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Original article

Heritability of the timing of food intake

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SUMMARY

Background & aims: While environmental factors are presumed to be primary drivers of food timing, preliminary evidence suggests that genetics may be an additional determinant. The aim was to explore the relative contribution of genetics and environmental factors to variation in the timing of food intake in a Spanish twin population. Because chronotype, bedtime and wake time are related to food timing, covariance with food timing was further assessed.

Methods: In this observational study, 53 pairs of adult (mean (SD) = 52 (6.03) years) female twins (28 monozygotic; 25 dizygotic) were recruited from the Murcia Twin Register. Zygosity was determined by DNA-testing. Timing of the three main meals of the day was assessed via 7-day dietary records, and the midpoint of food intake was computed by calculating the midpoint between breakfast and dinner times. Chronotype, bedtime and wake time were self-reported. Heritability of food timing and related traits were estimated by comparing monozygotic and dizygotic twin correlations and fitting genetic structural equation models to measured variables.

Results: We observed genetic influences for food timing, with highest heritability for the midpoint of food intake (64%) in an overweight/obese population (BMI = 26.01 ± 3.77). Genetic factors contributed to a higher degree to the timing of breakfast (56%) than the timing of lunch (38%) or dinner (n.s.). Similarly, heritability estimates were larger in related behavioral traits earlier on in the day (i.e. wake time, (55%)), than those later on in the day (i.e. bedtime, (38%)). Bivariate analyses revealed a significant genetic overlap between food timing and bedtime and chronotype (rG between 0.78 and 0.91).

Conclusions: Genetic influences appear to account for a significant proportion of the variability in food timing, particularly breakfast. Thus, interventions related to food timing may be more effective when targeting afternoon/evening traits, such as lunch or dinner times. Furthermore, our data suggest shared genetic architecture underlying food timing and phenotypically related traits.

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Abbreviations: CLOCK, Circadian Locomotor Output Cycles Kaput; MZ, Monozygotic; DZ, Dizygotic; MTR, Murcia Twin Register; BMI, Body Mass Index; ME, Morningness-Eveningness; SEM, Structural Equation Models; FIML, Full Information Maximum Likelihood.

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1. Introduction

Secular trends from national surveys indicate shifts in the timing of food intake toward later timing [1]. This late eating habit has been associated with adverse health outcomes such as estimated higher odds of being overweight/obese [2,3] and impaired glucose tolerance and insulin secretion [4,5]. Moreover, later consumption of the main meals of the day, as determined by self-reported food timing, has also been shown to hinder weight loss during a dietary intervention [6,7] and following bariatric surgery [8]. Adverse effects of later meals have also been suggested by experimental studies. In randomized, crossover studies, it was shown that a later lunch decreases resting-energy expenditure, fasting carbohydrate oxidation and glucose tolerance [5,9,10], later dinner times worsens postprandial glucose profiles for the following morning's breakfast [11], and later consumption of the main meal of the day inverts the salivary microbiota 24-rhythm [12]. Moreover, to include a high-energy breakfast plus a low-energy dinner reduced metabolic risk compared with a meal pattern with a low-energy breakfast plus a high-energy dinner [13].

These recent findings emphasize the importance of food timing as a novel dimension in nutrition science [6,14,15]. Indeed, the timing of food intake is newly proposed as a modifiable risk factor for weight management and chronic disease prevention [16]. As food timing is likely a complex trait, like food composition [17], elucidating the genetic and environmental components that contribute to the variability in food timing for individual eating episodes is necessary. Unraveling those components is relevant in designing more effective and individually tailored therapeutic strategies related to food timing and developing public health initiatives tackling later food intake and understanding biological pathways regulating decisions related to food timing [18,19]. Whereas environmental determinants of food timing such as chronotype, caloric density [20], and sleep [21–24], have been explored in epidemiological studies, genetics remains under-investigated [9].

Thus far, only a single study has investigated the heritability of food timing [25]. In that twin study from the United States, the highest heritability was observed for the timing of breakfast (24%), while lunch and dinner timing showed lower heritability estimates (ranging from 18 to 22%) [25]. Other related studies provide additional support for the putative genetic component of food timing. For instance, genetic influences have also been suspected for night eating syndrome (NES) and sleep-related eating disorder (SRED), two eating disorders with evening eating preference [26]. We have previously reported an association between a genetic variant in *CLOCK* (rs4580704) and lunch time [6]. Moreover, we reported that food timing modifies the association between a genetic variant in the *PLIN* locus and the efficacy of a weight loss intervention [27]. In addition, no study to our knowledge has investigated the genetics of food timing along with closely related heritable traits that may explain the metabolic implications of later food intake and unravel shared genetic architecture among those traits.

Findings from twin studies have indicated that genetics plays a major role in several diet-related phenotypes including energy and macronutrient intakes, dietary patterns, and the intake of specific foods [28]. Twins provide a naturally unique case–control experiment whereas the classical twin design compares the similarity of identical/monozygotic (MZ) and dizygotic (DZ) twins. Genetics are implicated in the investigated trait when MZ twins are observed to be considerably more similar than DZ twins. The aim of our current investigation was to explore the relative contribution of genetics and environmental factors to variation in the timing of the three main meals of the day (i.e., breakfast, lunch and dinner) in a twin population. Because chronotype, bedtimes and wake times are related to food timing, co-variation with these traits was further assessed.

2. Methods

2.1. Subjects

In this observational study, a sample of female twins selected from the Murcia Twin Register (MTR) participated in this study. The MTR is a population-based registry of people born between 1940 and 1966 in the region of Murcia, southeast Spain. The twin pairs that form the MTR are assumed to be representative of the general Spanish population [29]. The registry has collected information from >2200 individual twins. More detailed description regarding characteristics and procedures of the MTR can be found elsewhere [30,31]. Written informed consent was obtained from all participants. The Committee of Research Ethics of the University of Murcia has approved MTR data collection procedures and management; the protocol follows national regulations regarding personal data protection.

Using a regional health system database, female pairs living within the same geographical area, and within a 30-km radius from the recruitment center, and free from severe health condition that may impede or hinder participation such as cognitive disorders, diabetes mellitus, chronic renal failure, hepatic diseases or cancer were selected for inclusion in this study. A total of 118 twin pairs were recontacted (between 2012 and 2014), and a total of 53 pairs of adult female twins ($n = 106$) volunteered for this study (28 MZ; 25 DZ). This sample size has been shown to be enough to assess the heritability of chronotype and other related features [32,33]. Zygosity was confirmed by DNA testing.

2.2. Timing of food intake

The primary outcome of the present study was the timing of food intake. The timing of food intake was self-reported *via* a 7-day food record. Specifically, participants recorded the start time, finish time, and duration of individual food intake episodes during 5 weekdays and 2 weekend days. Midpoint of intake was ascertained by calculating the midpoint between breakfast and dinner times (first and last eating episode). Participants were instructed and trained on how to accurately complete the food records at the start of the study, and collected data were later reviewed with a technician.

2.3. Sleep and chronotype

Participants also recorded information related to sleep including bed-time and wake-time during the same 7-day period. Chronotype was assessed using the Morningness-Eveningness (ME) questionnaire, a 19-item scale developed by Horne and Östberg, and an ME score was computed [34]. A higher ME score reflects more morning (earlier) chronotype.

2.4. General characteristics of the sample/subjects and procedures

Body weight was estimated in barefooted participants wearing light clothes using a digital scale accurate to the nearest 0.1 kg. Height was determined using a portable stadiometer (rank, 0.14–2.10) and participants were positioned upright, relaxed, and with the head in the Frankfort plane. Body Mass Index (BMI) was calculated by weight (kg) divided by height (m^2). Total body fat was determined by bioelectrical impedance, using TANITA TBF-300 (Tanita Corporation of America, Arlington Heights, IL, USA) equipment. In addition, waist to hip ratio was calculated using waist

circumference (cm), at level of the umbilicus, and hip circumference (cm) [35].

2.5. Statistical analyses

First, differences between MZ and DZ general characteristics were assessed by *t*-test. Heritability analysis was based on the basic logic of twin studies and can be summarized as follows: MZ twins (identical) share 100% of their genetic makeup, while DZ twins (non-identical) share on average 50% of their segregating genes [36]. Comparing the resemblance (correlation) of MZ twins for a trait with the resemblance of DZ twins for that trait the total variance of a trait can be partitioned into genetic and environmental factors, following a variance components approach. Observed MZ and DZ correlations generally reflect a combination of additive (A; i.e., summed allelic effects across multiple genes) and non-additive (D; i.e., genetic dominance, possibly including epistasis) genetic factors, as well as shared (C; i.e., common/family environment) and individual (E; i.e., idiosyncratic experiences, including measurement error) environmental factors. A greater phenotypic resemblance in MZ twin pairs compared with DZ twin pairs must be due to genetic influences (A or D components), considering the assumption that both MZ and DZ twins are exposed to equal shared environments during childhood [37]. It is not possible to estimate C and D simultaneously in a classical twin model and the choice of modeling C or D depends on the pattern of MZ and DZ correlations; usually C is estimated if the DZ twin correlation is more than half of the MZ twin correlation (ACE model), and D is estimated if the DZ twin correlation is less than half of the MZ correlation (ADE model) [38].

Structural equation models (SEM) offer a precise way to estimate the variance explained by each of the latent components (A, C, D and E) and determines the combination that best matches the observed data. For each variable, the full models (ACE/ADE) were estimated and tested against nested sub-models, where A component, C/D component or both (AC/AD) were fixed to zero. The log-likelihood ratio test (LRT) was used to compare the fit of the different models and sub-models. The difference in *minus* two times the log-likelihood (−2LL) between two models has a $\chi 2$ distribution with the degrees of freedom (df) equaling the difference in df between the two models. Additionally, model fit was evaluated using Akaike's information criterion (AIC) which is a parsimony-adjusted statistic used to select among competing models.

In the present study, all SEM were fitted to the raw data employing the full information maximum likelihood (FIML) method within the Open-Mx package v2.7.9 [39] for R v3.3.3 [40]. The accuracy of the obtained parameters was assessed using likelihood-based 95% confidence intervals. Effect of age was regressed out from the raw scores using also the FIML procedure in Open-Mx. Subsequently, SEM were fitted to the residual scores. Data preparation and descriptive analyses were performed in SPSS v19 [41].

3. Results

The MTR population included in the present study comprised of 53 adult female twin pairs ($n = 106$) with overweight/obesity (BMI = 26.01 ± 3.77) and their general characteristics are presented in Table 1. Mean age of the selected participants was 52 years (SD: 6.0; Range: 46–69). Mean timing of food intake was 8:43 ± 00:53 for breakfast, 14:53 ± 00:31 for lunch, and 21:29 ± 00:41 for dinner. The mean midpoint of intake was estimated at 15:20 ± 00:36. Significant weekday and weekend differences were observed for breakfast timing only. The timing of breakfast was significantly earlier on weekdays (8:33 ± 1:03) compared to weekends (9:12 ± 1:06) ($P = 0.001$). No significant differences were observed between MZ and DZ twins for food timing. Furthermore, no differences were observed between the two groups for anthropometric measures, sleep timing, and chronotype.

Table 1
General characteristics of 53 twin pairs.

	Monozygotic (n = 56)	Dizygotic (n = 50)	*p* values
Age (years)	51 ± 6	53 ± 6	0.066
Weight (kg)	64.12 ± 8.56	63.44 ± 7.91	0.370
Height (cm)	156.43 ± 6.84	157.52 ± 5.61	0.369
BMI (kg/m)	26.30 ± 3.89	25.66 ± 3.65	0.404
Body fat (%)	32.99 ± 5.89	32.96 ± 6.72	0.979
Waist (cm)	90.56 ± 8.76	90.08 ± 10.66	0.805
Hip (cm)	103.68 ± 7.15	102.39 ± 8.10	0.379
WHR	1.15 ± 0.06	1.14 ± 0.09	0.742
Timing of food intake			
Breakfast	08:49 ± 00:54	08:36 ± 00:52	0.209
Lunch	14:31 ± 00:33	14:32 ± 00:30	0.904
Dinner	21:36 ± 00:40	21:22 ± 00:41	0.072
Midpoint of intake	15:16 ± 00:32	15:23 ± 00:40	0.335
Sleep			
Wake-time (hh:mm)	07:33 ± 01:09	07:38 ± 01:00	0.684
Bed-time (hh:mm)	24:18 ± 00:56	24:28 ± 00:59	0.288
Chronotype score (MEQ)	55.21 ± 8.67	56.44 ± 7.56	0.442

Data are represented as means ± SD.
Abbreviations: BMI, body mass index, WHR, waist-to-hip ratio, MEQ, morning-evening questionnaire.

MZ twins showed higher intra-pair correlations than DZ twins for breakfast and lunch timing, but not for dinner timing. In addition, MZ twins showed higher intra-pair correlations than DZ twins for wake and bed times and chronotype (Table 2). AE models, where phenotypic variance is explained by additive genetic and non-shared environmental factors, showed the best fit in every case. The only exception was for dinner timing, where a CE model showed a better fit accordingly to the higher DZ correlation compared to MZ (Table 3).

Higher heritability was observed for investigated traits made earlier on in the day (Fig. 1). Indeed, heritability was higher for the timing of breakfast (56%) compared to lunch (38%), and the timing of dinner was not determined to be heritable. Similarly, the heritability of wake time was higher (55%) compared to bedtime (38%).

Table 2
Twin intra-pair correlations with 95% CI for timing of food intake and related traits.

	Intra-pair correlation coefficients	
	r MZ (CI 95%)	r DZ (CI 95%)
Timing of food intake		
Breakfast	0.56 (0.26, 0.74)	0.29 (−0.12, 0.59)
Lunch	0.40 (0.06, 0.63)	0.15 (−0.26, 0.50)
Dinner	0.36 (−0.03, 0.63)	0.42 (0.08, 0.66)
Midpoint of intake	0.64 (0.39, 0.79)	0.44 (−0.16, 0.61)
Sleep		
Wake-time	0.54 (0.26, 0.73)	0.37 (−0.06, 0.65)
Bed-time	0.42 (0.10, 0.65)	0.02 (−0.36, 0.40)
Chronotype score (MEQ)	0.42 (0.11, 0.64)	0.23 (−0.22, 0.57)

r MZ: monozygotic intra-pair correlation coefficient, r DZ: dizygotic intra-pair correlation coefficient, CI (95%): confidence interval, MEQ: Morning-Evening Questionnaire.

Table 3
Model-fitting results for univariate models, and proportions of variance (parameter estimates) explained by additive genetic influences (A), shared-environmental (C) and residual variation (E) with 95% confidence intervals (CI).

	Goodness-of-fit index							Parameter estimates (CI = 95%)		
	Model	*−2LL*	*df*	*AIC*	ΔX^2	Δ*df*	*p*	A	C/D	E
Breakfast timing	ACE	258.11	101	56.11	–			0.53 (0, 0.74)	0.02 (0, 0.59)	0.45 (0.26, 0.74)
	AE	**258.12**	**102**	**54.12**	**0.003**	**1**	**0.954**	**0.56 (0.28, 0.74)**	–	**0.44 (0.26, 0.72)**
	CE	259.78	102	55.78	1.67	1	0.196			1
	E	271.01	103	65.01	12.89	1	<0.001			1
Lunch timing	ADE	155.69	101	−46.31				0.21 (0, 0.62)	0.19 (0, 0.63)	0.60 (0.37, 0.92)
	AE	**155.74**	**102**	**−48.26**	**0.05**	**1**	**0.830**	**0.38 (0.07, 0.62)**	–	**0.62 (0.38, 0.93)**
	E	161.46	103	−44.54	5.72	1	0.017			1
Dinner timing	ACE	205.29	101	3.29	–			0 (0, 0.60)	0.39 (0, 0.60)	0.61 (0.37, 0.86)
	AE	206.97	102	2.97	1.68	1	0.195	1		
	CE	**205.29**	**102**	**1.29**	**< 0.01**	**1**	**1**	–	**0.39 (0.14, 0.59)**	**0.61 (0.40, 0.86)**
	E	214.17	103	8.17	8.88	1	0.003			1
Midpoint of intake	ADE	168.56	101	−33.44				0.57 (0, 0.79)	0.07 (0, 0.85)	0.36 (0.21, 0.60)
	AE	**168.57**	**102**	**−35.43**	**0.008**	**1**	**0.930**	**0.64 (0.40, 0.79)**	–	**0.36 (0.21, 0.60)**
	E	187.79	103	−18.21	19.225	1	<0.0001	–	–	1
Wake-time	ACE	299.92	101	97.92	–			0.34 (0, 0.72)	0.20 (0, 0.64)	0.46 (0.27, 0.73)
	AE	**300.17**	**102**	**96.17**	**0.25**	**1**	**0.618**	**0.55 (0.29, 0.73)**	–	**0.45 (0.27, 0.70)**
	CE	300.64	102	96.64	0.72	1	0.395			1
	E	314.24	103	108.24	14.08	1	<0.001			1
Bed-time	ADE	287.83	101	85.83				0 (0, 0.61)	0.42 (0, 0.65)	0.58 (0.35, 0.91)
	AE	**288.44**	**102**	**84.44**	**0.61**	**1**	**0.436**	**0.38 (0.06, 0.63)**	–	**0.62 (0.37, 0.94)**
	E	293.80	103	87.80	5.36	1	0.021			1
Chronotype score (MEQ)	ACE	729.27	101	527.27	–			0.38 (0, 0.64)	0.04 (0, 0.55)	0.58 (0.35, 0.88)
	AE	**729.28**	**102**	**525.28**	**0.01**	**1**	**0.922**	**0.43 (0.13, 0.64)**	–	**0.57 (0.35, 0.86)**
	CE	729.90	102	525.90	0.62	1	0.430			1
	E	736.99	103	530.99	7.09	1	0.008			1

−2LL: twice negative log-likelihood; *df*: degrees of freedom; AIC: Akaike Information Criterion; ΔX2: difference in X2 to full model; Δ*df*: difference in degrees of freedom to full model. Bold values indicate best fitting model, MEQ: morning-evening questionnaire.

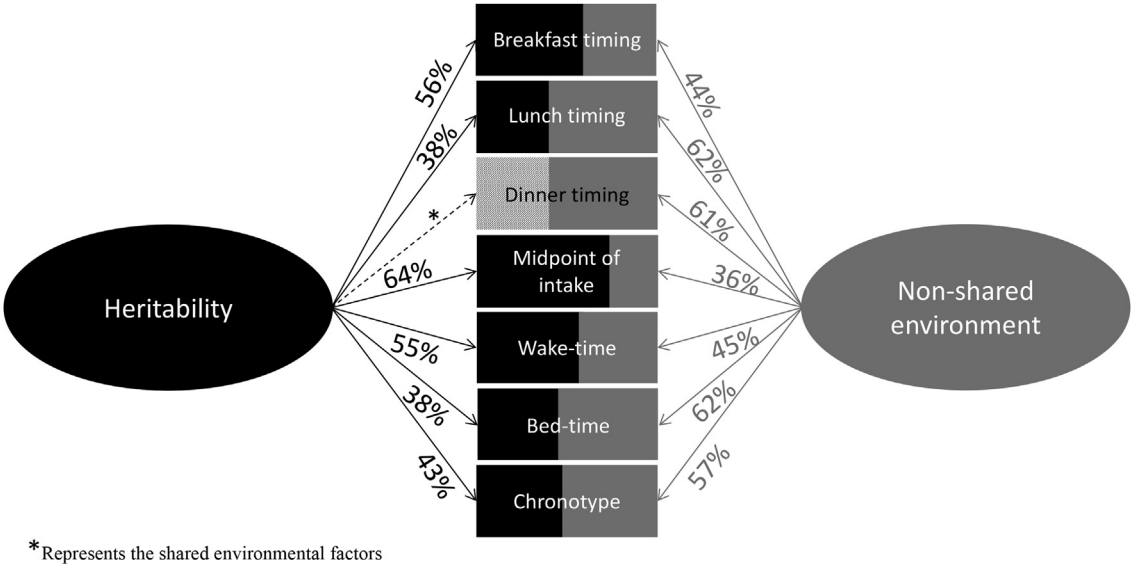

Fig. 1. Broad heritability and environmental effect estimates for food timing and related variables analyzed. The rectangles represent the contribution (percentage) of heritability (A: additive genetic factor + D: non-additive genetic factors) in black and non-shared environmental factors (E) in gray of the different variables. The asterisk represents the share environmental factors (C) in diagonals lines.

Furthermore, we observed the highest overall heritability for the midpoint of food intake (64%).

Further bivariate analyses for midpoint of food intake and the other timing-related factors – sleep timing and chronotype – rendered high genetic correlation estimates in the range of 0.78 and 0.91. Environmental correlations, however, were smaller and non-significant (Table 4). Hence 85% of the covariance between midpoint of intake and chronotype could be attributed to common genetic variation. Genetic contribution to covariance between midpoint of intake and wake and bed time was 90% and 75%, respectively.

4. Discussion

The present study provides supporting evidence that the timing of food intake is indeed heritable, and thus has an underlying genetic component. We observe that the estimated heritability of food timing varies by meal, and ranges from 56% for breakfast to

Table 4
Phenotypic (rPh), genetic (rG), and unique environmental (rE) correlations from bivariate AE models for midpoint food intake and circadian-timing related traits.

	Midpoint of food intake		
	rPh (CI 95%)	rG (CI 95%)	rE (CI 95%)
Sleep			
Wake-time	0.56 (0.40, 0.69)	0.79 (0.53, 1.00)	0.15 (−0.17, 0.45)
Bed-time	0.53 (0.37, 0.66)	0.78 (0.41, 1.00)	0.28 (−0.55, 0.56)
Chronotype score (MEQ)	−0.45 (−0.60, −0.27)	−0.91 (−1.00, −0.60)	0.23 (−0.10, 0.49)

MEQ: morning–evening questionnaire.

non-detectable heritability for dinner. Heritability estimates are higher for meals earlier on in the day (breakfast), than later on in the day (lunch and dinner). Similarly, heritability plays a larger role in other behaviors specific to the morning, such as wake times. Conversely, the environmental component is larger for the timing of dinner and other evening behaviors, such as bedtime. This variation in heritability suggests that interventions geared toward modifying behaviors later on in the day, may be more successful. Lastly, bivariate analyses for midpoint of food intake and sleep timing and chronotype suggest shared genetic architecture and likely common biological pathways underlying those phenotypically related traits.

Our data support the simultaneous interplay between genetics and environmental factors in contrast to earlier presumptions that the timing of food intake is determined by cultural factors alone. In twin studies, any learned habit should have an equal effect on MZ and DZ pairs and as such should have produced a significant effect of common (familial) environment in the analysis. Because the adult twin participants in the present study live separately and away from the familial environment, the higher intra-pair correlations found in MZ siblings suggests that food timing, like food composition [17], is a heritable trait.

Our results show that the timing of intake for breakfast, lunch, and dinner are differentially influenced by genetics with higher heritability for meals earlier on in the day, confirming previous results [25]. Consistent with the timing of meals, we also observe that other traits related to later on in the day tend to be more driven by environmental factors. Secular trends from US national surveys indicate shifts in food timing. For example, data from the National Health and Nutrition Examination Survey analyzing over a 40-year span from 1971–1974 to 2009–2010 observed later intakes of breakfast, snacks between breakfast and lunch, lunch, and snacks between lunch and dinner (among men), in addition to earlier intakes of snacks after dinner in 2009–2010 compared to 1971–1974 [42]. Our heritability results suggest that intervening for the purpose of advancing late lunch and dinner may be more achievable than changing breakfast time. Moreover, it is not clearly demonstrated that breakfast timing may impact health, but rather the prolongation of an overnight fast, which depends on the timing of both the first and the last meal, may be beneficial [43]. Our previous study on weight loss showed that a delayed breakfast time was not significantly associated with lower weight loss effectiveness [6]. Nevertheless, other breakfast habits such as skipping breakfast [44–46] or a lower energy intake at breakfast relative to at dinner [13,47] may yield adverse metabolic consequences. Thus, targeting the timing of breakfast intake might be less effective than targeting the timing of lunch and dinner for the purpose of achieving overall health: first because of its genetic influence and, second due to its unclear health benefits. By contrast, targeting the timing of lunch and dinner may be crucial as the timing of lunch has been observed to associate with weight loss success [6], and late or night-time eating was found to be linked to night-time hunger, body image distortions, and mood disorders [48], as well as elevated fasting blood levels of insulin and glucose that characterize metabolic syndrome [49].

In the current work, we aimed to study the relationship between the heritability of the timing of food intake and other phenotypically related traits, particularly sleep timing and chronotype. These traits have been associated with food timing in epidemiological studies [6,24,42,50]. There is also evidence for the heritability of sleep rhythms [51] and chronotype [52,53]. Our results confirm the importance of genetic factors for sleep timing phenotypes and chronotype. We detect moderate heritability for wake and bed times (55% and 38%, respectively), and for chronotype (43%), corroborating previous studies [52–56]. Furthermore, when analyzing the genetic and environmental contribution to covariation between those variables, we find high and significant genetic correlations (0.78–0.91) of the timing of food intake (midpoint) with sleep timing and chronotype. The genetic contribution to phenotypic correlation was 3–5 fold larger than that of the environment. Such outcome indicates that it is likely that a common set of genes underlies timing decisions regarding food intake, sleep timing and chronotype. Thus, future analyses in population-based studies equipped with genome-wide genetic data are warranted to confirm these genetic correlations.

Our results on the high heritability in food timing may be surprising, considering anecdotal evidence that food timing is driven primarily by cultural factors. However, studies performed under laboratory conditions with a protocol that controlled for several behaviors, including meal content and sleep periods, showed that the internal circadian clock controls the temporality of hunger and appetite independent of other behaviors [57]. Moreover, a recent study has demonstrated that adipose tissue specific deletion of *BMAL1*, a core molecular clock component, is able to impact the timing of food intake in mice [58]. Both studies indicate that the temporality of food intake is influenced by the internal circadian clock.

The present findings on the relative contribution of genetics and environmental factors to the timing of different meals may have relevance to the prevention and treatment of metabolic disorders considering the emerging evidence implicating food timing with metabolic diseases. Later timing of food intake has been associated with: (a) a substantial increase in the odds of being overweight/obese [2]; whereas, a later endogenous circadian timing of food intake, relative to melatonin onset, has been associated with increased body fat [3] (b) weight loss impairment during a dietary intervention [6,47] and following bariatric surgery [8]; (c) and decreased insulin sensitivity [6,47]. In addition, a large epidemiological study performed in 61,364 participants showed that late-night dinner consumption is associated with hyperglycemia, independent of relevant confounders, including BMI [59].

Some limitations need to be considered when interpreting the results of our study. Food and sleep timing were self-reported and are prone to measurement error, however these self-reported measures were previously found to be associated with metabolic diseases and weight-loss difficulty [6,8,9]. Furthermore, timing is a single dimension of diet. Finally, our study was limited to adult female twin pairs in Spain, and thus findings may not be generalizable to individuals of different gender, age, and BMI groups.

In conclusion, our data support that genetics may account for a large proportion of the variation in food timing, particularly for breakfast, whereas the environment appears to be a more important determinant of a more important determinant of variation in

lunch and dinner timing. These results suggest that intervention studies targeting food timing may be most effective if focused on modifiable factors later on in the day, such as lunch or dinner, rather than breakfast. In addition, future efforts should attempt to unravel specific genetic variants associated with food timing and disclose the shared genetic architecture underlying food timing and phenotypically related traits.

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Statement of authorship

Jesus Lopez-Minguez conducted research, analyzed data and wrote the paper; **Hassan S Dashti** analyzed data and wrote the paper, **Juan J Madrid-Valero** performed statistical analyses, **Juan A Madrid** analyzed data, **Richa Saxena** wrote the paper, **Frank AJL Scheer** wrote the paper, **Juan R Ordoñana** designed research, performed statistical analyses and wrote the paper, **Marta Garaulet** designed research, analyzed data and wrote the paper.

Conflict of interest

The authors declare no conflict of interest.

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