HHS Public Access

Author manuscript

Eur J Clin Nutr. Author manuscript; available in PMC 2017 September 01.

Published in final edited form as:

Eur J Clin Nutr. 2016 September ; 70(9): 1022–1027. doi:10.1038/ejcn.2016.32.

Ethnic Admixture Affects Diabetes Risk in Native Hawaiians: The Multiethnic Cohort

Gertraud Maskarinec[1], **Yukiko Morimoto**[1], **Simone Jacobs**[2], **Andrew Grandinetti**[3], **Marjorie K. Mau**[4], and **Laurence N. Kolonel**[1]

[1]University of Hawaii Cancer Center, Honolulu, HI

[2]German Institute of Human Nutrition, Potsdam-Rehbrücke, Germany

[3]Office of Public Health Studies, University of Hawaii, Honolulu, HI

[4]Department of Native Hawaiian Health, JABSOM, University of Hawaii, Honolulu, HI

Abstract

Background/Objectives—Obesity and diabetes rates are high in Native Hawaiians (NH) who commonly have mixed ancestries. Persons of Asian ancestry experience a high risk of type 2 diabetes despite the relatively low body weight. We evaluated the impact of ethnic admixture on diabetes risk among NH in the Multiethnic Cohort (MEC).

Methods/Subjects—Based on self-reports, 11,521 eligible men and women were categorized into NH/white, NH/other, NH alone, NH/Asian, and the most common three ancestry admixture, NH/Chinese/white. Cox proportional hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated with the NH/white category as the reference group; covariates included known confounders, i.e., body mass index (BMI), dietary and other life-style factors.

Results—The NH alone category had the highest proportion of overweight and obese individuals and the NH/Asian category the lowest proportion. During 12 years of follow-up after cohort entry at 56 years, 2,072 incident cases were ascertained through questionnaires and health plan linkages. All NH categories had higher HRs than the NH/white category before and after adjustment for BMI. In fully-adjusted models, the NH/Asian category showed the highest risk (HR=1.45; 95%CI: 1.27–1.65), followed by NH/other (HR=1.20; 95%CI: 1.03–1.39), NH/Chinese/white (HR=1.19; 95%CI: 1.04–1.37), and NH alone (HR=1.19; 95%CI: 1.03–1.37). The elevated risk by Asian admixture was more pronounced in normal weight than overweight/obese individuals.

Conclusions—These findings indicate that Asian admixture in NHs is associated with higher risk for type 2 diabetes independent of known risk factors and suggest a role for ethnicity-related genetic factors in the development of this disease.

Address for correspondence: Gertraud Maskarinec, MD, PhD, University of Hawaii Cancer Center, 701 Ilalo Street, Honolulu, HI 96813, Phone: (808) 586-3078, FAX: (808) 586-2982, gertraud@cc.hawaii.edu.

Conflict of Interests. None of the authors have a conflict of interest to declare.

Introduction

With increasing obesity rates, the number of people with type 2 diabetes is rising across the world,[1;2] but the incidence is considerably higher in Asians and Pacific Islanders than in whites.[3;4] In the United States, 20–40% higher rates of type 2 diabetes were reported for Asian Americans than whites after adjustment for body mass index (BMI).[3;5] High rates of obesity among Native Hawaiians (NH) and other Pacific Islanders appear to account for their elevated risk,[6–8] whereas the high incidence of diabetes in Asian Americans is independent of BMI.[8–11] After Captain Cook reached the Hawaiian Islands in 1778, the NH population was decimated due to diseases resulting from contact with Westerners, for which NHs had no immunity.[12;13] As a result of intermarriage with members of other ethnic groups, in particular white and Chinese, the number of Hawaii residents reporting Part-Hawaiian ethnicity increased to 289,970 by 2010.[14] Individuals reporting Hawaiian/Pacific Islander cultural identity have significantly higher BMIs than those reporting Asian American cultural identity that could be only partially explained by variations in educational attainment, socioeconomic status, and lifestyle factors.[15] From research within the Multiethnic Cohort (MEC), it appears that persons with multiple ancestries are more likely to have excess body weight.[16;17] For example, the prevalence of overweight/obesity in the NH/Asian admixture group was higher than the average of the prevalence estimates for NHs and Asians.[16] Given previous studies showing that Asian Americans experience a 2–3 fold higher risk of type 2 diabetes than whites at the same levels of BMI,[8;18] we examined whether the incidence of diabetes varies in NHs by admixture. The issue of admixed populations is of global importance since racial and ethnic admixture has become widespread as travel to distant countries from one's place of origin has become exceedingly common. Indeed, increasing mixed racial and ethnic ancestry has been documented across the United States[19], Mexico,[20] and Malaysia.[21] Understanding genetic ancestry and differences in racial admixture within regions has the potential to better inform public health officials to respond to projected health risks of a growing sector of the population, which is interracially mixed.[19;20]

Methods

Study Population

The MEC was established in 1993–1996 to study diet and cancer among different ethnic groups in Hawaii and California,[22] but the current analysis was restricted to the Hawaii component of the cohort.[8] All participants provided informed consent at cohort entry. Although whites, Japanese American, and NHs were targeted for recruitment in Hawaii, a substantial number of participants reported other Asian or multiracial backgrounds. A comparison of white, Japanese, and NH cohort members with census data indicated that the MEC represents all levels of education although cohort members were somewhat better educated than the general population.[22] Subjects entered the cohort by completing a 26-page, self-administered mailed survey that asked about demographic background, medical conditions, anthropometric measures, diet, and lifestyle factors. The participants marked all applicable ethnic backgrounds (African American, Chinese, Filipino, NH, Japanese, Korean, Latino, white, and other). Diet was assessed using a validated quantitative food frequency

questionnaire (FFQ) and an extensive nutritional database.[22;23] After identifying a total of 11,521 persons who reported NH ancestry, we created the following admixture categories: NH alone, NH/white, NH/Asian, i.e., one or more reports of Chinese, Korean, Japanese, or Filipino ancestry, NH/white/Chinese, a common combination for historic reasons,[13] and NH/other, i.e., combinations of Latino, African American, Other with white and/or Asian.

Identification of Diabetes Cases

Of the original 103,898 members within the Hawaii component of the MEC, 10,028 (9.7%) who reported a diagnosis of diabetes at baseline and 10 subjects with missing information were excluded. Information on diabetes status was available at three time points, i.e., from a follow-up questionnaire in 1999–2003, a medication questionnaire administered during 2003–2006, and a linkage with Hawaii's two major health plans in 2007.[8] Specific algorithms based on multiple claims for diabetes-related treatment services were used to identify cases with the purpose to be included in diabetes care registries.[8] One of the two health plans used an algorithm based on multiple claims for diabetes-related treatment services over a 2-year time span. Single diagnostic claims were not classified as cases in order to avoid false positives; physicians had the opportunity to refer or remove cases. One path identified a specified set of diagnosis codes associated with treatment services (inpatient, emergency, or outpatient professional); another path examined outpatient prescription drugs or supplies for diabetes. The other health plan, a health maintenance organization, based their case identification on clinical information (HbA_{1c} testing), pharmacy records (insulin, sulfonylurea drugs, metformin, and blood glucose testing supplies), hospital discharge diagnoses reflecting the presence of diabetes, and outpatient encounters. The Institutional Review Boards of the University of Hawaii and Kaiser Permanente approved the study protocol and all participants signed a consent form at cohort entry. When the health plans did not provide a diagnosis date, the estimated date of diagnosis was the midpoint in time between the last report of not having diabetes and the first indication of diabetes. For incident cases, the follow-up time was calculated as the time between the baseline questionnaire and an estimated diagnosis date. For non-cases, the follow-up time was calculated as the time between the date of the baseline questionnaire and any of the following events if applicable: the date of death, the last date when data on diabetes status was available, i.e., the date of the questionnaire or health plan linkage.

Statistical analysis

The average annual incidence rates were computed by ethnic group as the sum of the number of newly diagnosed diabetes cases divided by the sum of person-years of follow-up. Cox proportional regression using PROC PHREG in the SAS software package, version 9.3 (SAS Institute, Inc., Cary, NC) was applied to estimate diabetes risk by admixture group with NH/white as the reference category because they had the lowest incidence rate. In the first model, we calculated hazard ratios (HR) and 95% confidence intervals (CI) using models stratified by age at cohort entry (continuous) and adjusted for sex and years of education (<12, 12, 13–15, and ≥16 years). The second model also included BMI (<22.5, 22.5–<25, 25–<30, and ≥30 kg/m^2). In a third model, we further adjusted for all variables that were previously found to be associated with diabetes risk in this dataset, i.e., smoking status and physical activity, as well as intake of total energy, alcohol, red meat, dietary fiber,

sugared sodas, and coffee.[24;25] Physical activity was expressed as metabolic equivalents (METs) and 711 missing values were replaced with the means for each sex-ethnic group. Daily intakes of dietary fiber and red meat were expressed per 4,184 kJ of energy intake; logarithmic transformations were applied to intakes of dietary fiber, red meat, total energy as well as METs. For alcohol (<1, 1- to <1, ≥1 drink/day), coffee (almost never, <1, 1, 2, and ≥3 cups/day), and sugared soda (0, <2, and ≥2 sodas/week) consumption, categorical variables were created. To evaluate the importance of admixture as compared to the NH/white group independent of BMI, stratified models by BMI category were performed.

Results

After excluding subjects with prevalent diabetes or missing data at baseline, 11,521 cohort members (4,961 men and 6,560 women) who reported any Native Hawaiian ancestry were part of this analysis (Table 1). The mean age of the participants was 56 years at cohort entry and the mean follow-up time was 11.9±3.4 years. When divided into admixture groups (Table 2), 23% were NH/white, 16% NH/other, 21% NH/white/Chinese, 16% NH alone, and 24% NH/Asian. The distribution of BMI, smoking (except in men), education, physical activity, and dietary intakes differed significantly across admixture groups. The overall incidence of new cases was 16.0 per 1,000 pyrs with the highest rates in NH/Asian (18.3 per 1,000 pyrs) and NH alone (17.8 per 1,000 pyrs) and the lowest rate in the NH/white group (13.4 per 1,000 pyrs). After stratification by BMI category, the incidence was approximately 2-fold higher in the overweight and 5-fold higher in the obese participants when compared to the normal weight category. Within each BMI category, the pattern by admixture group was similar as in the total study population: the incidence rates were lowest for the NH/white group (3.8, 12.3, and 26.4 per 1,000 pyrs across BMI categories) and highest for those with Asian ancestry (8.8, 18.8, and 34.7 per 1,000 pyrs). For those who reported NH alone, the respective rates were similar to the NH/Asian group (6.7, 13.3, and 31.3 per 1,000 pyrs).

The basic Cox regression models stratified by age at cohort entry and adjusted for sex and years of education showed a similar pattern of ethnic differences as the incidence rates (Table 2). When compared to the NH/white group, the risk estimates for NH/Asian and NH alone were 1.36 and 1.27, respectively, in the simple model, whereas the risk was elevated by 12 and 16% for the NH/other and NH/Chinese/white admixture groups. Including BMI into the models primarily increased the relative risk for the NH/Asian group (HR=1.52; 95%CI: 1.34–1.73), while further adjustment for diet and lifestyle factors only modified the risk estimates minimally. Stratification by BMI category again indicated that the NH/Asian admixture group experienced the highest risk of diabetes as compared to other admixture groups; the respective HRs for normal weight, overweight, and obese participants were 2.33 (95%CI: 1.60–3.39), 1.54 (95%CI: 1.25–1.90), and 1.30 (95%CI: 1.08–1.56), whereas the HRs for the other admixture groups were 1.66–1.83 in the normal weight individuals, 1.07–1.26 among overweight persons, and 1.10–1.20 in the obese category.

Discussion

Within this population of more than 10,000 middle-aged and elderly individuals with NH ancestry who were followed for nearly 12 years, the incidence and risk of developing type 2

diabetes varied distinctly according to self-reported admixture. NH participants who reported only white admixture were least likely to develop diabetes even after adjustment for potential confounders, whereas any Asian ancestry predicted a more than 2-fold higher risk to be diagnosed with diabetes. As shown in previous reports,[8;18] the incidence rates for participants with white and Asian ancestry in the MEC were 9.9 and 17.9–21.3 per 100,000 pyrs, respectively; this indicates that the incidence in the NH/white group (13.4 per 100,000 pyrs) was one third higher than in whites, while incidence rate for the NH/Asian category (18.3 per 100,000 pyrs) was similar to the different Asian groups.[18] The type of admixture influenced diabetes risk more among normal weight than overweight/obese participants. One possible explanation, which needs to be explored in future research, is that genetic factors in Asians predispose to type 2 diabetes and other weight-related diseases,[26] but heavier Asian individuals develop diabetes primarily as a consequence of excess body weight.

A limited number of studies have described the effects of ethnic admixture. In addition to the obesity studies within the MEC,[16;17] the prevalence of type 2 diabetes in two rural communities in Hawaii was not significantly associated with the percentage of Hawaiian ancestry after adjusting for age,[27] but in a later report from the same population, a higher Hawaiian blood quantum was significantly associated with higher fasting glucose, BMI, and waist-to-hip ratio.[28] When descent from other ethnic groups was examined, full-Hawaiians had significantly higher fasting glucose concentrations than part-Hawaiians. In contrast, part-Hawaiians of predominantly Asian ancestry had the highest 2-hour postprandial glucose concentrations.

A number of different conditions have been investigated in relation to admixture among other ethnic groups. The percentage of African ancestry improved the prediction of lung function and allowed more accurate disease severity classification.[29] Similarly, significant associations of indigenous ancestry in admixed mestizos across Mexico with lung function have been reported.[20] In Latina women, the proportion of European vs. Indigenous American ancestry was associated with higher breast cancer risk after adjustment for known risk factors.[30] Among Latinos in the south western United States[31] and among Tohono O'odham (Pima) Indians in Arizona,[32] a higher proportion of Native American blood quantum was associated with a higher risk of type 2 diabetes.

The presence of insulin resistance syndrome among NHs was shown to be similar as in other high risk indigenous populations, e.g., individuals from the Tohono O'odham tribe in Arizona.[32] However, Asians are more likely to manifest a β-cell secretion defect measured as elevated post-glucose challenge hyperglycemia.[33] Thus, NHs who are an admixture of NH and Asian ancestry may be at higher risk because of a "double whammy" metabolic load of both insulin resistance and β-cell secretion defects within the same individual, who is unable to compensate for hyperinsulinemia and, thus, presents with elevated fasting and postprandial hyperglycemia defined as diabetes mellitus.

Given the history of NHs who probably started to settle on the Hawaiian Islands in 1025–1120 as seafarers migrating from East Asia across the Pacific,[34;35] the population genetic structure of NHs is highly complex. In a group of NH MEC participants, 27% East Asian, 27% European, and 46% NH ancestry was detected.[36] In a later analysis using high-

resolution genome-wide SNP data and mitochondrial genomes of 148 and 160 NHs,[35] the genome of NHs who self-reported full NH heritage contained 78% NH, 12% European, and 8% Asian ancestry. The estimated proportions of NH ancestry for those who reported mixed ancestry were found to be consistent with their self-reported heritage.[35] However, the impact of genetic markers to predict onset of type 2 diabetes has been thus far poorly delineated; therefore, self-report ethnic and racial ancestry has been most useful in terms of clinical care and prevention strategies.

This analysis had several strengths including the large sample size, the long follow-up, the detailed ethnicity information, the availability of self-reported data on dietary and lifestyle factors, and the multiple data sources to establish a diabetes diagnosis. A number of limitations need to be noted. Given the design of the questionnaire, we were not able to compute the percentage of NH or various Asian ancestries. Ideally, the self-reported ethnic background would be compared to genetic markers but these are not available for this large number of NH participants. Although no validations of self-reports and diabetes care registries are available for Hawaii, self-reported diabetes diagnoses[37;38] and care registries were found to be valid in other studies.[39] Given the age distribution of the MEC, which did not include persons less than 45 years of age at cohort entry, the current report pertains primarily to older individuals and excludes cases with early onset diabetes. The increasing number of cases diagnosed in younger Asians,[40] may have different etiologic features and/or genetic predispositions that need to explored in appropriate study populations.

The current findings are of interest to other populations because fine-scale ancestry patterns are critical for medical studies in many countries with highly admixed populations.[20] Efforts to develop more accurate disease prediction models based on genetic and lifestyle risk factors may have to consider the inclusion of differentiated ancestries instead of using simple self-reported categories.[20] The potential influence on public health and clinical care lies in targeted prevention approaches in high-risk populations.

The current findings among a study population aged 50 years and older indicate that NHs with Asian admixture are at higher risk for type 2 diabetes than those with white admixture or NH alone independent of BMI status, diet, and other known lifestyle related factors. Several complex biologic mechanisms for the high susceptibility of Asians to type 2 diabetes have been proposed,[41;42] including a role of visceral adipose tissue in glucose metabolism[43] and higher insulin sensitivity combined with lower insulin response in East Asians compared with whites.[42] These findings confirm that even among high risk populations, a broad range of risk assessment approaches may inform strategies to delay the onset of diabetes, can be tailored to individuals at highest risk, and should be implemented at earlier stages of the disease process, such as lower BMI and earlier age.

Acknowledgments

The Multiethnic Cohort Study has been supported by US Public Health Service (National Institutes of Health) grants R37CA54281 and UM1CA164973 and the diabetes project by R21DK073816. MKM was supported in part by NIH funding P20 MD 000173.

References

1. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. JAMA. 2002; 288:1723–1727. [PubMed: 12365955]
2. International Diabetes Federation. IDF Diabetes Atlas. 6. IDF; Bruxelles, Belgium: 2014. www.idf.org/diabetesatlas [3-2-2015]
3. Karter AJ, Schillinger D, Adams AS, Moffet HH, Liu J, Adler NE, et al. Elevated rates of diabetes in Pacific Islanders and Asian subgroups: The Diabetes Study of Northern California (DISTANCE). Diabetes Care. 2013; 36:574–579. [PubMed: 23069837]
4. Maskarinec G, Takata Y, Pagano I, Carlin L, Goodman MT, Le ML, et al. Trends and dietary determinants of overweight and obesity in a multiethnic population. Obesity (Silver Spring). 2006; 14:717–726. [PubMed: 16741275]
5. Lee JW, Brancati FL, Yeh HC. Trends in the prevalence of type 2 diabetes in Asians versus whites: results from the United States National Health Interview Survey, 1997–2008. Diabetes Care. 2011; 34:353–357. [PubMed: 21216863]
6. Grandinetti A, Kaholokula JK, Theriault AG, Mor JM, Chang HK, Waslien C. Prevalence of diabetes and glucose intolerance in an ethnically diverse rural community of Hawaii. Ethn Dis. 2007; 17:250–255. [PubMed: 17682354]
7. Davis J, Busch J, Hammatt Z, Novotny R, Harrigan R, Grandinetti A, et al. The relationship between ethnicity and obesity in Asian and Pacific Islander populations: a literature review. Ethn Dis. 2004; 14:111–118. [PubMed: 15002930]
8. Maskarinec G, Erber E, Grandinetti A, Verheus M, Oum R, Hopping BN, et al. Diabetes incidence based on linkages with health plans: the multiethnic cohort. Diabetes. 2009; 58:1732–1738. [PubMed: 19258435]
9. Burchfiel CM, Curb JD, Rodriguez BL, Yano K, Hwang LJ, Fong KO, et al. Incidence and predictors of diabetes in Japanese-American men. The Honolulu Heart Program. Ann Epidemiol. 1995; 5:33–43. [PubMed: 7728283]
10. Araneta MR, Wingard DL, Barrett-Connor E. Type 2 diabetes and metabolic syndrome in Filipina-American women : a high-risk nonobese population. Diabetes Care. 2002; 25:494–499. [PubMed: 11874936]
11. Staimez LR, Weber MB, Narayan KM, Oza-Frank R. A systematic review of overweight, obesity, and type 2 diabetes among Asian American subgroups. Curr Diabetes Rev. 2013; 9:312–331. [PubMed: 23590534]
12. Blaisdell RK. The impact of disease on Hawaii's history. Hawaii Med J. 2001; 60:295–296. [PubMed: 11797498]
13. Nordyke, EC. The peopling of Hawaii. University of Hawaii Press; Honolulu: 1989.
14. Hawaii Department of Business and Economic Development. [1-1-0015] 2013 State of Hawaii data book: a statistical abstract. 2014. http://dbedt.hawaii.gov/economic/databook/db2013/
15. Brown DE, Hampson SE, Dubanoski JP, Murai AS, Hillier TA. Effects of ethnicity and socioeconomic status on body composition in an admixed, multiethnic population in Hawaii. Am J Hum Biol. 2009; 21:383–388. [PubMed: 19213005]
16. Albright CL, Steffen AD, Wilkens LR, Henderson BE, Kolonel LN. The prevalence of obesity in ethnic admixture adults. Obesity (Silver Spring). 2008; 16:1138–1143. [PubMed: 18356848]
17. Albright CL, Steffen A, Wilkens LR, Henderson BE, Kolonel LN. Body mass index in monoracial and multiracial adults: results from the multiethnic cohort study. Ethn Dis. 2007; 17:268–273. [PubMed: 17682357]
18. Maskarinec G, Jacobs S, Morimoto Y, Chock M, Grandinetti A, Kolonel LN. Disparity in diabetes risk across Native Hawaiians and different Asian groups: the multiethnic cohort. Asia Pac J Public Health. 2015; 27:375–384. [PubMed: 25164594]
19. Bryc K, Durand EY, Macpherson JM, Reich D, Mountain JL. The genetic ancestry of African Americans, Latinos, and European Americans across the United States. Am J Hum Genet. 2015; 96:37–53. [PubMed: 25529636]

20. Moreno-Estrada A, Gignoux CR, Fernandez-Lopez JC, Zakharia F, Sikora M, Contreras AV, et al. Human genetics. The genetics of Mexico recapitulates Native American substructure and affects biomedical traits. Science. 2014; 344:1280–1285. [PubMed: 24926019]
21. Deng L, Hoh BP, Lu D, Saw WY, Twee-Hee OR, Kasturiratne A, et al. Dissecting the genetic structure and admixture of four geographical Malay populations. Sci Rep. 2015; 5:14375. [PubMed: 26395220]
22. Kolonel LN, Henderson BE, Hankin JH, Nomura AMY, Wilkens LR, Pike MC, et al. A multiethnic cohort in Hawaii and Los Angeles: baseline characteristics. Am J Epidemiol. 2000; 151:346–357. [PubMed: 10695593]
23. Stram DO, Hankin JH, Wilkens LR, Henderson B, Kolonel LN. Calibration of the dietary questionnaire for a multiethnic cohort in Hawaii and Los Angeles. Am J Epidemiol. 2000; 151:358–370. [PubMed: 10695594]
24. Steinbrecher A, Morimoto Y, Heak S, Ollberding NJ, Geller KS, Grandinetti A, et al. The preventable proportion of type 2 diabetes by ethnicity: the multiethnic cohort. Ann Epidemiol. 2011; 21:526–535. [PubMed: 21497517]
25. Doo T, Morimoto Y, Steinbrecher A, Kolonel LN, Maskarinec G. Coffee intake and risk of type 2 diabetes: the Multiethnic Cohort. Public Health Nutr. 2014; 17:1328–1336. [PubMed: 23442347]
26. Razak F, Anand SS, Shannon H, Vuksan V, Davis B, Jacobs R, et al. Defining obesity cut points in a multiethnic population. Circulation. 2007; 115:2111–2118. [PubMed: 17420343]
27. Grandinetti A, Chang HK, Mau MK, Curb JD, Kinney EK, Sagum R, et al. Prevalence of glucose intolerance among Native Hawaiians in two rural communities. Native Hawaiian Health Research (NHHR) Project. Diabetes Care. 1998; 21:549–554. [PubMed: 9571341]
28. Grandinetti A, Keawe'aimoku KJ, Chang HK, Chen R, Rodriguez BL, Melish JS, et al. Relationship between plasma glucose concentrations and Native Hawaiian Ancestry: The Native Hawaiian Health Research Project. Int J Obes Relat Metab Disord. 2002; 26:778–782. [PubMed: 12037647]
29. Kumar R, Seibold MA, Aldrich MC, Williams LK, Reiner AP, Colangelo L, et al. Genetic ancestry in lung-function predictions. N Engl J Med. 2010; 363:321–330. [PubMed: 20647190]
30. Fejerman L, John EM, Huntsman S, Beckman K, Choudhry S, Perez-Stable E, et al. Genetic ancestry and risk of breast cancer among U.S. Latinas. Cancer Res. 2008; 68:9723–9728. [PubMed: 19047150]
31. Parra EJ, Hoggart CJ, Bonilla C, Dios S, Norris JM, Marshall JA, et al. Relation of type 2 diabetes to individual admixture and candidate gene polymorphisms in the Hispanic American population of San Luis Valley, Colorado. J Med Genet. 2004; 41:e116. [PubMed: 15520398]
32. Mau MK, Grandinetti A, Arakaki RF, Chang HK, Kinney EK, Curb JD. The insulin resistance syndrome in native Hawaiians. Native Hawaiian Health Research (NHHR) Project. Diabetes Care. 1997; 20:1376–1380. [PubMed: 9283783]
33. Fujimoto WY, Bergstrom RW, Boyko EJ, Chen K, Kahn SE, Leonetti DL, et al. Type 2 diabetes and the metabolic syndrome in Japanese Americans. Diabetes Res Clin Pract. 2000; 50(Suppl 2):S73–S76. [PubMed: 11024587]
34. Wilmshurst JM, Hunt TL, Lipo CP, Anderson AJ. High-precision radiocarbon dating shows recent and rapid initial human colonization of East Polynesia. Proc Natl Acad Sci U S A. 2011; 108:1815–1820. [PubMed: 21187404]
35. Kim SK, Gignoux CR, Wall JD, Lum-Jones A, Wang H, Haiman CA, et al. Population genetic structure and origins of Native Hawaiians in the multiethnic cohort study. PLoS ONE. 2012; 7:e47881. [PubMed: 23144833]
36. Wang H, Haiman CA, Kolonel LN, Henderson BE, Wilkens LR, Le ML, et al. Self-reported ethnicity, genetic structure and the impact of population stratification in a multiethnic study. Hum Genet. 2010; 128:165–177. [PubMed: 20499252]
37. Molenaar EA, Van Ameijden EJ, Grobbee DE, Numans ME. Comparison of routine care self-reported and biometrical data on hypertension and diabetes: results of the Utrecht Health Project. Eur J Public Health. 2007; 17:199–205. [PubMed: 16887835]
38. Okura Y, Urban LH, Mahoney DW, Jacobsen SJ, Rodeheffer RJ. Agreement between self-report questionnaires and medical record data was substantial for diabetes, hypertension, myocardial

infarction and stroke but not for heart failure. J Clin Epidemiol. 2004; 57:1096–1103. [PubMed: 15528061]

39. Saydah SH, Geiss LS, Tierney E, Benjamin SM, Engelgau M, Brancati F. Review of the performance of methods to identify diabetes cases among vital statistics, administrative, and survey data. Ann Epidemiol. 2004; 14:507–516. [PubMed: 15301787]
40. Yeung RO, Zhang Y, Luk A, Yang W, Sobrepena L, Yoon KH, et al. Metabolic profiles and treatment gaps in young-onset type 2 diabetes in Asia (the JADE programme): a cross-sectional study of a prospective cohort. Lancet Diabetes Endocrinol. 2014; 2:935–943. [PubMed: 25081582]
41. Ma RC, Chan JC. Type 2 diabetes in East Asians: similarities and differences with populations in Europe and the United States. Ann N Y Acad Sci. 2013; 1281:64–91. [PubMed: 23551121]
42. Kodama K, Tojjar D, Yamada S, Toda K, Patel CJ, Butte AJ. Ethnic differences in the relationship between insulin sensitivity and insulin response: a systematic review and meta-analysis. Diabetes Care. 2013; 36:1789–1796. [PubMed: 23704681]
43. Hayashi T, Boyko EJ, McNeely MJ, Leonetti DL, Kahn SE, Fujimoto WY. Visceral adiposity, not abdominal subcutaneous fat area, is associated with an increase in future insulin resistance in Japanese Americans. Diabetes. 2008; 57:1269–1275. [PubMed: 18299316]

Table 1

Characteristics of participants with Native Hawaiian (NH) ancestry, Multiethnic Cohort *

	Male (N=4,961)						Female (N=6,560)					
	NH/white	NH/other	NH/Chinese/white	NH alone	NH/Asian	*P* value^	NH/white	NH/other	NH/Chinese/white	NH alone	NH/Asian	*P* Value^
N	1,078	714	983	905	1,281		1,554	1,084	1,489	926	1,507	
Age at cohort entry (years)	57.6±9.0	53.7±7.6	55.6±8.4	57.2±8.6	56.6±8.6	<0.001	57.4±8.8	52.8±7	55.0±8.5	57.3±8.7	56.7±8.7	<0.001
Follow-up time (years)	11.1±3.9	11.5±3.7	11.3±3.7	10.3±4.2	10.8±4.0		11.5±3.7	11.8±3	11.6±3.6	11.1±3.9	11.4±3.6	
Body mass index (kg/m^2)						<0.001						<0.001
<22	72 (6.7)	40 (5.6)	70 (7.1)	58 (6.4)	105 (8.2)		237 (15.3)	188 (17.3)	223 (15.0)	119 (12.9)	282 (18.7)	
22–<25	222 (20.6)	133 (18.6)	179 (18.2)	139 (16.4)	296 (23.1)		306 (19.7)	252 (23.3)	334 (22.4)	181 (20.0)	385 (25.6)	
25–<30	471 (43.7)	326 (45.7)	484 (49.2)	363 (40.1)	539 (42.1)		528 (34.0)	364 (33.6)	502 (33.7)	288 (31.1)	495 (32.8)	
≥30	313 (29.0)	215 (30.1)	250 (25.4)	345 (38.1)	341 (26.6)		483 (31.1)	280 (25.8)	430 (28.9)	338 (36.5)	345 (22.9)	
Smoking						0.09						<0.001
Never	333 (30.9)	207 (29.0)	334 (34.0)	304 (33.6)	455 (35.5)		658	469 (43.3)	667 (44.8)	368 (39.7)	802 (53.2)	
Past	498 (46.2)	345 (48.3)	440 (44.8)	392 (43.3)	538 (42.0)		517	350 (32.3)	475 (31.9)	299 (32.3)	404 (26.8)	
Current	247 (22.9)	162 (22.7)	209 (21.3)	209 (23.1)	288 (22.5)		379	265 (24.5)	347 (23.3)	259 (28.0)	301 (20.0)	
Education (years)						<0.001						<0.001
<12	135 (12.5)	63 (8.8)	75 (7.6)	179 (19.8)	154 (12.0)		151 (9.7)	57 (5.3)	104 (7.0)	177 (19.1)	158 (10.5)	
12	371 (34.4)	236 (33.1)	327 (33.3)	370 (40.9)	516 (40.3)		715	425 (39.2)	641 (43.1)	426 (46.0)	694 (46.1)	
13–15	354 (32.8)	244 (34.2)	349 (35.5)	233 (26.8)	364 (28.4)		467	384 (35.4)	476 (31.8)	195 (10.2)	391 (26.0)	
≥16	218 (22.0)	171 (24.0)	232 (23.6)	123 (12.4)	247 (19.3)		221	218 (20.1)	271 (18.2)	128 (13.8)	264 (17.5)	
Physical activity (METs)												
N with missing values	1.7±0.4 (57)	1.7±0.4 (20)	1.7±0.4 (44)	1.8±0.4 (75)	1.7±0.4 (69)	0.04	1.6±0.3 (105)	1.6±0.3 (50)	1.6±0.3 (90)	1.6±0.3 (102)	1.6±0.3 (99)	0.01
Total energy intake (kcals)	2763 ±1227	2803 ±1317	2772 ±1295	3007 ±1510	2791 ±1359	0.01	2309 ±1175	2284± 1161	2390 ±1210	2791 ±1359	2373 ±1265	<0.001
Dietary fiber (g/4,184 kJ/day)	9.2±3.8	8.6±3.4	8.9±3.5	9.3±4.0	8.6±3.5	<0.001	11.2±4.2	10.6±4.0	10.8±4.1	11.0±4.4	10.8±4.0	0.01
Red meat (g/4,184 kJ/day)	31.0±16.5	32.2±15.8	32.3±15.8	31.1±16.3	31.6±15.3	0.08	27.1±15.7	28.6±15.5	29.2±15.7	27.6±16.2	28.4±15.1	<0.001
Coffee (cups/day)	1.3±1.2	1.3±1.3	1.2±1.3	1.2±1.3	1.2±1.3	0.01	1.2±1.3	1.1±1.2	1.2±1.2	1.1±1.3	1.1±1.2	<0.001
Alcohol intake (g/day)	18.4±32.8	16.4±38.0	17.3±34.9	16.4±41.8	14.3±35.7	<0.001	6.3±22.1	4.1±14.7	5.7±22.3	4.2±14.2	3.3±15.4	<0.001

	Male (N=4,961)						Female (N=6,560)					
	NH/white	**NH/other**	**NH/Chinese/white**	**NH alone**	**NH/Asian**	***P* value^**	**NH/white**	**NH/other**	**NH/Chinese/white**	**NH alone**	**NH/Asian**	***P* Value^**
Sugared soda (g/day)	218±491	238±540	215±448	292±663	298±613	<0.001	186±522	160±438	188±500	235±605	178±456	<0.001

*Data are presented as mean±SD or N (%); percentages may not add to 100 due to rounding.

^Comparisons of ethnic groups within each sex were performed using generalized linear model on log-transformed, continuous variables (except for age at cohort entry) or chi-square test on categorical variables.

Table 2

Diabetes incidence rates and risk by Native Hawaiian (NH) admixture across body mass index (BMI) categories*

Variable/Admixture group	NH/white	NH/other	NH/Chinese/white	NH alone	NH/Asian	All
Number of participants	2,632	1,798	2,472	1,831	2,788	11,521
Incident diabetes cases	400	317	438	348	569	2,072
Diabetes incidence (1,000 pyrs)						
All	13.4	15.1	15.4	17.8	18.3	16.0
Normal weight	3.8	5.7	5.5	6.7	8.8	6.2
Overweight	12.3	14.9	13.9	13.3	18.8	14.7
Obese	26.4	29.3	32.0	31.3	34.7	30.6
Diabetes risk						
Model I*	1.00	1.12 (0.97–1.30)	1.16 (1.01–1.32)	1.27 (1.10–1.46)	1.36 (1.20–1.54)	
Model I plus BMI^	1.00	1.23 (1.06–1.42)	1.23 (1.07–1.40)	1.19 (1.03–1.37)	1.52 (1.34–1.73)	
Model I plus BMI, diet, and lifestyle¶	1.00	1.20 (1.03–1.39)	1.19 (1.04–1.37)	1.19 (1.03–1.37)	1.45 (1.27–1.65)	
By BMI category						
Normal weight (<25 kg/m^2)	1.00	1.81 (1.16–2.83)	1.66 (1.09–2.53)	1.83 (1.16–2.91)	2.33 (1.60–3.39)	
Overweight (25–<30 kg/m^2)	1.00	1.26 (0.99–1.62)	1.15 (0.92–1.44)	1.07 (0.83–1.39)	1.54 (1.25–1.90)	
Obese (≥30 kg/m^2)	1.00	1.10 (0.89–1.36)	1.20 (0.99–1.45)	1.19 (0.99–1.44)	1.30 (1.08–1.56)	

*Hazard ratios and 95% confidence intervals were obtained by Cox proportional hazard models, stratified by age at cohort entry and adjusted for, sex, and years of education (<12, 12, 13–15, and ≥16 years)

^Additionally adjusted for BMI (<22, 22–<25, 25–<30, and ≥30 kg/m^2)

¶Further adjustment for physical activity (log-transformed metabolic equivalents, 711 missing values were replaced with means for each sex-ethnic group), smoking (never, past, and current), years of education (<12, 12, 13–15, and ≥16 years), total energy intake (log-transformed), coffee (almost never, <1, ≥1, 2, and ≥3 cups/day) sugared soda (0, <2, and ≥2/week), alcohol (<1 drink/month, ≥1 drink/month to <1 drink/day, ≥1 drink/day), and intake of dietary fiber and red meat (both variables were log-transformed continuous, density per 4,184 kJ/day)

Eur J Clin Nutr. Author manuscript; available in PMC 2017 September 01.