Safer (MOMS) Initiative. Available at: http://www.acog.org/About_ACOG/ACOG_Departments/Government_Relations_and_Outreach/MOMs. Accessed July 8, 2013.

50. Angood PB, Armstrong EM, Ashton D, et al; Transforming Maternity Care Symposium Steering Committee. Blueprint for action: steps toward a high-quality, high-value maternity care system. *Womens Health Issues*. 2010;20(1 suppl):S18–S49.