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A post-mortem stereological study of striatal cell number in human obesity

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Abstract

Objective—Neuroimaging studies have revealed abnormalities in brain structure, including the striatum, in obese people. We aimed to investigate the cellular and parenchymal basis for these findings in post-mortem brain tissue.

Design and Methods—Design-based (unbiased) stereology combined with histochemical and immunocytochemical staining were used to quantify total number of neurons and astrocytes in post-mortem striatal brain samples from 9 obese (BMI 40.2±6.1 kg*m^{-2}) and 8 lean (BMI 24.4±1.0 kg*m^{-2}) donors. Total numbers of Nissl-stained neurons and GFAP-immunopositive astrocytes were counted in ten systematic-random sections starting from the frontal pole of the striatum.

Results—There were no differences in mean total numbers of neurons (obese: 7.60E+06; SD 2.50E+06; lean: 7.85E+06; SD 8.26E+05; $p<0.78$) or astrocytes (obese: 7.42E+06; SD 2.27E+06; lean: 7.43E+06; SD 2.50E+06; $p<0.99$). A higher variance was found for number of neurons ($p<0.007$) but not astrocytes ($p<0.72$) in the obese group. Neuron/glia ratios were similar in both groups (obese: 1.07; SD 0.39; lean: 1.15; SD 0.37; $p<0.70$) with an overall striatal neuron/glia ratio of 1.11 (SD 0.37) across the entire study population (n=17).

Conclusion—We found no difference in the average numbers of neurons and astrocytes in the anterior striatum between lean and obese people. The morphological basis for structural brain changes in obesity requires further investigation.

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Conflicts of Interest Statement

The authors declare that there are no conflicts of interest.

Keywords

obesity; brain; striatum; stereology; post-mortem

Introduction

Recent studies indicate that the prevalence of obesity in the United States exceeds 35% in the adult population (1). Consequently, there has been increasing interest in the neurological basis for the behaviors that promote obesity. Dysfunction of the brain's reward system including dopaminergic mesolimbic and mesocortical pathways may play a role by promoting unhealthy food choices and causing compensatory overconsumption (2). Human neuroimaging studies have repeatedly shown an important role for the striatum in reward related behavior and obesity (3–6). In healthy humans, striatal volume was positively associated with inhibitory control and negatively associated with reward sensitivity (7), on the other hand lower striatal gray matter volume has been reported in obese subjects (8). Similar obesrvations have been made on a functional level with reduced striatal dopamine D2 receptor (DRD2) (9) and dopamine transporter (DAT) (10) in obese subjects compared to normal weight subjects. In addition lower striatal DRD2 density in obese subjects might be involved in reduced inhibitory control and salience attribution (11). In humans it is not known whether these abnormalities precede obesity, and therefore represent neural markers of increased propensity to gaining weight, or occur as a consequence of chronic obesity, even though rodent studies suggest a progressive decline of striatal dopaminergic function with increasing obesity (12).

Since all previous reports on structural brain alterations in obese humans are based on in-vivo imaging techniques, the underlying histological causes remain unknown. This seems particularly important, as a potentially reduced number of neurons (inherent or acquired) in reward related brain regions such as the striatum could provide additional explanations for known difficulties in losing weight or maintaining a stable body weight after weight loss (13). Hence, new approaches are required to enhance our understanding of the neurobiology of obesity. Based on the above delineated results from *in-vivo* studies, we hypothesized that post-mortem striatal samples of obese donors would exhibit lower density of neurons and glial cells (i.e. astrocytes) than lean donors. Brain samples were analyzed using computerized stereology as previously detailed for applications to human brains (14–21).

Methods and Procedures

Tissue Samples

Brains were acquired in compliance with requirements of the institutional review committee of the Harvard Brain Tissue Resource Center (wwww.brainbank.mclean.org). Brains were fixed in 10% formalin and coronally dissected in a standardized procedure by the donating institution. Inclusion criteria for this study included no evidence of psychiatric or neurological diseases. Groups were divided based on body mass index (BMI) calculated as pre-mortem body weight divided by pre-mortem height squared (lean: mean BMI=24.4 ± 1.0 kg/m^2; obese: 40.2 ± 6.1 kg/m^2). Brain samples for obese and lean subjects were

matched using predefined inclusion criteria (age, postmortem-interval, time in formalin fixation) and screened for evidence of neuropathology. Based on matching criteria and availability of tissue and anthropometric data, striatal samples were obtained from a total on n=18 cases. A single case in the obese group was excluded from the statistical analysis on the basis of atypical histological appearance expected for striatum (Figure 1), leaving a total of n=17 cases (9 obese and 8 lean).

Tissue Preparation

Blocks of formalin-fixed post-mortem human brain containing the most anterior 5 mm of the striatum ("striatal cap", caudate and putamen) were dehydrated through graded ethanol and xylenes, and then embedded in paraffin. Paraffin blocks were serially sectioned in the coronal plane at an instrument setting of 25 μm. With a random start in the first series of 6 sections (interval: 150 μm), the 1st and 2nd sections in each series of 6 serial sections were mounted separately on 50×75 mm Superfrost Plus microscope slides (1 section per slide, 10 slides per set, 2 sets per brain) and stained with cresyl violet and GFAP-immunocytochemistry, respectively (for details see supplementary material).

Stereology

Trained personnel blind to group used a computerized stereology system (*Stereologer*, Stereology Resource Center, Tampa, FL, for specifications see supplementary material) to quantify total numbers of neurons and astrocytes in n=10 sections sampled in a systematic-random manner through the striatal cap. Specifically, these studies used the optical fractionator method (22), as previously applied by our group to human brains (17, 23) (for recent stereology reviews, see 19–21). Briefly, the striatum was outlined at low power (4x) on each section, followed by counting neurons and astrocytes on thin focal plane scanning at high magnification (60x, 1.4 na) in the z-axis. Neuronal somas of all sizes were included in the count if they met the inclusion criteria: well-formed nucleus, nucleolus, and nuclear membrane with evidence of some cytoplasm (Figure 2B). Cells immunopositive for GFAP were counted as astrocytes (Figure 2C). Neurons and astrocytes were counted if they fell within the 3-D disector or intersected the inclusion planes without touching the exclusion planes on the unbiased counting frame. This unbiased counting method was repeated at 100 to 200 systematic-random locations across all n=10 sections for each case to achieve a high stringency level, as evidenced by a coefficient of error less than 10% (CE < 0.10) for both lean and obese groups (for more details see supplementary material).

Statistical Analyses

SAS Software (SAS Institute Inc, version 9.2, Cary, NC) was used for all statistical analyses. Two-sample t-test and table analyses were applied for group comparisons using either pooled or Satterthwaite 95% intervals whenever appropriate and Levene's test for equality was used for additional analyses of variance. Pearson or Spearman correlation were used for correlational analyses whenever appropriate.

Results

Sample

Table 1 summarizes clinical data on brain donors and donated tissue. All brains were from male donors with the exception of a single female (case #10) in the obese group. As shown in Table 2, the only significant difference between lean and obese subjects is BMI ($p<0.0001$). Additional analyses excluding the 3 subjects with BMI>25 and the female subject did not significantly change the results as described below.

Microscopic Appearance

Striatal samples showed the typical patch matrix mosaic of the striatum at low power (Figure 2A). The microscopic appearance of Nissl-stained cell bodies at high power (60x oil immersion) covered a range of soma sizes (small, medium, large) with a predominance of medium-sized cell bodies (Figure 2B).

Stereology Counts of Neurons

For the total of 17 cases in this study, the overall mean total number (Total N) of cresyl violet-stained neurons in the anterior striatum was 7.72E+06 (SD 1.85E+06). Statistical analysis revealed no differences in mean Total N of Nissl-stained neurons for the lean (7.85E+06: SD 8.26E+05) and obese (7.60E+06; SD 2.50E+06) groups ($p=0.78$; Satterthwaite approximation for unequal variances) in the anterior striatum (Figure 3A). However, the Levene's test for equality showed significantly larger variation in neuronal cell counts for the obese group ($p=0.007$).

Stereology Counts of Astrocytes

Statistical analyses of astrocyte findings showed a mean Total N of 7.42E+06 (SD 2.31E +06) GFAP-positive astrocytes in anterior striatum. As shown in Figure 3B, there was no difference ($p = 0.99$) in mean Total N of astrocytes in anterior striatum for the lean group (7.43E+06; SD 2.50E+06) and the obese group (7.42E+06; SD 2.27E+06). Variance of astrocyte counts did not differ between lean and obese groups according to Levene's test for equality of variation ($p=0.72$).

Correlational Analyses and Neuron/Glia Ratio

Using the Pearson product moment statistic, there were no significant correlations between total N for neurons and astrocytes for both groups analyzed together ($p=0.12$; $r=0.40$); or within lean ($p=0.16$; $r=0.55$) and obese ($p=0.26$; $r=0.43$) groups analyzed separately. Correlational analyses of BMI with neuron or astrocyte counts were also not significant (data not shown). No differences were found for neuron/glia ratios (i.e. quotient of mean neuronal and glial cell counts) between the groups [entire sample 1.11 (SD 0.37); lean 1.15 (SD 0.37); obese 1.07 (0.39); $p=0.70$].

Discussion

This is the first study in postmortem human brains to assess a possible histological basis for obesity related brain abnormalities. It has been hypothesized that reduced functioning of the

brain reward system might facilitate the development of obesity, with cumulative evidence pointing to an important role of the striatum in reward and reward-related behavior (3–6). Despite previous neuroimaging reports suggesting morphological and functional differences in the striatum of obese people, we found no histological differences in numbers of either neurons or astrocytes in striatum from lean and obese people. The only significant finding was a significantly higher variance for counts of neurons but not astrocytes in striatum of obese compared to lean people.

Previous neuroimaging findings differ with regard to specific locations and even the directionality of the respective alterations in obesity. Pannaciulli et al. (2006) reported reduced striatal gray matter volume but increased striatal white matter volume in obese subjects (8). A second group of investigators reported similar white matter increases in the dorsal striatum of obese people (24). An imaging study by Schäfer et al. (2010) reported a negative relationship between BMI and volume of dorsal and ventral striatal gray matter (25). Conversely, ventral and dorsal striatal gray matter was positively associated with BMI in a sample of 122 comparably young lean, overweight and obese adults (26).

Apart from structural differences in striatum, functional neuroimaging studies also indicate neurochemical and functional alterations in human obesity (3, 27, 28). In particular, striatal dopaminergic function appears to play a key role in promoting incentive salience, thus increasing the “wanting” aspect of food-related behavior (29). This concept is supported by findings from radiotracer studies showing reduced availabilities of striatal DRD2 dopamine receptors (9) and dopamine transporter (DAT) in obese subjects (10). More recent results, however, do not support these earlier findings (30, 31). The discrepancies with regard to alterations of reward-related brain functions in obesity, including striatal responses to food cues, has lead some authors to propose more complicated models of reward dysfunction. Burger et al. suggested a dynamic vulnerability model in which hyper-responsivity to food leads to overconsumption, followed by downregulation of dopaminergic receptors (32). The result in this case would be striatal hypo-responsivity to food reward with compensatory increases in food intake. A dynamic model with varying degrees of dopaminergic function may explain the above inconsistencies in striatal findings in neuroimaging studies. This in combination with lack of information about duration of obesity might also provide a possible explanation for the here observed higher variance of striatal neurons in obese humans.

To date few stereological studies exist that investigated neuronal and glial counts of the healthy human brain, and none prior have been carried out in any part of the striatum. Beside no differences in numbers of neurons and astrocytes for obese and lean groups, we report close to a 1:1 balance between the neurons (mean 7.72 ± 1.85 million) and astrocytes (mean 7.42 ± 2.31 million) in striatum. These findings agree with recent studies challenging the widespread supposition that the human brain contains approximately ten times more glia cells than neurons (33). Across all regions of cortical gray matter, Azevedo et al. (2009) reported a ratio between 1 and 1.5 for neuronal (mean 86.1 billion) and non-neuronal cells (mean 84.6 billion) (34).

A few caveats need to be acknowledged. First, working with human brain tissue is challenging in numerous ways. Particularly the limited availability of tissue and the resulting small sample sizes need to be acknowledged. Also, the only structure in the donor brains consistently available for unbiased systematic-random sampling was the anterior part of the striatum ("striatal cap"). Systematic-random sampling through the entire striatum could yield different findings than reported here. A second consideration is that besides basic anthropological data such as height and weight, no additional data was available with regard to onset, duration of obesity, and length of weight stability. Third, although our records search did not indicate any known addictive or psychiatric disorders, heterogeneity in causes of death and potential comorbidities could have influenced the results. Rather than BMI, recent studies indicate that variables such as free fat mass and waist circumference more closely correlate with changes in brain volumes (35, 36).

Conclusion

To our knowledge, this is the first quantitative histological study of neurons and astrocytes from the brains of otherwise healthy obese and lean subjects. Using computerized stereology we found no differences in mean neuronal and astrocyte density in the striatum but a significantly higher variance in striatal neuronal counts in obese subjects. We confirmed a neuron/glial ratio close to 1.0 for the striatum, in agreement with ratios reported for cerebral cortex. These results indicate a need to further explore the underlying biology and histology of neuroimaging findings.

Acknowledgments

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- The striatum plays a key role in eating and reward related behaviour
- Neuroimaging studies have revealed structural abnormalities of the striatum in human obesity
- This is the first post-mortem study of brain samples from obese and lean people
- Despite a higher variance in neuronal cell counts for the obese group, no differences in mean striatal numbers of neurons and astrocytes were observed between lean and obese

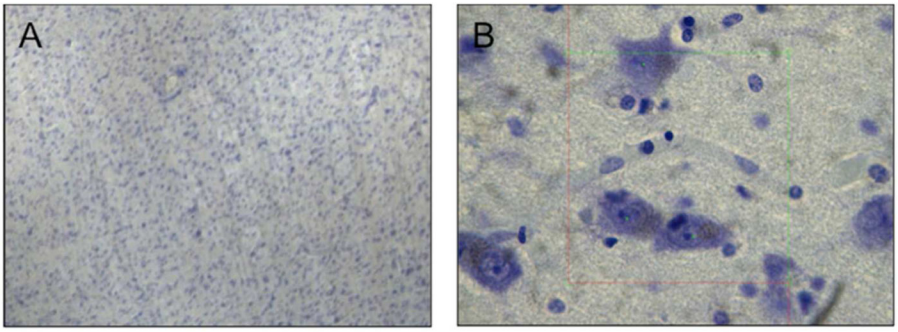

Figure 1.

Histological appearance of tissue from excluded case showing lack of the patch matrix mosaic at low mag (4x, left), lack of predominant medium-sized neurons at high magnification and an atypically high number of large, pigmented cells (60x, right). The Total N of neurons and astrocytes for this case were 5.41E+05 and 1.10E+06, respectively.

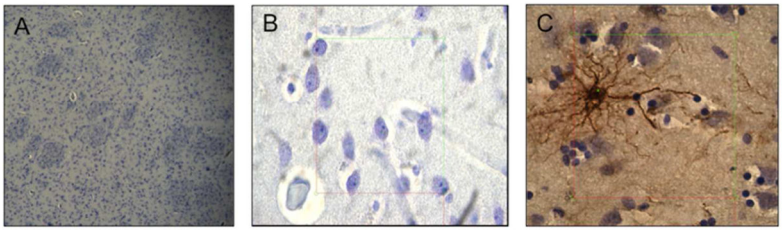

Figure 2.

Typical patch matrix in Nissl-stained striatal section at low (4x) power (A). Images of typical cresyl violet-stained neurons (B) and GFAP-immunopositive astrocyte (C) in striatum (60x oil).

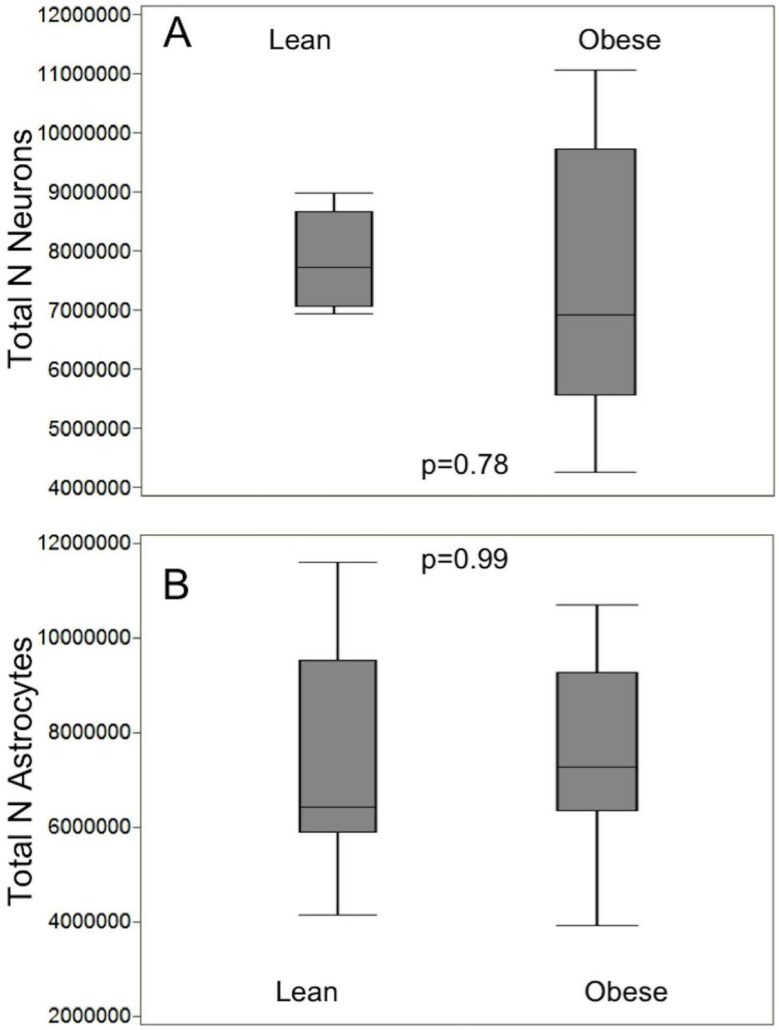

Figure 3.

Boxplots of mean neuronal (A) and astrocytal (B) counts in lean and obese subjects and corresponding p-values (two-sample t-Test). Comparisons between obese and lean groups reveal no statistical differences for neurons ($p<0.78$) or astrocytes ($p < 0.99$).

Table 1

Clinical characteristics of study population.

Group	Case	Age (years)	sex	BMI (kg*m^{-2})	Cause of death	Time in formalin (days)	Postmortem Interval (hours)	Comorbidities
Lean	1	63	m	22.4	CPA	1499	17.9	HLP
	2	56	m	24.4	CPA	2033	28.8	HLP, HTN
	3	68	m	24.4	AMI	1616	24.2	Kidney stones
	4	65	m	24.3	CPA	1420	26.2	None reported
	5	60	m	23.8	AMI	2187	22.1	None reported
	6	56	m	25.1	AMI	2049	26.0	CAD
	7	57	m	25.4	RA	1372	21.8	CHF, DM2, COPD
	8	65	m	25.4	AMI	1563	22.9	CAD, HLP, HTN
Obese	9	55	m	31.5	CPA	1198	23.9	HTN, gastric ulcer
	10	63	f	33.1	CPA	1612	26.8	HTN, psoriasis
	11	47	m	38.1	AMI	804	26.8	HTN, HU, sleep apnea
	12	68	m	37.3	CPA	1142	16.8	AP
	13	57	m	39.1	CPA	808	25.0	HTN, AF,
	14	62	m	44.1	CPA	2272	21.7	Varicosis
	15	56	m	42.0	AMI	1786	26.2	DM2, HLP
	16	62	m	46.2	CPA	2302	25.6	DM2, CRI, pHT
	17	65	m	50.5	AMI	1644	23.2	HTN, COPD, Prostate-Ca

Abbreviations: CPA, cardiopulmonary arrest; AMI, acute myocardial infarction; RA respiratory arrest; HLP, hyperliproteinemia; HTN, arterial hypertension; CAD, coronary artery disease; CHF, chronic heart failure; DM2 type 2 diabetes mellitus; COPD, chronic obstructive pulmonary disease; HU, hyperuricemia; AP, angina pectoris; AF, atrial fibrillation; CRI, chronic renal failure; pHT, pulmonary hypertension;

Table 2

Group comparison of lean and obese

	Obese	Lean	p
Sex (m/f)*	8/1	8/0	n.s.
Age (years)	59.4 (±6.3)	61.2 (±4.7)	n.s.
BMI ($kg*m^{-2}$)	40.2 (±6.1)	24.4 (±1.0)	<0.0001
Postmortem Interval (hours)	24.0 (±3.2)	23.7 (±3.3)	n.s.
Time in Formalin (days)	1508 (±563)	1714 (±320)	n.s.
Hemisphere (L/R)*	6/3	7/1	n.s.

All results apart from*are presented as mean ± SD