Research on the Link Between Obesity and Cardiovascular Disease Risk

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Abstract. This research examines the complex relationship between obesity and the risk of cardiovascular disease (CVD). Using a comprehensive dataset, we conducted a one-sample t-test to determine the significance of 17 variables related to obesity. The results showed that all variables, including demographic, lifestyle, and behavioral factors, significantly deviated from zero, indicating their importance in obesity studies. An ordinal logistic regression analysis was conducted to explore the predictors of obesity classification. The analysis revealed complex interactions among various factors. The study highlights the multifactorial nature of obesity and its impact on cardiovascular disease (CVD), providing insights for healthcare policies and future research directions. It particularly emphasizes the need to understand the role of less significant factors and the influence of cultural, environmental, and socioeconomic contexts on obesity. The implications of this research are significant for both the medical field and public health policy. The findings emphasize the intricate interplay of multiple factors in obesity and its correlation with the risk of cardiovascular disease. This comprehension could lead to more effective and targeted interventions, preventive measures, and policy formulations. For future research, it opens avenues to explore the role of less significant factors and to understand the impact of cultural, environmental, and socio-economic contexts on obesity. This study highlights the necessity of a comprehensive strategy to tackle the worldwide issue of obesity and its related health hazards.

Keywords: Obesity; cardiovascular disease; ordinal logistic regression.

1. Introduction

Obesity has become a global health crisis affecting both wealthy and poor nations. It is the result of multiple factors like unhealthy diets, sedentary lifestyles, and genetic predispositions [1]. Excessive fat accumulation characterizes obesity and threatens health. From 1975 to 2016, obesity rates tripled worldwide, with over 650 million obese adults. In 2016, approximately 39% of adults over 18 were overweight and 13% were obese [2]. In 2020 and 2016 respectively, 39 million children under 5 and over 340 million children and adolescents aged 5-19 were obese. The WHO predicts over half of the world's adults are overweight or obese. Obesity rates continue rising rapidly in many regions, and current trends forecast 18% of men and over 21% of women will be obese by 2025, heavily burdening individuals, societies, and healthcare [3]. Increased consumption of high-calorie, nutrient-poor foods and physical inactivity have been linked to the growth in obesity. Imbalances between calorie intake and expenditure, influenced by more processed and sugary foods and less activity, drive the increase in obesity and overweight. These shifts often result from development-related environmental and social changes combined with inadequate health, agriculture, transportation, urban planning, and education policies. The obesity epidemic has critical health implications, leading to various chronic illnesses like diabetes, heart disease, and certain cancers [4].

The development of cardiovascular disease (CVD), which includes heart disease and stroke, is closely linked to obesity and is the leading cause of death worldwide. The relationship between obesity and CVD is complex and multifaceted, including metabolic syndrome, hypertension, diabetes mellitus, and many other factors that are influenced by obesity. The impact of obesity on cardiovascular health is much more than just being overweight. Adipose tissue plays a key role in cardiovascular pathology, especially in dysfunctional states. It is an active endocrine organ that secretes adipokines, which have important effects on inflammation, vascular function and homeostasis in the body [5]. These adipokines create an inflammatory environment conducive to

atherosclerosis, hypertension, and other cardiovascular diseases [6], thereby affecting human cardiovascular health. In addition, the indirect effect of obesity on CVD is demonstrated by its association with the metabolic syndrome, which includes insulin resistance, hyperglycemia, dyslipidemia, and hypertension [7]. The interaction between obesity-related metabolic disorders and cardiovascular health involves complex biochemical and physiological changes that exacerbate endothelial dysfunction and alter cardiac structure [8]. Together, these conditions increase the risk of developing cardiovascular disease, and this paper aims to delve into this complex relationship by exploring how obesity contributes to cardiovascular risk, the mechanisms underlying this link, and the implications for prevention and treatment. This review draws on recent research and data analysis to provide a comprehensive overview of this important public health issue.

The "Guideline for the Management of Overweight and Obesity in Adults" defines overweight as having a body mass index (BMI) ≥25 and <30 kg/m2 and obesity as a BMI ≥30 kg/m2 using World Health Organization criteria [9]. BMI strongly correlates with body fat percentage across different populations, though its ability to predict an individual's body fat is limited and varies widely by sex, age, and ethnicity. Location-specific BMI cutoffs have been set for Asian subgroups (e.g. China) at 24 kg/m2 for overweight and 28 kg/m2 for obesity. Waist circumference, signaling abdominal obesity, has more impact on cardiovascular health than BMI metrics. In particular, visceral fat independently associates with adverse cardiovascular outcomes [10]. Regardless of BMI or overall adiposity, the American Heart Association notes a robust link between total obesity and abdominal obesity [11]. This association persists despite variations in subcutaneous versus dangerous intra-abdominal or visceral adipose tissue (VAT) between overweight or obese individuals [12]. Individuals with less VAT within elevated BMI categories often have more favorable cardiovascular disease (CVD) risk profiles, sometimes called “metabolically healthy obesity.” Some may have abdominal obesity without overall obesity or vice versa [13]. Those with excess visceral fat exhibit higher CVD risk than the metabolically healthy obese. The growing evidence for visceral fat marking cardiovascular risk stresses the need for precise body composition measures when gauging CVD risk, beyond simple BMI.

The aim of this research is to analyze the relationship between obesity and the risk of cardiovascular disease. It is important to understand this connection in the current medical context due to the increasing global prevalence of obesity, which is leading to a rise in cardiovascular diseases. This study aims to enhance our understanding of how obesity affects cardiovascular health by integrating data analysis with current scientific knowledge. The significance of this research lies in its potential to inform better clinical practices and public health strategies to mitigate these risks. This is particularly important given that cardiovascular diseases remain a leading cause of mortality worldwide.

2. Methodology

2.1. Data Source

The obesity and cardiovascular disease risk data utilized in this paper was obtained from the Kaggle website. The original information was stored in a CSV file format. The data was made publicly available on Kaggle by Fabio Mendoza Palechor and Alexis de la Hoz Manotas, researchers from the Universidad de la Costa (CUC) in Colombia. To restate, the obesity and heart disease risk data was sourced from Kaggle, having been contributed by Palechor and Manotas of the CUC university in Colombia. It was provided on Kaggle in the CSV format.

2.2. Variable Introduction

The dataset contains information gathered from 2,111 anonymous participants ages 14-61 in Mexico, Peru, and Colombia. The data was collected through an online survey covering participants' eating habits, physical condition, medical history across 17 variables in Table 1.

Table 1. Different types of variables

Term	Type	Meaning
Gender	Nominal variable	gender relates to reproductive functions and sexual traits.
Age	numeric	mean of Lifespan
Height	numeric	Height is the vertical distance from base to top of an organism.
Weight	numeric	weight refers to the mass of an organism
family_history_with_overweight	nominal variable	family history of overweight or obesity.
FAVC	nominal variable	regular intake of high-calorie food.
FCVC	numeric	vegetable consumption frequency.
NCP	numeric	mean of main meal frequency evaluation.
CAEC	nominal variable	consumption of Food Between Meals
SMOKE	nominal variable	mean of smoking status
CH2O	numeric	mean of water intake or hydration level.
SCC	nominal variable	mean of self-monitoring of calorie intake.
FAF	numeric	mean of frequency of physical activity
TUE	numeric	mean of time using electronic devices.
CALC	nominal variable	mean of alcohol intake assessment.
MTRANS	nominal variable	mean of travel habit assessment.
NObeyesdad	nominal variable	the classification of obesity

2.3. Method Introduction

A scatter plot utilizes an X-Y coordinate plane to visually depict values for two variables, allowing exploration of the relationship between those variables. As a basic analytic tool, scatter plots are critical for statistical analysis.

The One-Sample t-Test examines whether the average of a small sample differs significantly from a population average. It requires the sample data be approximately normal. This test works well when assessing smaller sample sizes against known or expected mean values.

Ordinal logistic regression, also known as the ordered logit model, is a statistical method used for modeling the relationship between an ordinal dependent variable and one or more independent variables. This model is widely used in social sciences, medical research, market research, and other fields where outcomes are ordinal. It helps in understanding how changes in predictor variables affect the likelihood of different outcomes on an ordinal scale.

3. Results and Discussion

3.1. One-Sample t-Tests

The one-sample t-tests revealed that all 17 variables—Gender, Age, Height, Weight, family history with overweight, FAVC, FCVC, NCP, CAEC, SMOKE, CH2O, SCC, FAF, TUE, CALC, MTRANS, and NObeyesdad-differ significantly from the null value ($p < 0.05$). This indicates that the mean

values of these variables are statistically distinct from zero, suggesting that they are measurable and significant features within the study population.

The one-sample t-test conducted against the null hypothesis value of 0.0 shows in Table 2 that all variables are significantly different ($p < 0.05$). Key findings include Age and Weight: Showed a substantial deviation from the null value, indicating their pronounced presence in the study cohort. Gender: Demonstrated a significant mean difference from zero, possibly reflecting the proportion of gender representation in the sample. Lifestyle Choices (FAVC, FCVC, NCP, CAEC, SMOKE, CH2O, SCC): Each of these dietary and behavioral variables displayed meaningful differences from the null value, suggesting their relevance in the context of obesity. Physical and Digital Activity (FAF, TUE): Both variables were significantly different from zero, underlining their potential impact on obesity. Family History with Overweight: Showed a notable difference, highlighting the genetic or familial component in obesity risk. Obesity Classification (NObeyesdad): As expected, this variable significantly differed from zero, reflecting the varied obesity levels within the sample. CALC and MTRANS: These variables also exhibited significant deviations, implying their role in the obesity context.

Table. 2 Single sample t test analysis results

Variable	Mini	Max	AVG	SD	t	p
Gender	0	1	0.494	0.5	45.394	0.000**
Age	14	61	24.313	6.346	176.026	0.000**
Height	1.45	1.98	1.702	0.093	837.948	0.000**
Weight	39	173	86.586	26.191	151.893	0.000**
family_history_with_overweight	0	1	0.818	0.386	97.259	0.000**
FAVC	0	1	0.884	0.32	126.769	0.000**
FCVC	1	3	2.419	0.534	208.164	0.000**
NCP	1	4	2.686	0.778	158.595	0.000**
CAEC	1	4	2.859	0.469	280.385	0.000**
SMOKE	0	1	0.021	0.143	6.702	0.000**
CH2O	1	3	2.008	0.613	150.516	0.000**
SCC	0	1	0.045	0.208	10.026	0.000**
FAF	0	3	1.01	0.851	54.572	0.000**
TUE	0	2	0.658	0.609	49.638	0.000**
CALC	1	4	3.269	0.515	291.325	0.000**
MTRANS	1	5	3.365	1.261	122.574	0.000**
NObeyesdad	1	7	4.016	1.952	94.514	0.000**

3.2. Ordinal Logistic Regression

This study conducts an ordinal logistic regression analysis to elucidate the predictors of obesity classification ('NObeyesdad'). Table 3 shows that the dependent variable is ordered. Including a wide array of variables, such as demographic, behavioral, and lifestyle factors, the model demonstrates a McFadden pseudo-R-squared value of 0.099, suggesting that the independent variables can explain 9.9% of the variability in obesity classification.

The analysis reveals that age, weight, family history of overweight, certain eating habits (CAEC), and caloric consumption behaviors (SCC) are positively associated with higher levels of obesity classification, with odds ratios exceeding 1. This indicates an increased likelihood of higher obesity classification with the rise in these predictors.

Conversely, gender, height, avoidance of high-calorie food (FAVC), frequency of vegetable consumption (FCVC), and the number of main meals (NCP) are negatively associated with obesity classification, implying a reduced likelihood of higher obesity classification with these factors. Variables such as SMOKE, CH2O, FAF, and TUE did not exhibit a significant relationship with 'NObeyesdad'.

Table 3. Analyze the Frequency Distribution of the Dependent Variable

name	dependent variable	frequency	percentage
NObeyesdad	Insufficient_Weight	272	12.88%
	Normal_Weight	287	13.60%
	Obesity_Type_I	351	16.63%
	Obesity_Type_II	297	14.07%
	Obesity_Type_III	324	15.35%
	Overweight_Level_I	290	13.74%
	Overweight_Level_II	290	13.74%
	total	2111	100

The study identifies several key determinants that significantly contribute to obesity classification. The findings highlight the complex interplay of genetics (family history), physiological factors (age and weight), and lifestyle choices (dietary habits and alcohol consumption). The notable absence of a significant effect from variables like smoking, water intake, and physical activity frequency warrants further investigation to disentangle the intricate dynamics of obesity risk factors. This research adds to the understanding of obesity's multifactorial nature and underscores the need for personalized and comprehensive approaches in obesity management and prevention strategies (Table 4).

Table 4. Analysis results of ordered Logistic regression model.

Variable	β	SE	z -value	Wald $\chi 2$	P -value	OR-value
Gender	-0.226	0.108	-2.099	4.407	0.036	0.797
Age	0.061	0.009	7.002	49.021	0	1.063
Height	-3.92	0.683	-5.743	32.98	0	0.02
Weight	0.031	0.002	13.453	180.996	0	1.032
family_history_with_overweight	1.043	0.125	8.311	69.072	0	2.837
FAVC	-0.711	0.133	-5.365	28.788	0	0.491
FCVC	-0.356	0.082	-4.361	19.016	0	0.701
NCP	-0.277	0.053	-5.218	27.223	0	0.758
CAEC	0.934	0.096	9.715	94.376	0	2.544
SMOKE	-0.511	0.28	-1.821	3.315	0.069	0.6
CH2O	0.093	0.067	1.379	1.9	0.168	1.097
SCC	0.786	0.197	3.983	15.864	0	2.196
FAF	-0.098	0.051	-1.91	3.646	0.056	0.907
TUE	0.022	0.068	0.319	0.102	0.75	1.022
CALC	0.114	0.031	3.678	13.526	0	1.121
MTRANS	0.122	0.041	2.967	8.803	0.003	1.129

4. Conclusion

The analysis uncovered significant associations between various demographic, lifestyle, and behavioral factors and obesity classification ('NObeyesdad'). Age, weight, family_history_with_overweight, CAEC, SCC, CALC, and MTRANS had a significant positive impact on obesity classification. In contrast, factors such as height, FAVC, FCVC, NCP, and SMOKE showed a significant negative association with obesity levels. Gender, CH2O, FAF, and TUE did not have a significant impact on obesity classification. The Odds Ratios (OR) and their 95% Confidence Intervals (CI) provided detailed insights into the strength and precision of these associations.

The findings emphasize the multifactorial nature of obesity and the need for a holistic approach in research and policymaking. Future research should focus on unraveling the complex interactions

among the variables, including genetic predispositions, dietary habits, and lifestyle choices. Policies aimed at obesity prevention and management should consider these diverse influences. The absence of significant impact from certain variables, such as gender and water consumption, indicates the need for further investigation. This could result in more targeted interventions.

Healthcare strategies should be customized to address the various causes of obesity, including personalized dietary and exercise plans. Public health campaigns could benefit from emphasizing the significance of lifestyle changes, particularly in diet and physical activity, along with awareness of genetic risk factors. Further studies are recommended to explore the roles of non-significant factors and to understand the impact of cultural, environmental, and socioeconomic factors on obesity. Lastly, collaboration among healthcare professionals, policymakers, and researchers is crucial to develop effective, evidence-based strategies to combat the growing obesity epidemic.

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