# Racial-ethnic Disparities in Postpartum Hemorrhage in Native Hawaiians, Pacific Islanders, and Asians

Scott A. Harvey MD, MS; Eunjung Lim PhD; Krupa R. Gandhi MPH; Jill Miyamura PhD; and Kazuma Nakagawa MD

## Abstract

*The objective of this study was to assess racial-ethnic differences in the prevalence of postpartum hemorrhage (PPH) among Native Hawaiians and other Pacific Islanders (NHOPI), Asians, and Whites. We performed a retrospective study on statewide inpatient data for delivery hospitalizations in Hawai'i between January 1995 and December 2013. A total of 243,693 in-hospital delivery discharges (35.0% NHOPI, 44.0% Asian, and 21.0% White) were studied. Among patients with PPH, there were more NHOPI (37.1%) and Asians (47.6%), compared to Whites (15.3%). Multivariable logistic regression was used to assess the impact of maternal race-ethnicity on the prevalence of PPH after adjusting for delivery type, labor induction, prolonged labor, multiple gestation, gestational hypertension, gestational diabetes, preeclampsia, chorioamnionitis, placental abruption, placenta previa, obesity, and period with different diagnostic criteria for preeclampsia. In the multivariable analyses, NHOPI (adjusted odds ratio [aOR], 1.40; 95% confidence interval [CI], 1.32-1.48) and Asians (aOR, 1.45; 95% CI, 1.37-1.53) were more likely to have PPH compared to Whites. In the secondary analyses of 12,142 discharges with PPH, NHOPI and Asians had higher prevalence of uterine atony than Whites (NHOPI: 77.2%, Asians: 73.9% vs Whites: 65.1%, P < .001 for both comparisons).*

## Keywords

*Postpartum hemorrhage, Native Hawaiian and Pacific Islanders*

## Introduction

Postpartum hemorrhage (PPH) is one of the leading causes of maternal mortality and morbidity with 140,000 annual deaths estimated worldwide.[1,2] PPH is defined as an estimated blood loss of ≥500 mL after a vaginal delivery or ≥1000 mL after a cesarean delivery, or by a postpartum hematocrit reduction of more than 10%.[1,3] Common causes of PPH include uterine atony, genital tract trauma, retained products of conception, coagulation disorders, uterine inversion, and implantation of placenta into the lower uterine segment.[4] Its incidence is likely underestimated since the clinician must rely on visual estimation of blood loss to make the diagnosis. Furthermore, PPH could occur either immediately or up to 6-12 weeks postpartum,[1,5] which makes public reporting difficult.

Racial-ethnic disparities are persistent problems in women's health and obstetric outcomes in the United States and are largely associated with disparate socioeconomic and insurance status.[6] Several studies have shown significant racial-ethnic disparities in pregnancy-related morbidity and mortality.[7-10] Predominantly studied populations are Hispanics, African Americans, and Asians/Pacific Islanders who have significantly higher risk of PPH than non-Hispanic Whites.[9,11-15] Although Native Hawaiians and other Pacific Islanders (NHOPI) have been reported to have higher rates of severe PPH,[16] this finding was limited to California data.[11] To date, there are no studies assessing the racial-ethnic differences in PPH among NHOPI and Asians in Hawai'i. Furthermore, NHOPI have been historically aggregated with Asians into a single racial-ethnic category in the prior studies, which masks differences that may, at times, be substantial. Therefore, we sought to assess racial-ethnic disparities in the prevalence of PPH among a population in Hawai'i that predominantly consists of NHOPI, Asians, and Whites.

## Methods

### Data Source

We conducted a retrospective study using Hawai'i Health Information Corporation (HHIC) inpatient data from January 1995 to December 2013. HHIC maintains the largest healthcare database in Hawaii including inpatient administrative, emergency department, and financial data. The HHIC inpatient database has detailed, all visit, discharge data from every non-federal hospital in Hawai'i by all payers, and includes race-ethnicity, sex, age, insurance type, and *International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9)* primary and secondary diagnoses and procedure codes.[17] This study was approved by the Institutional Review Board of the University of Hawai'i at Manoa.

### Patients

We received a de-identified dataset from HHIC that included 286,556 discharges (age >16) who had the primary diagnosis of vaginal or cesarean delivery. As this study is specifically evaluating the ethnic population of Hawai'i, non-Hawai'i residents (n=1,571 discharges), and records lacking race-ethnicity data or with race-ethnicity other than NHOPI, Asian, and White (n=41,292 discharges) were excluded (total 42,863 discharges). In the final analyses, a total of 243,693 delivery discharges were included.

### Variables

The primary outcome variable was discharge diagnosis of PPH as defined by ICD-9 Codes of 666.x and 641.11. These ICD-9 codes for PPH are well-validated, showing a positive predictive value of 80%.[19] The primary independent variable for this study was race-ethnicity, which was obtained from the HHIC race-ethnicity classifications. HHIC includes only one primary race-ethnicity, reported by each hospital from the patient's self-report at admission. The seven largest groups were Native Hawaiian, Japanese, Filipino, Chinese, other Pacific Islander,

other Asian, and White, accounting for more than 90% of the inpatient database. We classified race-ethnicity into three groups: NHOPI (Native Hawaiian and other Pacific Islander), Asian (Japanese, Filipino, Chinese, and other Asian), and White for this study, and used Whites as the reference group. We considered the following potential pre-specified confounders: age, delivery type (cesarean with labor, cesarean without labor, operative vaginal delivery, and spontaneous vaginal delivery), history of cesarean delivery, smoking, obesity, substance abuse, multiple gestation, multiparity, chronic hypertension, gestational hypertension, pre-gestational diabetes, gestational diabetes, pre-eclampsia, labor induction, polyhydramnios, chorioamnionitis, placental abruption, placenta previa, and prolonged labor.[18-22] We also categorized the period with different diagnostic criteria for preeclampsia (before year 2000, between 2000 and 2013 November, after 2013 November). In secondary analyses, we investigated racial-ethnic disparities in uterine atony, transfusion, and hysterectomy among patients who were diagnosed with PPH. All ICD-9 codes for clinical variables are described in the Appendix.

### Statistical Analysis

Demographic and clinical characteristics are represented by frequency, mean, and standard deviation. Bivariate associations between the variables and PPH were assessed using chi-squared tests for categorical variables and two-sample *t*-test for maternal age. A multivariable logistic regression using all factors in Table 1 and stepwise selection method was performed with significance level of 0.01 for entry and staying to find the best model to investigate the impact of race-ethnicity on PPH. C-statistic was assessed to measure the accuracy of the final model in predicting PPH. Chi-squared tests were used to assess racial/ethnic differences in the rate of uterine atony, hysterectomy, and transfusion. Due to the large data, $P < .01$ was considered statistically significant. All analyses were performed in SAS 9.4 (SAS institute, Cary NC).

## Results

A total of 243,693 in-hospital delivery discharges (35.0% NHOPI, 44.0% Asian, and 21.0% White) were analyzed. The average maternal age was 28.5 years (SD=6.3). Overall, there were 12,141 (5.0%) discharge diagnoses of PPH. A bivariate analysis demonstrated risk factors that were significantly associated with PPH (Table 1). Among patients with PPH, there were more NHOPI (37.1%) and Asians (47.6%), compared to Whites (15.3%). A multivariable logistic regression was conducted using all the factors in Table 1 and stepwise selection was used to find the best model to investigate the impact of race-ethnicity on PPH. Table 2 represents the results from the final logistic regression model for PPH, with moderate level of prediction (c-statistic = 0.657, 95% confidence interval [CI], 0.652-0.662). After controlling for the confounding variables, race-ethnicity remained significant. Compared to Whites, NHOPI (adjusted odds ratio [aOR], 1.40; 95% CI, 1.32-1.48) and Asians (aOR, 1.45; 95% CI, 1.37-1.53) were more likely to have PPH.

Furthermore, labor induction (aOR, 1.45; 95% CI, 1.37-1.52), prolonged labor (aOR, 1.26; 95% CI; 1.12-1.42), multiple gestation (aOR, 2.72; 95% CI, 2.22-3.32), gestational hypertension (aOR, 1.37; 95% CI, 1.27-1.48), gestational diabetes (aOR, 1.15; 95% CI, 1.07-1.23), preeclampsia (aOR, 2.11; 95% CI, 1.95-2.28), chorioamnionitis (aOR, 2.15; 95% CI, 2.01-2.30), placental abruption (aOR, 1.78; 95% CI, 1.54-2.07), placenta previa (aOR, 66.01; 95% CI, 59.30-73.48), and obesity (aOR, 1.56; 95% CI, 1.38-1.77) were associated with PPH (Table 2). Patients who delivered vaginally were more likely to have PPH than the patients who went through cesarean section delivery with labor (operative vaginal: aOR, 1.74; 95% CI, 1.63-1.87; spontaneous vaginal: aOR, 1.36; 95% CI, 1.04-1.77). Patients who delivered after year 2000 were more likely to be diagnosed with PPH than patients who delivered before year 2000 (between 2000 and 2013 November: aOR, 1.13; 95% CI, 1.08-1.18; after year 2013 November: aOR, 1.09; 95% CI, 1.02-1.17). In addition, to account for correlated multiple deliveries by the same patients, a separate multilevel model was conducted using hospital record identifier or master patient identifier to link patient-level encounter data. The model presents similar results in the magnitude and significance of the estimates (results not shown).

Table 3 represents prevalence of uterine atony, transfusion and hysterectomy by race-ethnicity among patients who had PPH. NHOPI (77.2%) and Asians (73.9%) had a higher proportion of uterine atony and PPH, compared to Whites (65.1%) ($P < .001$). Whites (13.1%) were more likely to receive transfusion than NHOPI (9.5%) and Asians (7.9%) after PPH ($P < .001$).

## Discussion

Using Hawai'i statewide claims data over an 18-year period, we demonstrated that NHOPI and Asians may have higher prevalence of PPH compared to Whites after adjusting for known risk factors. This study also validated prior studies that Asians/Pacific Islanders have higher rates of PPH, independent of the known risk factors for PPH.[11] Additionally, this study demonstrated a higher prevalence of uterine atony in NHOPIs and Asians than Whites, among those with PPH.

PPH is a clinical diagnosis yielded by the imprecise visual estimation of blood loss that may be diluted in other fluids (ie, amniotic fluid, urine, saline irrigation, etc) after the completion of the delivery of the fetus and placenta. The majority of blood loss is typically derived from the prior placental attachment site in the uterus, as the placenta avulses from its vascular bed, spiral arteries, and veins.[23] These vessels that were used to provide blood flow to the placenta and baby are mechanically compressed with a firm contraction of the uterus after placental delivery, and in conjunction with the coagulation system, these vessels are clotted to prevent further bleeding. Postpartum uterine bleeding may be in conjunction with genital tract lacerations of the cervix and vagina that may significantly contribute to blood loss and hemorrhage. Failure of the uterus to contract postpartum (uterine atony), abnormal vasculature of the uterus, incomplete evacuation of a portion of the placenta

| Table 1. Clinical Characteristics of Patients with and without Postpartum Hemorrhage | | | |
|---|---|---|---|
| **Variable** | **Total** | **Postpartum Hemorrhage, n (%)** | |
| | | **No (n=231,551)** | **Yes (n=12,142)** |
| **Age (yr.), mean ± SD** | 28.5 ± 6.3 | 28.5 ± 6.2 | 28.7 ± 6.4 |
| **Maternal Race-ethnicity** | | | |
| NHOPI | 85,178 (35.0%) | 80,670 (34.8%) | 4,508 (37.1%) |
| Asian | 107,256 (44.0%) | 101,477 (43.8%) | 5,779 (47.6%) |
| White | 51,259 (21.0%) | 49,404 (21.3%) | 1,855 (15.3%) |
| **Type of Delivery** | | | |
| Spontaneous Vaginal | 183,584 (75.3%) | 174,192 (75.2%) | 9,392 (77.4%) |
| Operative Vaginal | 1,657 (0.7%) | 1,597 (0.7%) | 60 (0.5%) |
| Cesarean without Labor | 31,060 (12.8%) | 29,524 (12.8%) | 1,536 (12.7%) |
| Cesarean with Labor | 27,382 (11.2%) | 26,238 (11.3%) | 1,154 (9.4%) |
| **Labor Induction** | 30,195 (12.4%) | 28,036 (12.1%) | 2,159 (17.8%) |
| **Prolonged Labor** | 5,049 (2.1%) | 4,728 (2.0%) | 321 (2.6%) |
| **Previous Cesarean** | 30,658 (12.6%) | 29,458 (12.7%) | 1,200 (9.9%) |
| **Multiple Gestation** | 1,286 (0.5%) | 1,161 (0.5%) | 125 (1.0%) |
| **Multiparity** | 2,991 (1.2%) | 2,810 (1.2%) | 181 (1.5%) |
| **Chronic Hypertension** | 371 (0.2%) | 344 (0.2%) | 27 (0.2%) |
| **Gestational Hypertension** | 11,427 (4.7%) | 10,641 (4.6%) | 786 (6.5%) |
| **Pre-gestational Diabetes** | 2.061 (0.9%) | 1,931 (0.8%) | 130 (1.1%) |
| **Gestational Diabetes** | 16,985 (7.0%) | 15,938 (6.9%) | 1,047 (8.7%) |
| **Preeclampsia** | 9,217 (3.8%) | 8,320 (3.6%) | 897 (7.4%) |
| **Polyhydramnios** | 1,733 (0.7%) | 1,611 (0.7%) | 122 (1.0%) |
| **Chorioamnionitis** | 12,134 (5.0%) | 11,030 (4.8%) | 1,104 (9.1%) |
| **Placental Abruption** | 2,408 (1.0%) | 2,175 (0.9%) | 233 (1.9%) |
| **Placenta Previa** | 1,882 (0.8%) | 612 (0.3%) | 1,270 (10.5%) |
| **Smoking** | 6,101 (2.5%) | 5,725 (2.5%) | 376 (3.1%) |
| **Obesity** | 3,469 (1.4%) | 3,176 (1.4%) | 293 (2.4%) |
| **Substance Abuse** | 5,307 (2.2%) | 4,981 (2.2%) | 326 (2.7%) |
| **Period with Different Diagnostic Criteria for Preeclampsia** | | | |
| Before year 2000 | 61,694 (25.3%) | 58,845 (25.4%) | 2,849 (23.5%) |
| Between year 200 and year 2013 November | 153,940 (63.2%) | 146,023 (63.1%) | 7,917 (65.2%) |
| After year 2013 November | 28,059 (11.5%) | 26,683 (11.5%) | 1,376 (11.3%) |

NHOPI = Native Hawaiian or other Pacific Islander. Column percentage. All were $P < .01$ except chronic hypertension ($P = .042$).

or other products of conception, deficits within a patient's coagulation system, and large lacerations in to the genital tract place postpartum women at risk for PPH.[23]

There have been several studies linking a familial or genetic predisposition to PPH. A large California database study was able to identify a higher risk of PPH among Asians/Pacific Islanders as a cohort, but did not distinguish rates of PPH among the distinct ethnicities.[11] A recent study in the Swedish Database of vaginal births identified families with higher rates of PPH than the general population;[24] these studies suggest a familial predisposition to PPH. In an attempt to identify genetic entities that place an individual at risk for PPH, a cohort of 3,219 Italian women was used to study genetic polymorphisms to determine a biochemical rationale for women at risk for PPH.[25] Aside from the aforementioned studies, there is a paucity in data identifying genetic and ethnic entities as potential independent risk factors for PPH. Interestingly, in a sub-analysis of our study, we found a statistically higher rate of uterine atony as the cause of PPH among each distinct ethnicity of Asian and Pacific Islander when compared to Whites in Hawai'i. This may suggest a genetic predisposition for defects of uterine contraction in the postpartum state in these individuals, placing them at higher risk for hemorrhage. Our study also showed an unexpected finding of modestly higher rates of transfusion among Whites compared to NHOPI and Asians. Due to the retrospective nature of the study, the reason for this apparent association is unclear and bears further investigation.

As postpartum hemostasis is multifaceted and relies on the coordination of multiple systemic factors, other genetic predispositions to include defects in coagulation, prostaglandin synthesis, or tissue pliability (degree of laceration) may also contribute to the higher PPH incidence in Asians/Pacific Island-

| Table 2. Final Model by Stepwise Multivariable Logistic Regression for Patients with Postpartum Hemorrhage | | |
|---|---|---|
| **Variable** | **Unadjusted OR (95% CI)** | **Adjusted Odds Ratio (95% CI)** |
| **Maternal Race-ethnicity** | | |
| NHOPI | 1.52 (1.44-1.60)*** | 1.40 (1.32-1.48)*** |
| Asian | 1.49 (1.41-1.57)*** | 1.45 (1.37-1.53)*** |
| White | Reference | Reference |
| **Type of Delivery** | | |
| Operative Vaginal | 1.23 (1.15-1.31)*** | 1.74 (1.63-1.87)*** |
| Spontaneous Vaginal | 0.86 (0.66-1.11) | 1.36 (1.04-1.77)* |
| Cesarean without Labor | 1.18 (1.09-1.28)*** | 0.97 (0.89-1.07) |
| Cesarean with Labor | Reference | Reference |
| **Labor Induction** | 1.57 (1.50-1.65)*** | 1.45 (1.37-1.52)*** |
| **Prolonged Labor** | 1.30 (1.16-1.46)*** | 1.26 (1.12-1.42)*** |
| **Multiple Gestation** | 2.07 (1.72-2.49)*** | 2.72 (2.22-3.32)*** |
| **Gestational Hypertension** | 1.44 (1.33-1.55)*** | 1.37 (1.27-1.48)*** |
| **Gestational Diabetes** | 1.28 (1.20-1.36)*** | 1.15 (1.07-1.23)*** |
| **Preeclampsia** | 2.14 (1.99-2.30)*** | 2.11 (1.95-2.28)*** |
| **Chorioamnionitis** | 2.00 (1.88-2.13)*** | 2.15 (2.01-2.30)*** |
| **Placental Abruption** | 2.06 (1.80-2.37)*** | 1.78 (1.54-2.07)*** |
| **Placenta Previa** | 44.07 (39.95-48.63)*** | 66.01 (59.30-73.48)*** |
| **Obesity** | 1.78 (1.58-2.01)*** | 1.56 (1.38-1.77)*** |
| **Period with Different Diagnostic Criteria for Preeclampsia** | | |
| Before year 2000 | Reference | Reference |
| Between year 200 and year 2013 November | 1.12 (1.07-1.17)*** | 1.13 (1.08-1.18)*** |
| After year 2013 November | 1.07 (1.00-1.14) | 1.09 (1.02-1.17)* |

NHOPI = Native Hawaiian or other Pacific Islander. C-statistic was 0.657 (95% CI = 0.652-0.662). *$P$ < .05; **$P$ < .01; ***$P$ < .001

| Table 3. Rate of Uterine Atony, Transfusion and Hysterectomy among Patients with Postpartum Hemorrhage by Race-ethnicity, n (%) | | | |
|---|---|---|---|
| **Race-ethnicity** | **Uterine Atony (n=8,962; 73.8%)** | **Transfusion (n=1,591; 8.7%)** | **Hysterectomy (n=218; 1.8%)** |
| NHOPI | 3,482 (77.2%)*** | 429 (9.5%)*** | 72 (1.6%) |
| Asian | 4,273 (73.9%)*** | 459 (7.9%)*** | 109 (1.9%) |
| White | 1,207 (65.1%) | 242 (13.1%) | 37 (2.0%) |

NHOPI = Native Hawaiian or other Pacific Islander. Chi-square test was conducted to compare White. *$P$ < .05; **$P$ < .01; ***$P$ < .001

ers. Asians have twice the odds of having a 3rd or 4th degree perineal laceration postpartum, which can lead to significant PPH even after controlling for known risk factors such as maternal age, operative vaginal delivery rate (forceps or vacuum), birth weight, and episiotomy rate.[8,26] Further study is needed to better assess which specific biological factor is driving the observed racial-ethnic differences in the prevalence of PPH.

The strengths of our study include a large sample size of over 240,000 deliveries and detailed ethnicity information for a diverse, multiethnic population. The study findings were also consistent with prior studies and support a multitude of risk factors that increase the risk of PPH. There are several limitations to our study. First, estimation of blood loss is most often "qualitative" rather than a "quantitative" assessment, which may introduce bias from the diagnosing clinician. There may also be reporting bias to either over report or underreport PPH in this population. Second, the data on gestational age at delivery and birth weight were not available, and thus their impact could not be assessed. Third, race-ethnicity was based on the patient's self-report and the algorithm used by HHIC to assign a patient to a single race-ethnicity may underestimate White race. Fourth, since the data was limited to the state of Hawai'i, our results may not be generalizable to other populations within the mainland United States. Fifth, this data assessed the "in-hospital" deliveries for the specific hospital admission and did not account for outside of institution births or patients who were readmitted to the hospital for a "delayed PPH" possibly impacting our observation. Sixth, our study was limited by what variables were available. Future studies should consider other potential confounders such as BMI, prior history of pre-

eclampsia, breastfeeding, or oxytocin administered postpartum which can stimulate uterine contractions. Finally, similar to the other claims databases, misclassification or miscoding of ICD-9 codes could occur due to physician coding or data entry errors.

## Conclusion

PPH prevalence was higher in NHOPI and Asians compared to Whites in the state of Hawai'i. PPH is a major contributor to morbidity and mortality both worldwide and in the United States and further delineating distinct ethnicity data as independent risk factors may be important for patient counseling, prevention, and treatment. This epidemiological data will also aid in identifying future research studies aimed at genetics/genomics within families and ethnicities at risk for PPH. Currently, as the etiology and pathophysiology for PPH is multifactorial, we are not able to assert a specific rationale for why those of Asian and Pacific Islander ethnicity have higher rates of PPH, even when controlling for known risk factors. With the delineation of race to be an additional risk factor for PPH, further preventative tactics may be employed to effectively reduce morbidity and mortality from this disease.[27] Further studies to delineate the causal relationship between PPH and ethnicity are needed to aid in the counseling, prevention, and treatment of these patients as well as to confirm our findings.

## Conflict of Interest

None of the authors identify any conflict of interest.


## Acknowledgments

Dr. Nakagawa was supported in part by the research grant from the National Institute on Minority Health and Health Disparities of the National Institutes of Health (NIH) (P20MD000173). Dr. Lim and Ms. Gandhi were partially supported by three grants U54MD007584, G12MD007601, and P20GM103466 from the NIH. The content is solely the responsibility of the authors and does not necessarily reflect the official opinion of the NIH.



Authors' Affiliations:
- The Queen's Medical Center, Honolulu, HI (SAH, KN)
- Department of Obstetrics and Gynecology and Women's Health, John A. Burns School of Medicine, University of Hawai'i, Honolulu, HI (SAH)
- Office of Biostatistics and Quantitative Health Sciences, John A Burns School of Medicine, University of Hawai'i, Honolulu, HI (EL, KRG)
- Hawai'i Health Information Corporation, Honolulu, HI (JM)
- Department of Medicine, John A. Burns School of Medicine, University of Hawai'i, Honolulu, HI (KN)

Correspondence to:
Scott A Harvey MD, MS; Kapi'olani Medical Center, 1319 Punahou St., #824, Honolulu, HI 96826; Email: sharvey@hawaii.edu


## References


1. Bulletins—Gynecology ACoP. ACOG Practice Bulletin. Clinical management guidelines for obstetrician-gynecologists, Number 72, May 2006: Vaginitis. *Obstetrics and gynecology.* 2006;107(5):1195-1206.
2. *WHO Recommendations for the Prevention and Treatment of Postpartum Haemorrhage*. Geneva2012.
3. Gynecologists RCoOa. Postpartum hemorrhage: prevention and management. 2011; https://www.rcog.org.uk/globalassets/documents/guidelines/gt52postpartumhaemorrhage0411.pdf. Accessed November 29, 2016.
4. Oyelese Y, Ananth CV. Postpartum hemorrhage: epidemiology, risk factors, and causes. *Clin Obstet Gynecol.* 2010;53(1):147-156.
5. Rath WH. Postpartum hemorrhage--update on problems of definitions and diagnosis. *Acta Obstet Gynecol Scand.* 2011;90(5):421-428.
6. Women ACoHCfU. ACOG committee opinion. Number 317, October 2005. Racial and ethnic disparities in women's health. *Obstetrics and Gynecology.* 2005;106(4):889-892.
7. Magann EF, Evans S, Hutchinson M, Collins R, Howard BC, Morrison JC. Postpartum hemorrhage after vaginal birth: an analysis of risk factors. *South Med J.* 2005;98(4):419-422.
8. Bryant AS, Worjoloh A, Caughey AB, Washington AE. Racial/ethnic disparities in obstetric outcomes and care: prevalence and determinants. *American Journal of Obstetrics and Gynecology.* 2010;202(4):335-343.
9. Briley A, Seed PT, Tydeman G, et al. Reporting errors, incidence and risk factors for postpartum haemorrhage and progression to severe PPH: a prospective observational study. *BJOG : An International Journal of Obstetrics and Gynaecology.* 2014;121(7):876-888.
10. Louis JM, Menard MK, Gee RE. Racial and ethnic disparities in maternal morbidity and mortality. *Obstetrics and Gynecology.* 2015;125(3):690-694.
11. Bryant A, Mhyre JM, Leffert LR, Hoban RA, Yakoob MY, Bateman BT. The association of maternal race and ethnicity and the risk of postpartum hemorrhage. *Anesth Analg.* 2012;115(5):1127-1136.
12. Cabacungan ET, Ngui EM, McGinley EL. Racial/ethnic disparities in maternal morbidities: a statewide study of labor and delivery hospitalizations in Wisconsin. *Matern Child Health J.* 2012;16(7):1455-1467.
13. Creanga AA, Berg CJ, Syverson C, Seed K, Bruce FC, Callaghan WM. Race, ethnicity, and nativity differentials in pregnancy-related mortality in the United States: 1993-2006. *Obstetrics and Gynecology.* 2012;120(2 Pt 1):261-268.
14. Creanga AA, Bateman BT, Kuklina EV, Callaghan WM. Racial and ethnic disparities in severe maternal morbidity: a multistate analysis, 2008-2010. *American Journal of Obstetrics and Gynecology.* 2014;210(5):435 e431-438.
15. Grobman WA, Bailit JL, Rice MM, et al. Racial and ethnic disparities in maternal morbidity and obstetric care. *Obstetrics and Gynecology.* 2015;125(6):1460-1467.
16. Lyndon A, Lee HC, Gilbert WM, Gould JB, Lee KA. Maternal morbidity during childbirth hospitalization in California. *J Matern Fetal Neonatal Med.* 2012;25(12):2529-2535.
17. Corporation HHI. Hawaii Health Information Corporation. http://www.hhic.org/. Accessed January 16, 2015.
18. Kramer MS, Berg C, Abenhaim H, et al. Incidence, risk factors, and temporal trends in severe postpartum hemorrhage. *American Journal of Obstetrics and Gynecology.* 2013;209(5):449 e441-447.
19. Lao TT, Sahota DS, Cheng YK, Law LW, Leung TY. Advanced maternal age and postpartum hemorrhage - risk factor or red herring? *J Matern Fetal Neonatal Med.* 2014;27(3):243-246.
20. Magann EF, Evans S, Hutchinson M, Collins R, Lanneau G, Morrison JC. Postpartum hemorrhage after cesarean delivery: an analysis of risk factors. *South Med J.* 2005;98(7):681-685.
21. Wetta LA, Szychowski JM, Seals S, Mancuso MS, Biggio JR, Tita AT. Risk factors for uterine atony/postpartum hemorrhage requiring treatment after vaginal delivery. *American Journal of Obstetrics and Gynecology.* 2013;209(1):51 e51-56.
22. Driessen M, Bouvier-Colle MH, Dupont C, et al. Postpartum hemorrhage resulting from uterine atony after vaginal delivery: factors associated with severity. *Obstetrics and Gynecology.* 2011;117(1):21-31.
23. Cunningham FG, Williams JW. *Williams Obstetrics.* 23rd ed. New York: McGraw-Hill Medical; 2010.
24. Oberg AS, Hernandez-Diaz S, Frisell T, Greene MF, Almqvist C, Bateman BT. Genetic contribution to postpartum haemorrhage in Swedish population: cohort study of 466,686 births. *BMJ.* 2014;349:g4984.
25. Biguzzi E, Franchi F, Acaia B, et al. Genetic background and risk of postpartum haemorrhage: results from an Italian cohort of 3219 women. *Haemophilia.* 2014;20(6):e377-383.
26. Combs CA, Murphy EL, Laros RK, Jr. Factors associated with postpartum hemorrhage with vaginal birth. *Obstetrics and Gynecology.* 1991;77(1):69-76.
27. Haeri S, Dildy GA, 3rd. Maternal mortality from hemorrhage. *Semin Perinatol.* 2012;36(1):48-55.