UCSF
UC San Francisco Previously Published Works

Title

Obesity and metabolic outcomes in a safety-net health system

Permalink

Journal

ISSN

Authors

Huynh, Michael P
Bradshaw, Patrick T
Tana, Michele M
et al.

Publication Date

DOI

Peer reviewed

Obesity and Metabolic Outcomes in a Safety-Net Health System

Michael P. Huynh[a,*], **Patrick T. Bradshaw**[b], **Michele M. Tana**[c,d], **Carly Rachocki**[c], **Ma Somsouk**[c,e]

[a]School of Public Health, UCLA Center for Health Policy Research, Los Angeles, CA, USA;

[b]School of Public Health, Division of Epidemiology and Biostatistics, University of California, Berkeley, Berkeley, CA, USA;

[c]Department of Medicine, Division of Gastroenterology, University of California, San Francisco, San Francisco, CA, USA;

[d]UCSF Liver Center, San Francisco, CA, USA;

[e]Center for Vulnerable Populations, University of California, San Francisco, San Francisco, CA, USA

Abstract

In the United States, obesity has increased in prevalence over time and is strongly associated with subsequent outcomes such as diabetes mellitus (DM) and nonalcoholic fatty liver disease (NAFLD). It is unclear, however, as to how the magnitude of NAFLD risk from obesity and DM is increased in safety-net health system settings. Among the San Francisco Health Network (SFHN) patients (N=47,211), we examined the association between Body Mass Index (BMI) and elevated liver enzyme levels, including interaction by DM status. Our findings revealed that 32.2 percent of SFHN patients were obese, and Pacific Islanders in the safety-net had the highest rates of obesity compared to other racial groups, even after using higher race-specific BMI cutoffs. In SFHN, obesity was associated with elevated liver enzymes, with the relationship stronger among those without DM. Our findings highlight how obesity is a stronger factor of NAFLD in the absence of DM, suggesting that practitioners consider screening for NAFLD among safety-net patients with obesity even if DM has not developed. These results highlight the importance of directing efforts to reduce obesity in safety-net health systems and encourage researchers to further examine effect modification between health outcomes in such populations.

Keywords

obesity; biomeasures; epidemiology; state and local demography; race/ethnicity

*Correspondence details: Michael P. Huynh, MPH, 10960 Wilshire Blvd, Suite 1550, Los Angeles, CA 90024, mphuynh@ucla.edu.

Introduction

Obesity is a major cause of morbidity and mortality in the United States, and the epidemic has continued to increase in prevalence over time (Mokdad et al. 2003). Between 1976–1980, the percentage of people in the U.S. with obesity was 14.5 percent (Mokdad et al. 1999), and by 2011–2014, the prevalence among U.S. adults had risen to 36.5 percent (Ogden et al. 2015). Within the U.S., California has also experienced similar trends. Between 2001–2012, obesity in California increased among adults from 19.3 percent to 24.8 percent (Wolstein, Babey, and Diamant 2015).

The epidemic of obesity has been strongly associated with the epidemic of metabolic syndrome and subsequent manifestations of diabetes mellitus (DM), cardiovascular disease, and fatty liver disease (Paschos and Paletas 2009). Obesity is a major risk factor for nonalcoholic fatty liver disease (NAFLD), which involves fat deposition in the liver that may result from developed insulin resistance and increased body fat mass (Shifflet and Wu 2009). NAFLD is associated with various adverse health consequences, including cirrhosis, hepatocellular cancer, and impaired quality of life (Sanyal 2011, Golabi et al. 2016). It has become the most common liver disorder in the United States, accounting for 75 percent of chronic liver disease between 2005–2008 (Younossi et al. 2011), and the prevalence of NAFLD has steadily increased over time in step with obesity trends between 1988–2008. Type-2 DM has also been shown to be a risk factor for NAFLD and to be associated with higher Body Mass Index (BMI) (Younossi et al. 2011, Davila et al. 2005), suggesting a potential interaction between obesity, type-2 DM, and NAFLD.

It is unclear whether health consequences of the obesity prevalence in the general U.S. population are similar to that in "safety-net" health system patients—vulnerable groups of people who are uninsured, low-income, or qualified to receive care from public assistance programs (Burt and Arispe 2004). Because obesity disproportionately afflicts lower socioeconomic populations (Levine 2011, McLaren 2007), it could underlie important health disparities in the safety-net setting. Therefore, our objective was to describe obesity among patients in a large integrated safety-net system and evaluate the obesity burden as manifested by DM and NAFLD within the safety-net.

We hypothesized that DM would be an intermediate factor of the pathway between obesity and liver disease for safety-net patients. One first aim of this cross-sectional study was to determine the prevalence of obesity between May 2015 and June 2017 among patients in the San Francisco Health Network (SFHN). To better understand potential implications of the obesity burden in safety-net settings, we also examined whether BMI was associated with elevated liver enzyme levels, a marker of liver disease, in the SFHN population, after adjusting for demographic factors, smoking status, and health insurance. Finally, we investigated whether DM modified the relationship between BMI and liver enzyme levels.

Methods

Study Setting and Population

We used existing medical records data from patients in the SFHN, a population that primarily consists of low-income patients, the majority of whom access health care services through Medicaid, Medicare, and other public health assistance programs. We included patients ages 18 and older who had at least one encounter with their primary care clinic between May 2015 and June 2017.

The University of California, Berkeley Committee for the Protection of Human Subjects agreed to rely on the University of California, San Francisco Committee on Human Research approval of the protocol #17–22760 for this study.

Study Variables

We used the electronic health record system to retrieve patient demographic variables (i.e., sex, race/ethnicity, and age), measured height and weight, smoking status, and health insurance for the SFHN population. Laboratory variables that were extracted included HbA1c (glycated hemoglobin) percentage, AST (aspartate aminotransferase) and ALT (alanine aminotransferase) values in U/L (enzyme unit per liter), hepatitis B surface antigen status, and hepatitis C antibody status.

Obesity was defined using BMI, calculated as weight in pounds divided by height in inches squared, multiplied by 703 (CDC 2008). BMI classification was based on the World Health Organization (WHO) standard weight status categories for adults: underweight (< 18.5), normal (18.5–24.9), overweight (25.0–29.9), and obese (30.0) (Organization 2000b). The standard BMI cut points for overweight and obese BMI among adults may not apply universally to all groups of people, as these cutoffs do not correspond to similar levels for cardiometabolic outcomes, such as DM, in all racial/ethnic groups (Gallagher et al. 1996, Organization 2004). Due to these differences, the following weight status categories were used for adults who identified as Asian: underweight (< 18.5), normal (18.5–22.9), overweight (23.0–27.4), and obese (27.5) (Jih et al. 2014). In contrast, Pacific Islanders have significantly lower levels of body fat at higher BMI levels than Whites, which prompted our use of the following categories for Pacific Islanders: underweight (< 18.5), normal (18.5–25.9), overweight (26.0–31.9), and obese (32.0) (Swinburn et al. 1999, Organization 2000a). These race-specific categories were applied to the safety-net population.

From the SFHN electronic health records, the two variables that were used to determine DM status were physician diagnosis and HbA1c, or glycated hemoglobin, which reflects average blood glucose levels over the prior three months (Basu 2018). Because blood glucose tests can fluctuate, depending on whether a person ate a meal at a certain time before the test, HbA1c results were chosen as a more stable, consistent measurement for diagnosing DM (Committee 2009). Established cut points for HbA1c-defined diabetes were delineated as: no diabetes at baseline (HbA1c < 5.7 percent and did not have a DM diagnosis, or had no HbA1c results), pre-diabetes (HbA1c of 5.7–6.49 percent and no DM diagnosis), diabetes

(HbA1c of 6.5 – 7.99 percent or a diagnosis of DM), and uncontrolled diabetes (HbA1c of 8 percent or greater) (Association 2015).

The likelihood of NAFLD is increased in the setting of abnormal AST and ALT in the absence of viral hepatitis, absence of alcoholic pattern of liver injury, and presence of consistent abnormal levels of liver enzymes (Chalasani et al. 2018). Because ultrasound and imaging data were not available for all patients, we used abnormal AST and ALT in the absence of other typical etiologies of liver disease to establish suspected NAFLD (Clark, Brancati, and Diehl 2003, Huang et al. 2006, Hall and Cash 2012). To minimize the contribution of other health conditions to abnormal liver function tests, we excluded patients who tested positive for the Hepatitis B surface antigen (HBsAg) or Hepatitis C virus antibody (anti-HCV), as well as those with an AST/ALT ratio greater than 1 (Amarapurkar et al. 2007, Sorbi, Boynton, and Lindor 1999). Liver enzymes were considered abnormal if AST ≥ 37 U/L or ALT ≥ 40 U/L in men and AST or ALT ≥ 31 U/L in women (Ong, Pitts, and Younossi 2008). Patients were omitted if they had AST or ALT values greater than 500, as these marked elevations have been previously observed to indicate the presence of vital hepatitis (Gowda et al. 2009).

Statistical Analysis

To estimate the burden of obesity, we first calculated the proportions of the four BMI groups (underweight, normal, overweight, and obese) in the SFHN, as well as the subgroups within this population. We then used unadjusted and adjusted logistic regression models to examine whether BMI and DM were associated with elevated AST/ALT levels, with normal weight as the reference group. We adjusted for sex, age in years, race/ethnicity, smoking status, and health insurance as covariates in the analysis. Previous studies have found that smokers tend to have a lower body weight and an increased risk of developing NAFLD (Hu 2008, Hamabe et al. 2011, Liu et al. 2013), suggesting that smoking status acts as a confounder in the relationship between BMI and elevated AST/ALT levels. Additionally, eligibility criteria for certain health insurance plans, such as Medicaid and Medicare, require specific income levels or disability status, which can affect an individual's access to care and subsequent health outcomes (Levy and Meltzer 2004). To determine whether DM modified the effect of BMI on elevated AST/ALT levels, we conducted a likelihood ratio test to compare the model that included the main effects (BMI and DM) and their multiplicative interaction to the model including only the main effects for these variables. In order to compare subgroups with sufficient cell sizes, a binary variable for DM status was constructed to test for effect modification.

A total of 18,286 patients (27.9 percent of the total sample) were excluded from the SFHN dataset due to missing data on height and weight measurements, being less than 18 years of age, and missing demographic information between May 2015 and June 2017 (Figure 1). In examining the associations between BMI and elevated AST/ALT levels, a total of 15,372 patients (32.6 percent) were excluded due to missing AST and ALT data (Figure 1).

Results were considered statistically significant for p-values < 0.05. All analyses were conducted in Stata version 14.2.

Results

Study Population

Among the overall SFHN safety-net population (N=47,211), the majority of people were female (N=25,069), 50–64 years old (N=17,253), of Hispanic/Latino (N=13,267) or Asian (N=13,506) race/ethnicity, non-smokers (N=30,879), or Medicaid patients (N=24,281) (Table 1). In the total population, 32.2 percent of patients from SFHN were obese, while 36.4 percent were overweight. A higher prevalence of obesity was seen among Latino (42.0 percent), Black (38.2 percent), Native American (23.9 percent), and Pacific Islander (54.3 percent) ethnic groups.

Obesity, Liver Injury, and Diabetes Mellitus

For SFHN patients, greater BMI was associated with elevated AST and ALT levels (Table 2). While 9.0 percent of SFHN patients in the normal BMI category had elevated AST and ALT levels, this percentage increased to 16.4 percent in the overweight category and 24.0 percent in the obese category ($p < 0.001$). A dose-response relationship was also observed between BMI and DM (Table 2). For example, the percentage of SFHN patients who were categorized as having DM increased with greater BMI level: 7.9 percent for normal weight, 13.4 percent for overweight, and 20.0 percent for obese ($p < 0.001$). After stratifying by DM status, we found that patients with obesity and DM had the highest percentage of elevated AST/ALT levels at 24.8 percent (Table 3). With the exception of patients who were underweight, a dose-response relationship between BMI and elevated AST/ALT was observed in both patients without DM ($p < 0.001$) and with DM ($p < 0.001$) (Table 3).

In the first adjusted model examining the association between BMI and elevated AST and ALT levels, compared to normal weight individuals, those with obesity were most likely to have elevated liver enzyme levels [3.02 (2.76, 3.31)] (Supplementary Table 1). DM status was also associated with elevated liver enzyme levels, with the highest odds among people with uncontrolled diabetes in the unadjusted model [1.61 (1.44, 1.80)]. When both BMI and DM were included in an adjusted model, higher levels of BMI and DM were more associated with elevated liver enzyme levels, compared to normal weight and no DM as the reference group. Having an underweight BMI was not significantly associated with liver injury for any of the models.

Relative to normal weight individuals with no DM, those at higher BMI levels were more likely to have elevated liver enzyme levels (Supplementary Table 2). This relationship was compounded by DM, as individuals with both obesity and DM were most likely to have elevated AST and ALT levels, compared to individuals with normal weight and no DM [3.74 (3.32, 4.21)]. When examining stratum-specific groups, those without DM to those with DM, we found when comparing individuals who are obese to individuals with normal weight, the relationship between BMI and elevated liver enzyme levels was stronger among those without DM [3.07 (2.78, 3.39)] than among those with DM [2.18 (1.77, 2.69), p-interaction < 0.001].

Discussion

Using electronic medical records from SFHN, we observed results that revealed patients of Pacific Islander, Hispanic or Latino, and Black race/ethnicity were the groups most likely to have obesity within the San Francisco safety-net population. We also found that in SFHN, higher BMI levels were associated with DM status and elevated AST/ALT levels. Given the size of this study, our findings highlight the importance of considering cardio-metabolic outcomes in safety-net settings and health services that directly pertain to such clinical care.

Our findings are in agreement with those of previous studies of obesity among racial/ethnic groups in the United States. Prior data analyses from the Behavioral Risk Factor Surveillance System (BRFSS) in 2006–2008 showed that non-Hispanic blacks (35.7%) and Hispanics (28.7%) had a greater prevalence of obesity, compared with non-Hispanic whites (23.7%) (Pan et al. 2009). Additionally, the prevalence of obesity was found to be consistently higher among Pacific Islanders compared to other ethnic groups among adults in Hawaii and California (Mau et al. 2009). These earlier studies also pointed out the challenges of sufficient sampling of Pacific Islanders. Despite that they are one of the highest-risk groups for obesity, they comprise less than 1 percent of the population, which makes recruitment and sub-analysis of these individuals more difficult (Mau et al. 2009).

To our knowledge, our study was the first to investigate differences in the effects of BMI on elevated liver enzyme levels by DM. A surprising finding from our analysis was that obesity was more strongly related to abnormal liver enzymes in non-diabetic patients. This implies that DM may be a moderator in the relationship between BMI and liver enzymes, as our results suggest that obesity is a stronger factor of NAFLD in the absence of diabetes resistance. One potential explanation to our findings is that people with DM are already introduced to complications with insulin resistance (Bhatt and Smith 2015), so that obesity would not contribute as much additional magnitude to the development of fatty liver. In one study, interactions between BMI and DM indicated higher risks for cardiovascular mortality among individuals with lower BMI and DM than those with higher BMI and DM (Ma et al. 2012). Other researchers argue that the relationship between DM and NAFLD can be complex and bidirectional, suggesting that NAFLD may lead to DM (Anstee, Targher, and Day 2013). A cohort study used elevated ALT in the absence of excessive alcohol consumption to indicate the presence of NAFLD, and over 11 years of follow-up, subjects with NAFLD were more likely to be obese and had an increased risk of developing DM (Adams et al. 2009). At the same time, the presence of DM is considered to be a risk factor for NAFLD in current clinical management practices (Chalasani et al. 2018), which prompts further inquiry as to how these metabolic outcomes affect one another.

The strengths of our study include having a large sample size of a low-income population. We accounted for BMI measurement differences in Asian and Pacific Islander adults by applying appropriate cutoffs from WHO recommendations. All of the information for SFHN patients came from medical records, rather than self-reported data, which allowed us to use biomarker assessments for more objective results and reduce potential for information bias. This included the use of HbA1c, a reflection of elevated glucose over time, to define the presence of DM for greater reliability instead of blood glucose, which is more susceptible to

random fluctuations. When examining the relationship between BMI and elevated liver enzyme levels, we did not adjust for metabolic syndrome conditions, such as hypertension and dyslipidemia, because these factors have been proposed as intermediate variables between obesity and fatty liver, which decreased our potential for overadjustment bias (Hu 2008).

Limitations of our work include a lack of temporality between the health indicators of BMI, DM, and elevated AST/ALT, due to the cross-sectional nature of the study design. As a variable, BMI is an indirect measure of obesity, since it does not capture relevant estimates of body composition, which may lead to misclassification (Rothman 2008). Additionally, we could not distinguish between type-1 and type-2 DM status in our analysis. However, the prevalence of type-1 DM is relatively low in the United States, at a percentage up to 0.34 percent from the National Health and Nutrition Examination Survey (NHANES) in 1999–2010 (Menke et al. 2013), so all patients with DM who met the inclusion criteria were included in our analysis. We also acknowledge the possibility of unmeasured and residual confounding from factors that were not measured in this study. These include dietary and lifestyle factors, such as physical activity and degree of alcohol consumption, as the SFHN medical records did not have reliable or consistent data for such variables. Because our study was focused on the safety-net population of San Francisco, we cannot generalize these findings to the external U.S. population, and a disproportionate number of Hispanic or Latino patients in SFHN had missing data.

Our results suggest that health implications of obesity include an increased likelihood of NAFLD in safety-net health systems. These findings indicate potential avenues for future public health research by stratifying analyses for low-income groups to compare population differences. Our work also suggests the importance of state- and national-level surveys to capture adequate numbers of Pacific Islanders for research in obesity and metabolic health, since they are one of the highest-risk groups for obesity but remain underrepresented in public health research. Future work could examine mediating effects between these variables and detailed analysis of effect modification in cohort studies to test for causal relationships, as well as enhanced biomarker assessments for fatty liver in medical records. In practice, clinical providers could apply this research to enhance obesity management of patients. Our findings suggest that primary care physicians (PCP), for instance, should check liver enzymes in patients with obesity, even if, and perhaps particularly if, there is no presence of DM. Without this research, PCPs might be tempted to only screen for NAFLD in patients who also have DM, given the known association between NAFLD and insulin resistance. It is imperative that clinical practitioners also partner with local schools and communities to implement interventions that support structural changes in the environment, which have shown to be the most effective strategies for preventing obesity in populations of low socioeconomic position (Beauchamp et al. 2014). As the obesity epidemic continues to increase over time, it is essential to reduce its prevalence by directing public health interventions to vulnerable patient populations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Funding Details:

References

Adams LA, Waters OR, Knuiman MW, Elliott RR, and Olynyk JK. 2009 "NAFLD as a risk factor for the development of diabetes and the metabolic syndrome: an eleven-year follow-up study." Am J Gastroenterol 104 (4):861–7. doi: 10.1038/ajg.2009.67. [PubMed: 19293782]

Amarapurkar D, Kamani P, Patel N, Gupte P, Kumar P, Agal S, Baijal R, Lala S, Chaudhary D, and Deshpande A. 2007 "Prevalence of non-alcoholic fatty liver disease: population based study." Ann Hepatol 6 (3):161–3. [PubMed: 17786142]

Anstee Quentin M, Giovanni Targher, and Christopher P Day. 2013 "Progression of NAFLD to diabetes mellitus, cardiovascular disease or cirrhosis." Nature reviews Gastroenterology & hepatology 10 (6):330. [PubMed: 23507799]

Association American Diabetes. 2015 "Standards of medical care in diabetes-2015 abridged for primary care providers." Clin Diabetes 33 (2):97–111. doi: 10.2337/diaclin.33.2.97. [PubMed: 25897193]

Basu Rita. 2018 "Insulin Resistance & Prediabetes." National Institute of Diabetes and Digestive and Kidney Diseases

Beauchamp A, Backholer K, Magliano D, and Peeters A. 2014 "The effect of obesity prevention interventions according to socioeconomic position: a systematic review." Obesity reviews 15 (7):541–554. [PubMed: 24629126]

Bhatt Harikrashna B, and Robert J Smith. 2015 "Fatty liver disease in diabetes mellitus." Hepatobiliary surgery and nutrition 4 (2):101. [PubMed: 26005676]

Burt Catharine W, and Irma E Arispe. 2004 "Characteristics of emergency departments serving high volumes of safety-net patients; United States, 2000."

CDC. 2008 "About Adult BMI." Department of Health and Human Services, Centers for Disease Control and Prevention.

Chalasani Naga, Younossi Zobair, Lavine Joel E, Charlton Michael, Cusi Kenneth, Rinella Mary, Harrison Stephen A, Brunt Elizabeth M, and Sanyal Arun J. 2018 "The diagnosis and management of nonalcoholic fatty liver disease: practice guidance from the American Association for the Study of Liver Diseases." Hepatology 67 (1):328–357. [PubMed: 28714183]

Clark Jeanne M, Brancati Frederick L, and Diehl Anna Mae. 2003 "The prevalence and etiology of elevated aminotransferase levels in the United States." The American Journal of Gastroenterology 98 (5):960–967. [PubMed: 12809815]

Committee, International Expert. 2009 "International Expert Committee report on the role of the A1C assay in the diagnosis of diabetes." Diabetes care 32 (7):1327–1334. [PubMed: 19502545]

Davila JA, Morgan RO, Shaib Y, McGlynn KA, and El-Serag HB. 2005 "Diabetes increases the risk of hepatocellular carcinoma in the United States: a population based case control study." Gut 54 (4):533–539. [PubMed: 15753540]

Gallagher Dympna, Visser Marjolein, Sepulveda Dennis, Pierson Richard N, Tamara Harris, and Heymsfield Steven B. 1996 "How useful is body mass index for comparison of body fatness across age, sex, and ethnic groups?" American journal of epidemiology 143 (3):228–239. [PubMed: 8561156]

Golabi Pegah, Otgonsuren Munkhzul, Cable Rebecca, Felix Sean, Koenig Aaron, Sayiner Mehmet, and Younossi Zobair M. 2016 "Non-alcoholic fatty liver disease (NAFLD) is associated with impairment of health related quality of life (HRQOL)." Health quality of life outcomes 14 (1):18. [PubMed: 26860700]

Gowda Shivaraj, Desai Prakash B, Hull Vinayak V, Math Avinash A K, Vernekar Sonal N, and Kulkarni Shruthi S. 2009 "A review on laboratory liver function tests." The Pan african medical journal 3.

Hall P, and Cash J. 2012 "What is the real function of the liver 'function' tests?" Ulster Med J 81 (1):30–6. [PubMed: 23536736]

Hamabe A, Uto H, Imamura Y, Kusano K, Mawatari S, Kumagai K, Kure T, Tamai T, Moriuchi A, Sakiyama T, Oketani M, Ido A, and Tsubouchi H. 2011 "Impact of cigarette smoking on onset of nonalcoholic fatty liver disease over a 10-year period." J Gastroenterol 46 (6):769–78. doi: 10.1007/s00535-011-0376-z. [PubMed: 21302121]

Hu Frank. 2008 Obesity epidemiology: Oxford University Press.

Huang Xing-Jiu, Choi Yang-Kyu, Im Hyung-Soon, Yarimaga Oktay, Yoon Euisik, and Kim Hak-Sung. 2006 "Aspartate aminotransferase (AST/GOT) and alanine aminotransferase (ALT/GPT) detection techniques." Sensors 6 (7):756–782.

Jih Jane, Mukherjea Arnab, Vittinghoff Eric, Nguyen Tung T, Tsoh Janice Y, Fukuoka Yoshimi, Bender Melinda S, Tseng Winston, and Kanaya Alka M. 2014 "Using appropriate body mass index cut points for overweight and obesity among Asian Americans." Preventive medicine 65:1–6. [PubMed: 24736092]

Levine James A. 2011 Poverty and obesity in the US. Am Diabetes Assoc.

Levy Helen, and Meltzer David. 2004 "What do we really know about whether health insurance affects health." Health policy and the uninsured:179–204.

Liu Y, Dai M, Bi Y, Xu M, Xu Y, Li M, Wang T, Huang F, Xu B, Zhang J, Li X, Wang W, and Ning G. 2013 "Active smoking, passive smoking, and risk of nonalcoholic fatty liver disease (NAFLD): a population-based study in China." J Epidemiol 23 (2):115–21. [PubMed: 23399520]

Ma SH, Park BY, Yang JJ, Jung EJ, Yeo Y, Whang Y, Chang SH, Shin HR, Kang D, Yoo KY, and Park SK. 2012 "Interaction of body mass index and diabetes as modifiers of cardiovascular mortality in a cohort study." J Prev Med Public Health 45 (6):394–401. doi: 10.3961/jpmph.2012.45.6.394. [PubMed: 23230470]

Mau MK, Sinclair K, Saito EP, Baumhofer KN, and Kaholokula JK. 2009 "Cardiometabolic health disparities in native Hawaiians and other Pacific Islanders." Epidemiol Rev 31:113–29. doi: 10.1093/ajerev/mxp004. [PubMed: 19531765]

McLaren Lindsay. 2007 "Socioeconomic status and obesity." Epidemiologic reviews 29 (1):29–48. [PubMed: 17478442]

Menke A, Orchard TJ, Imperatore G, Bullard KM, Mayer-Davis E, and Cowie CC. 2013 "The prevalence of type 1 diabetes in the United States." Epidemiology 24 (5):773–4. doi: 10.1097/EDE.0b013e31829ef01a. [PubMed: 23903880]

Mokdad AH, Ford ES, Bowman BA, Dietz WH, Vinicor F, Bales VS, and Marks JS. 2003 "Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001." Jama 289 (1):76–9. [PubMed: 12503980]

Mokdad AH, Serdula MK, Dietz WH, Bowman BA, Marks JS, and Koplan JP. 1999 "The spread of the obesity epidemic in the united states, 1991–1998." JAMA 282 (16):1519–1522. doi: 10.1001/jama.282.16.1519. [PubMed: 10546690]

Ogden Cynthia L, Carroll Margaret D, Fryar Cheryl D, and Flegal Katherine M. 2015 Prevalence of obesity among adults and youth: United States, 2011–2014: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics.

Ong JP, Pitts A, and Younossi ZM. 2008 "Increased overall mortality and liver-related mortality in non-alcoholic fatty liver disease." J Hepatol 49 (4):608–12. doi: 10.1016/j.jhep.2008.06.018. [PubMed: 18682312]

Organization World Health. 2000a The Asia-Pacific perspective: redefining obesity and its treatment. Sydney: Health Communications Australia.

Organization, World Health. 2000b Obesity: preventing and managing the global epidemic: World Health Organization.

Organization World Health. 2004 "Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies." Lancet 363 (9403):157–63. doi: 10.1016/s0140-6736(03)15268-3. [PubMed: 14726171]

Pan L, Galuska DA, Sherry B, Hunter AS, Rutledge GE, Dietz WH, and Balluz LS. 2009 "Differences in prevalence of obesity among black, white, and hispanic adults-United States, 2006–2008." Morbidity Mortality Weekly Report 58 (27):740–744. [PubMed: 19609247]

Paschos P, and Paletas K. 2009 "Non alcoholic fatty liver disease and metabolic syndrome." Hippokratia 13 (1):9. [PubMed: 19240815]

Rothman KJ 2008 "BMI-related errors in the measurement of obesity." Int J Obes (Lond) 32 Suppl 3:S56–9. doi: 10.1038/ijo.2008.87. [PubMed: 18695655]

Sanyal Arun J. 2011 "NASH: a global health problem." Hepatology Research 41 (7):670–674. [PubMed: 21711426]

Shifflet Allison, and Wu George Y. 2009 "Non-alcoholic steatohepatitis: an overview." Journal of the Formosan Medical Association 108 (1):4–12. [PubMed: 19181602]

Sorbi D, Boynton J, and Lindor KD. 1999 "The ratio of aspartate aminotransferase to alanine aminotransferase: potential value in differentiating nonalcoholic steatohepatitis from alcoholic liver disease." Am J Gastroenterol 94 (4):1018–22. doi: 10.1111/j.1572-0241.1999.01006.x. [PubMed: 10201476]

Swinburn BA, Ley SJ, Carmichael HE, and Plank LD. 1999 "Body size and composition in Polynesians." International journal of obesity 23 (11):1178. [PubMed: 10578208]

Wolstein Joelle, Babey Susan H, and Diamant Allison L. 2015 "Obesity in California." UCLA Center for Health Policy Research: Los Angeles.

Younossi Zobair M, Stepanova Maria, Afendy Mariam, Fang Yun, Younossi Youssef, Mir Hesham, and Srishord Manirath. 2011 "Changes in the prevalence of the most common causes of chronic liver diseases in the United States from 1988 to 2008." Clinical Gastroenterology and Hepatology 9 (6):524–530.e1. [PubMed: 21440669]
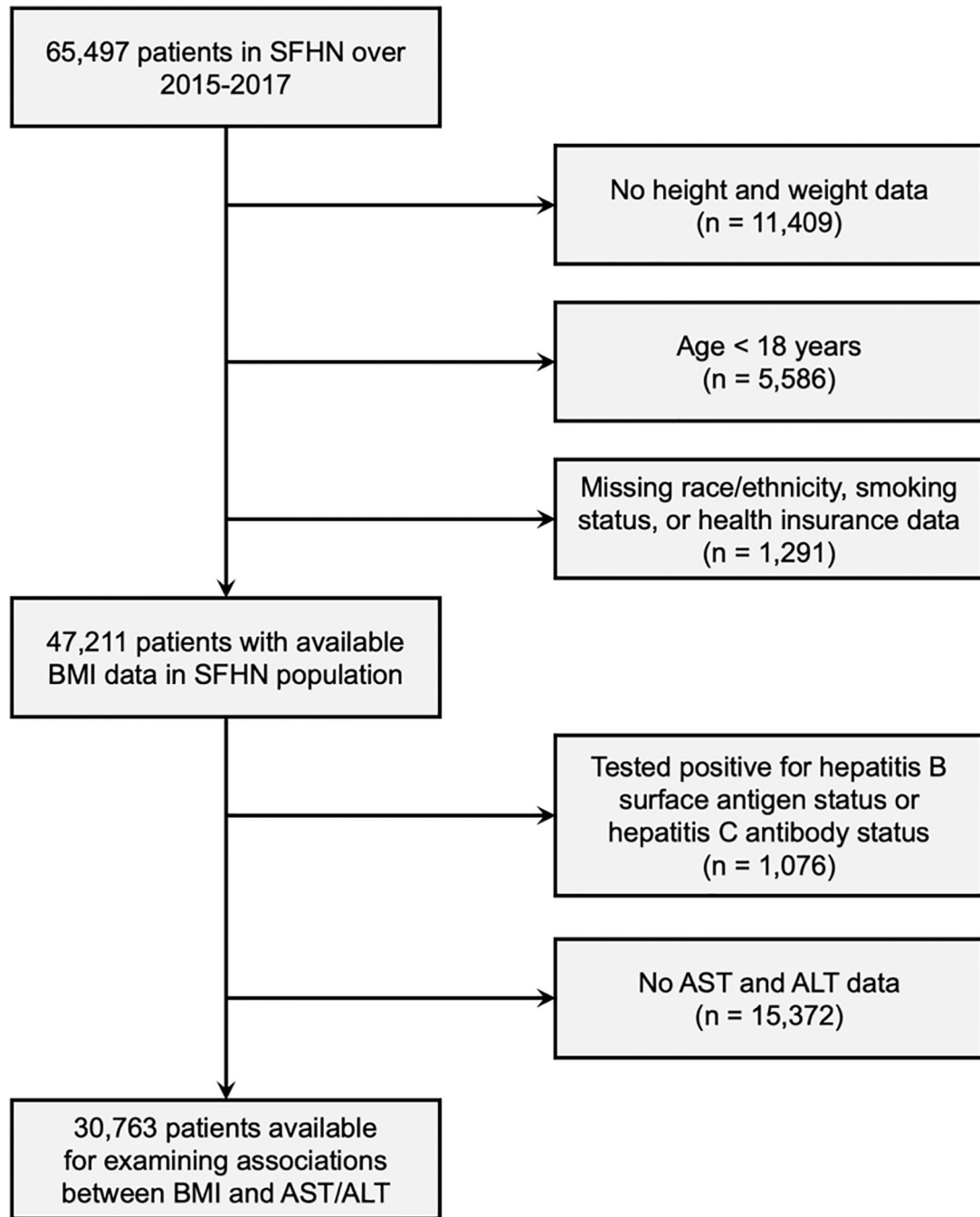
Figure 1.
Flow diagram of patients in the San Francisco Health Network (SFHN) included in the study

Table 1.

Demographic characteristics and BMI groups of patients in the San Francisco Health Network (SFHN)

	Underweight %	Normal %	Overweight %	Obese %	N
All	2.2	29.2	36.4	32.2	47,211
Sex					
Male	1.8	29.5	39.6	29.1	22,142
Female	2.6	28.9	33.6	34.9	25,069
Age in Years					
18–34	2.5	37.9	31.8	27.8	9,523
35–49	1.2	25.9	35.4	37.5	11,371
50–64	2.2	27.0	38.0	32.7	17,253
65+	3.1	28.3	39.6	29.1	9,064
Race/Ethnicity					
White	2.6	37.3	32.7	27.5	8,397
Hispanic or Latino	0.7	20.3	36.9	42.0	13,267
Black or African American	2.7	30.0	29.1	38.2	6,870
Asian	3.1	31.2	43.5	22.2	13,506
Native Hawaiian or Pacific Islander	1.0	17.6	27.1	54.3	387
Native American or Alaska Native	2.9	42.2	31.1	23.9	306
Other or Unknown	2.4	33.2	33.1	31.4	4,478
Smoking Status					
Never	1.9	27.6	37.2	33.3	30,879
Past	2.2	27.2	37.0	33.5	6,479
Current	3.3	35.6	32.9	28.2	9,249
Passive	2.8	35.1	42.2	19.9	604
Health Insurance					
Commercial	1.4	32.4	36.4	29.8	352
Medicaid	2.3	31.3	34.2	32.3	24,281
Medicare	3.0	27.6	37.7	31.7	8,046
Public [a]	1.4	25.4	40.7	32.5	12,186
Uninsured	2.7	32.3	33.3	31.8	2,346

[a]Public includes the following health insurance plans: Healthy Kids, Healthy San Francisco, and Healthy Worker.

Table 2.

Clinical outcomes by BMI group of patients in the San Francisco Health Network (SFHN)

	N	No Elevated AST & ALT %	Elevated AST & ALT %	No Diabetes %	Prediabetes %	Diabetes %	Uncontrolled Diabetes %
All	30,763	83.2	16.8	58.4	20.6	14.1	7.0
BMI Group							
Underweight	662	92.6	7.4	77.5	14.5	6.2	1.8
Normal	8,309	91.0	9.0	72.4	15.7	7.9	4.1
Overweight	11,069	83.6	16.4	58.8	21.6	13.4	6.2
Obese	10,723	76.0	24.0	45.9	23.7	20.0	10.4

Chi-square analysis was used to examine the association between BMI and elevated AST and ALT levels: $X^2 = 799.11$, $p < 0.001$

Chi-square analysis was used to examine the association between BMI and DM status: $X^2 = 1.6 \times 10^3$, $p < 0.001$

Table 3.

Elevated AST and ALT levels by diabetes mellitus status and BMI group of patients in the San Francisco Health Network (SFHN)

| | No Diabetes | | Diabetes | |
| | | Elevated AST & ALT | | Elevated AST & ALT |
BMI	N	%	N	%
Underweight	609	6.6	53	17.0
Normal	7,313	8.6	996	12.1
Overweight	8,897	16.2	2,172	17.1
Obese	7,461	23.6	3,262	24.8

Chi-square analysis was used to examine the association between the following:

BMI and elevated AST and ALT levels among patients without diabetes: $X^2 = 665.52$, $p < 0.001$

BMI and elevated AST and ALT levels among patients with diabetes: $X^2 = 96.31$, $p < 0.001$