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Association of past obesity and BMI trajectories with cancer mortality: a prospective cohort study

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Abstract

Objective This study aimed to assess the association of past obesity and past BMI trajectories with cancer mortality in National Health and Nutrition Examination Survey.

Methods Past obesity was identified based on past maximum body weight, and trajectories of past BMI change were determined by latent class trajectory modeling (LCTM). Cox regression was used to assess the association of past obesity and past BMI trajectories with cancer mortality.

Results A total of 4,058 cancer patients participated in this study, of which 46.3% were past obesity, resulting in a significantly lower risk of cancer mortality compared to participants who were not past obesity (HR = 0.92, 95% CI: 0.92–0.93, $P < 0.01$). The LCTM identified five trajectories of past BMI, and compared with participants whose BMI remained in the normal range, the risk of death was 17% and 23% lower for participants in the "Long-term overweight" and "Long-term obesity" trajectory groups, respectively. In contrast, participants in the "Recent weight gain" and "Recent weight loss" trajectory groups had an increased risk of cancer death of 19% and 40%, respectively.

Conclusions This study found that past obesity is consistent with the "obesity paradox." In furtherance, a moderately elevated and stable BMI might be associated with lower cancer mortality.

Keywords Past obesity, BMI trajectory, Cancer mortality, Obesity paradox, NHANES

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Text box 1. Contributions to the literature

- Whether past obesity and past BMI trajectories in cancer patients are consistent with the "obesity paradox" has not been adequately investigated.
- Past obesity may improve survival rates for cancer patients.
- Cancer patients who have experienced significant changes in body weight in the past should be given special attention.

Introduction

Obesity affects 38% of the world's population and is expected to exceed 50% by 2035, and is one of the most serious public health problems for health care systems [1]. Correspondingly, the increased prevalence of obesity has led to an increased burden of obesity-related cancers [2]. Chronic inflammation is at the core of obesity, and this subclinical inflammatory state sets the stage for cancer development and progression. It has long been widely accepted that elevated BMI is associated with a poorer cancer prognosis [3], as well as guidelines have affirmed it [4]. However, some emerging research has identified the existence of an "obesity paradox" that higher BMI improves the survival rate of cancer patients [5–7]. The unexpected association between overweight/obesity with cancer mortality compared to normal-weight cancer patients may be due to the differences in nutritional reserve, tumor aggressiveness, and response to immunotherapy [8].

Furthermore, we note that recent researchers have found that obesity, as an environmental factor, trains innate immune cells (e.g., monocytes and macrophages) into an inflammatory phenotype that is retained through "epigenetic memory" [9]. Since tumors are closely related to immunity and inflammation, it suggests that our assessment of only the current BMI of tumor patients may not correctly reflect their relationship with survival. The lack of consideration of prior BMI (e.g., early adulthood, middle age) and the failure to adequately differentiate between pre-diagnostic, at-diagnostic, and post-diagnostic BMI may have contributed to the discrepancy in the results of some of the current studies [10, 11].

As the current discussion of prior BMI remains insufficient and conclusions are not entirely consistent, we proposed to use data from the National Health and Nutrition Examination Survey (NHANES) 1999–2018 to explore whether the effect of past obesity on tumor-related survival is similarly consistent with the "obesity paradox". In addition, dynamic changes in BMI across the lifecycle are similarly associated with disease and health outcomes [12, 13]. Some studies have done something similar to explore this, but have only generalized the population with elevated BMI into obese and/or overweight, which clearly ignores the health effects of the trend and magnitude of BMI changes over time. Therefore, this study will also identify the trajectory of past BMI changes over the life cycle of cancer patients by latent class trajectory model (LCTM) using past BMI at multiple time points, and further explore the impact of past BMI trends on cancer prognosis.

Methods

Data collection and research population

Data for our study came from the National Health and Nutrition Examination Survey (NHANES) 1999–2018. NHANES is a continuous survey created and conducted by the National Center for Health Statistics (NCHS) of the Centers for Disease Control and Prevention (CDC). A total of 111,797 participants were enrolled in 10 cycles from 1999 to 2018, excluding missing cancer-related data ($n = 4{,}178$), missing survival data ($n = 52{,}733$), missing past and current weight ($n = 5{,}651$) and patients with non-malignant tumors ($n = 44{,}644$). To make sure the results were reliable, we further excluded participants who died in accidents ($n = 45$). Participants with a BMI too high or too low suggesting that the participant may be in an extreme state or that the data are biologically implausible were excluded, so participants with a BMI < 15 kg/m^2 or >45 kg/m^2 were excluded ($n = 288$). In addition, to ensure consistency in the time series of prior BMI (25 years and 10 years ago), participants 35 years and younger were excluded ($n = 200$). The final inclusion of 4,058 eligible subjects (Fig. 1).

Past BMI

Past obesity was defined based on self-reported maximum weight and height in the weight history. In addition, NHANES asked respondents about their weight at age 25, 10 years ago, and 1 year ago, and also included height at age 25 to account for the possibility that height decreases with age [14]. Height at age 25 was used to calculate BMI at age 25, and height measured at the time of examination was used to calculate BMI 10 years ago and 1 year ago.

Mortality

Death status and cause of death were determined by National Death Index (NDI) records through December 31, 2019, which are linked to the NHANES dataset [15]. ICD-10 identifies cause-specific mortality. Cancer deaths are defined as ICD-10 codes C00-C97.

Covariates

Our study also extracted factors that potentially influence the prognosis of patients with malignant tumors, including family demographic information, lifestyle habits, and self-reported health status. Family demographic information, including gender, age, ethnicity, educational level

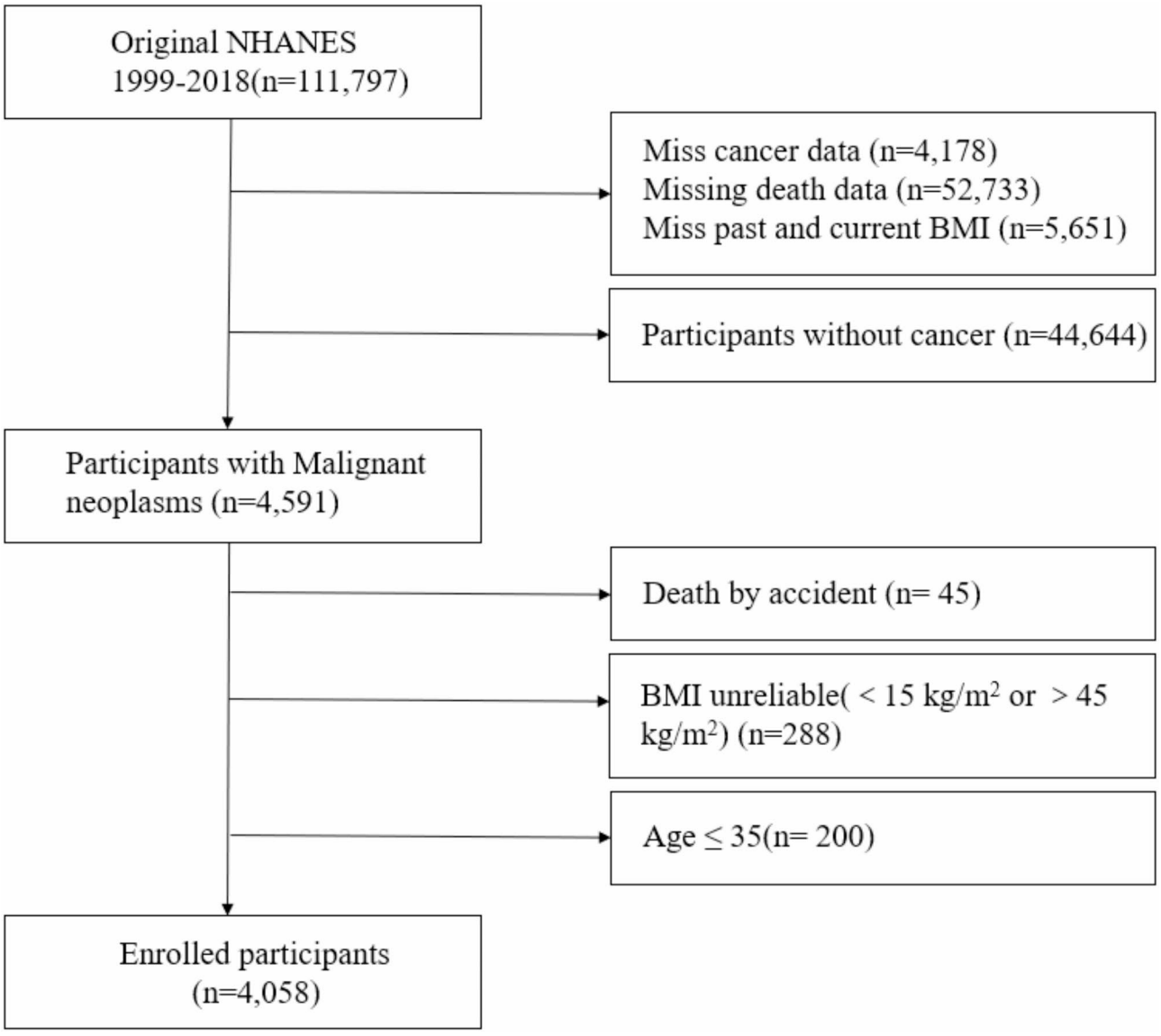

Fig. 1 Flow chart for inclusion and exclusion of the study population

and poverty-income ratio (PIR, < 1.3/≥1.3, <1.5/≥3.5). Drinking status is categorized as: Never (had < 12 drinks in lifetime); Former (had ≥ 12 drinks in 1 year and did not drink last year, or did not drink last year but drank ≥ 12 drinks in lifetime); mild (had ≥ 1 drinks per day for females, ≥ 2 drinks per day for males); moderate (had ≥ 2 drinks per day for females, ≥ 3 drinks per day for males, or binge drinking ≥ 2 days per month); heavy (had ≥ 3 drinks per day for females, ≥ 4 drinks per day for males). Smoking status is categorized as: never (smoked less than 100 cigarettes in life); former (smoked more than 100 cigarettes in life and smoke not at all now); now (smoked moth than 100 cigarettes in life and smoke some days or every day). Current medical conditions were based on self-reported outcomes including diabetes mellitus (yes/impaired fasting glucose (IFG)/impaired glucose tolerance (IGT)/no), hypertension (yes/no). Past obesity: past maximum BMI ≥ 30 kg/m^2; current obesity: BMI ≥ 30 kg/m^2 at the time of measurement. Participants were categorized into 4 groups based on past and current BMI at the time of the joint analysis: Keep normal(Both past maximal BMI and BMI at time of measurement were less than 30 kg/m^2);Past obesity only (Past maximal BMI ≥ 30 kg/m^2, BMI at time of measurement<30 kg/m^2);Currently obesity only (Past maximal BMI<30 kg/m^2, BMI at time of measurement ≥ 30 kg/m^2); Keep obesity (Past maximal BMI ≥ 30 kg/m^2, BMI at time of measurement ≥ 30 kg/m^2).

Statistical analysis

Descriptive analysis

Data were acquired and calculated using R 4.2.1 software, and descriptive analyses were performed using SPSS 26.0 software. To account for the complex survey design of the NHANES data, we followed the instructions for the survey and used appropriate sampling weights. In our study we used mean ± standard deviation (mean ± SD) to describe quantitative data with a normal distribution, and one-way analysis of variance (F) was used to test for differences between groups. Whereas quantitative data with non-normal distribution were expressed as median and quartiles [M (Q1, Q3)], and comparisons between groups were made using the Mann-Whitney U test. Percentages were used to describe categorical variables, and comparisons between groups were made using the chi-square test ($\chi 2$). P values < 0.05 were considered statistically significant.

Latent class trajectory model (LCTM)

To identify trajectories of change in past BMI (25 years, 10 years ago, 1 year ago), we modeled LCTM using M-Plus Version 8.3 (Muthen & Muthen, Los Angeles, CA, USA). LCTM is a special type of finite mixture modeling designed to find potential populations that exhibit similar trends over time. The final number of classes was determined by: (1) evaluation of model fit metrics, including Akaike information criterion (AIC) and Bayesian information criterion (BIC), where smaller values indicate better model fit; (2) Lo-Mendell-Rubin likelihood ratio test (LMRLRT) and Bootstrap likelihood ratio test (BS-LRT), which compare k and k-1 class models, where k is the number of potential classes; (3) model interpretability; (4) The average posterior probability of each class exceeds 70% while size ≥ 2% [16].

Subsequently cox proportional risk regression models were used to estimate associations between past and current obesity, trajectories of past BMI change, and risk of cancer death, calculating hazard ratios (HR) and 95% confidence intervals (CIs), with $p < 0.05$ considered statistically significant.

Results

Basic characteristics of the participants

Table 1 shows the basic characteristics of the participants. There were 4,058 participants, 9.0% of whom died of malignant tumors. Compared to cancer survivors, participants who died from malignant tumors were disproportionately male, less educated, had lower incomes, smoked more frequently, and were more likely to have comorbid hypertension and diabetes. Survivors and participants who died from cancer were 46.7% and 42.5% previously obesity, 33.0% and 28.9% currently obesity, respectively.

Relationship between past obesity and cancer mortality

Table 2 shows the association between past obesity and the risk of cancer death. The hazard ratio for past obesity was 0.92(95% CI 0.92–0.93) compared with participants who were no past obesity. Results remained stable across age and sex subgroups.

In the joint analysis, we further categorized the participants into 4 categories of Keep normal, Past obesity only, Currently obesity only and Keep obesity. Participants with Past obesity only and Keep obesity had a significantly lower risk of cancer death compared to participants who kept their BMI normal, with HRs of 0.96 (95% CI 0.96–0.97) and 0.78 (95% CI 0.77–0.78), respectively. In contrast, Currently obesity only participants had a 39% increased risk of cancer death.

Past BMI trajectory

The LCTM tested 6 trajectories of past BMI changes, and based on the fit indices of the models (Supplementary Table 1), class5 was ultimately identified as the optimal model (Fig. 2). Class 1 participants had a BMI that was almost stable in the overweight range, hence the name "Long-term overweight" (31.7%). Class 2 participants had a sharp increase in BMI over 10 years, referred to as "Recent weight gain" (3.3%). All classes had varying increases in BMI from the age of 25 years, while only Class 3 participants had a recent (10 years ago – 1 year ago) rapid decrease in BMI, hence the name "Recent weight loss" (3.1%). Class 4 participants consistently had a BMI in the normal range and were named "Normal" (57.2%). Class 5 participants maintained a BMI greater than 30, named "Long-term obesity" (4.7%). Baseline characteristics of the different categories are listed in Supplementary Table 2.

Association of past BMI trajectories with cancer mortality

Table 3; Fig. 3 show the association between prior BMI trajectories and risk of cancer death. Compared with participants in the "Normal" trajectory group, the hazard ratios for the "Long term overweight" and "Long term obesity" trajectory groups were 0.83 (95% CI 0.83–0.84) and 0.77 (95% CI 0.76–0.78), respectively. On the contrary, participants in the "Recent weight gain" and "Recent weight loss" trajectory groups had an increased risk of cancer death by 19% ($P < 0.01$) and 40.0% ($P < 0.01$), respectively.

Discussion

This study explored the association between past obesity and past BMI trajectories with tumor-related mortality in cancer patients. Our study suggests that past obesity is a protective factor for the prognosis of patients with malignant tumors. The trajectory of past BMI changes suggests that cancer patients who remain chronically overweight

Table 1 Baseline characteristics of participants.[1]

Variable	Total	Death status of malignant neoplasm		*p*-value
		No (91.0%)	Yes (9.0%)	
Age (median (IQR))	66.0(55.0,75.0)	65.0(54.0,75.0)	70.0(62.0,78.0)	< 0.01
Sex (n, %)				< 0.01
Female	2026, 55.4	1843, 56.6	183, 42.9	
Male	2032, 44.6	1716, 43.4	316, 57.1	
Race (n, %)				< 0.01
Non-Hispanic Black	560, 5.2	463, 4.8	97, 9.3	
Non-Hispanic White	2888, 87.6	2541, 87.9	347, 84.5	
Mexican American	242, 1.8	219, 1.8	23, 1.3	
Other Hispanic	188, 2.0	173, 2.0	15, 1.6	
Other race	180, 3.4	163, 3.4	17, 3.4	
Education (n, %)				< 0.01
Less Than 9th Grade	379, 4.8	323, 4.6	56, 6.7	
9-11th Grade	473, 8.6	388, 8.0	85, 14.5	
High School Grad/GED	958, 22.6	830, 22.1	128, 27.5	
Some College or AA degree	1174, 30.3	1044, 30.4	130, 29.4	
College Graduate or above	1071, 33.6	972, 34.8	99, 21.9	
PIR(%)				< 0.01
<1.3	799, 13.7	671, 13.0	128, 20.9	
1.3–3.5	1542, 36.3	1340, 35.7	202, 42.1	
≥ 3.5	1382, 50.0	1241, 51.4	141, 37.0	
Smoking status (%)				< 0.01
Never	1779, 44.6	1631, 46.2	148, 28.4	
Former	1699, 40.2	1439, 39.0	260, 52.1	
Now	578, 15.2	487, 14.8	91, 19.5	
Drinking status(%)				< 0.01
Never	469, 10.2	420, 10.2	49, 10.2	
Former	945, 20.8	775, 19.5	170, 34.3	
Mild	1573, 45.3	1409, 46.2	164, 35.6	
Moderate	398, 14.2	363, 14.6	35, 10.2	
Heavy	297, 9.6	258, 9.6	39, 9.8	
Hypertension (%)				< 0.01
No	1397, 40.6	1247, 41.3	150, 33.9	
Yes	2660, 59.4	2312, 58.7	348, 66.1	
DM (%)				< 0.01
DM	1023, 20.7	891, 20.6	132, 22.4	
IFG	224, 5.8	197, 5.6	27, 7.6	
IGT	141, 3.0	130, 3.1	11, 2.1	
No	2667, 70.4	2339, 70.7	328, 68.0	
Past obesity (%)				< 0.01
No	2145, 53.7	1865, 53.3	280, 57.5	
Yes	1913, 46.3	1694, 46.7	219, 42.5	
Current obesity (%)				< 0.01
No	2737, 67.3	2378, 67.0	359, 71.1	
Yes	1321, 32.7	1181, 33.0	140, 28.9	

[1]All results were survey-weighted except for sample counts

PIR, Poverty income ratio; DM, Diabetes Mellitus; IFG, Impaired Fasting Glucose; IGT, Impaired glucose tolerance

or obese conform to the "obesity paradox," meaning that cancer mortality declines, whereas the opposite is true for cancer patients who have recently gained weight.

Our results suggest that past obesity reduces cancer mortality in cancer patients. An interesting recent study drew our attention to the fact that high-fat diet-induced obese mice had increased expression of inflammatory genes even when they returned to normal weight afterward [9]. These obesity-associated inflammatory cytokines (e.g., Tumor Necrosis Factor) selectively induce

Table 2 Associations between past obesity and cancer mortality.[1]

Variables	*n*, %	HR (95% CI)	*p*-value
Past obesity *(ref: past non-obesity)*	1913, 46.3	0.92(0.92,0.93)	< 0.01
Gender subgroup			
Male (ref: past non-obesity)	2032, 44.6	0.95 (0.94,0.96)	< 0.01
Female (ref: past non-obesity)	2026, 55.4	0.90(0.90,0.91)	< 0.01
Age subgroup			
<60 (ref: past non-obesity)	967, 34.8	0.71(0.70,0.71)	< 0.01
≥ 60 (ref: past non-obesity)	3091, 65.2	0.94(0.93,0.94)	< 0.01
Joint analysis			
Keep normal	2065, 51.9	ref.	-
Past obesity only	672, 15.4	0.96(0.96,0.97)	< 0.01
Currently obesity only	80, 1.8	1.39(1.38,1.41)	< 0.01
Keep obesity	1241,30.9	0.78(0.77,0.78)	< 0.01

[1]All results were survey-weighted except for sample counts

n = unweighted sample size; HR = hazard ratio; CI = confidence interval; ref = reference group;

Model was adjusted for age, sex, race, poverty income ratio, education level, drinking status, smoking status, current obesity, hypertension and diabetes

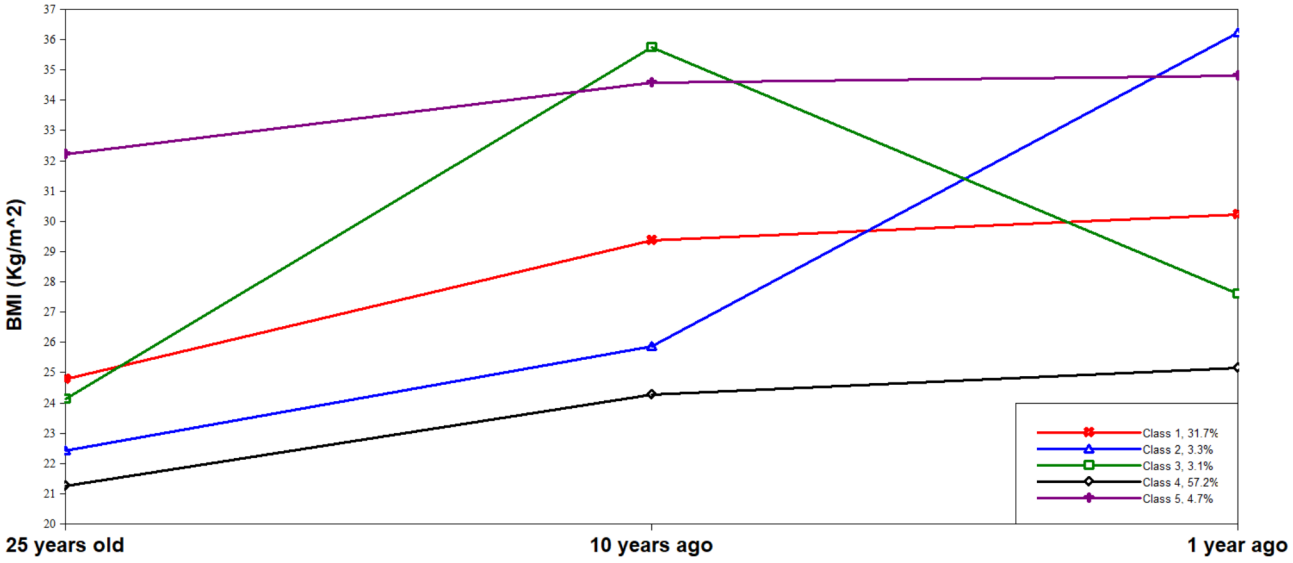

Fig. 2 Past body mass index trajectories in cancer patients

Table 3 Associations between BMI trajectory patterns and cancer mortality. [1]

BMI trajectory patterns	*n*, %	HR (95% CI)	*p*-value
Normal	2397,59.5	ref.	< 0.01
Long-term overweight	1237,30.1	0.83(0.83,0.84)	< 0.01
Recent weight gain	123,3.4	1.19(1.17,1.20)	< 0.01
Recent weight loss	120,2.5	1.40(1.39,1.41)	< 0.01
Long-term obesity	181,4.5	0.77(0.76,0.78)	< 0.01

[1]All results were survey-weighted except for sample counts

n = unweighted sample size; HR = hazard ratio; CI = confidence interval; ref = reference group;

Model was adjusted for age, sex, race, poverty income ratio, education level, drinking status, smoking status, hypertension and diabetes

PD-1 expression on tumor-associated macrophages (TAMs), which ultimately impairs tumor immune surveillance [17]. Therefore, not only current obesity, but past obesity may also conform to the "obesity paradox". Despite previous obesity in increasing the prevalence of cancer, potentially it also offers cancer patients the possibility of a better response to immunotherapy, as well as the exploration of new approaches to cell-specific therapies based on epigenetics [18].

In addition to cancer, the major comorbidities of obesity, such as type 2 diabetes mellitus and cardiovascular diseases, are closely associated with cancer, sharing

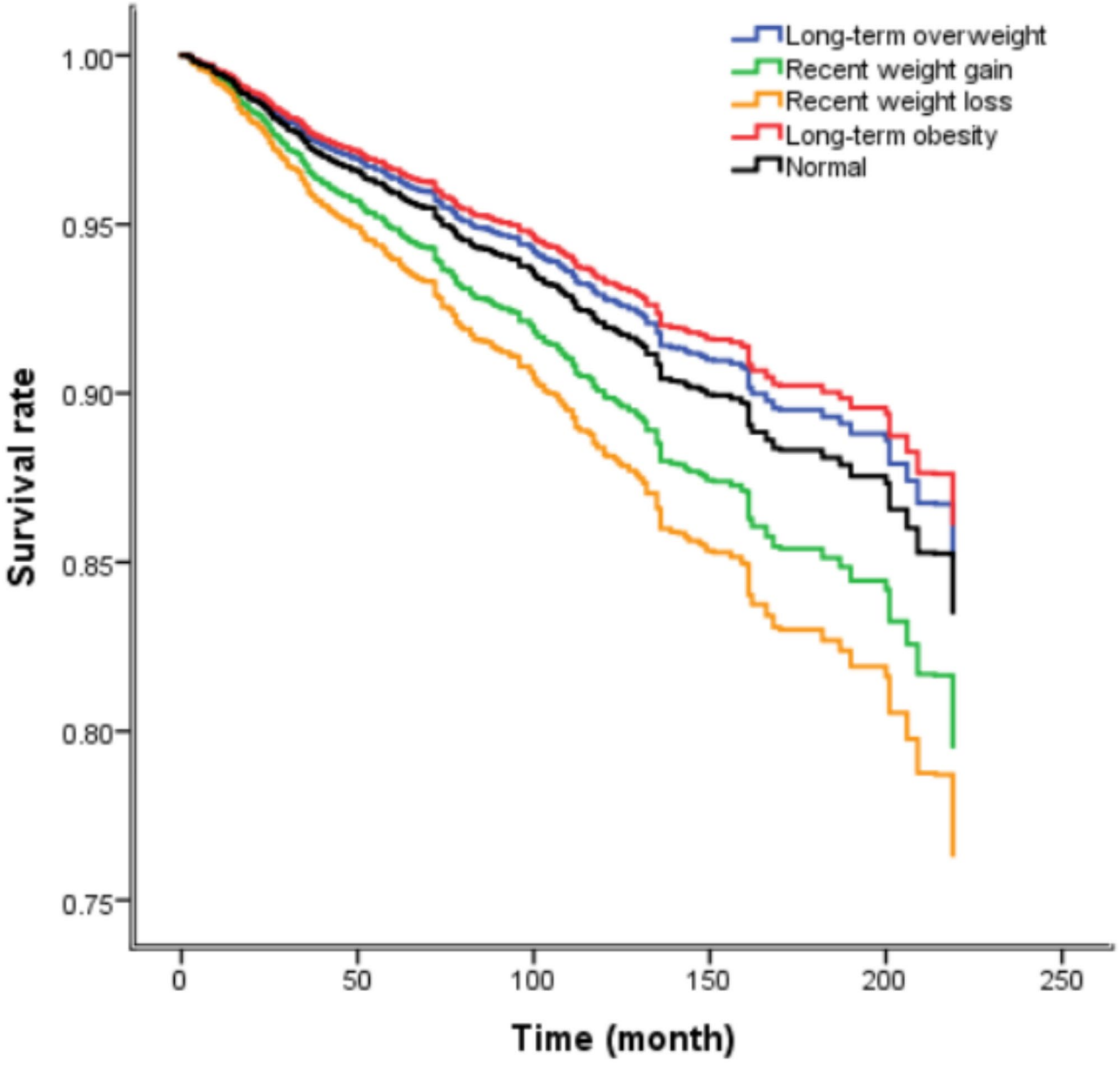

Fig. 3 The impact of past BMI trajectory on the survival rate of cancer patients. Black: Normal; Blue: Long-term overweight; Green: Recent weight gain; Orange: Recent weight loss; Red: Long-term obesity

overlapping mechanisms such as chronic low-grade inflammation, oxidative stress, and metabolic dysregulation [19, 20]. Given the shared mechanisms between obesity-related comorbidities and cancer, inflammatory indices such as the advanced lung cancer inflammation index (ALI)—which incorporates BMI, albumin, and the neutrophil-to-lymphocyte ratio—have been proposed as potential tools to assess cancer mortality risk. However, a recent cross-sectional study demonstrated that ALI was not associated with cancer mortality in diabetic patients [21], suggesting that comprehensive indices focusing on specific aspects may be more suitable for risk assessment. Further research is needed to explore these indices in the context of obesity-related comorbidities. Additionally, a meta-analysis found a weak but significant inverse correlation between BMI and serum vitamin D levels [22]. Vitamin D can modulate the tumor inflammatory microenvironment and immune properties through various mechanisms, which are crucial for inhibiting tumor invasion [23]. Most studies on cancer patients indicate that higher levels of vitamin D are associated with a lower risk of mortality [24]. A cross-sectional study based on NHANES also found that vitamin D insufficiency/deficiency had an additive effect on increased all-cause

mortality in individuals of normal weight, overweight, and obesity [25]. Interestingly, the same study observed that vitamin D excess was associated with an increased risk of cancer mortality in obese subjects, although this result should be interpreted with caution due to the small sample size.

Current research on the effect of past/pre-diagnosis BMI on cancer survival remains controversial [12, 26–28], with only the idea that cancer patients with weight loss have a worse prognosis being relatively widely accepted [8]. Our finding that recent weight gain is a risk factor for cancer survival does not appear to be consistent with the "obesity paradox". Participants in the "Recent weight gain" trajectory group already had a BMI that met the criteria for morbid obesity(BMI ≥ 35 kg/m^2), and extreme BMIs have been associated with lower overall survival in cancer patients [29], which is also consistent with the U-shaped association between BMI and cancer survival. Overly obese cancer patients have more difficulty with early surgery and are more likely to have metastasis and recurrence than those with a normal BMI [30]. An Austrian cohort study showed that overweight cancer patients had a better overall prognosis, while obesity was not associated with prognosis in cancer patients [28]. Simply categorizing participants as obese and non-obese, ignoring past weights and its trend and morbid obesity, may be one of the reasons why some studies have found this zero association.

In contrast to past BMI trajectories in which weight fluctuated, our results found that long-term maintenance of moderately high body weight is critical for the prognosis of cancer patients. Although we did not have access to the time of the participant's cancer diagnosis, it is likely that the participant's weight 1 year earlier had been affected by factors such as the tumor, surgery, and chemotherapy because of the close time interval. A study by Thivat et al. [31] showed weight fluctuations of more than 5% during chemotherapy in breast cancer patients were associated with poorer prognosis and higher mortality. Using a similar model to ours, a recent study in China found that participants in the "containing BMI" group had a higher quality of life and a better prognosis after radical gastric cancer surgery [32]. In addition, Chen et al. [33] investigated the association between weight change and mortality in adulthood using NHANES and found that the pattern of weight change was associated with all-cause mortality, but not with cancer mortality. In this regard, we emphasize that the study population should be more homogeneous and that observation of cancer mortality in the whole population is inevitably confounded by potentially unknown confounding factors. Overall, the safest body type trajectory is a stable weight, and weight loss is not recommended for everyone [34]. As a study in China found [35], for people with low weight early in life, modest weight gain and maintenance of stability can reduce overall mortality in later life. However, similar to these studies, we did not assess other obesity-related metrics such as waist circumference and visceral fat. A retrospective study demonstrated that while there was no difference in survival rates among colorectal cancer patients when obesity was classified based on BMI, significant differences in survival were observed for stage II and III patients when classified according to visceral fat levels [36]. A multicenter prospective study found that a lower visceral fat area (VFA) was associated with poorer survival in cancer patients [37]. Compared to BMI, VFA is a more representative indicator of body fat content, and the discrepancies observed in previous studies may also be attributed to BMI's inability to accurately reflect body composition [37, 38]. A recent significant report also emphasized that the definition and diagnostic criteria for clinical obesity should incorporate additional metrics such as waist circumference, waist-to-height ratio, and visceral fat area, alongside BMI [39]. Although BMI is highly convenient, future research should utilize comprehensive indicators to assess obesity more precisely for accurate risk evaluation.

This study explored the association of past obesity and past BMI trajectories with cancer survival, and it has several strengths, NHANES obtained a representative sample of Americans through sampling and adhered to strict study protocols with very reliable quality control. We used the LCTM to identify trajectories of past BMI changes over the lifespan of cancer patients, which preserves the trend and extent of BMI changes compared to simply categorizing BMI into obese and non-obese. Although early BMI may be less relevant to cancer risk and mortality due to the temporal distance. However, we believe that a long-term BMI trajectory with BMI at age 25 as a baseline provides a clearer picture of weight history, which may still provide valuable insights into cancer risk and mortality. At the same time there are some limitations of our study. First, the failure to obtain information on the treatment received by the participants may have affected the stability of the results. Second, the determination of previous BMI was based on questionnaires, which may produce recall bias. In addition, we recognize that the three time points may not fully capture the dynamic nature of BMI over time, which may underestimate the relevance of BMI trajectories to cancer mortality [40]. Third, LCTM is a data-driven exploratory analysis, which may have some limitations when generalizing to other populations.

Conclusion

The study found that past obesity also appears to be consistent with the "obesity paradox," and that maintaining a moderately elevated BMI for a long period of time

might be associated with reduced cancer mortality. These results emphasize the importance of maintaining weight stability in cancer patients, and more attention should be paid in the future to the role of past weight changes.

Abbreviations

BMI	Body mass index
CI	Confidence intervals
DM	Diabetes Mellitus
HR	Hazard ratio
IFG	Impaired Fasting Glucose
LCTM	Latent class trajectory model
NHANES	National Health and Nutrition Examination Survey
PIR	Poverty Income Ratio

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13690-025-01576-6.

Supplementary Material 1

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Author contributions
ZH, ZJL, SY, SL and HZ conceived and designed the study. ZH, ZJL, WC, ZJ, ZX and LD completed the data extraction and statistical analysis.SY, SL and HZ interpreted the results. ZH and ZLJ drafted the manuscript. All authors discussed the results, revised the manuscript and contributed to the final version. All authors have read and approved the manuscript.

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Data availability
All data of the study are presented in the text or supplementary materials. The datasets analyzed during the current study were publicly available from the NAHNES. Data from the NHANES can be found at https://wwwn.cdc.gov/nchs/nhanes/Default.aspx.

Declarations

Ethics approval and consent to participate
As the data used in this study were obtained from publicly available NHANES data, all data-related research had previously received approval from their respective ethical review committees and had obtained written informed consent from the participants. Consequently, this study does not require additional ethical approval.

Consent for publication
We used anonymous de-identified data that are publicly available from the NHANES. The NHANES obtained informed consent from all participants. Information related to this process can be found at We used anonymous de-identified data that are publicly available from the NHANES. The NHANES obtained informed consent from all participants.

Competing interests
The authors declare no competing interests.

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