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Anthropometric factors in relation to risk of glioma

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Abstract

Introduction—Increased height and greater adiposity have been linked to an increased risk of many cancer types, though few large studies have examined these associations in glioma. We examined body weight and height as potential risk factors for glioma in a large US-based case–control study.

Methods—The analysis included 1,111 glioma cases and 1,096 community controls. In a structured interview, participants reported their height and weight at 21 years of age, lowest and highest weight in adulthood, and weight 1–5 years in the past.

Results—Being underweight at age 21 ($\text{BMI} < 18.5 \text{ kg/m}^2$) was inversely associated with the risk of glioma development. This protective association was observed in both men and women, but reached statistical significance in women only (multivariate OR 0.68; 95 % CI 0.48, 0.96). When BMI at age 21 was assessed as a continuous variate, a small but significant increase in risk was observed per unit increase in kg/m^2 (OR 1.04; 95 % CI 1.02, 1.07). Adult height, recent body weight, and weight change in adulthood were not associated with glioma risk. All results were similar among never smokers and were consistent after stratifying by glioma subtype.

Conclusion—The present data suggest that a low body weight in early adulthood is associated with a reduced risk of glioma later in life. Results are consistent with previous studies in showing no material association of glioma risk with usual adult body weight. The present study does not support any association of adult stature with glioma risk.

Keywords

Glioma; Glioblastoma; BMI; Risk; Anthropometrics; Epidemiology

Introduction

Brain tumors are relatively rare tumors, with an estimated 23,910 adults diagnosed with brain or nervous system cancers in 2012 and 13,700 cancer-related deaths [1]. Despite advances in treatment, glioma remains one of the most aggressive human tumors and is associated with a median survival of 12–15 months [2]. The incidence of glioma is reported more frequently in Caucasians than in other races and occurs more often in males than in females [3]. With the exception of ionizing irradiation exposure [4], environmental or lifestyle risk factors remain largely unknown.

Obesity is an important risk factor for a wide spectrum of cancers [5], whereas the relationship between body size and glioma risk is unclear. Results from prospective cohort studies indicate no consistent association with adult body mass index (BMI) or measures of body fat distribution including waist circumference and waist-to-hip ratio [6–8]. In one prospective study, obesity at age 18 was associated with a significantly increased risk of developing glioma later in life [8].

Increasing adult height has been associated with the risk of a spectrum of cancers [9], reflecting genetic as well as environmental influences. A pooled analysis of individual data from 15 cohort and case–control studies based on 1,354 glioma cases and 4,734 controls provided suggestive evidence of a *U*-shaped relationship with a shorter and taller stature associated with increased risk and that a positive association with height may be stronger for glioblastoma than other glioma subtypes [10].

To shed further light on the association of anthropometric factors with the risk of glioma, we examined information on self-reported height and body weight from a large case–control study conducted in the Southeastern United States.

Subjects and methods

Study population

Subjects in the analysis were enrolled in a clinic-based case–control study examining risk factors for glioma. All individuals in the present analysis were aged 25 or older and had a

recent (within 3 months) primary diagnosis of glioma. Glioma cases were identified in neurosurgery and neuro-oncology clinics in the Southeastern United States including Vanderbilt University Medical Center (Nashville, TN), Moffitt Cancer Center (Tampa, FL), University of Alabama at Birmingham (Birmingham, AL), Emory University (Atlanta, GA), and the Kentuckiana Cancer Institute (Louisville, KY). Cases were enrolled between December 2004 and June 2012. Eighty-seven percent of eligible glioma patients were enrolled in the study and completed the study interview, a median of 1.0 month following the glioma diagnosis (interquartile range: 2 weeks–1.7 months). Controls were identified from white page listings with frequency matching to cases on age, gender, race, and state of residence [11]. An estimated 50 % of contacted eligible households yielded a participating control.

The Investigational Review Boards of each participating institution approved the study, and written informed consent was obtained from all participants.

Data collection

Demographic data and information on known and suspected glioma risk factors were collected in structured interviews. Subjects were asked to report height and usual body weight at age 21 and also body weight approximately 5 years before the interview (or 1 year before interview among those enrolled during the pilot phase of the study), as well as the lowest (excluding periods of brief illness) and highest (excluding pregnancy among women) weight as adults. BMI, calculated as body weight in kilograms (kg)/meter² (m²), was determined at each time point of interest. Reliability of interview responses was evaluated among 148 control subjects interviewed a second time, 2–4 months following an initial interview, with high correlations found for adult height (Spearman $r = 0.99$), weight at age 21 (Spearman $r = 0.92$), and recent weight (Spearman $r = 0.96$), as well as maximum (Spearman $r = 0.99$) and minimum (Spearman $r = 0.89$) adult weight.

Statistical analysis

Unconditional logistic regression was used to estimate odds ratios (OR) and 95 % confidence intervals (CI) relating anthropometric factors with glioma risk. Glioma risk related to height and BMI was modeled as categorical and continuous variables. We considered glioma risk according to World Health Organization cut points defining underweight (BMI < 18.5 kg/m²), overweight (BMI 25–29.9 kg/m²), and obese (BMI ≥ 30 kg/m²) [12], with a normal body weight (BMI 18.5–24.9 kg/m²) serving as referent. We further examined whether BMI at age 21, and lowest and highest adult body weight was associated with glioma risk. Weight change was analyzed by taking the difference in weight between age 21 and recent pre-morbid body weight and grouping into one of three categories: loss of >10 lbs, weight maintained within ±10 lbs, or gain of >10 lbs. Multivariate models included terms for age at interview (5 year categories), state of residence, race (Caucasian/nonCaucasian), gender, and education (high school or less, some college, college graduate, graduate education). To test for linear trend, anthropometric variables were included in the model as ordinal variables. Statistical analysis was performed using SAS, version 9.3 (SAS Institute, Inc., Cary, NC). A p value <0.05 was considered statistically significant, and all statistical tests were two-sided.

Results

Characteristics of the study population are shown in Table 1. The majority of interviews were completed by the participant, whereas fifteen case interviews (1.35 %) were completed by a proxy respondent. The mean age of glioma cases at diagnosis was 54 years (range 25–92), and the mean age of controls at enrollment was 57 years (range 25–89). Consistent with

reported incidence rates [3], males comprised approximately 60 % of the case group and the majority (95 %) of subjects were Caucasian. Among the cases, histological subtypes of glioma included glioblastoma multiforme ($n = 694$, 62.5 %), lower grade astrocytoma ($n = 224$, 20.2 %), oligodendroglioma or mixed oligoastrocytoma ($n = 150$, 13.5 %), and glioma with unspecified histology ($n = 43$, 3.87 %). Somewhat more cases than controls completed high school or less and more controls than cases attained a college degree.

Results for BMI at age 21, in the recent past, and for maximum adult weight are shown in Table 2. For body weight at age 21, in both men and women we found no evidence of excess risk associated with overweight or obesity when compared to a normal body weight. In contrast, a reduced risk was observed among subjects reporting a low body weight (BMI < 18.5 kg/m²) at age 21 (multivariate OR 0.67; 95 % CI 0.51, 0.88). The result was consistent in men and women, though it reached statistical significance only in women (multivariate OR 0.68; 95 % CI 0.48, 0.96). When body mass at age 21 was modeled as a continuous variate, the relative risk of glioma increased 4 % for each unit increase in BMI ($p = 0.0007$). A positive trend with increasing BMI was observed in men and women, though it was statistically significant only in women ($p = 0.0046$). Obesity at age 21 was suggestively associated with an excess risk in women (multivariate OR 1.66; 95 % CI 0.85, 3.23) though not in men (multivariate OR 0.77; 95 % CI 0.45, 1.31), although few subjects reported obese body weights at age 21 and these results were imprecise. Relative body weight in the recent past (1–5 years before interview) was unrelated to glioma risk (Table 2).

We observed no association between adult height and glioma risk when assessed according to categories of tallness or as a continuous variate (Table 3): The relative risk of glioma per one inch increment in height was 1.02 (95 % CI 0.98, 1.07) in men and 1.01 (95 % CI 0.96, 1.06) in women.

In further analyses, we considered whether weight change in adult life was associated with glioma risk. We observed no association with weight change from age 21 to recent prediagnostic body weight or with lowest and highest reported BMI in adulthood, adjusting for age, gender, race, state of residence, and body weight at age 21 (not shown).

All results were similar among never smokers and for histological subtypes of glioma although data in some exposure categories were limited. (Results for height and BMI restricting the case group to GBM are shown in Supplemental Tables 1 and 2.) Results were also similar after excluding cases with tumors involving the temporal lobe who may have had disturbances in long-term memory. A history of diabetes as reported in the interview was not associated with glioma risk (multivariate OR 0.96; 95 % CI 0.74, 1.24); results for BMI 1–5 years prior to interview and at age 21 were unchanged after further adjustment for diabetes.

Discussion

In this case–control study of more than 1,000 glioma patients and controls, a low body weight in early adulthood was associated with a significantly reduced risk of glioma later in life. We also observed a small, yet significant increase in glioma risk with increasing relative body weight at the age of 21, a pattern observed in both men and women, but reaching statistical significance in women only. Our results indicate no association with glioma risk for midlife body weight, weight change over the course of adult life, or weight fluctuations in adulthood. Adult height was not associated with glioma risk in men or women.

To the authors' knowledge, only two previous studies evaluated whether early adult body weight is associated with glioma risk [8, 13]. Moore et al. [8] in the prospective NIH-AARP

Diet and Health Study found that for weight reported at the age of 18, a low body weight was associated with a reduced risk of glioma, and an obese body weight, an increased risk of glioma later in life, though only the latter finding attained statistical significance. In the present study, obesity at age 21 was not significantly associated with an excess risk. However, we did observe a positive trend in risk with increasing body weight at age 21 (OR per unit increase in kg/m^2 1.04; 95 % CI 1.02, 1.07; $p = 0.0007$), driven mainly by a nonsignificant excess risk associated with obesity in women (OR 1.67), which was not observed in men (OR 0.77). However, these results were based on relatively sparse data and were imprecise. The magnitude of risk reduction associated with a low body weight ($\text{BMI} < 18.5 \text{ kg/m}^2$) in early adult life was similar in the study by Moore et al. (OR 0.69) and the current study (OR 0.67). In the study of Moore et al., BMI after age 18 and weight gain in adulthood were unrelated to glioma risk, similar to the current findings. In a study based on the prospective Harvard Alumni Health Study in which body weight at the time of college enrollment was available for approximately 20,000 men matriculated between 1914 and 1952 and followed over succeeding decades, Gray et al. [13] found no association between early adult (mean age 18.4 years) or midlife (mean age 46.1 years) BMI and subsequent mortality from brain cancer based on 85 brain tumor-related deaths. However, as metastases to the brain from systemic cancers are up to 10 times more common than primary brain tumors [14], some portion of deaths attributed to primary brain tumor in death records used to assign cause of death may have represented metastatic brain tumors. Taken together, the available evidence suggests that energy balance early in life may represent a modifiable risk factor for glioma, a hypothesis that should be explored in future studies.

Increasing adult stature has been associated with risk of a spectrum of cancers [9] thought to reflect genetic influences combined with childhood nutrition and health. In a pooled analysis of individual-level data from 15 studies (“GliomaScan”) in which information was available from a combined total of 1,354 glioma cases and 4,734 controls, Kitahara and colleagues [10] reported a modest and nonsignificant 3 % increase in glioma risk per 5-cm increase in height (age- and gender-adjusted OR 1.03; 95 % CI 0.98, 1.08) in men and women combined. In a meta-analysis that included all studies of height in relation to glioma/CNS tumors, including GliomaScan, the authors observed a stronger positive association with height (OR 1.07; 95 % CI 1.05, 1.09 per 5-cm increase in height). In the GliomaScan studies, there was suggestive evidence that both a shorter and taller stature increase risk. Furthermore, an excess risk with increasing height was restricted to glioblastoma in women. While neither of these patterns was clearly evident in the current data (as shown in Supplemental Table 2), our study does not rule out complex associations of glioma risk with height.

Inherent limitations in the current study arise from the use of self-reported body measurements and retrospectively collected information on body weight in the recent and distant past. Quality of the data in controls is suggested in our reliability study which showed excellent agreement of responses to height and weight questions in interviews conducted several months apart. Likewise, findings were similar after excluding subjects enrolled during the pilot phase who reported body weight 1 year prior to diagnosis (in the main study, subjects reported weight 5 years prior to diagnosis). However, it is possible that misclassification of body weight was greater among glioma cases as a consequence of the brain tumor. While this is difficult to rule out, we note that all results were similar after excluding the ~35 % of cases with tumors involving the temporal lobe, the seat of long-term memory in the brain. Among the cases, preclinical disease and weight gain from glucocorticoid treatment (a standard component of care in these patients) could also have affected body weight after diagnosis and distorted the participant’s memory of recent prediagnostic body weight; however, exceedingly rapid enrollment of cases in the study (median: 1 month following diagnosis) reduces this concern. Furthermore, results were

similar after excluding glioma cases reporting long-standing headache or other symptoms with an onset predating the glioma diagnosis by 6 months or more that might have affected diet and physical activity levels and therefore body weight in the recent past. Few case interviews (<2 %) were completed by proxy.

As this is a case–control study, we cannot rule out effects of selection bias in the data. In particular, a response rate among community controls of ~50 % is lower than optimal. However, correspondence of present results with prospective data on lifetime body weight provides assurance that selective enrollment of controls was unlikely to materially influence the current findings. A principal strength of the current study is its large size, especially when considering the low prevalence of glioma. To the knowledge of the authors, this is one of the largest individual case–control studies of glioma, and by its size, rapid enrollment of cases and the pathologic confirmation and clinical annotation available for all cases offers the potential for novel insight into the origin of glioma and modifiable lifestyle or environmental risk factors. Our series of glioma cases well represents the documented incidence rate of glioma by sex, age, and race in the US population [3].

In conclusion, our findings are similar to those of Moore and colleagues [8] in suggesting that energy balance in early adulthood may influence glioma risk later in life. Findings support the need for a better understanding of glioma risk in relation to early adulthood BMI, and the environmental and genetic influences on body size which may also influence neural development and brain tumor risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Descriptive characteristics in glioma cases and controls

	All		Men		Women	
	Cases (n = 1,111) (%)	Controls (n = 1,096) (%)	Cases (n = 646) (%)	Controls (n = 554) (%)	Cases (n = 465) (%)	Controls (n = 542) (%)
Age (years)						
< 40	18.7	12.1	18.7	12.4	18.7	11.8
40– < 45	8.01	8.39	7.74	6.14	8.39	10.7
45– < 50	9.36	10.7	10.2	10.3	8.17	11.1
50– < 55	12.2	11.8	11.6	11.2	13.1	12.4
55– < 60	11.5	12.5	10.7	12.1	12.7	12.9
60– < 65	14.0	13.3	13.8	13.0	14.4	13.6
65– < 70	11.1	13.0	11.8	14.4	10.1	11.6
70+	15.0	18.2	15.5	20.4	14.4	15.8
Education						
High school or less	36.3	26.8	34.8	24.01	38.3	29.7
Some college	26.5	26.5	27.2	24.91	25.6	28.2
College degree	21.7	28.5	23.1	29.78	19.8	27.1
Graduate degree	15.5	18.1	14.9	21.3	16.3	14.9
Race						
Caucasian	94.6	94.3	94.1	97.1	95.3	93.4
African American	4.68	4.56	4.95	2.71	4.30	6.46
Other	0.72	0.18	0.93	0.18	0.43	0.18

Table 2

Gender-stratified associations of BMI with glioma risk

	All			Men			Women		
	Cases n (%)	Controls n (%)	OR (95% CI)*	Cases n (%)	Controls n (%)	OR (95% CI)*	Cases n (%)	Controls n (%)	OR (95% CI)*
BMI (in kg/m ²)									
At age 21 years									
< 18.5	103 (9.44)	154 (14.13)	0.67 (0.51, 0.88)	34 (5.35)	42 (7.61)	0.67 (0.41, 1.09)	69 (15.16)	112 (20.82)	0.68 (0.48, 0.96)
18.5–24.9	715 (65.54)	715 (65.60)	1.00 (ref)	391 (61.48)	341 (61.78)	1.00 (ref)	324 (71.21)	374 (69.52)	1.00 (ref)
25–29.9	221 (20.26)	172 (15.78)	1.23 (0.97, 1.56)	182 (28.62)	137 (24.82)	1.16 (0.89, 1.52)	39 (8.57)	35 (6.51)	1.39 (0.85, 2.27)
30+	52 (4.77)	49 (4.50)	1.06 (0.70, 1.60)	29 (4.56)	32 (5.80)	0.77 (0.45, 1.31)	23 (5.05)	17 (3.16)	1.66 (0.85, 3.23)
Per 1 kg/m ² increase			1.04 (1.02, 1.07)			1.03 (1.00, 1.07)			1.05 (1.02, 1.09)
Test for trend			<i>p</i> = 0.0007			<i>p</i> = 0.0539			<i>p</i> = 0.0046
Recent past									
< 18.5	18 (1.63)	18 (1.64)	1.12 (0.56, 2.22)	8 (1.24)	3 (0.54)	2.47 (0.63, 9.70)	10 (2.17)	15 (2.77)	0.80 (0.34, 1.87)
18.5–24.9	336 (30.46)	384 (35.07)	1.00 (ref)	133 (20.68)	142 (25.63)	1.00 (ref)	203 (44.13)	242 (44.73)	1.00 (ref)
25–29.9	447 (40.53)	427 (39.00)	1.07 (0.87, 1.31)	311 (48.37)	260 (46.93)	1.26 (0.94, 1.69)	136 (29.57)	167 (30.87)	0.95 (0.70, 1.29)
30+	302 (27.38)	266 (24.29)	1.15 (0.91, 1.45)	191 (29.70)	149 (26.90)	1.26 (0.91, 1.75)	111 (24.13)	117 (21.63)	1.11 (0.80, 1.54)
Per 1 kg/m ² increase			1.01 (0.99, 1.02)			1.01 (0.98, 1.03)			1.01 (0.98, 1.03)
Test for trend			<i>p</i> = 0.5664			<i>p</i> = 0.6745			<i>p</i> = 0.6343
Maximum									
< 18.5	2 (0.18)	3 (0.27)	0.79 (0.13, 4.91)	0 (0.00)	1 (0.18)	NA	2 (0.43)	2 (0.37)	1.15 (0.15, 8.65)
18.5–24.9	169 (15.39)	197 (17.97)	1.00 (ref)	53 (8.31)	65 (11.73)	1.00 (ref)	116 (25.22)	132 (24.40)	1.00 (ref)
25–29.9	401 (36.52)	421 (38.41)	1.02 (0.79, 1.32)	261 (40.90)	232 (41.88)	1.38 (0.91, 2.09)	140 (30.43)	189 (34.94)	0.86 (0.61, 1.22)
30+	526 (47.91)	475 (43.34)	1.15 (0.89, 1.48)	324 (50.78)	256 (46.21)	1.43 (0.94, 2.16)	202 (43.91)	218 (40.30)	1.07 (0.77, 1.48)
Per 1 kg/m ² increase			1.00 (0.99, 1.01)			0.99 (0.97, 1.01)			1.01 (1.00, 1.02)
Test for trend			<i>p</i> = 0.7509			<i>p</i> = 0.1787			<i>p</i> = 0.2925

* Odds Ratios (OR) and 95% Confidence Intervals (CI) adjusted for age, race, education and state of residence, and in combined models, gender

Table 3

Gender-stratified associations of height with glioma risk

	Cases <i>n</i> (%)	Controls <i>n</i> (%)	OR (95 % CI)*
Height: male (inches)			
< 69	134 (20.74)	116 (20.94)	0.89 (0.64, 1.26)
69–70	171 (26.47)	147 (26.53)	1.00 (ref)
71–72	178 (27.55)	171 (30.87)	0.87 (0.64, 1.19)
73	163 (25.23)	120 (21.66)	1.17 (0.84, 1.63)
Per 1-inch increase			1.02 (0.98, 1.07)
Test for trend			<i>p</i> = 0.3144
Height: female (inches)			
<63	94 (20.22)	97 (17.90)	1.35 (0.92, 1.97)
63–64	116 (24.95)	161 (29.70)	1.00 (ref)
65–66	139 (29.89)	149 (27.49)	1.33 (0.95, 1.88)
67	116 (24.95)	135 (24.91)	1.26 (0.88, 1.79)
Per 1-inch increase			1.01 (0.96, 1.06)
Test for trend			<i>p</i> = 0.7688

* Odds Ratios (OR) and 95% Confidence Intervals (CI) adjusted for age, race, education and state of residence