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Sex-specific association between later circadian timing of food intake and adiposity among Chinese young adults living in real-world settings

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Abstract

Timing of food intake is an emerging aspect of nutrition; however, there is a lack of research accurately assessing food timing in the context of the circadian system. The study aimed to investigate the relation between food timing relative to clock time and endogenous circadian timing with adiposity and further explore sex differences in these associations among 151 young adults aged 18–25 years. Participants wore wrist actigraphy and documented sleep and food schedules in real time for 7 consecutive days. Circadian timing was determined by dim-light melatonin onset (DLMO). The duration between last eating occasion and DLMO (last EO-DLMO) was used to calculate the circadian timing of food intake. Adiposity was assessed using bioelectrical impedance analysis. Of the 151 participants, 133 were included in the statistical analysis finally. The results demonstrated that associations of adiposity with food timing relative to circadian timing rather than clock time among young adults living in real-world settings. Sex-stratified analyses revealed that associations between last EO-DLMO and adiposity were significant in females but not males. For females, each hour increase in last EO-DLMO was associated with higher BMI by 0·51 kg/m^2 ($P = 0·01$), higher percent body fat by 1·05 % ($P = 0·007$), higher fat mass by 0·99 kg ($P = 0·01$) and higher visceral fat area by 4·75 cm^2 ($P = 0·02$), whereas non-significant associations were present among males. The findings highlight the importance of considering the timing of food intake relative to endogenous circadian timing instead of only as clock time.

Keywords: **Chrono-nutrition: Circadian rhythm: Food timing: Adiposity: Young adults**

Obesity represents a growing public health challenge[1], and it is particularly detrimental during early young adulthood as it increases the risk of developing chronic diseases later in life[2]. Obesity is typically defined through global metrics such as BMI, however, is a non-specific marker of body fat and provides no insight in body fat distribution[3]. Specifically, Asians generally have a higher body fat than white people of the same age, sex and BMI category[4]. Efforts to combat the obesity pandemic and its related metabolic diseases have mainly targeted traditional risk factors, e.g. excess energy intake and insufficient physical activity. However, traditional risk factors cannot fully explain the rapid rise of obesity during the past decades. Over the past 10 years, many causes of obesity and metabolic diseases have been identified, such as the timing of food intake[5–7].

Evidence for the importance of food timing for obesity comes from animal models[8–10] as well as observational[11,12] and experimental[13,14] studies in humans, although not all studies are consistent[15,16]. Previous observational studies linking late eating with higher obesity risk revealed that food timing per se might alter body weight without a significant difference in energy intake and expenditure[17,18]. However, most studies have utilised clock time to illustrate food timing[7], which fails to accurately characterise food timing in the context of the circadian timing system. It is well established that there are

Abbreviations: DLMO, dim-light melatonin onset; EO, eating occasion; FM, fat mass; VFA, visceral fat area.

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individual differences in endogenous circadian timing relative to clock hour[19,20], and that can vary by up to 10 h among college students[21]. This potential mismatch between clock and circadian timing may account for the contradictory associations between food timing and obesity in human studies.

Experimental evidence suggests the alignment between food timing and circadian rhythms, as with some forms of time-restricted eating or feeding, may prevent weight gain and promote metabolic health[22]. However, food timing relative to endogenous circadian timing has not yet been thoroughly examined in human studies. McHill and colleagues[21] examined the association between food timing relative to objective markers of circadian rhythms with adiposity in USA college students. The available evidence emphasises that it may be more physiologically relevant to consider the timing of food intake relative to endogenous circadian timing instead of only as clock time[21].

Sex differences in the circadian system have been well characterised. For example, females have a shorter intrinsic circadian period and an earlier circadian phase than males[23,24]. Given previous studies indicated that metabolic response to a meal depends on the timing of food intake relative to the individual's circadian rhythm[21,25,26], it is essential to explore the different metabolic responses to later circadian timing of food intake in males and females, which has not been investigated in previous research.

Hence, the objective of the present study was twofold: First, to examine the associations of food timing relative to clock time and endogenous circadian timing with general and visceral adiposity among Chinese college students and second to explore whether these associations differed by sex.

Methods

Participants

Participants were recruited from undergraduate students at local universities in Hefei, Anhui Province (China), using a multi-method approach spanning paper posters, flyers around campus and online advertisement (e.g. social media). The inclusion criteria included the following: (i) having the ability to download phone applications, (ii) not being engaged in a weight loss program, (iii) not taking any medication and (iv) not being engaged in night work within a 12-month period or traveling across time zones within a 3-month period before and during the procedure[27]. This study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects were approved by the Ethics Committee of the Anhui Medical University (No. 20180082). Written informed consent was obtained from all subjects.

Procedure

Pre-Laboratory procedures. Participants wore a wrist actigraphy and documented sleep and food schedules in real -time for 7 consecutive days.

Laboratory procedures. For calculation of circadian timing, participants were admitted to the laboratory for 1 night, arriving at approximately 14.00 h. After admission, participants completed the bioelectrical impedance analysis to assess adiposity. The lights were dimmed (< 10 lux) at 18.00 h. Salivary melatonin was sampled hourly from 19.00 h until 01.00 h. The following morning, participants were asked to void, and then their height and weight were measured in light clothing without shoes before eating or drinking anything. After completion of the laboratory procedures, participants were given breakfast and transported to school.

Measures

Demographics. At inclusion, participants filled in a set of questionnaires on socio-demographics and lifestyle, including age, sex, smoking status (current, former and never), alcohol status (current, former and never), consumption of sugar-sweetened beverages (never, 1–3 times/week, 4–6 times/week and ≥ 1 time/day) and screen time (< 2 h/d, 2–5 h/d and ≥ 5 h/d).

Dim-Light melatonin onset. Melatonin concentrations were determined from serial saliva samples (approximately 2 ml) collected hourly using Salivettes (Starstedt, Nümbrecht), beginning at 19.00 and ending at 01.00 h (seven measurements per subject) under dim-light conditions (< 10 lux), as verified using a portable illuminance meter (TES-1339R; Taishi Corp.). During the sample period, participants were seated in comfortable chairs, watched a dimmed TV (< 10 lux), talked to each other, had access to toilet facilities (also < 10 lux) and a research assistant was present at all times to ensure they did not fall asleep. Snacks and water were offered following each sampling, and participants rinsed their mouths with water 20 min before each sample collection. The saliva sample was centrifuged at 3300 ***g*** for 5 min. If there was less than 2 ml of saliva after centrifugation, the sample was centrifuged again at 3500 g for 5 min. Samples were frozen and stored at −80°C before being analysed. Melatonin levels were determined using a commercially available enzyme-linked immunosorbent assay kit from IBL (IBL International GmbH). Dim-light melatonin onset (DLMO) was calculated as the linear interpolated clock time at which evening salivary melatonin concentrations crossed and maintained above a 5 pg/ml threshold[27].

Clock time of food intake. Each participant was asked by Tencent Document (https://docs.qq.com/mall/index) to complete a 7-day (5 weekdays and 2 weekend days) ecological momentary assessment of food timing, based on a signal-contingent approach at semifixed intervals (i.e., 07.00–10.00, 10.00–13.00, 13.00–16.00, 16.00–19.00 and 19.00–22.00)[28]. To synchronise participants' food timing information while minimising the burden on participants, students received notifications five times a day via Tencent Document, and each data entry took approximately 1–2 min. Participants reported the start timing of breakfast, lunch, dinner and first and last eating occasion in real-time, with an eating occasion (EO) defined as food or drink containing any amount of calories[12]. Eating midpoint was defined as the midpoint between the first and last EO[29]. Eating jetlag was calculated in hours as follows: Eating midpoint on weekends − Eating midpoint on weekdays, and all

analyses were conducted using the absolute value of the estimated eating jetlag[29]. An experienced research staff instructed study participants on how to accurately complete the food records at the start of the study and reviewed these data with the participants every day to ensure the correctness of self-reported data.

Circadian timing of food intake. The duration between the average clock time of the last EO and DLMO (last EO-DLMO) was used to calculate the circadian timing of food intake. DLMO is a marker of the circadian phase and the beginning of an individual's biological night[30]. Therefore, the last EO-DLMO was conducted in an attempt to capture the EO that may have occurred during the biological night when energy expenditure was lower[31] and diet-induced thermogenesis tended to be lower as well[32].

Sleep. For 7 consecutive days, participants were instructed to wear an actigraphy (ActiGraph GT3X+) on the non-dominant wrist for 24 h per day (except when bathing or swimming) and to complete an electronic sleep diary. Sleep onset, offset, duration and sleep fragmentation index were manually scored using the Sadeh algorithm in ActiLife software version 6·13·3 (Actigraph) from actigraphy and corroborated with sleep diary times[33]. The midsleep point refers to the midpoint between actigraphy-derived sleep onset and offset.

BMI. BMI (kg/m^2) was estimated using laboratory measurements: height and weight (without shoes and light clothing) were taken using standard protocols, without eating or drinking after the morning void.

Body composition. Body composition, including percent body fat (%), fat mass (FM, kg) and visceral fat area (VFA, cm^2), were measured using an InBody S10 body composition analyser (Inbody Co., Seoul, Korea), a bioelectrical impedance analysis device that is portable, non-invasive and non-radiation. The VFA measured by bioelectrical impedance analysis using Inbody ® (Inbody Co., Seoul, Korea) demonstrated a good correlation with VFA observed by the gold standard method – computed tomography[34–36].

Analysis. Categorical variables were compared using the χ^2 test and continuous variables using paired or unpaired t tests. Possible linear and non-linear relationships between last EO (clock time/circadian timing) and adiposity were evaluated with multivariable-adjusted restricted cubic splines. Multiple linear regression models were used to test whether last EO (clock time/circadian timing) and sleep measures predicted adiposity. Further, sex-stratified analyses were performed to explore the sex differences of these associations. Scatter plots and Pearson correlations were used to examine the sex-specific association between later circadian timing of food intake with general and visceral adiposity. Age, sex, alcohol status, sugar-sweetened beverage consumption, screen time and sleep duration were included as covariates in multivariate linear regression models and restricted cubic splines models. Analyses were performed with R Software version 4·2·1 (R Foundation for Statistical Computing) and STATA 16 statistical software (Stata Corporation). Alpha < 0·05 (two-sided) was deemed statistically significant.

Results

General characteristics of the study participants

Of the 151 participants, 133 were included in the statistical analysis finally. Of the eighteen who were not included, five were not included because DLMO was not observed during the sampling period and thirteen did not have a complete 7-day food record (see Flow Chart, Fig. 1). The average age of the study population was 20·7 (SD, 0·8) years, 84 were females.

The main characteristics of study participants were presented by sex in Table 1. Compared with the male participants, female participants were more likely to have slightly earlier DLMO (female: 21:53, male: 22:06, $P = 0·40$) and last EO (clock time) of the day (19:40 *v.* 19:43, $P = 0·83$). When considering the circadian timing of last EO, females have the later circadian timing of last EO (i.e. last EO closer to DLMO) than males (–2·2 *v.* –2·4, $P = 0·61$). Female participants had higher percent body fat (28·0 *v.* 18·2, $P < 0·001$), FM (15·6 *v.* 12·8, $P = 0·012$) and VFA (65·9 *v.* 50·6, $P = 0·004$) than male participants, while male participants had higher BMI (20·9 *v.* 22·6, $P = 0·003$).

Linear and nonlinear associations between last eating occasion (clock time/circadian timing) with general and visceral adiposity

Multivariable-adjusted restricted cubic splines demonstrated significant positive linear associations between last EO-DLMO with adiposity (BMI: $P_{overall} = 0·03$, $P_{nonlinear} = 0·08$; percent body fat: $P_{overall} = 0·02$, $P_{nonlinear} = 0·35$; FM: $P_{overall} = 0·03$, $P_{nonlinear} = 0·21$; VFA: $P_{overall} = 0·02$, $P_{nonlinear} = 0·14$) (Fig. 2 (e)–(h)). In addition, sex-stratified analyses revealed that these linear associations were significant in females but not males (online Supplementary Fig. 1 (a)–(h)).

Association between last eating occasion (clock time/circadian timing) with general and visceral adiposity by sex

As shown in Table 2, the results demonstrated no association between the clock time of last EO with general and visceral adiposity, subanalyses after stratification for sex also revealed similar trends. However, when considering the circadian timing of last EO, significant associations were observed, such that individuals with their last EO closer to DLMO had higher BMI, percent body fat, FM and VFA (Table 2). Furthermore, sex-stratified analyses revealed that these associations were significant in females but not males (Fig. 3, Table 2). For females, each hour increase in last EO-DLMO was associated with higher BMI by 0·51 kg/m^2 (95 % CI: 0·12, 0·91; $P = 0·01$), higher percent body fat by 1·05 % (95 % CI: 0·30, 1·80; $P = 0·007$), higher FM by 0·99 kg (95 % CI: 0·21, 1·77; $P = 0·01$) and higher

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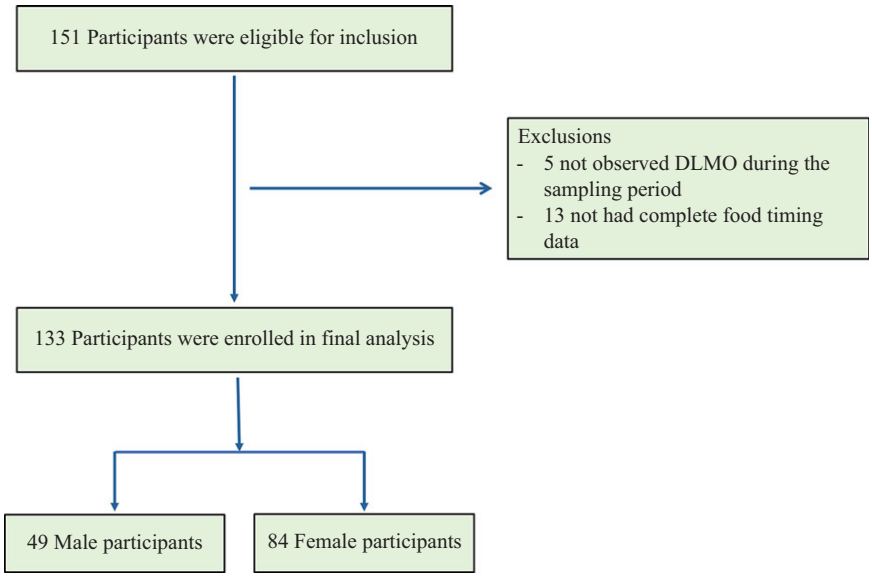

Fig. 1. Participant flow chart.

Table 1. Descriptive characteristics of participants by sex among 133 young adults (Numbers and percentages; mean values and standard deviations)

	Total (*n* 133)				Males (*n* 49)				Females (*n* 84)				
Characteristics	No.	%	Mean	SD	No.	%	Mean	SD	No.	%	Mean	SD	*P*
Age (years)	133		20·7	0·8	49		20·6	0·6	84		20·8	0·9	0·27
Alcohol status (current, %)	25	18·8			16	32·7			9	10·7			**0·002**
Sugar-sweetened beverages consumption (1–3 times/day, %)	104	78·2			35	71·4			69	82·1			0·08
Screen time (≥ 5 h/d, %)	119	89·5			44	89·8			75	63·0			0·93
Chronobiology													
DLMO (h:min)	133		21:58	1:22	49		22:06	1:22	84		21:53	1:21	0·40
Midsleep point (h:min)	133		3:52	0:32	49		4:01	0:26	84		3:47	0:35	**0·02**
Sleep duration (h)	133		7·3	0·7	49		7·1	0·7	84		7·4·	0·7	**0·02**
Eating timing													
First EO (h:min)	133		9:28	1:14	49		9:16	1:13	84		9:34	1:15	0·19
Last EO (h:min)	133		19:42	1:20	49		19:43	1:24	84		19:40	1:19	0·83
Last EO-DLMO (h)	133		−2·3	1·7	49		−2·4	1·9	84		−2·2	1·6	0·61
Eating midpoint (h:min)	133		14:35	1:01	49		14:30	1:05	84		14:37	0:58	0·50
Eating jetlag (h)	133		0·9	0·7	49		0·8	0·8	84		0·9	0·7	0·58
Adiposity													
BMI (kg/m^2)	133		21·6	3·2	49		22·6	3·4	84		20·9	2·9	**0·003**
PBF (%)	133		24·4	7·6	49		18·2	6·5	84		28·0	5·7	< **0·001**
FM (kg)	133		14·6	6·2	49		12·8	6·5	84		15·6	5·8	**0·012**
VFA (cm^2)	133		60·2	29·7	49		50·6	29·1	84		65·9	28·8	**0·004**

PBF, percent body fat; FM, fat mass; VFA, visceral fat area; DLMO, dim-light melatonin onset; EO, eating occasion; Last EO-DLMO, local time of the last EO minus DLMO.
Data are mean (SD) or *n* (%) for continuous and categorical variables, respectively. For last EO-DLMO, higher values denote last EO closer to, or after, DLMO.
Bold font is used to indicate statistically significant *P* value ($P < 0·05$).

VFA by 4·75 cm^2 (95 % CI: 0·84, 8·65; $P = 0·02$), whereas no similar associations were observed among males.

Association between sleep duration and sleep quality with general and visceral adiposity by sex

The results of the multiple linear regression models examining the associations of sleep duration and sleep quality with general and visceral adiposity are displayed in online Supplementary Tables 1 and 2. There were no significant associations between sleep duration and sleep quality with adiposity and results of stratified analyses by sex were consistent with overall results (both $P > 0·05$).

Discussion

The findings demonstrated that the timing of food intake relative to endogenous circadian timing rather than clock time was associated with general and visceral adiposity among young adults living in real-world settings. Strong linear associations between later circadian timing of food intake and adiposity were found among young females but not males. The findings

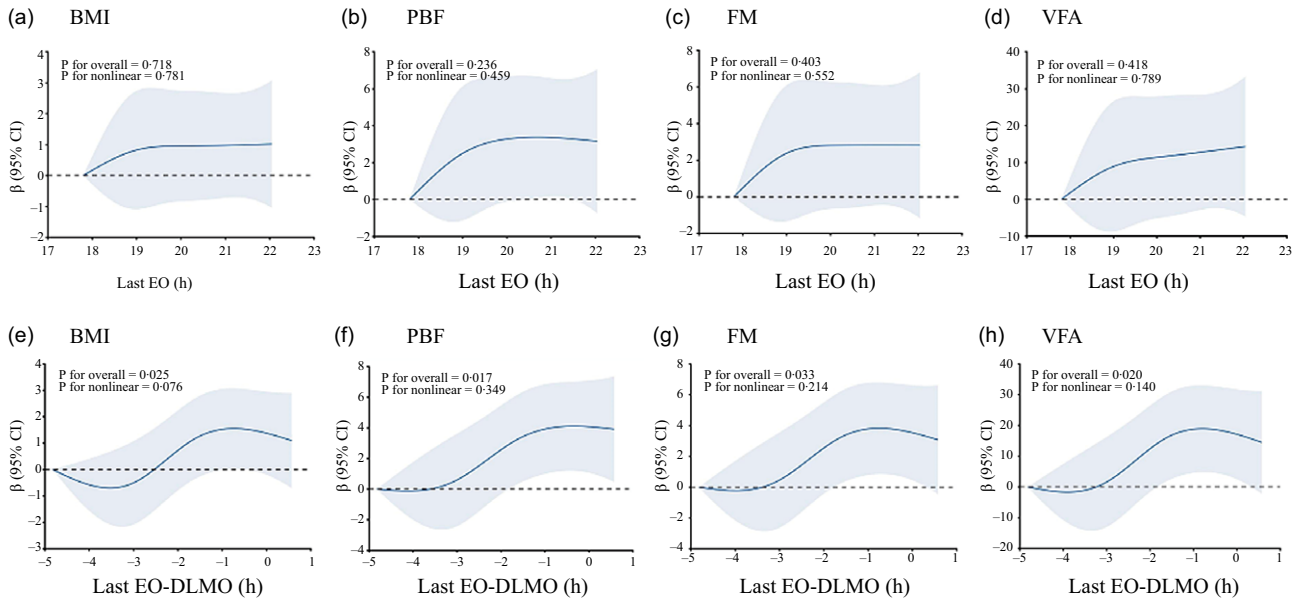

Fig. 2. Restricted cubic splines representing the association between last EO (clock time/circadian timing) with general and visceral adiposity among 133 young adults. Heavy central line represents the estimated adjusted beta, with shaded ribbons denoting 95 % confidence interval. For last EO-DLMO, higher values denote last EO closer to, or after, DLMO. Splines were adjusted for age, sex (female, male), alcohol status (current, former and never), sugar-sweetened beverages consumption (never, 1–3 times/week, 4–6 times/week and ≥ 1 time/d), screen time (< 2 h/d, 2–5 h/d and ≥ 5 h/d) and sleep duration (h). DLMO, dim-light melatonin onset; EO, eating occasion; FM, fat mass; Last EO-DLMO, local time of the last EO minus DLMO; PBF, percent body fat; VFA, visceral fat area.

Table 2. Association between last EO (clock time/circadian time) with general and visceral adiposity by sex among 133 young adults (Unstandardised beta and 95 % confidence intervals)

	Total (*n* 133)			Males (*n* 49)			Females (*n* 84)		
Adiposity	B	95 % CI	*P*	B	95 % CI	*P*	B	95 % CI	*P*
Last EO (clock time)									
BMI (kg/m^2)	0·19	−0·21, 0·60	0·35	−0·23	−1·00, 0·55	0·55	0·47	−0·02, 0·96	0·06
PBF (%)	0·65	−0·12, 1·43	0·10	0·47	−0·98, 1·93	0·52	0·83	−0·12, 1·77	0·09
FM (kg)	0·54	−0·26, 1·33	0·18	0·01	−1·46, 1·47	0·99	0·94	−0·03, 1·91	0·06
VFA (cm^2)	2·96	−0·79, 6·71	0·12	0·67	−5·88, 7·21	0·84	4·83	0·03, 9·64	0·05
Last EO–DLMO (circadian timing)									
BMI (kg/m^2)	0·33	0·02, 0·65	**0·04**	0·16	−0·43, 0·74	0·59	0·51	0·12, 0·91	**0·01**
PBF (%)	0·88	0·28, 1·48	**0·004**	0·85	−0·22, 1·92	0·12	1·05	0·30, 1·80	**0·007**
FM (kg)	0·75	0·14, 1·37	**0·02**	0·62	−0·47, 1·71	0·26	0·99	0·21, 1·77	**0·01**
VFA (cm^2)	3·67	0·74, 6·61	**0·02**	3·07	−1·76, 7·90	0·21	4·75	0·84, 8·65	**0·02**

PBF, percent body fat; FM, fat mass; VFA, visceral fat area; DLMO, dim-light melatonin onset; EO, eating occasion; Last EO-DLMO, local time of the last EO minus DLMO; B = unstandardised beta.
Adjusted for age, sex (female, male), alcohol status (current, former and, never), sugar-sweetened beverages consumption (never, 1–3 times/week, 4–6 times/week and ≥ 1 time/day), screen time (< 2 h/d, 2–5 h/d and ≥ 5 h/d) and sleep duration (h). For last EO-DLMO, higher values denote last EO closer to, or after, DLMO.
Bold font is used to indicate statistically significant *P* value ($P < 0·05$).

highlight the importance of considering the timing of food intake relative to endogenous circadian timing instead of only as clock time. Nutrition strategies aimed to improve metabolic health should consider the circadian timing of food intake, particularly as our society moves toward personalised healthcare strategy.

Research on associations of food timing with adiposity is scant among young adults and has produced mixed results[37]. One possible reason is that the clock time of food intake depends more on cultural and environmental factors such as work and school schedules, which may not accurately reflect the physiological impact of food timing on health. The present study indicated that later circadian timing of food intake (i.e. the timing of food intake relative to DLMO) was linked with higher general and visceral adiposity, while there were no differences when expressed in clock hours. These findings are consistent with a recent study conducted by McHill *et al.*[21] and support the notion that examining the circadian timing of food intake rather than clock time could be more relevant when investigating associations with adiposity in young adults.

One potential mechanism for increased adiposity in response to later circadian timing of food intake is through effects on the thermic effect of food. Thermic effect of food is the energy expenditure in response to food intake[32,38,39], which is lower in the biological evening than morning due to the influence of the circadian system[32]. Given the association of adiposity with food timing relative to circadian timing rather than clock time, food

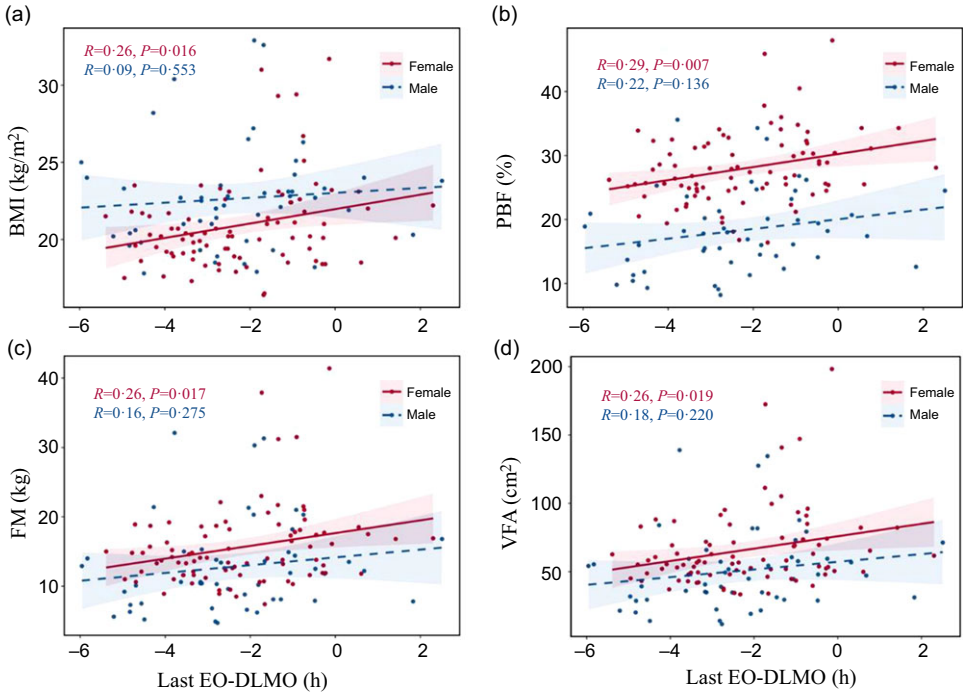

Fig. 3. Scatter plots showing correlation between last EO-DLMO with BMI (a), PBF (b), FM (c) and VFA (d) by sex among 133 young adults. Pearson's correlation coefficients and *P* value for each analysis are shown in the corresponding graph. For last EO-DLMO, higher values denote last EO closer to, or after, DLMO. DLMO, dim-light melatonin onset; EO, eating occasion; FM, fat mass; Last EODLMO, local time of the last EO minus DLMO; PBF, percent body fat; VFA, visceral fat area.

intake closer to or after DLMO is a decrease in thermic effect of food, contributing to a positive energy balance and weight gain over time.

Another explanatory hypothesis is that the concurrence of food intake with elevated melatonin concentrations increases the probability of impaired glucose tolerance[40,41]. It is known that postprandial glucose concentrations are higher in the biological evening than in the biological morning[42,43], and melatonin may further suppress the release of insulin in the biological evening[44]. Hence, eating earlier in the daytime to align with circadian rhythms may be preferred for improved blood glucose management, body weight control and related health outcomes, which will limit the concurrence of elevated glucose levels (due to postprandial state) and melatonin concentrations. Furthermore, later circadian timing of food intake may lead to a desynchronisation between the central and peripheral clocks and possibly result in metabolic disturbances[45,46]. In addition to the function of the circadian system, pathway analyses of the adipose tissue gene expression profiles revealed that late (circadian) eating modified lipid metabolic pathways in a direction consistent with increased adipogenesis and reduced lipolysis, contributing to increased lipid storage[47].

An interesting observation of the present study is the sex-specific association between later circadian timing of food intake with higher general and visceral adiposity in females but not males. One possible explanation for this observation is that the different metabolic response depends on the timing of food intake relative to an individual's circadian system. Duffy and colleagues[24] precisely examined the intrinsic circadian period in 157 healthy adults (33·1 % female) who underwent the gold-standard forced desynchrony protocol, and the results demonstrated that females have shorter intrinsic circadian periods than males and a greater proportion of females had intrinsic circadian periods shorter than 24 h. These sex-related circadian timing may explain the differential metabolic effects of food schedules among females *v.* males. However, whether and how sex modulates the adverse metabolic effects of later circadian timing of food intake is far less investigated. Further studies are needed to confirm these findings and investigate the underlying mechanism.

The absence of associations between sleep duration and sleep quality with adiposity in our results was unexpected, because insufficient sleep and poor sleep quality have been linked to higher weight[48–51]. However, individuals with insufficient sleep may tend to increase food intake later in the day[52–54] and later timing of food intake (closer to the biological night) is related to weight gain and obesity[21].

Limitations of the study should be noted. There were no repeated assessments of circadian timing, thereby limiting interpretations of potential changes in circadian alignment with food timing, though DLMO is an extremely precise measure of circadian timing, and robust stability in DLMO time has been demonstrated in young adults living in real-world settings[55]. A further key limitation of this study is the lack of data on dietary composition, which would have enhanced the analysis. Energy intake, macronutrient composition of the diet and overall diet quality may also interact synergistically with later circadian timing of food intake to influence adiposity[18], so that the

observed effects may partly elated to the later circadian timing of food intake. However, the 7-day ecological momentary assessment of food records in this study provides more reliable and accurate data on food timing than questionnaire methods about usual mealtimes[56]. In addition, we acknowledge that the population of university students may not be representative of the entire population in circadian timing of food intake. Older populations are more likely to have an earlier endogenous circadian phase and therefore may be more vulnerable to the effect of eating at a later clock time[57] because it would be closer to their earlier DLMO. Other populations with known late eating at biological night are night shift workers, who have an increased risk of obesity[58] and metabolic syndrome[59]. Examination of the timing of food intake relative to DLMO in diverse populations is needed to extend our findings. Finally, the sample sizes differed between males and females in the current study, with males having a smaller sample size relative to females, which may affect the associations between later circadian timing of food intake and adiposity in males. Although the sex-specific association between later circadian timing of food intake with higher general and visceral adiposity is an important observation, comparable groups of males and females and longitudinal follow-up studies are needed to demonstrate the causality and potential mechanisms underlying this relationship.

Conclusions

In summary, the synthesis of real-world observations and precise laboratory data in the present study identified a new potential risk factor for weight gain and disease: food intake relative to endogenous circadian timing. The finding is particularly important for young adults and even teenagers, whose behaviour tends to be more evening-oriented. Future studies are needed to examine the interactions between circadian timing of food intake and energy intake, macronutrient profile and diet quality to influence weight trajectories, energy balance and metabolism over time. Furthermore, the sex-specific association observed in this study highlights an important, but under-explored, area of research – sex differences in later circadian timing of food intake effects, as well as the need to further investigate the underlying mechanisms.

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Supplementary material

For supplementary material/s referred to in this article, please visit https://doi.org/10.1017/S0007114524001636

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