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Association of Binge Eating Behavior with Total and Abdominal Adipose Tissue in a Large Sample of Participants Starting a Weight Loss or Maintenance Program

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ABSTRACT

Objective: It is not clear whether binge eating (BE) behavior is associated with body composition independently of body mass index (BMI). Our aim has been to evaluate the BMI-independent contribution of BE severity and BE status on the total amount of fat mass and abdominal fat distribution in a large sample of participants.

Method: We performed a cross-sectional study among 8524 participants followed at a nutritional center. BMI and waist circumference (WC) were measured, body fat (BF) was estimated by skinfold measurement, and abdominal visceral (VAT) and subcutaneous (SAT) adipose tissues were measured by ultrasonography. BE was assessed using the Binge Eating Scale (BES). The association between the continuous BES score (BE severity) and adiposity was assessed in the whole sample after adjustment for BMI and other confounders. The effect of BE status on adiposity was also assessed by matching binge eaters (BES $\geq$ 18), for sex, age, and BMI, with non-binge eaters (BES $<$ 18).

Results: We found that 17.7% of the participants were binge eaters. Continuous BES score was associated with increasing WC (0.03 cm, 95% confidence interval [CI], 0.02 to 0.05 every 1 BES unit, $p < 0.001$) and decreasing BF (0.01%, 95% CI, −0.02 to −0.00 every 1 BES unit, $p = 0.003$). No association was found between BE severity and VAT and SAT. After matching, the BF of binge eaters was 0.29% (95% CI, −0.50 to −0.07, $p = 0.01$) lower than that of non-binge eaters.

Conclusions: Given the very small effect size, BE severity and status are not associated in a biologically meaningful manner with BF content and distribution.

Abbreviation: BE, binge eating; BED, binge eating disorder; BES, Binge Eating Scale; BF, body fat; BH, body height; BMI, body mass index; BW, body weight; CEM, coarsened exact matching; CI, confidence interval; GLM, generalized linear model; SAT, subcutaneous adipose tissue; VAT, visceral adipose tissue; WC, waist circumference

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Introduction

Approximately 17% of individuals starting a weight loss or maintenance program engage in binge eating (BE) (1), a behavior characterized by eating in a discrete period of time an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances, accompanied by a sense of lack of control over eating (2). According to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*, when such behavior occurs at least once weekly for 3 months without compensatory behavior, it is to be considered a binge eating disorder (BED) (2). BE is also a characteristic behavior of other eating disorders like bulimia nervosa, binge/purge anorexia nervosa, and other specified feeding or eating disorder (2). Women, young people, and people with obesity are at greater risk for BE (1).

Compared to non-binge eaters, binge eaters have a higher risk of obesity as the result of excess energy intake during binge episodes and the food consumed being highly palatable and typically high in sucrose and fat (3,4). Both obesity and the excessive consumption of such foods are risk factors for metabolic abnormalities. Several cross-sectional and longitudinal studies focused on the metabolic consequences of BE have shown that binge eaters are at greater risk for both cardiometabolic risk factors like dyslipidemia, hypertension, and impaired fasting glucose and chronic diseases like diabetes (5–10). However, whether this increased risk is attributable to BE behavior or is only a consequence of the greater prevalence of overweight and obesity in binge eaters remains a controversial and debated issue (11,12).

The total amount of fat mass and its abdominal distribution, as visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT), are known risk factors for cardiometabolic disease (13–15). Most studies show that VAT contributes more to

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cardiometabolic risk than SAT (13–15). However, information about the body composition of binge eaters is limited. Indeed, it is not known whether binge eaters have a different fat mass amount and distribution than non-binge eaters with similar body mass index (BMI). An evaluation of the BMI-independent relationship between BE and total and abdominal fat is useful to understand whether binge eaters have a body composition different from that of non-binge eaters, which could predispose them to greater cardiometabolic risk.

Our study was aimed at evaluating the contribution of BE severity and BE status to fat mass and its abdominal distribution in a large sample of participants with a wide range of BMI after adjustment for confounders known to affect both BE and body composition like sex, age, and nutritional status.

Materials and methods

Study design

We performed a cross-sectional study among 9421 consecutive Caucasian adults who self-referred to the International Center for the Assessment of Nutritional Status (University of Milan, Milan, Italy) between September 2010 and March 2017 in order to participate in a structured weight loss or weight maintenance program.

The statistical analysis was conducted using two different approaches:

Approach 1: Using regression modeling, we studied the association of BE severity with fat mass and its abdominal distribution in the whole sample.

Approach 2: Using a case-control design, participants identified as binge eaters were matched, for sex, age, and BMI, with non-binge eaters.

Study procedures

On the same day, the participants underwent a clinical examination, an anthropometric assessment, and an ultrasound measurement of VAT and SAT. A structured interview was performed to obtain information about marital status, education, smoking status, and structured physical activity. The latter was investigated asking participants the following questions: "Do you practice any structured physical activity?" and "How many hours per week do you spend on this activity?" Participants who spent $\geq$ 2 hours per week in any structured physical activity were considered as active (1). All patients completed the Binge Eating Scale (BES) questionnaire in order to evaluate the presence of BE (16,17). Excluded from the study were participants younger than 18 years; those with diagnosed infective, neurological, gastrointestinal, cardiac, renal, or pulmonary disease; those using medications known to cause lipodystrophy (e.g., steroids and antiretroviral agents); those with scars in the VAT measurement area; and those who were unable to understand and fill in the questionnaire. From the 9421 participants initially recruited, we excluded those with missing values on the BES questionnaire (n = 876, 9%) or on one of the variables of interest (n = 21). This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human participants were approved by the ethics committee of the University of Milan (report n. 23/2016). Written informed consent was obtained from all participants.

Anthropometric measurements

Anthropometric measurements were collected by trained registered dieticians following standard guidelines (18). Body weight (BW, kg) was measured to the nearest 100 g with a column scale (Seca 700 balance, Seca Corporation) and with participants wearing only light underwear and after bladder emptying. Body height (BH, cm) was measured to the nearest 0.1 cm using a vertical stadiometer. BMI was calculated using the formula: BMI (kg/m^2) = BW (kg)/BH^2 (m^2) and classified according to the World Health Organization cutoffs (19). Waist circumference (WC) was measured to the nearest 0.5 cm midway between the lower rib margin and the superior anterior iliac with a nonstretch tape (18). Skinfold thicknesses were measured using a Holtain Tanner/Whitehouse Skinfold Caliper (Holtain Ltd). Four skinfolds were measured: biceps, triceps, subscapular, and suprailiac. Each skinfold was measured 3 times and the mean was used for analysis. The intra-observer variation for the skinfold measurement ranged from 2.5% to 2.9%. Body density and body fat (BF, %) were estimated by Durnin and Womersley's (20) and Siri's formulas (21), respectively.

Abdominal ultrasonography

Abdominal ultrasonography was performed among fasting participants by the same operator using a Logiq 3 Pro instrument equipped with a 3.5-MHz convex-array probe and with a 7.5-MHz linear probe (GE Healthcare). VAT and SAT were measured 1 cm above the umbilicus. The examination was performed at end-expiration and applying a standardized probe pressure. SAT was measured with the 7.5-MHz linear probe as the distance between the epidermis and the external face of the rectus abdominis muscle; VAT was measured with the 3.5-MHz convex-array probe as the distance between the anterior wall of the aorta and the posterior surface of the rectus abdominis muscle (14,22). Each measurement was performed 3 times and the mean of the 3 measurements was used for analysis. The within-day intra-operator coefficient of variation for repeated measures of VAT and SAT in our laboratory is 0.8% (14,15,23).

Psychological assessment

Eating behavior was evaluated using the Italian version of the BES (16,17). The BES consists of 16 forced-choice questions, each with a set of 3 or 4 answers. The BES gives a score ranging from 0 to 46. Participants with a BES score $\geq$ 18 were identified as binge eaters (24).

Statistical analysis

Most continuous variables had non-Gaussian distributions, and all are reported as 25th, 50th, and 75th percentiles. Discrete variables are reported as counts and proportions. The

association of BE to fat mass and its abdominal distribution was assessed using two different approaches.

In approach 1, we evaluated the association between the BES score (continuous) and the outcomes of interest. We used a generalized linear model (GLM) with a Gaussian family, an identity link, and robust confidence intervals (CIs). This method does not assume that the dependent variable is normally distributed but assumes the normality of residuals (25,26). Sex (discrete; 0 = female, 1 = male), age (continuous, years), BMI (continuous, kg/m^2), marital status (discrete; 0 = single, widower, or separated, 1 = married/cohabiting), education (discrete; 0 = low degree [diploma or lower degree], 1 = high degree [graduated or higher degree]), smoking (discrete; 0 = nonsmoker, 1 = ex-smoker, 2 = smoker), and physical activity (discrete; 0 = no, 1 = yes) were considered as potential confounders.

In approach 2, we used a case-control design to evaluate the association between BE status (dichotomous) and the outcomes of interest. Coarsened exact matching (CEM) (27) was used to match binge eaters and non-binge eaters on sex (same), age (same), and BMI ($\Delta BMI_{max} = \pm 0.5$ kg/m^2). Continuous measurements of binge eaters and non-binge eaters were compared using a GLM with a Gaussian family, an identity link, and BE status (0 = no; 1 = yes) as predictors. Matching was taken into account using CEM-related weights and robust 95% CIs in all GLMs. Confounders were included in the models as described above under approach 1.

Marginal means were obtained from all models. A p value $\leq$ 0.05 was considered statistically significant. Statistical analysis was performed using STATA version 12.0 (StataCorp).

Results

Table 1 shows the sociodemographic, anthropometrical, and lifestyle characteristics of the 8524 participants who met the inclusion criteria according to the BE status. Table S1 reports the characteristics of the sample according sex. Overall, 72.2% of participants were women and 27.8% were men; 21.8% were normal weight, 38.4% were overweight, and 39.8% were obese. It was observed that 17.7% of participants were identified as binge eaters.

Table 2 shows the association between the BES score and the outcomes of interest. The BES score was significantly associated with WC and BF. A 1-point increment of the BES score was

Table 1. Sociodemographic, anthropometrical, and lifestyle characteristics of the recruited sample according to binge eating status.

	Non-Binge Eaters (n = 7017)			Binge Eaters (n = 1507)			Total (n = 8524)		
	P25	P50	P75	P25	P50	P75	P25	P50	P75
Age (years)	37	47	56	34	43	52	37	46	55
BMI (kg/m^2)	25.3	28.3	32.0	26.4	29.9	34.1	25.4	28.6	32.4
Waist circumference (cm)	86.4	95.6	106.0	87.5	98.0	107.8	86.5	96.0	106.3
VAT (cm)	3.3	4.9	7.3	3.4	4.8	6.9	3.3	4.9	7.2
SAT (cm)	1.9	2.6	3.5	2.2	3.0	3.9	2.0	2.7	3.5
Body fat (%)	32.8	37.4	40.9	35.0	39.0	42.1	33.1	37.7	41.2
BES score	4	8	12	19	22	26	5	9	15
	n	%		n	%		n	%	
Sex									
Women	4851	69.1		1303	86.5		6154	72.2	
Men	2166	30.9		204	13.5		2370	27.8	
Age classes									
18–19 years	91	1.3		32	2.1		123	1.4	
20–29 years	640	9.1		201	13.3		841	9.9	
30–39 years	1368	19.5		369	24.5		1737	20.4	
40–49 years	2029	28.9		455	30.2		2484	29.1	
50–59 years	1652	23.5		284	18.8		1936	22.7	
60–69 years	952	13.6		133	8.8		1085	12.7	
$\geq$ 70 years	285	4.1		33	2.2		318	3.7	
BMI classes									
Normal weight	1605	22.9		256	17		1861	21.8	
Overweight	2766	39.4		504	33.4		3270	38.4	
Obesity 1 class	1788	25.5		439	29.1		2227	26.1	
Obesity 2 and 3 class	858	12.2		308	20.4		1166	13.7	
Marital status									
Single	3230	46.0		809	53.7		4040	47.4	
Married	3787	54.0		698	46.3		4484	52.6	
Education									
Low degree	4072	58.0		951	63.1		5023	58.9	
High degree	2945	42.0		556	36.9		3501	41.1	
Smoking									
Nonsmoker	3785	53.9		771	51.2		4556	53.4	
Ex-smoker	1672	23.8		340	22.5		2012	23.6	
Smoker	1560	22.3		396	26.3		1956	23.0	
Physical activity									
No	3826	54.5		903	59.9		4729	55.5	
Yes	3191	45.5		604	40.1		3795	44.5	

P25 = 25th percentile; P50 = median/50th percentile; P75 = 75th percentile; VAT = visceral adipose tissue; SAT = subcutaneous adipose tissue; BES = Binge Eating Scale; BMI = body mass index.

Table 2. Association Between BES Score and Amount and Distribution of Body Fat

	WC (cm)	VAT (cm)	SAT (cm)	BF (%)
BES score	0.03***	0.00	0.00	−0.01**
	[0.02 to 0.05]	[−0.00 to 0.01]	[−0.00 to 0.00]	[−0.02 to −0.00]
Sex (male)	9.34***	2.19***	−0.30***	−8.53***
	[9.08 to 9.60]	[2.10 to 2.28]	[−0.35 to −0.25]	[−8.69 to −8.36]
Age (years)	0.16***	0.05***	−0.02***	0.15***
	[0.15 to 0.17]	[0.05 to 0.05]	[−0.02 to −0.02]	[0.14 to 0.15]
BMI (kg/m^2)	2.14***	0.28***	0.14***	0.61***
	[2.11 to 2.17]	[0.27 to 0.29]	[0.14 to 0.15]	[0.60 to 0.63]
Marital status (married)	0.32**	0.04	−0.02	0.53***
	[0.08 to 0.56]	[−0.04 to 0.12]	[−0.07 to 0.02]	[0.39 to 0.66]
Education (high degree)	0.47***	−0.19***	−0.02	−0.22**
	[0.24 to 0.69]	[−0.26 to −0.11]	[−0.06 to 0.02]	[−0.35 to 0.08]
Smoking (ex-smoker)	0.55***	0.09*	−0.04	−0.01
	[0.27 to 0.83]	[0.00 to 0.19]	[−0.09 to 0.01]	[−0.17 to 0.15]
Smoking (smoker)	0.68***	0.17***	−0.01	−0.22**
	[0.41 to 0.95]	[0.08 to 0.26]	[−0.06 to 0.04]	[−0.39 to −0.06]
Physical activity (yes)	−0.78***	−0.33***	−0.04	−0.55***
	[−1.00 to −0.55]	[−0.40 to −0.26]	[−0.08 to 0.00]	[−0.68 to −0.42]
Constant	23.88***	−5.69***	−0.27***	14.69***
	[23.03 to 24.73]	[−5.96 to −5.43]	[−0.43 to −0.12]	[14.16 to 15.23]
Observations	8524	8524	8524	8524

Values are regression coefficients and 95% confidence intervals in brackets.
BES = Binge Eating Scale; WC = waist circumference; VAT = visceral adipose tissue; SAT = subcutaneous adipose tissue; BF = total body fat; BMI = body mass index.
*$p < 0.05$. **$p < 0.01$. ***$p < 0.001$.

associated with an increment of 0.03 cm (95% CI, 0.02 to 0.05) in WC and with a reduction of 0.01% (95% CI, −0.02 to −0.00) in BF. No association was found between BE severity and VAT and SAT.

We then tested whether BE status was associated with fat mass and its abdominal distribution using a case-control approach. We performed CEM to match binge eaters and non-binge eaters for sex, age, and BMI. With this procedure, 1227 binge eaters were matched with 2718 non-binge eaters. As required by the study design, binge and non-binge eaters were similar in sex (89.3% vs. 89.3% female), age (43 years, 95% CI, 43 to 44 vs. 43 years, 95% CI, 43 to 44), and BMI (29.3 kg/m^2, 95% CI, 29.0 to 29.5 vs. 29.3 kg/m^2, 95% CI, 29.1 to 29.5).

Table 3 gives the GLMs used to test whether the amount of fat mass and the distribution of abdominal adipose tissue differed between binge and non-binge eaters. BF was 0.29% lower in binge eaters compared to non-binge eaters. No differences in VAT and SAT were observed.

The marginal means of the outcomes of interest in function of the BE status are reported in Table 4.

Discussion

In this study, we evaluated the contribution of BE severity and BE status on fat mass and its abdominal distribution. We found that BE severity was associated with WC and BF estimated by skinfold measurement, but not with ultrasound measurement

Table 3. Association Between Binge Eating Status and Amount and Distribution of Body Fat

	WC (cm)	VAT (cm)	SAT (cm)	BF (%)
Binge eating (yes)	0.12	0.05	0.02	−0.29**
	[−0.28 to 0.52]	[−0.07 to 0.17]	[−0.05 to 0.09]	[−0.50 to −0.07]
Sex (male)	9.58***	2.32***	−0.28***	−8.30***
	[8.95 to 10.21]	[2.08 to 2.55]	[−0.41 to −0.15]	[−8.73 to −7.88]
Age (years)	0.16***	0.05***	−0.02***	0.16***
	[0.14 to 0.18]	[0.04 to 0.05]	[−0.02 to −0.01]	[0.15 to 0.17]
BMI (kg/m^2)	2.16***	0.26***	0.16***	0.62***
	[2.11 to 2.22]	[0.25 to 0.28]	[0.15 to 0.17]	[0.59 to 0.65]
Marital status (married)	0.44	0.13	−0.11**	0.25*
	[−0.03 to 0.91]	[−0.00 to 0.26]	[−0.19 to −0.03]	[0.03 to 0.47]
Education (high degree)	0.45*	−0.19**	0.01	−0.24*
	[0.02 to 0.88]	[−0.31 to −0.06]	[−0.06 to 0.09]	[−0.46 to −0.01]
Smoking (ex-smoker)	0.71**	0.09	−0.02	−0.20
	[0.18 to 1.23]	[−0.08 to 0.26]	[−0.11 to 0.08]	[−0.47 to 0.08]
Smoking (smoker)	0.77**	0.19**	−0.07	−0.49***
	[0.26 to 1.28]	[0.05 to 0.33]	[−0.16 to 0.03]	[−0.76 to −0.21]
Physical activity (yes)	−0.29	−0.20**	−0.08	−0.46***
	[−0.71 to 0.13]	[−0.32 to −0.08]	[−0.16 to 0.00]	[−0.69 to −0.23]
Constant	23.44***	−5.12***	−0.77***	14.16***
	[21.83 to 25.05]	[−5.60 to −4.64]	[−1.07 to −0.48]	[13.22 to 15.10]
Observations	3945	3945	3945	3945

Values are regression coefficients and 95% confidence intervals in brackets.
WC = waist circumference; VAT = visceral adipose tissue; SAT = subcutaneous adipose tissue; BF = total body fat; BMI = body mass index.
*$p < 0.05$. **$p < 0.01$. ***$p < 0.001$.

Table 4. Body Fat Amount and Distribution of Abdominal Adipose Tissue in Function of Binge Eating Status

	WC (cm)	VAT (cm)	SAT (cm)	BF (%)
Non-binge eaters	95.2 [95.0 to 95.5]	4.9 [4.8 to 5.0]	3.0 [2.9 to 3.0]	38.2 [38.0 to 38.3]
Binge eaters	95.3 [95.0 to 95.6]	4.9 [4.9 to 5.0]	3.0 [2.9 to 3.0]	37.9 [37.7 to 38.1]
Observations	3945	3945	3945	3945

Values are predicted means and 95% confidence intervals in brackets obtained from multivariable generalized linear models.
WC = waist circumference; VAT = visceral adipose tissue; SAT = subcutaneous adipose tissue; BF = total body fat.

of VAT and SAT. It should be noted, however, that the effect size was very small, with a theoretical maximal difference between BES extremes of 1.4 cm for WC and 0.5% for BF. Moreover, when we compared binge eaters to non-binge eaters, the two groups differed only by 0.3% in BF. These statistically significant associations, which are lower than the measurement errors, are presumably caused by the large sample size (28) and are not of biological interest.

We obtained these results using two different approaches. The first approach allowed us to study the relationship between BE severity and adiposity parameters. The evaluation of relationships between continuous variables via a regression model avoids loss of information and power and is more biologically plausible than more or less arbitrary dichotomization (29). Nevertheless, a clinical audience may be more interested in knowing the differences attributable to BE status. Therefore, we have included a comparison between the two groups using a case-control design, controlling for known confounders influencing both BE and body composition.

Our findings agree with those of a previous study performed among a small sample of obese women (30). The authors found no difference in the anthropometric measurements and body composition between binge eaters and non-binge eaters and attributed this lack of relationship to the limited range of weight considered in the study (30). We found that, even increasing the BMI range (from normal weight to severe obesity), there is no difference in the amount of fat mass between the two groups. However, these results disagree with the findings of a previous work conducted among obese persons with BED, who had greater amounts of fat mass compared to obese persons without BED (31). This discrepancy may be explained by the fact that the authors focused on persons affected by BED, while BE is a behavior common in different eating disorders. Concerning abdominal adipose tissue, a longitudinal study found that women with a greater percentage of abdominal fat, measured by dual-energy x-ray absorptiometry, were at the highest risk for loss-of-control eating, one of the features of BE (32). However, we did not find any association of BE severity or status with the ultrasound measurements of VAT and SAT, similar to a previous cross-sectional study that found similar VAT and SAT areas between binge eaters and non-binge eaters (6). In addition to confirming, in a larger sample, that BE status does not affect body composition, our study shows that there is no relationship between the severity of BE and the total amount of fat mass and the thickness of VAT and SAT. Thus, there is no apparent pattern of body fat that would seem to place binge eaters at increased risk for metabolic abnormalities and chronic diseases as compared with non-binge eaters. This confirms the results of our previous work focused on assessing the relative contribution of BE to major cardiometabolic risk factors (12). Previous cross-sectional and longitudinal studies have reported a higher risk of dyslipidemia, hypertension, impaired fasting glucose, and diabetes in binge eaters compared to non-binge eaters (5–10). However, some of these studies did not consider BMI as a confounding factor. On the contrary, our study showed that after adjusting for nutritional status and some lifestyle variables, there was no association between BE severity and metabolic parameters, and binge eaters had the same cardiometabolic risk as non-binge eaters (12). Therefore, we speculate that BE increases the risk of obesity and this, in turn, increases cardiometabolic risk.

The main strength of this study is its large sample size. To the best of our knowledge, this is the largest study focused on assessing the association between BE and body composition. Second, different from previous studies that focused only on the contribution of BE status, we first tested the relationship between BE severity and adiposity parameters. Third, when we assessed the contribution of BE status, we matched binge eaters and non-binge eaters for sex, age, and BMI. This allowed better control of the confounders. Finally, we did not limit our analysis to fat mass but also investigated the effect of BE on the distribution of abdominal adipose tissue.

However, our study is not free of limitations. First, its cross-sectional nature does not allow the establishment of a cause–effect relationship. Second, we conducted the study in participants seeking a weight loss or maintenance program, and this could affect the generalizability of our results. Third, we did not use gold standard methods for the evaluation of total and abdominal fat. However, anthropometric measurements, skinfold measurement, and ultrasonography are the most used techniques in clinical practice and epidemiological studies. Fourth, BE was assessed using only a self-reported questionnaire, without a structured interview. This may have led to a less accurate identification of cases of BE. Fifth, we did not include some confounders in our analysis—for example, income—that may be associated with BE. Finally, as in any observational study, potential residual confounding could not be ruled out.

Conclusion

Binge eaters do not appear to have a different nutritional phenotype compared to non-binge eaters. Both total fat mass and abdominal fat distribution are similar between groups, suggesting the same cardiometabolic risk. However, the screening and the treatment of BE are issues of clinical relevance for the prevention of obesity and its metabolic comorbidities.

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Disclosure of potential conflicts of interest

No potential conflicts of interest were disclosed.

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Author contributions

AL, AB, and SB conceived and designed the experiments; AL, LV, RDA, VP, VB, and AV performed the experiments; AL managed and checked the data; AL and GB analyzed the data; and AL wrote the paper. All the authors have seen and approved the final version of the manuscript.

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