HHS Public Access
Author manuscript
Am J Public Health. Author manuscript; available in PMC 2015 August 25.

Published in final edited form as:
Am J Public Health. 2013 October ; 103(10): e65–e72. doi:10.2105/AJPH.2013.301469.

Fraction of Gestational Diabetes Mellitus Attributable to Overweight and Obesity by Race/Ethnicity, California, 2007–2009

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Abstract

Objectives—We calculated the racial/ethnic-specific percentages of gestational diabetes mellitus (GDM) attributable to overweight and obesity.

Methods—We analyzed 1 228 265 records of women aged 20 years or older with a live, singleton birth in California during 2007 to 2009. Using logistic regression, we estimated the

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Contributors: S. Y. Kim created the study, analyzed the data, led the writing, and supervised all aspects of its implementation. C. Saraiva and H. G. Wilson analyzed the data and synthesized the analysis. M. Curtis, J. Troyan, L. England, and A. J. Sharma synthesized the analysis. All authors helped conceptualize ideas, interpret findings, and review drafts of the article.

Human Participant Protection: Institutional review board approval was obtained from the Committee for the Protection of Human Subjects, California Health and Human Services Agency and the Centers for Disease Control and Prevention.

magnitude of the association between prepregnancy body mass index and GDM and calculated the percentages of GDM attributable to overweight and obesity overall and by race/ethnicity.

Results—The overall estimated GDM prevalence ranged from 5.4% among White women to 11.9% among Asian/Pacific Islander women. The adjusted percentages of GDM deliveries attributable to overweight and obesity were 17.8% among Asians/Pacific Islander, 41.2% among White, 44.2% among Hispanic, 51.2% among Black, and 57.8% among American Indian women. Select Asian subgroups, such as Vietnamese (13.0%), Asian Indian (14.0%), and Filipino (14.2%), had the highest GDM prevalence, but the lowest percentage attributable to obesity.

Conclusions—Elevated prepregnancy body mass index contributed to GDM in all racial/ethnic groups, which suggests that decreasing overweight and obesity among women of reproductive age could reduce GDM, associated delivery complications, and future risk of diabetes in both the mother and offspring.

Gestational diabetes mellitus (GDM) is defined as carbohydrate intolerance leading to hyperglycemia with onset or first recognition during pregnancy. GDM is associated with increased maternal and infant complications, including infant macrosomia, birth trauma, hypoglycemia, and cesarean section.[1–4] Offspring of mothers with GDM are at increased risk for metabolic syndrome and type 2 diabetes in adulthood.[5] GDM prevalence estimates range from 1% to 14% of all pregnancies in the United States, depending on the population studied and the diagnostic tests employed.[6] Although some women diagnosed with GDM have abnormal glycemia that persists, most women will revert to normal carbohydrate metabolism after delivery.[7] Women with a history of GDM are at increased risk of GDM in future pregnancies,[8] and more than 50% will develop type 2 diabetes later in life.[9]

Previous studies have shown that GDM prevalence estimates differ vastly by race/ethnicity. American Indians, Hispanics, and Asians have the highest estimates of GDM,[10,11] and these differences have not been fully explained by prepregnancy body mass index (BMI).[12,13] California is the most populous state in the United States, with an estimated 39.1 million residents in 2010. Its population is diverse, with an estimated 42% White, 37% Hispanic, 12% Asian, 6% Black, 2% multiracial, 0.6% American Indian, and 0.4% Native Hawaiian/ Pacific Islander. Recent trends in the racial/ethnic composition of California's population predicts a continuing decline in the White population and an increase in the Hispanic population through 2020, when Hispanics are projected to become the largest racial/ethnic group in California.[14] In addition, California records more than 500 000 births in a given year, half of which are to Hispanic women. In California, during the next 10 years, births to Black women will likely decrease by almost 4%, and births to Asian, American Indian, and Pacific Islander women will likely increase by 6%, 6%, and 16%, respectively.[8] Hispanic women are expected to have the largest numerical increase in births during the next 10 years.[15]

In a recent study of diabetes during pregnancy in California, it was reported that prevalence of maternal diabetes had risen from 4.6% of births in 1999 to 6.5% in 2005, with preexisting diabetes increasing by 28% and GDM increasing by 44%.[16] The authors suggested that the increase in GDM might be attributable to recent increases in overweight and obesity among women of reproductive age. The purpose of the present study was to estimate the contribution of BMI to GDM risk across different racial/ethnic groups by calculating the race/ethnicity-specific percentages of GDM attributable to pre-pregnancy overweight and obesity among women giving birth in California during 2007 to 2009.

Methods

Annually, maternal and infant inpatient hospital discharge records for deliveries are linked to birth certificate records using probabilistic methods representing approximately 98% of all California births.[17–19] The present analysis includes all records for California resident women aged 20 years or older with a live singleton birth during 2007 to 2009 whose hospital delivery record was linked to the birth certificate. There were a total of 1402 186 live singleton births to California resident adult women (which excluded 149 961 births to women aged < 20 years). We restricted to adults to have a more consistent definition of adult BMI. Institutional review board approval was obtained from the Committee for the Protection of Human Subjects, California Health and Human Services Agency and the Centers for Disease Control and Prevention (CDC). The human subjects' coordinator at the CDC deemed this study to be nonresearch, in accordance with the CDC's Guidelines for Defining Public Health Research and Public Health Non-Research,[20] because the California Department of Public Health analyzed all data and only aggregate results were shared with the CDC.

Maternal Characteristics

Maternal characteristics, such as age at time of delivery, education, race/ethnicity, expected principle source of payment for delivery (Medi-Cal [California's Medicaid] or other), parity, any smoking during pregnancy, nativity (foreign-born vs US-born), height, prepregnancy weight, and diabetes status are recorded on the birth certificate. Maternal race/ethnicity categories on California's birth certificate are White, Black, American Indian or Alaska Native, Asian Indian, Cambodian, Chinese, Filipino, Hmong, Japanese, Korean, Laotian, Thai, Vietnamese, Other Asian, Native Hawaiian, Guamanian, Samoan, Other Pacific Islander, or other. Maternal Hispanic origin is collected separately with the following categories: Mexican, Central/South American, Puerto Rican, Cuban, and other. For our initial analysis, we combined Asian/Pacific Islander subgroups and Hispanic subgroups, and categorized race/ethnicity as non-Hispanic White, non-Hispanic Black, non-Hispanic Asian/ Pacific Islander, non-Hispanic American Indian, and Hispanic and referred to these groups as White, Black, Asian/Pacific Islander, American Indian, and Hispanic. Because of uncertainty regarding the make-up of the “other” racial/ethnic group, we excluded women in this group from all but the descriptive analyses. In addition, we conducted separate ethnicity-specific analyses by grouping Asian/Pacific Islander women as Chinese, Japanese, Korean, Vietnamese, Other Southeast Asian (Cambodian, Laotian, or Thai), Hmong, Asian Indian, Filipino, Pacific Islander (Guamanian, Hawaiian, Samoan, or Other Pacific Islander) and Hispanic women by their origin as Mexican, Central/South American, Puerto Rican, or Cuban.

Prepregnancy BMI, defined as maternal weight in kilograms divided by the square of height in meters, was calculated from height and prepregnancy weight reported on the birth certificate. We excluded extreme values for height and weight: specifically, women with heights of less than 4 feet, 0 inches or equal to or taller than 6 feet, 11 inches and with prepregnancy weights of less than 75 pounds or equal to or more than 400 pounds. For our main analysis, we classified women as underweight (BMI < 18.5 kg/m^2), normal weight (BMI =18.5–24.9 kg/m^2), overweight (BMI = 25.0–29.9 kg/m^2), class I obese (BMI = 30.0–34.9 kg/m^2), class II obese (BMI = 35.0–39.9 kg/m^2), class III obese (BMI ≥ 40.0 kg/m^2), and total overweight/obese (BMI ≥ 25.0 kg/m^2).[21] Because some Asian populations have elevated risk of diabetes at lower BMIs, we examined additional cutoff points for BMI in a subanalysis of Asian/Pacific Islanders, in whom we classified women as underweight (BMI <18.5 kg/m^2), normal weight (BMI =18.5–22.9 kg/m^2), overweight I (BMI = 23.0–24.9 kg/m^2), overweight II (BMI = 25.0–29.9 kg/m^2), and obese (BMI ≥ 30.0 kg/m^2), as recommended by the World Health Organization (WHO).[22]

Diabetes Status

To determine diabetes status during pregnancy, we used information from both the birth certificate and hospital discharge data. The birth certificate includes 16 complications and procedures of pregnancy and concurrent illnesses, 1 of which is diabetes. Diabetes status is indicated on the birth certificate as prepregnancy diabetes (diagnosis before this pregnancy) or gestational diabetes (diagnosis in this pregnancy). For this study, a birth record that indicated both prepregnancy and gestational diabetes in the same pregnancy was classified as prepregnancy diabetes. On the hospital discharge record, a principal diagnosis and up to 24 other diagnoses are reported; these conditions are coded according to the *International Classification of Diseases, Ninth Revision, Clinical Modifications* (*ICD-9-CM*).[23] We defined preexisting diabetes as presence of *ICD-9-CM* codes for pregestational diabetes (250) or diabetes mellitus (648.0), whereas GDM was defined as glucose intolerance complicating pregnancy, childbirth, or postpartum (*ICD-9-CM* code 648.8). Hospital discharge records indicating both preexisting diabetes and GDM were classified as preexisting diabetes.

GDM status was ascertained using data from both the birth certificate and hospital discharge record. These sources demonstrated moderate agreement in classifying diabetes status during pregnancy ($\kappa = 0.51$). GDM cases were deliveries in which either the birth certificate or hospital discharge record indicated gestational diabetes. Pregnancies without diabetes were those in which both sources indicated no diabetes (neither preexisting nor gestational). Deliveries indicating preexisting diabetes were excluded from the analysis because the mothers were not at risk for GDM. After excluding 159 537 records (or 10.3%) with missing race/ethnicity or BMI information, and 14 384 records (or 0.9%) that indicated preexisting diabetes mellitus, our final data set included 1 228 265 live singleton births to California resident women aged 20 years or older during 2007 to 2009 (96 361 GDM cases and 1 131 904 births with no indication of diabetes before or during pregnancy).

Statistical Analysis

We described the distribution of maternal demographic and behavioral characteristics overall and by race/ethnicity. Next, we estimated the prevalence of GDM and its 95% confidence intervals (CIs) by BMI category for each racial/ethnic group. We selected potential confounders to include in the logistic models on the basis of results reported in published literature, and when we included the potential confounder, we changed the crude odds ratio (OR) by 10%. Because we saw evidence of confounding by nativity in some race groups, we included nativity as a covariate in the final adjusted models. Although none of the potential confounders changed the ORs by more than 10%, we included age and parity as covariates because they were found to be independently associated with both BMI and GDM in previous studies.[24,25] To determine whether race/ethnicity modified the association between BMI and GDM, we assessed interaction between BMI and race/ethnicity using a likelihood ratio test; a *P* value of <.05 was considered statistically significant.

Using logistic regression for the entire population and for each racial/ethnic subgroup separately, we computed relative risks (RRs) and their 95% CIs according to methods described by Flanders and Rhodes[26] with normal BMI as the referent group. In addition, we estimated the population attributable fraction (PAF) and the 95% CIs of GDM births associated with overweight and obesity overall and for overweight and obese BMI categories. The overall PAF is exactly equal to the sum of the categorical PAFs; these estimates were based on adjusted logistic regression using methods described by Graubard and Fears.[27] The PAF incorporates both a measure of association between overweight/obesity and GDM and the prevalence of overweight/obesity in the population as a whole. We interpreted each PAF estimate to be the reduction in GDM prevalence that would be expected to occur if all women in the overweight or obese BMI categories had a GDM risk equal to that of women in the normal BMI category.[28] Finally, we calculated RRs and PAFs for specific Asian and Hispanic subgroups and used Asian-specific BMI categories for Asian subgroups following the WHO recommendations.[15]

Results

Maternal demographic characteristics overall and by race/ethnicity are described in Table 1. Only half (49.5%) of the women in our study population entered pregnancy at a normal weight; 26.1% were overweight and 20.5% were obese. Prevalence of overweight and obesity increased during our study period, from 45.6% in 2007 to 47.3% in 2009 ($P< .001$; data not shown). Prevalence of overweight/obesity was higher among American Indian (61.7%), Black (56.5%), and Hispanic (56.3%) women compared with White (38.7%) and Asian/Pacific Islander (22.9%) women ($P< .001$). GDM prevalence estimates that we used to calculate the RR and PAF are shown in Table A (available as a supplement to this article at http://www.ajph.org). The estimated prevalence of GDM using information from both the birth certificate and hospital discharge data was 7.8% overall, increasing from 7.6% in 2007 to 8.3% in 2009 ($P< .001$; data not shown). GDM prevalence was lowest among White women (5.4%) and highest among Asian/Pacific Islander women (11.9%). However, when specific BMI categories were examined, GDM prevalence was lowest in non-Hispanic Black women in all categories ($P< .001$). Overall, GDM prevalence by BMI category was as follows: underweight, 4.0%; normal weight, 5.1%; overweight, 8.8%; class I obese, 12.2%; class II obese, 15.7%; and class III obese, 18.8% (Table A). In addition, we found that 2.0% of women with GDM were underweight, 32.4% were normal weight, 29.2% were overweight, and 36.5% were obese (19.6% class I obese, 10.0% class II obese, and 6.9% class III obese).

A test for interaction between BMI and race/ethnicity was highly significant (*P*< .001). In the model that included all races combined, we found that the adjusted RRs of developing GDM were 0.69 (95% CI = 0.66, 0.72) among underweight women, 1.8 (95% CI = 1.8, 1.9) among overweight women, 2.7 (95% CI = 2.6, 2.7) among class I obese women, 3.7 (95% CI = 3.6, 3.7) among class II obese women, and 4.6 (95% CI = 4.5, 4.7) among class III obese women (Table 2). Although the estimates were different across the race/ethnicity subgroups, the trends across all racial/ethnic subgroups were similar when using the standard cut points for BMI, with the adjusted RR for class III obesity highest among White women (RR = 5.9; 95% CI = 5.6, 6.2) and lowest among Asian/Pacific Islander women (RR = 2.6; 95% CI = 2.3, 2.9). The adjusted fractions of GDM attributable to overweight and obesity were 38.6% overall, 17.8% among Asian/Pacific Islander, 41.2% among White, 44.2% among Hispanic, 51.2% among Black, and 57.8% among American Indian women (Table 2).

Among Asian/Pacific Islander women, GDM prevalence varied by subgroup: Chinese (12.1%), Vietnamese (13.0%), Other Southeast Asian (9.5%), Asian Indian (14.0%), Filipino (14.2%), and Pacific Islander women (10.5%; *P*< .001; Table 3). Using the Asian cutoff point of BMI more than 23 for overweight, the adjusted fraction of GDM attributable to overweight and obesity combined in Asian/Pacific Islander women was 26.3% overall and ranged from 12.5% among Vietnamese women to 48.4% among Pacific Islander women. When we examined just overweight class I, the PAF was highest in Japanese women (9.7%) and lowest in Pacific Islander women (2.5%), and when we examined just obesity, the PAF was highest in Pacific Islander women (35.4%) and lowest in Chinese women (2.8%).

Among Hispanic women, GDM prevalence varied by subgroups: Mexican (8.7%), Central/South American (7.4%), Puerto Rican (6.6%), and Cuban (5.5%) women (*P*< .001). The adjusted fraction of GDM attributable to overweight and obesity among Hispanic women overall was 44.2%, and ranged from 38.5% among Central/South American women to 65.7% among Cuban women (Table 4). When we examined overweight alone, the PAF was highest in Cuban women (21.1%) and lowest in Mexican women (14.0%), and when we examined just obesity class III, the PAF was highest in Puerto Rican women (11.8%) and lowest in Central/South American women (4.1%).

Discussion

We estimated that more than one third of GDM cases in California could be prevented if expectant mothers entered pregnancy with a normal BMI. Our overall PAF estimate was consistent with the results of a similar analysis using data from Florida (2004–2007), which indicated that 41.1% of GDM cases were attributable to overweight and obesity.[13] Because of the large number of Asian and Hispanic women in the population, we were able to examine a greater diversity of Asian and Hispanic subpopulations in this study than in previous studies. Consistent with previous studies, we found that Asian/Pacific Islanders overall had the highest GDM prevalence estimates; however, prepregnancy BMI did not contribute uniformly to GDM risks across Asian/Pacific Islander subgroups. In American Indian and Black women, high prepregnancy BMI contributed to the prevalence of GDM to a greater extent than for other racial/ethnic groups. Obesity prevention strategies to reduce the risk of GDM might be most effective if targeted to appropriate subgroups of women with high PAF estimates, whereas different strategies might be needed for some Asian ethnic subgroups that have high GDM prevalence but low BMI.

Differences in GDM and obesity prevalence by race/ethnicity might reflect genetics differences, cultural factors (even within the same racial/ethnic group), acculturation, psychosocial stressors, time and duration in the United States, as well as differences in health care systems and reporting practices. Furthermore, patterns of body fat distribution differ by race and ethnicity, and BMI is not a perfect measure of body fat or central adiposity, which are stronger predictors of diabetes.[29] For example, Asians are more prone to develop abdominal obesity, and differences in fat distribution are considered a major contributor to observed excessive prevalence of insulin resistance and diabetes.[30] Cultural differences include diet, physical activity, and perceptions of weight. Furthermore, in our study, approximately half of the population was foreign-born; therefore, environmental changes in immigrant women, including diet and physical activity and the amount of time spent in the United States, might play a role in influencing diabetes status and obesity among this population.[10,31,32]

California's diverse Asian/Pacific Islander population allowed us to examine the contribution of BMI to GDM in more Asian ethnic subgroups than in previous studies. In our study, subgroups of Asian women, such as Asian Indian, Filipino, Chinese, and Vietnamese, had GDM prevalence estimates as high as 12% to 14%, which was consistent with the rise in type 2 diabetes among the general Asian population.[33] However, because of the low prevalence of overweight and obesity among Chinese and Vietnamese women, their PAF estimates were much lower than for all Asians/Pacific Islanders combined, whereas subgroups with higher prepregnancy BMI, such as Asian Indian and Filipino women, had much higher PAF estimates. By contrast, Hmong and Pacific Islander women had relatively lower GDM prevalence but much higher prepregnancy BMI; therefore, their PAF estimates were high relative to other Asian subgroups. Asian women might need targeted interventions at lower BMI levels; however, it is not clear what interventions would be appropriate.

Hispanic women accounted for more than half of all births in California, where more than 80% were of Mexican origin and 10% were of Central/South American origin. Hispanic women experienced a high prevalence of GDM, contributing to more than half of all GDM cases in California. Overall, our results suggested that if all Hispanic women entered pregnancy at a normal weight, 44.2% of GDM cases among Hispanics and nearly 1 in 4 cases of GDM in California would be prevented.

Maternal obesity poses severe risks of adverse pregnancy outcomes, including GDM and maternal and infant morbidity and mortality, and has consequences for offspring that extend beyond the postpartum period.[34,35] Although obesity is undoubtedly multifactorial in origin, and will therefore likely require multiple interventions at the individual, group, and community level, preconception health counseling is a strategy aimed at reducing obesity. Recent guidelines emphasize the importance of maintaining a healthy weight throughout life and recommend that health care providers weigh patients regularly, openly discuss BMI status and its implications on reproductive and general health, and encourage women to think about proper nutrition and physical activity well before they get pregnant.[36] During pregnancy, clinicians need to help women focus on appropriate gestational weight gain because excess weight gain can make it harder to lose weight postpartum and puts women at a higher BMI for a subsequent pregnancy.[37] Furthermore, providers should continue to monitor women's health throughout the post-partum period to ensure women return to a healthy weight to improve overall health and outcomes of future pregnancies. In addition, women with a GDM-affected pregnancy need to be routinely screened for abnormal glucose levels to promptly diagnose chronic diabetes and provide ongoing clinical management.[6] The Affordable Care Act will soon expand access to these preventive health care services for all women of reproductive age.[38]

Limitations

Our study had a few limitations. First, height and prepregnancy weight were obtained from birth certificates and might be based on self-report and not on measurements obtained from clinical measurements. Estimates of obesity prevalence based on self-reported height and weight tend to be lower than those based on measured height and weight.[39] Therefore, we might have underestimated the prevalence of prepregnancy overweight and obesity, which could have resulted in an underestimation of the RR and the PAF.

Second, we likely underestimated GDM prevalence because previous studies showed that GDM data from birth certificates and hospital discharge records had low sensitivity. There was likely little bias in GDM screening or diagnosis by race because the American Congress of Obstetricians and Gynecologists recommends universal GDM screening. Third, our data might not be generalizable to women in states other than California. However, California is the most populous US state and has a high level of racial/ethnic diversity, making it a good source of data for studying racial/ethnic variations in the extent to which BMI is associated with GDM risk.

Finally, we decided to restrict our study to adults to have a more consistent definition of adult BMI; therefore, our results did not apply to births to adolescents. Hence, future studies should consider risk stratification based on age and race/ethnicity.

Conclusions

Elevated prepregnancy BMI contributed substantially to GDM risk in most racial/ethnic groups, which suggested that decreasing the prevalence of overweight and obesity among women of reproductive age could considerably reduce the prevalence of GDM and associated delivery complications. However, the contribution of overweight and obesity to GDM risk varied by race/ethnicity and was lowest among Asian women and their subgroups and highest among American Indian and Black women. The risk of GDM among Black, American Indian, Hmong, and Pacific Islander women might have been affected the most by strategies to reduce obesity, whereas different interventions might be needed for other Asian subgroups. Preconception health strategies that promote healthy weight, diet, and exercise could significantly reduce the overall burden of GDM and potentially reverse the upward trend in GDM prevalence in California. To identify effective prevention strategies, a better understanding is needed of the etiology of GDM among select Asian subgroups in which BMI is not a significant contributor to GDM.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Note. The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

References

1. Casey BM, Lucas MJ, Mcintire DD, Leveno KJ. Pregnancy outcomes in women with gestational diabetes compared with the general obstetric population. Obstet Gynecol. 1997; 90(6):869–873. [PubMed: 9397092]
2. Xiong X, Saunders LD, Wang FL, Demianczuk NN. Gestational diabetes mellitus: prevalence, risk factors, maternal and infant outcomes. Int J Gynaecol Obstet. 2001; 75(3):221–228. [PubMed: 11728481]
3. Barahona MJ, Sucunza N, Garcia-Patterson A, et al. Period of gestational diabetes mellitus diagnosis and maternal and fetal morbidity. Acta Obstet Gynecol Scand. 2005; 84(7):622–627. [PubMed: 15954869]
4. Jensen DM, Sorensen B, Feilberg-Jorgensen N, Westergaard JG, Beck-Nielsen H. Maternal and perinatal outcomes in 143 Danish women with gestational diabetes mellitus and 143 controls with a similar risk profile. Diabet Med. 2000; 17(4):281–286. [PubMed: 10821294]
5. Dabelea D, Mayer-Davis EJ, Lamichhane AP, et al. Association of intrauterine exposure to maternal diabetes and obesity with type 2 diabetes in youth: The SEARCH Case-Control Study. Diabetes Care. 2008; 31(7):1422–1426. [PubMed: 18375420]
6. ACOG Practice Bulletin. Clinical management guidelines for obstetrician-gynecologists. Number 30, September 2001 (replaces Technical Bulletin Number 200, December 1994). Gestational diabetes. Obstet Gynecol. 2001; 98(3):525–538. [PubMed: 11547793]
7. Bottalico JN. Recurrent gestational diabetes: risk factors, diagnosis, management, and implications. Semin Perinatol. 2007; 31(3):176–184. [PubMed: 17531899]
8. Getahun D, Fassett MJ, Jacobsen SJ. Gestational diabetes: risk of recurrence in subsequent pregnancies. Am J Obstet Gynecol. 2010; 203(5):467 e1–e6. [PubMed: 20630491]
9. Kim C, Newton KM, Knopp RH. Gestational diabetes and the incidence of type 2 diabetes: a systematic review. Diabetes Care. 2002; 25(10):1862–1868. [PubMed: 12351492]
10. Savitz DA, Janevic TM, Engel SM, Kaufman JS, Herring AH. Ethnicity and gestational diabetes in New York City, 1995-2003. BJOG. 2008; 115(8):969–978. [PubMed: 18651880]
11. Chu SY, Abe K, Hall LR, Kim SY, Njoroge T, Qin C. Gestational diabetes mellitus: all Asians are not alike. Prev Med. 2009; 49(2–3):265–268. [PubMed: 19596364]
12. Shai I, Jiang R, Manson JE, et al. Ethnicity, obesity, and risk of type 2 diabetes in women: a 20-year follow-up study. Diabetes Care. 2006; 29(7):1585–1590. [PubMed: 16801583]
13. Kim SY, England L, Sappenfield W, et al. Racial/ethnic differences in the percentage of gestational diabetes mellitus cases attributable to overweight and obesity, Florida, 2004-2007. Prev Chronic Dis. 2012; 9:E88. [PubMed: 22515970]
14. California Department of Finance. Race/Ethnic Population with Age and Sex Detail, 2000-2050. Sacramento, CA: California Department of Finance; 2007.
15. Johnson, HP. Birth Rates in California. San Francisco, CA: Public Policy Institute of California; 2007.

16. Fridman, M.; Gregory, KD.; Korst, LM.; Lu, MC.; Shah, S. Trends in Maternal Morbidities in California 1999-2005. Los Angeles, CA: California Department of Public Health, Maternal Child and Adolescent Health Division with the Regents of the University of California; 2010.
17. Gilbert WM, Young AL, Danielsen B. Pregnancy outcomes in women with chronic hypertension:a population-based study. J Reprod Med. 2007; 52(11):1046–1051. [PubMed: 18161404]
18. Glass HC, Pham TN, Danielsen B, Towner D, Glidden D, Wu YW. Antenatal and intrapartum risk factors for seizures in term newborns: a population-based study, California 1998-2002. J Pediatr. 2009; 154(1):24–28. [PubMed: 18760807]
19. Herrchen B, Gould JB, Nesbitt TS. Vital statistics linked birth/infant death and hospital discharge record linkage for epidemiological studies. Comput Biomed Res. 1997; 30(4):290–305. [PubMed: 9339323]
20. [Accessed June 18, 2013] Distinguishing Public Health Research and Public Health Nonresearch. 2010. Available at: http://www.cdc.gov/od/science/integrity/docs/cdc-policy-distinguishing-public-health-research-nonresearch.pdf
21. World Health Organization. [Accessed February 2, 2011] BMI classification. Available at: http://apps.who.int/bmi/index.jsp?introPage=intro_3.html
22. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet. 2004; 363(9403):157–163. [PubMed: 14726171]
23. International Classification of Diseases, Ninth Revision, Clinical Modification. Hyattsville, MD: National Center for Health Statistics; 1980. DHHS publication PHS 80-1260
24. Di Cianni G, Volpe L, Lencioni C, et al. Prevalence and risk factors for gestational diabetes assessed by universal screening. Diabetes Res Clin Pract. 2003; 62(2):131–137. [PubMed: 14581150]
25. Raatikainen K, Heiskanen N, Heinonen S. Transition from overweight to obesity worsens pregnancy outcome in a BMI-dependent manner. Obesity (Silver Spring). 2006; 14(1):165–171. [PubMed: 16493135]
26. Flanders WD, Rhodes PH. Large sample confidence intervals for regression standardized risks, risk ratios, and risk differences. J Chronic Dis. 1987; 40(7):697–704. [PubMed: 3597672]
27. Graubard BI, Fears TR. Standard errors for attributable risk for simple and complex sample designs. Biometrics. 2005; 61(3):847–855. [PubMed: 16135037]
28. Levine BJ. The other causality question: estimating attributable fractions for obesity as a cause of mortality. Int J Obes (Lond). 2008; 32(suppl 3):S4–S7. [PubMed: 18695651]
29. Abate N, Chandalia M. The impact of ethnicity on type 2 diabetes. J Diabetes Complications. 2003; 17(1):39–58. [PubMed: 12505756]
30. Khoo CM, Sairazi S, Taslim S, et al. Ethnicity modifies the relationships of insulin resistance, inflammation, and adiponectin with obesity in a multiethnic Asian population. Diabetes Care. 2011; 34(5):1120–1126. [PubMed: 21464462]
31. Rosenmöller DL, Gasevic D, Seidell J, Lear SA. Determinants of changes in dietary patterns among Chinese immigrants: a cross-sectional analysis. Int J Behav Nutr Phys Act. 2011; 8:42. [PubMed: 21592378]
32. Afable-Munsuz A, Ponce NA, Rodriguez M, Perez-Stable EJ. Immigrant generation and physical activity among Mexican, Chinese and Filipino adults in the US. Soc Sci Med. 2010; 70(12):1997–2005. [PubMed: 20378226]
33. Cockram CS. The epidemiology of diabetes mellitus in the Asia-Pacific region. Hong Kong Med J. 2000; 6(1):43–52. [PubMed: 10793402]
34. Report from 2002 to 2003 Maternal Death Reviews. Sacramento, CA: California Department of Public Health, Maternal Child and Adolescent Health Division; 2011. The California Pregnancy-Associated Mortality Review.
35. Chu SY, Callaghan WM, Kim SY, et al. Maternal obesity and risk of gestational diabetes mellitus. Diabetes Care. 2007; 30(8):2070–2076. [PubMed: 17416786]
36. 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. [PubMed: 16617292]

37. Nehring I, Schmoll S, Beyerlein A, Hauner H, von Kries R. Gestational weight gain and long-term post-partum weight retention: a meta-analysis. Am J Clin Nutr. 2011; 94(5):1225–1231. [PubMed: 21918221]

38. US Department of Health and Human Services. [Accessed April 13, 2012] Affordable Care Act. 2012. Available at: http://www.hrsa.gov/womensguidelines

39. Park S, Sappenfield WM, Bish C, Bensyl DM, Goodman D, Menges J. Reliability and validity of birth certificate prepregnancy weight and height among women enrolled in prenatal WIC program: Florida, 2005. Matern Child Health J. 2011; 15(7):851–859. [PubMed: 19937268]

Table 1

Maternal Demographic and Behavioral Characteristics: California, 2007-2009

		Race/Ethnicity					
Maternal Characteristics	**Total, No. (%)**	**Non-Hispanic White, No. (%)**	**Non-Hispanic Black, No. (%)**	**Non-Hispanic Asian/PI, No. (%)**	**Non-Hispanic American Indian, No. (%)**	**Hispanic, No. (%)**	**Non-Hispanic Other, No. (%)**
Total	1 228 265	350 697 (28.6)	60 685 (4.9)	168 933 (13.8)	4134 (0.3)	621187 (50.6)	22 629 (1.8)
Age at delivery, y							
20–34	995 384 (81.0)	266 525 (76.0)	51621 (85.1)	122 871 (72.7)	3628 (87.8)	532184 (85.7)	18 555 (82.0)
≥35	232 881 (19.0)	84 172 (24.0)	9064 (14.9)	46 062 (27.3)	506 (12.2)	89 003 (14.3)	4074 (18.0)
Education							
< 12th grade, no diploma	275 929 (22.9)	15 871 (4.6)	7137 (12.0)	7057 (4.3)	810 (20.1)	243 592 (40.0)	1462 (6.5)
High school graduate/GED	315 511 (26.2)	74 105 (21.5)	20 915 (35.1)	25 636 (15.5)	1535 (38.0)	187 653 (30.8)	5667 (25.4)
Some college credit	614 999 (51.0)	255 516 (74.0)	31614 (53.0)	133 023 (80.3)	1693 (41.9)	177 942 (29.2)	15 211 (68.1)
Expected principle source of payment at delivery							
Medi-Cal (California's Medicaid)	542 128 (44.2)	72 742 (20.8)	31110 (51.4)	34 451 (20.4)	2239 (54.3)	394 497 (63.6)	7089 (31.4)
Other	684 661 (55.8)	277 534 (79.2)	29 435 (48.6)	134 360 (79.6)	1883 (45.7)	225 939 (36.4)	15 510 (68.6)
Parity							
0	436 699 (35.6)	150 759 (43.0)	20 760 (34.2)	77 078 (45.6)	1174 (28.4)	177134 (28.5)	9794 (43.3)
1-2	632 875 (51.5)	171 775 (49.0)	29145 (48.1)	82 367 (48.8)	1989 (48.1)	337 062 (54.3)	10 537 (46.6)
>2	158 404 (12.9)	28 066 (8.0)	10 753 (17.7)	9449 (5.6)	969 (23.5)	106 877 (17.2)	2290 (10.1)
Nativity							
Foreign-born	567 784 (46.2)	45 544 (13.0)	6316 (10.4)	138 665 (82.2)	78 (1.9)	373 909 (60.2)	3272 (14.5)
US-born	660 024 (53.8)	304 996 (87.0)	54 318 (89.6)	30 090 (17.8)	4056 (98.1)	247 220 (39.8)	19 344 (85.5)
Any smoking during pregnancy							
Yes	31 574 (2.6)	18 818 (5.4)	3389 (5.7)	1405 (0.8)	495 (12.1)	5950 (1.0)	1517 (6.7)
No	1190 276 (97.4)	330 850 (94.6)	56 486 (94.3)	167 298 (99.2)	3607 (87.9)	611056 (99.0)	20 979 (93.3)
BMI (kg/m^2)							
Underweight (< 18.5)	48 448 (3.9)	14 265 (4.1)	2260 (3.7)	16 320 (9.7)	107 (2.6)	14 599 (2.4)	897 (4.0)
Normal (18.5-24.9)	608 234 (49.5)	200 630 (57.2)	24148 (39.8)	113 905 (67.4)	1475 (35.7)	256 591 (41.3)	11 485 (50.8)
Overweight (25.0-29.9)	320 346 (26.1)	77 777 (22.2)	16 331 (26.9)	27 607 (16.3)	1088 (26.3)	192 084 (30.9)	5459 (24.1)
Class 1 obesity (30.0-34.9)	154 453 (12.6)	33 861 (9.7)	9292 (15.3)	7985 (4.7)	776 (18.8)	99 853 (16.1)	2686 (11.9)

Maternal Characteristics	Total, No. (%)	Race/Ethnicity					
		Non-Hispanic White, No. (%)	Non-Hispanic Black, No. (%)	Non-Hispanic Asian/PI, No. (%)	Non-Hispanic American Indian, No. (%)	Hispanic, No. (%)	Non-Hispanic Other, No. (%)
Class II obesity (35.0-39.9)	61 636 (5.0)	15 236 (4.3)	4777 (7.9)	2227 (1.3)	395 (9.6)	37 756 (6.1)	1245 (5.5)
Class III obesity (≥ 40)	35 148 (2.9)	8928 (2.5)	3877 (6.4)	889 (0.5)	293 (7.1)	20 304 (3.3)	857 (3.8)
Overweight/obese (≥ 25)	571 583 (46.5)	135 802 (38.7)	34 277 (56.5)	38 708 (22.9)	2552 (61.7)	349 997 (56.3)	10 247 (45.3)
Obese (≥ 30)	251 237 (20.5)	58 025 (16.6)	17.946 (29.6)	11101 (6.6)	1464 (35.4)	157 913 (25.4)	4788 (21.2)
GDM							
Yes	96 361 (7.8)	18 806 (5.4)	3371 (5.6)	20 129 (11.9)	316 (7.6)	52 256 (8.4)	1483 (6.6)
No	1131 904 (92.2)	331 891 (94.6)	57 314 (94.4)	148 804 (88.1)	3818 (92.4)	568 931 (91.6)	21146 (93.4)

Note. BMI = body mass index; GDM = gestational diabetes mellitus; GED = general equivalency diploma; PI = Pacific Islander.

Table 2

Adjusted Relative Risks and Population Attributable Fraction (PAF) of Gestational Diabetes Mellitus (GDM) Associated With Body Mass Index (BMI), by Race/Ethnicity: California, 2007-2009

		Race/Ethnicity				
	Total RR (95% CI)	Non-Hispanic White RR (95% CI)	Non-Hispanic Black RR (95% CI)	Non-Hispanic Asian/PI RR (95% CI)	Non-Hispanic American Indian RR (95% CI)	Hispanic RR (95% CI)
RR BMI (kg/m^2)						
Underweight (< 18.5)	0.69 (0.66, 0.72)	0.8 (0.8, 0.9)	0.8 (0.6, 1.0)	0.6 (0.6, 0.7)	0.3 (0.0, 2.1)	0.6 (0.6, 0.7)
Normal (18.5-24.9; Ref)	1.0	1.0	1.0	1.0	1.0	1.0
Overweight (25.0-29.9)	1.8 (1.8, 1.9)	2.0 (2.0, 2.1)	2.0 (1.8, 2.2)	1.8 (1.7, 1.9)	2.2 (1.6, 3.1)	1.8 (1.8, 1.8)
Class I obesity (30.0-34.9)	2.7 (2.6, 2.7)	3.2 (3.1, 3.4)	2.8 (2.6, 3.2)	2.3 (2.2, 2.4)	3.1 (2.2, 4.3)	2.6 (2.5, 2.7)
Class II obesity (35.0-39.9)	3.7 (3.6, 3.7)	4.5 (4.3, 4.7)	3.9 (3.5, 4.3)	2.6 (2.4, 2.8)	4.2 (2.9, 6.1)	3.6 (3.5, 3.7)
Class III obesity (≥ 40)	4.6 (4.5, 4.7)	5.9 (5.6, 6.2)	5.2 (4.6, 5.8)	2.6 (2.3, 2.9)	5.7 (4.0, 8.2)	4.6 (4.4, 4.7)
PAF BMI (kg/m^2)						
Overweight (25.0-29.9)	13.5 (13.1, 13.8)	13.3 (12.6, 14.1)	13.8 (11.8, 15.7)	10.8 (10.1, 11.5)	13.6 (7.6, 19.5)	14.3 (13.7, 14.8)
Class I obesity (30.0-34.9)	12.4 (12.1, 12.6)	12.3 (11.7, 12.9)	13.9 (12.3, 15.5)	4.9 (4.5, 5.2)	16.6 (11.1, 22.1)	14.9 (14.5, 15.3)
Class II obesity (35.0-39.9)	7.4 (7.2, 7.6)	8.6 (8.2, 9.1)	10.9 (9.7, 12.1)	1.5 (1.3, 1.8)	13.3 (8.9, 17.7)	8.8 (8.6, 9.1)
Class III obesity (≥ 40)	5.4 (5.3, 5.6)	7.0 (6.6, 7.4)	12.6 (11.4, 13.9)	0.6 (0.5, 0.7)	14.4 (10.1, 18.6)	6.2 (5.9, 6.4)
Total overweight/obese (≥ 25.0)	38.6 (38.1, 39.1)	41.2 (40.1, 42.3)	51.2 (48.0, 54.4)	17.8 (17.0, 18.6)	57.8 (47.3, 68.4)	44.2 (43.3, 45.0)

Note. CI = confidence interval; PI = Pacific Islander; RR = relative risk. Risks adjusted for maternal age, parity, and nativity.

Table 3

Gestational Diabetes Mellitus (GDM) Prevalence, Adjusted Relative Risks and Population Attributable Fraction (PAF) of GDM Associated With Body Mass Index (BMI), by Asian Ethnicity: California, 2007-2009

Maternal Characteristics	All Asian/PI % (95% CI)	Chinese % (95% CI)	Japanese % (95% CI)	Korean % (95% CI)	Vietnamese % (95% CI)	Other Southeast Asian % (95% CI)	Hmong % (95% CI)	Asian Indian % (95% CI)	Filipino % (95% CI)	Pacific Islander % (95% CI)
GDM prevalence	11.9 (11.8, 12.1)	12.1 (11.7, 12.4)	6.8 (6.2, 7.5)	6.2 (5.8, 6.6)	13.0 (12.6, 13.5)	9.5 (8.8, 10.1)	8.1 (7.4, 8.9)	14.0 (13.6, 14.5)	14.2 (13.9, 14.6)	10.5 (9.7, 11.3)
RR BMI (kg/m^2)										
Underweight (< 18.5)	0.7 (0.7, 0.8)	0.8 (0.8, 0.9)	0.8 (0.6, 1.2)	0.5 (0.4, 0.7)	0.7 (0.7, 0.8)	0.7 (0.5, 1.0)	1.0 (0.4, 2.2)	0.5 (0.4, 0.7)	0.8 (0.6, 0.9)	0.8 (0.4, 1.8)
Normal (18.5-22.9; Ref)	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0	1.0
Overweight I (23.0-24.9)	1.5 (1.5, 1.6)	1.5 (1.4, 1.7)	2.0 (1.5, 2.5)	1.8 (1.5, 2.1)	1.4 (1.2, 1.5)	1.4 (1.2, 0.17)	1.4 (1.0, 2.0)	1.6 (1.5, 1.7)	1.4 (1.3, 1.6)	1.4 (1.0, 2.1)
Overweight II (25.0-29.9)	2.0 (2.0, 2.1)	2.1 (1.9, 2.2)	2.4 (1.9, 3.1)	2.4 (2.0, 2.9)	1.9 (1.7, 2.1)	1.8 (1.5, 2.1)	2.2 (1.6, 2.9)	1.9 (1.8, 2.1)	2.1 (1.9, 2.2)	1.7 (1.3, 2.4)
Obese (≥ 30)	2.7 (2.6, 2.8)	2.7 (2.4, 3.1)	4.7 (3.6, 6.3)	4.7 (3.8, 5.9)	2.8 (2.4, 3.3)	3.0 (2.5, 3.6)	3.9 (3.0, 5.2)	2.6 (2.3, 2.9)	2.7 (2.5, 2.9)	2.7 (2.0, 3.5)
PAF BMI (kg/m^2)										
Overweight I (23.0-24.9)	6.4 (5.8, 7.0)	6.2 (5.0, 7.5)	9.7 (5.5, 13.9)	8.3 (5.4, 11.3)	3.9 (2.6, 5.2)	5.0 (2.0, 8.0)	3.9 (-0.2, 8.1)	8.9 (7.2, 10.6)	6.0 (4.7, 7.3)	2.5 (-0.2, 5.1)
Overweight II (25.0-29.9)	12.3 (11.6, 13.0)	8.5 (7.4, 9.7)	11.5 (7.5, 15.5)	11.0 (8.2, 13.8)	6.4 (5.1, 7.6)	10.9 (7.4, 14.3)	19.5 (12.8, 26.2)	15.1 (13.2, 17.0)	16.0 (14.6, 17.5)	10.6 (5.3, 15.8)
Obese (≥ 30)	7.6 (7.1, 8.0)	2.8 (2.2, 3.4)	10.1 (7.0, 13.2)	6.9 (5.0, 8.7)	2.3 (1.7, 2.9)	11.0 (8.4, 13.6)	23.0 (17.9, 28.2)	7.0 (6.0, 8.0)	10.0 (9.0, 11.0)	35.4 (27.8, 43.0)
Total overweight/obese (≥23)	26.3 (25.2, 27.4)	17.6 (15.7, 19.5)	31.3 (24.6, 38.0)	26.2 (21.7, 30.8)	12.5 (10.6, 14.5)	26.9 (21.1, 32.7)	46.4 (35.5, 57.4)	31.0 (27.9, 34.1)	32.1 (29.6, 34.5)	48.4 (36.0, 60.8)

Note. CI = confidence interval; PI = Pacific Islander; RR = relative risks. Risks adjusted for maternal age, parity, and nativity.

Table 4

Gestational Diabetes Mellitus (GDM) Prevalence, Adjusted Relative Risks and Population Attributable Fraction (PAF) of GDM Associated With Body Mass Index (BMI), by Hispanic Ethnicity: California, 2007-2009

Maternal Characteristics	All Hispanics % (95% CI)	Mexican % (95% CI)	Central/South American % (95% CI)	Puerto Rican % (95% CI)	Cuban % (95% CI)
GDM prevalence	8.4 (8.3, 8.5)	8.7 (8.6, 8.8)	7.4 (7.2, 7.6)	6.6 (5.9, 7.4)	5.5 (4.5, 6.7)
RR BMI (kg/m^2)					
Underweight (< 18.5)	0.6 (0.6, 0.7)	0.7 (0.6, 0.7)	0.5 (0.3, 0.7)	0.6 (0.1, 2.2)	1.1 (0.1, 8.0)
Normal (18.5-24.9; Ref)	1.00	1.00	1.00	1.00	1.00
Overweight (25.0-29.9)	1.79 (1.75, 1.84)	1.8 (1.7, 1.8)	1.8 (1.7, 1.9)	2.7 (2.0, 3.8)	3.3 (1.8, 6.1)
Class I obesity (30-34.9)	2.6 (2.5, 2.7)	2.6 (2.5, 2.7)	2.4 (2.3, 2.6)	3.7 (2.6, 5.3)	6.1 (3.3, 11.3)
Class II obesity (35-39.9)	3.6 (3.5, 3.7)	3.6 (3.5, 3.7)	3.3 (3.0, 3.6)	6.0 (4.2, 8.7)	9.0 (4.5, 17.9)
Class III obesity (≥ 40)	4.6 (4.4, 4.7)	4.5 (4.4, 4.7)	4.3 (3.8, 4.9)	7.4 (5.0, 10.9)	9.8 (4.6, 20.9)
PAF BMI (kg/m^2)					
Overweight (25.0-29.9)	14.3 (13.7, 14.8)	14.0 (13.5, 14.6)	15.3 (13.4, 17.1)	18.8 (12.4, 25.2)	21.1 (10.1, 32.1)
Class I obesity (30.0-34.9)	14.9 (14.5, 15.3)	15.1 (14.6, 15.5)	12.7 (11.4, 14.0)	15.6 (10.5, 20.7)	22.5 (13.0, 31.9)
Class II obesity (35.0-39.9)	8.8 (8.6, 9.1)	8.9 (8.6, 9.1)	6.4 (5.5, 7.2)	13.9 (9.6, 18.2)	12.5 (5.4, 19.6)
Class III obesity (≥ 40)	6.2 (5.9, 6.4)	6.2 (5.9, 6.4)	4.1 (3.5, 4.7)	11.8 (7.9, 15.7)	9.6 (3.4, 15.9)
Total overweight/obese (≥ 25.0)	44.2 (43.3, 45.0)	44.1 (43.3, 45.0)	38.5 (35.8, 41.1)	60.1 (50.7, 69.6)	65.7 (51.1, 80.3)

Note. CI = confidence interval; RR = relative risks. Risks adjusted for maternal age, parity, and nativity.