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Association between obesity and non-alcoholic fatty liver disease (NAFLD) in adults aged 18 years and above – data from NHANES 2017–2018

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Abstract

Introduction and Objective. Studies have Increasingly addressed a possible association between obesity and non-alcoholic fatty liver disease (NAFLD), although it remains an issue of controversy. The aim in the study is to determine an association between obesity and NAFLD among 1,120 adults ≥ 18 years of age, through a cross-sectional analysis of the National Health and Nutrition Examination Survey from 2017–2018.

Materials and Method. Data analyzed in the study were obtained from the NHANES database which provides multitudinous information concerning the health and nutrition of the general population in the USA. Data obtained from the NHANES surveys, conducted between 2017–2018, was utilised. A multivariable logistic regression model was used to determine the association between obesity and NAFLD.

Results. Of the 1,120 participants enrolled, 434 (38.75%) had NAFLD. There was a positive association between obesity and NAFLD in all three models: model 1 – OR=1.80, 95% CI: 1.39–2.21; model 2 – OR=1.87, 95% CI: 1.41–2.33; model 3 – OR=1.76, 95% CI: 1.29–2.22). There was a positive association between BMI and NAFLD in all three models (model 1: OR=1.18, 95% CI: 1.13–1.23; model 2: OR=1.19, 95% CI: 1.14–1.25; model 3: OR=1.18, 95% CI: 1.12–1.24).

Conclusions. The study findings confirm a strong association between obesity and NAFLD in individuals aged 18 and older, leading to a significantly elevated risk of NAFLD among those who are obese. Therefore, it is concluded that prompt interventions by healthcare professionals are necessary to facilitate weight reduction and support the recovery of health in obese patients.

Key words

NAFLD, NHANES, adults, obesity, association

INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) constitutes a widespread chronic hepatic disorder characterized by excessive lipid accumulation, believed to stem from abnormal fat accumulation in the liver due to metabolic syndrome, obesity, and other related factors [1, 2]. Recently, NAFLD constitutes a pivotal contributor to the global burden of hepatic pathology. Epidemiological surveys from various regions have consistently revealed a high prevalence of NAFLD, notably in Western countries and certain Asian regions (e.g., China, Japan) [3–5]. Moreover, the escalating global obesity rates have contributed to an increasing prevalence of NAFLD [6]. Hence, NAFLD has evolved into a critical global public health concern, bearing significant clinical implications for both its prevention and treatment.

Obesity stands as one of the most closely associated metabolic diseases with NAFLD. Research indicates that obesity contributes to an elevated incidence of various metabolic disorders [7]. Not only are individuals with obesity more prone to NAFLD, but the progression and severity of their NAFLD are also typically more pronounced [8]. The link between obesity and NAFLD extends beyond excess body weight, encompassing diverse fat distribution and abnormal metabolic function [9]. Obesity triggers abnormal fat metabolism in the liver, hastening the accretion of fat in the liver and thus fostering the onset and progression of NAFLD [10, 11].

Studying the correlation between NAFLD and obesity enhances our understanding of the link between these two serious diseases. This is not only crucial for early detection and prevention of NAFLD, but also offers guidance for intervening and treating obesity. In-depth research on the association between NAFLD and obesity could establish a scientific foundation for developing personalized intervention strategies, advancing the management and treatment of obesity and related metabolic disorders. Thus, investigating the correlation between NAFLD and obesity holds significant guidance for modern medical research and clinical practice. This study analyzes obesity-NAFLD associations in adults (≥18 years) using covariate-adjusted NHANES 2017–2018 data.

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MATERIALS AND METHOD

Study population. The dataset utilized in this study was derived from the NHANES database, a nationally representative repository containing extensive health and nutritional information on residents of the USA. Analysis incorporated datasets from the 2017–2018 survey cycles, with all participants providing written informed consent for anonymous data usage. The study protocols received ethical approval from the National Center for Health Statistics Institutional Review Board.

The study cohort encompassed adults aged ≥18 years, with 9,254 eligible individuals listed in the NHANES database during the 2017–2018 cycles. participants aged less than 18 (n=3398), were excluded, as well as those with hepatitis B surface antibody positive,(?) and missing data (n=2,033), HCV antibody/HCV RNA positive and missing data (n=128), and excessive consumption of alcohol, and missing data (n=2437). Participants lacking valid measurements for fatty liver disease (n=83), glycohaemoglobin (n=3), BMI (n=7), or marital status (n=45) were also excluded. Following application of the exclusion criteria, 1,120 individuals comprised the final analytical sample (Fig. 1).

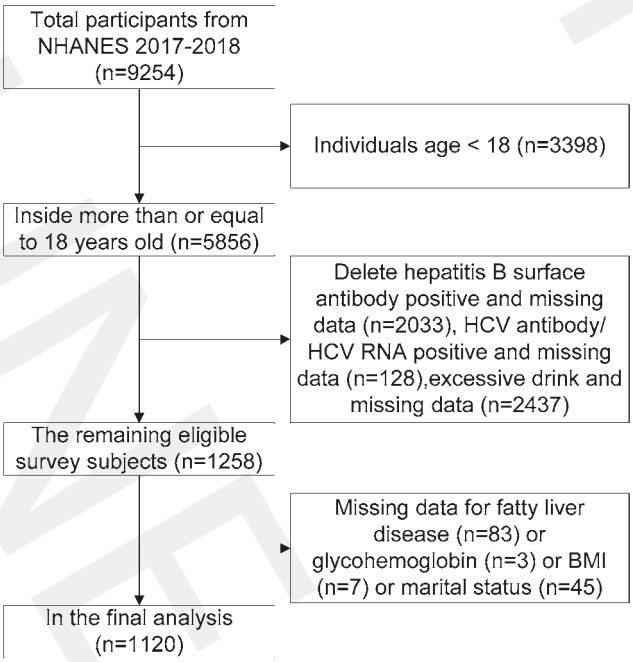

Figure 1. Flow chart of sample selection

The data collection protocols employed in the NHANES study are comprehensively outlined on the CDC's official platform (www.cdc.gov/nchs/nhanes/), while this section presents an abridged synopsis. Medical and physical examination, as well as the collection of blood samples, were conducted in the Mobile Examination Center. Demographic, health-related, and life style-related data were collected through in-home interviews.

Obesity status. BMI (kg/m^2) was derived by dividing body weight (kg) by height (m) squared. Participants with BMI ≥30 met the obesity classification threshold, while those below this value were categorized as non-obese [12].

NAFLD. The 2017–2018 NHANES employed vibration-controlled transient elastography (VCTE) to assess hepatic fibrosis via liver stiffness measurements (LSM), and quantify steatosis through controlled attenuation parameters (CAP) [13]. Examinations were conducted using the FibroScan® 502 V2 Touch system (Echosens, MA) with probe selection (medium/extra-large) guided by real-time automated recommendations, requiring ≥10 valid measurements per subject [14]. LSM values (1.5–75 kPa) and CAP scores (100–400 dB/m) were automatically calculated as median values with IQR, where elevated LSM indicates progressive fibrosis and increased CAP reflects higher hepatic lipid deposition [14, 15]. Hepatic steatosis was confirmed at CAP ≥285 dB/m (80% sensitivity, 77% specificity) [16], while NAFLD diagnosis excluded secondary causes of chronic liver disease and heavy alcohol intake (≥2 daily drinks for males, ≥1 for females) [17, 18].

Covariates. Data on potential confounding variables was sourced from the NHANES database. Quantitative measures comprised age, body mass index, and glycohaemoglobin levels. Nominal factors encompassed gender, ethnicity, marital status, and educational attainment. The NHANES portal (http://www.cdc.gov/nchs/nhanes/) provides methodological specifics regarding NAFLD classification, obesity assessment, and covariate collection protocols. Diabetes was assessed using glycohaemoglobin, if glycohaemoglobin was greater than or equal to 6.5%, the participant was categorized as having diabetes [19].

Statistical analysis. Multivariable logistic regression analysis assessed associations between obesity/BMI and NAFLD, reporting odds ratios (OR) with 95% confidence intervals (CI). Three analytical models were developed aligned with STROBE epidemiological guidelines [20]: Model 1 – unadjusted for covariates; Model 2 – adjusted for age, gender, ethnicity; and Model 3 – fully adjusted with all covariates. Statistical computations employed R 4.3.2, establishing significance at $p<0.05$.

RESULTS

Participants characteristics. Table 1 details participant baseline characteristics. Among the 1,120 subjects, 434 (38.75%) exhibited NAFLD and exhibited elevated age, BMI, and higher marriage/cohabitation rates, compared to non-NAFLD counterparts.

Associations of obesity with NAFLD. Table 2 demonstrates significant associations between obesity/BMI and NAFLD across all models. Initial unadjusted analyses revealed robust correlations (Model 1: obesity OR=1.80, 95%CI 1.39–2.21; BMI OR=1.18, 95%CI 1.13–1.23). These associations persisted after adjusting for age, gender, and ethnicity (Model 2: obesity OR=1.87, 1.41–2.33; BMI OR=1.19, 1.14–1.25) and remained significant in fully-adjusted models (Model 3: obesity OR=1.76, 1.29–2.22; BMI OR=1.18, 1.12–1.24). Consistent patterns emerged across gender subgroups.

Table 1. Weighted characteristics of study sample with and without NAFLD

Variables	NAFLD (n=434)	Non-NAFLD (n=686)	P
Age (years)	55.27 ± 0.89	49.88 ± 1.35	0.001
Gender (%)			0.035
Male	36.7	24.8	
Female	63.3	75.2	
Race (%)			0.036
Non-Hispanic White	73.4	67.4	
Non-Hispanic Black	7.9	12.0	
Mexican American	7.2	5.7	
Other race	11.5	14.9	
Educational level (%)			0.816
Less than high school	7.5	7.1	
High school	29.9	27.0	
More than high school	62.6	65.9	
Marital status			0.028
Married/Living with Partner	72.9	63.1	
Widowed/Divorced/Separated	18.9	21.9	
Never married	8.2	15.0	
Missing			
Body Mass Index (kg/m^2)	34.69 ± 0.65	27.45 ± 0.35	<0.001
Obesity (kg/m^2)			<0.001
Less than 30	28.5	70.7	
More than or equal to 30	71.5	29.3	
Diabetes			<0.001
Glycohaemoglobin less than 6.5%	77.0	96.8	
Glycohaemoglobin more than or equal to 6.5%	23.0	3.2	

Mean ± SD for continuous variables – P-value calculated by weighted linear regression model. % for categorical variables – P-value calculated by weighted chi-square test

Table 2. Associations between body mass index or obesity and NAFLD

	Model 1 OR (95% CI, P)	Model 2 OR (95% CI, P)	Model 3 OR (95% CI, P)
Total			
BMI (kg/m^2)	1.18 (1.13, 1.23) <0.001	1.19 (1.14, 1.25) <0.001	1.18 (1.12, 1.24) <0.001
Non-obesity	Ref.	Ref.	Ref.
Obesity	1.80 (1.39, 2.21) <0.001	1.87 (1.41, 2.33) <0.001	1.76 (1.29, 2.22) <0.001
Male			
BMI (kg/m^2)	1.27 (1.13, 1.43) <0.001	1.27 (1.13, 1.44) <0.001	1.30 (1.16, 1.45) <0.001
Non-obesity	Ref.	Ref.	Ref.
Obesity	2.08 (1.23, 2.93) <0.001	2.14 (1.21, 3.06) <0.001	2.23 (1.29, 3.17) <0.001
Female			
BMI (kg/m^2)	1.17 (1.12, 1.22) <0.001	1.17 (1.12, 1.23) <0.001	1.16 (1.10, 1.22) <0.001
Non-obesity	Ref.	Ref.	Ref.
Obesity	1.73 (1.29, 2.18) <0.001	1.75 (1.28, 2.23) <0.001	1.58 (1.09, 2.07) <0.001

Model 1 – no covariates were adjusted; Model 2 – age, gender, race were adjusted; Model 3 – age, gender, race, educational level, marital status and glycohaemoglobin were adjusted; BMI – body mass index

DISCUSSION

The study analyzes obesity-NAFLD associations in adults (≥18 years) using NHANES 2017–2018 data. NAFLD, defined by excessive hepatic fat deposition without significant alcohol exposure, represents a spectrum of metabolic liver diseases, from benign steatosis to progressive NASH, with potential progression to cirrhosis and hepatocellular carcinoma [21]. Generally, NAFLD is closely linked to such metabolic disorders as metabolic syndrome, obesity, hypertension, hyperlipidaemia, and diabetes [6, 22]. In clinical practice, the diagnosis of NAFLD primarily relies on liver ultrasound, CT or MRI scans, and liver tissue biopsy [23]. NAFLD constitutes a leading etiological factor in the worldwide burden of liver disease, highlighting its critical public health priority for preventive and therapeutic interventions [24].

Obesity is characterized by the excessive accumulation of body fat, leading to a body weight that exceeds the ideal range. Assessment and evaluation of obesity commonly involve the BMI. According to the WHO criteria, obesity is defined as BMI ≥30 kg/m^2, with overweight classified as 25–29.9 kg/m^2[12]. Additionally, obesity encompasses not only excessive body weight, but also the distribution of adipose tissue within the body and the biological traits of fat cells. Furthermore, obesity is frequently associated with various metabolic conditions, including hypertension, hyperlipidaemia, and diabetes, and elevates the risk of cardiovascular and cerebrovascular diseases, as well as neoplasms and musculoskeletal disorders [7, 25]. Given these circumstances, obesity has evolved into a global public health concern, underscoring its clinical importance in terms of prevention and management.

The findings of the current study indicate a significant association between obesity and NAFLD in adults aged 18 and above, with the correlation remaining statistically significant even after adjusting for age, gender, race, and other covariates. Notably, the prevalence of NAFLD is higher in the obese population, consistent with findings from prior research [26, 27]. This research is important in enhancing understanding of the association between these variables and the potential factors contributing to NAFLD development in individuals with obesity. Furthermore, it provides detailed insights into the association among adults aged 18 and above, which can assist in formulating targeted intervention strategies for preventing NAFLD in this demographic group.

The association between obesity and NAFLD encompasses a multitude of potential mechanisms which underscore their intricate interrelations. Obesity elicits an expansion in both the size and quantity of adipocytes, consequently heightening overall fat storage. Surplus adipose tissue can instigate lipid metabolism irregularities, encompassing disturbances in processes such as fat assimilation, synthesis, oxidation, and excretion, thus fostering the onset and progression of NAFLD [28].

Obesity can precipitate insulin resistance, further fostering hepatic fat accumulation and NAFLD development [29, 30]. Additionally, obesity has the potential to disrupt the balance of gut microbiota, augmenting the presence of deleterious bacteria while reducing beneficial strains, hence promoting the incidence of NAFLD [31]. Moreover, obesity triggers the generation of inflammatory mediators, including C-reactive protein (CRP) and tumour necrosis factor-α (TNF-α),

exacerbating hepatic inflammatory responses and fostering the occurrence and progression of NAFLD [32].

The intricate relationship between obesity and NAFLD involves various mechanisms. Obesity can trigger metabolic abnormalities such as insulin resistance and dyslipidaemia, leading tothe excessive accretion of fat in the body, thereby exacerbating the development of NAFLD [25, 33]. Furthermore, obesity stimulates inflammatory responses and the release of cytokines, thus promoting liver inflammation and accelerating the progression of NAFLD [32]. Changes in the gut microbiota due to obesity can impact food absorption and metabolism in the gut, consequently influencing the incidence and expansion of NAFLD [31]. Obesity-related dysfunctional adipose redistribution, particularly abdominal fat deposition, pathogenically links to NAFLD development [28]. Certain genetic variations have also been linked to the occurrence of both obesity and NAFLD, establishing a genetic basis for their correlation [34].

Limitations of the study. Initially, the utilization of cross-sectional data from NHANES 2017–2018 precluded the conduct of a longitudinal study, thereby impeding determination of the causal relationship between obesity and NAFLD. Moreover, the selection of participants was restricted to adults aged 18 and above, thereby restricting the generalizability of the findings to the paediatric population. Furthermore, the study failed to account for other potential influencing factors, including physical activity, socio-economic status, smoking, and alcohol consumption, potentially limiting the applicability of analysis of the results. In addition, the data were solely sourced from public databases, and the accessible information is limited. Therefore, in the future, large-sample, prospective, and multi-center studies will be conducted to collect more epidemiological and clinicopathological data, aiming to further analyze and validate the conclusions of the study undertaken.

CONCLUSIONS

The study findings confirm a strong association between obesity and NAFLD in individuals aged 18 and older, leading to a significantly elevated risk of NAFLD among those who are obese. Therefore, it is concluded that prompt interventions by healthcare professionals are necessary to facilitate weight reduction and support the recovery of health in obese patients. Moreover, it is imperative for governments and public health organizations to prioritize obesity as an issue, implement appropriate measures, devise relevant policies, and advocate for the widespread adoption of healthy lifestyles in order to decrease the prevalence of obesity and NAFLD.

In essence, the study furnishes scientific evidence regarding the connection between obesity and NAFLD in adults aged 18 and above, emphasizing the importance of interventions and policies targeting the reduction of adult obesity and NAFLD. Subsequent research endeavors could investigate the mechanisms underlying the correlation between obesity and NAFLD, as well as assess the effectiveness of diverse intervention strategies for the prevention and treatment of these conditions.

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