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The timing of energy intake

Giovanna Muscogiuri[1,2,3]

[1]*Dipartimento di Medicina Clinica e Chirurgia, Unità di Endocrinologia, Diabetologia e Andrologia, Università degli Studi di Napoli Federico II, Via Sergio Pansini 5, Naples 80131, Italy*

[2]*Centro Italiano per la cura e il Benessere del paziente con Obesità (C.I.B.O.), Dipartimento di Medicina Clinica e Chirurgia, Unità di Endocrinologia, Diabetologia e Andrologia, Università degli Studi di Napoli Federico II, Via Sergio Pansini 5, Naples 80131, Italy*

[3]*Cattedra Unesco 'Educazione alla Salute e Allo Sviluppo Sostenibile', Università degli Studi di Napoli Federico II, Via Sergio Pansini 5, Naples 80131, Italy*

The aim of the paper is to review the current evidence on the impact of 'the timing of energy intake' on the risk of developing obesity and obesity-related metabolic diseases. The prevalence of obesity is currently increasing worldwide thus becoming a severe health burden for most countries. Indeed, obesity represents a risk factor for several non-communicable diseases such as cancer, type 2 diabetes, dyslipidaemia, CVD and overall mortality. In order to treat obesity, several pharmacological approaches have been developed and are indicated for subjects with obesity with a BMI ≥ 30 kg/m^2 or ≥ 27 kg/m^2 and obesity-related comorbidities. For severe obesity (BMI ≥ 40 kg/m^2), bariatric surgery represents a promising approach. The most common bariatric surgical procedures are represented by the Roux-en-Y gastric bypass, laparoscopic adjustable band, laparoscopic gastric sleeve and biliopancreatic diversion with duodenal switch. Both anti-obesity pharmacological and surgical treatments require change in lifestyle. When a nutritional plan is established, attention is usually paid to macronutrient composition and energy intake, while 'the time of food' is not taken into account. Chronotype, which is the attitude of a subject to carry out most of their daily activities in the first (morning chronotype) or second half (evening chronotype) of the day, has been reported to have a role in the pathogenesis of obesity and obesity-related cardiometabolic diseases as well as eating speed. Thus, adopting a physiological timing of energy intake could be an additional strategy to potentiate the current anti-obesity approaches.

Key words: Obesity: Chronotype: Evening chronotype: Eating speed

Obesity is a chronic disease that is becoming a growing public health burden globally[1]. Indeed, the prevalence of obesity has been reported to have tripled since 1975[1]. Weight excess that characterises obesity results in negative consequences for health[2]. In particular, it has been highlighted that obesity could negatively influence the quality of life[3] not only because of mechanical impediment of weight excess but also because of the coexistence of obesity-related comorbidities such as type 2 diabetes, hypertension, CVD and non-alcoholic steatohepatitis[2]. However, obesity is still an underestimated disease[4]. According to data coming from the National Health and Nutrition Examination Survey 2011–2018, the advice to lose weight was given to only 40 % of adults with obesity or overweight[5]. In addition, in the U.S. national awareness, care, and treatment in

Corresponding author: Giovanna Muscogiuri, email giovanna.muscogiuri@gmail.com

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obesity management study, the diagnosis of obesity was made in only 55 % of subjects with obesity[6].

The pathogenesis of obesity involved several 'actors'. Indeed, it is a multifactorial, disease involving lifestyle behaviours, food intake, metabolism, energy expenditure and physical activity[7].

The current treatments for obesity include pharmacological, surgical and non-pharmacological approaches[8]. Several pharmacologic agents were approved by the U.S. Food and Drug Administration and the European Medicines Agency for weight management with the purpose of helping subjects with obesity achieve target weight loss by antagonising the body's adaptation mechanisms that counteract weight loss[9]. The indications for pharmacologic therapy are for subjects with obesity with a BMI ≥ 30 kg/m^2 or ≥ 27 kg/m^2 and an obesity-related comorbidity such as type 2 diabetes, hypertension, obstructive sleep apnoea or dyslipidaemia. Pharmacological therapy should be stopped if subjects with obesity do not experience a significant reduction in body weight after 3 months of treatment[9].

Aside from pharmacological treatment, bariatric surgery is also a promising approach for treating obesity and it is indicated when BMI is 40 kg/m^2 or higher[10]. The most common bariatric surgical procedures are the Roux-en-Y gastric bypass, laparoscopic adjustable band, laparoscopic gastric sleeve and biliopancreatic diversion with duodenal switch[10].

The non-pharmacologic approaches to obesity are mostly represented by dietary interventions[11]. Although there is a wide array of nutritional protocols, the aim of dietary interventions is to establish a negative energy balance needed for weight loss. However, patients can be guided in the selection of an appropriate diet, and the best diet intervention is the one to which the patient can adhere[11].

Although the macronutrient composition is taken into account when establishing the nutritional plan, attention is not paid to '*the time of the food*'. In recent years, several studies have highlighted how feeding times, eating speed and frequency, and chronotype can influence biological rhythms, determining subject's tendency to develop obesity and metabolic diseases[12]. Thus, the aim of the paper is to review the current evidence on the impact of 'the time of food' on the risk of developing obesity and obesity-related metabolic diseases.

Chronotype

Chronotype is a subject's attitude to carry out most of their daily activities in the first part (morning chronotype or 'lark') or in the second part (evening chronotype or 'owl') of the day[13]. Based on circadian behavioural phenotype variants, three categories of chronotypes could be identified: morning, evening and intermediate chronotypes. The morning chronotype prefers to wake up early and do their daily activities in the first half of the day. The evening chronotype prefers to wake up later in the morning and do most of their daily activities in the afternoon or evening. In an intermediate position between these two categories, there is an intermediate chronotype, who has habits that fall between morning and evening chronotypes without a clear preference between morning and evening[13].

The nutritional habit of evening chronotype is usually unhealthy and is characterised by delayed mealtimes, most of the daily food intake in the second half of the day and skipping breakfast[14,15]. In particular, we previously demonstrated in a cross-sectional study aiming to investigate the association between chronotype categories (morning, evening, intermediate) and adherence to Mediterranean diet in 172 middle-aged Italian adults, that evening chronotype showed the lowest adherence to Mediterranean diet compared to morning and intermediate chronotypes[16]. Given the role of the association of diet with chronotype we also suggested a specific cut-off of the Prevención con Dieta Mediterránea score, a validated questionnaire for assessing adherence to Mediterranean diet, which identifies subjects with the evening chronotype[17]. Of note, a Prevención con Dieta Mediterránea score <8 was the threshold for identifying subjects with the evening chronotype[17].

Almost all studies examining the aspect of mealtimes reported similar results, with earlier chronotypes having earlier mealtimes and later chronotypes having later mealtimes[18–21]. Thus, these results reflect the possibility that the norm of mealtime follows a person's own biological clock despite social clock limitations. Meule *et al.* suggested that the biochemical rhythm of appetite-regulating peptides (leptin and ghrelin) might underlie this behaviour, since the evening chronotype has a delayed release of ghrelin and leptin than the morning chronotype[22]. The results of recent research have added to the growing body of evidence supporting the link between chronotype and breakfast[21–23]. A genetic study was conducted on fifty-three pairs of female twins to investigate the influence of genetic inheritance on mealtimes[23]. They found that the time of breakfast (56 %) and lunch (38 %) was mainly genetically influenced, but not the time of dinner. In addition, the genetic heritability of waking time was also greater than sleeping time (55 *v.* 38 %)[23]. Hence, this could be the possible underlying mechanism behind the relationship between chronotype and breakfast habits, since chronotype was also associated with sleep–wake time[24,25]. Another interesting finding from the latest research is that only the morning chronotype benefited from higher energy intake earlier in the day (within 2 h of waking) and was protected from obesity, with a reduced likelihood of developing overweight or obesity[26]. Although many studies highlight the importance of breakfast[27,28], not everyone (especially the evening chronotype) may be able to reap its benefits. There are a limited number of studies on the effects of delaying meal timing in individuals with evening chronotypes. Delay in eating dinner or supper was less severe in evening chronotypes than in shift workers (range: 7:19 p.m. to 9:31 p.m.)[14]. Nonetheless, evening chronotypes remain subject to a milder form of chronodisruption (in terms of irregular and delayed mealtimes)[29]. The subjects belonging to different chronotype categories mostly

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differ in feeding timing, although they reported similar nutrient intake[14,15,30]. Indeed, it has been reported that subjects with evening chronotype were more prone to have less energy intake by 4–5 % in the morning and more energy intake by 6–7 % at evening compared to subjects with morning chronotype[30]. Thus, it highlights that although subjects belonging to different chronotype categories have the same amount of energy intake during the day, the breakdown of energy intake throughout the day is different. In addition, subjects with morning chronotype prefer to eat protein-rich meal in the first half of the day while subjects with evening chronotype prefer sucrose intake[26]. The preference to consume more alcohol and caffeine is another feature of subjects with evening chronotype[31] that can also contribute to the higher prevalence sleep disturbances in this category of subjects[32,33].

Among the several nutritional aspects addressed so far, it appears that chronotype also influences eating speed. In order to better characterise the eating habits of subjects with obesity belonging to three different chronotype categories, we previously investigated eating speed during the three main meals in eighty-one subjects with overweight/obesity in a cross-sectional study[34]. To investigate the duration of main meals, a dietary interview by a qualified nutritionist was conducted. Subjects with the evening chronotype spent significantly less time on lunch than subjects with the morning chronotype[34].

Consequently, the nutritional habits of subjects with evening chronotype increase the risk of developing obesity and obesity-related disease.

In this regard, chronotype seems to be also involved in metabolic comorbidities. Results of our previous research in 174 middle-aged Italian adults found out that the evening chronotype was linked to an increased risk of type 2 diabetes and CVD compared to morning chronotype after adjustment for sex, BMI, sleep quality and adherence to Mediterranean diet[35]. In a cross-sectional study in eighty-seven subjects with obesity aimed to investigate the association of non-alcoholic fatty liver diseases with chronotype categories, only individuals with the evening chronotype were above the threshold of non-alcoholic steatohepatitis evaluated as index of non-alcoholic steatohepatitis (≥50)[36]. In addition, subjects with the evening chronotype presented significantly higher indirect indices of non-alcoholic fatty liver diseases such as visceral adipose index, liver fatty equation, hepatic steatosis index and index of non-alcoholic steatohepatitis than morning chronotype, also after the adjustment for age, sex and BMI[36].

Besides as seen earlier a role in the development of metabolic comorbidities and in determining eating habits in obesity, chronotype also appears to influence the efficacy of nutritional approaches for obesity. In particular, existing literature suggests that very low-energy ketogenic diet can be an effective intervention for weight loss and improving metabolic health in individuals with obesity[37–39]. In this regard, we carried out a study to investigate whether chronotype categories can have a role in determining the efficacy of very low-energy ketogenic diet in terms of weight loss and changes of body composition in 248 women with overweight or obesity[40]. Women with evening chronotype experienced significantly less weight loss, reduced fat mass and increased fat-free mass than women with morning chronotype. Using a linear regression model, chronotype score was the main predictor of weight loss achieved with very low-energy ketogenic diet[40].

In conclusion, the circadian preference towards eveningness is associated with a delay in meal timing, the breakfast skipping habit, engagement with excessive consumption during night-time, lower protein and vegetables intake, as well as increased sucrose, sweets, caffeine and alcohol intake. A limited number of studies have also shown that the aforementioned chronotype was related to a lower intake of grains and fruits. Despite that, both morning and evening chronotypes relatively consumed the same number of energy, amount of carbohydrates, fat, cholesterol, fibre, legumes, meat, fish and dairy products. Nonetheless, more aspects of dietary behaviours, micronutrients and other food group intakes between chronotypes remain uncertain, and more studies are required to elucidate the relationship between chronotype and body weight in the long term.

Eating speed

Eating speed is the length of time spent eating. In particular it has been highlighted that lower energy intake is usually accompanied by slow-eating speed thus providing important evidence that slow eating is a fundamental component of healthy dietary pattern[41]. In addition, eating speed is of paramount importance because if it is too fast, it increases the risk of developing obesity and obesity-related cardiometabolic diseases[41,42]. This is due to the impact of eating speed on secretion of hormones that play a role in the regulation of satiety and appetite[43]. In fact, slow-eating speed results in an increase in the secretion of peptide YY and glucagon-like peptide-1, that in turn exert a beneficial effect on weight[43]. Eating speed is determined by mastication that could also have an impact on energy intake and body weight because it could modify the composition of the bolus thus making it more digestible andg havin a different effect on secretion of satiety and appetite hormones[43].

It is interesting to underline that eating speed could also have a role in determining the risk of developing obesity-related cardiometabolic complications[42,44–46]. However, in the assessment of patients with obesity, eating speed is not usually evaluated, just giving importance to BMI, waist circumference, waist:hip ratio and waist:height ratio, currently considered the main markers of health risk in obesity[8]. These parameters reflect the unhealthy long-term behaviours in the body[47,48]. In this context, eating speed might become an early marker that could predict long-term nutritional behaviours. Indeed, eating speed has been used in several Japanese cohort studies as a predictive factor for obesity[45,49–51]. However, in most of the studies carried out on obesity,

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especially outside of Asia, eating speed is often not taken into account, thus missing important information on nutritional habits. Thus, it would be of paramount importance to include eating speed in the assessment of patients with obesity because acting on this parameter could contribute to implement the efficacy of current anti-obesity treatments and in subjects at high risk to develop weight excess, it could contribute to prevent obesity.

Eating speed has not only been associated with obesity. Indeed, a cross-sectional study has been carried out aiming to investigate the link between eating speed and cardiovascular risk factors in 792 subjects previously enrolled in the Prevención con Dieta Mediterránea study[44]. Each subject self-reported eating speed thus was then categorised as slow, medium or fast. The main finding of this study was that fast-eating subjects had 59 % more chances to develop hypertriglyceridaemia compared to slow-eating subjects, also after adjustment for potential confounders[44]. We also carried out a similar cross-sectional study in eighty-seven subjects with obesity aimed to investigate the effect of eating speed at the main meals (breakfast, lunch and dinner) on the risk of developing cardiometabolic diseases (type 2 diabetes mellitus, dyslipidaemia and hypertension)[42]. A dietary interview was performed to collect information about meal duration and eating habits at the main meals. According to median value of meal duration, meals were classified into two groups: fast-eating group and slow-eating group. Of note, the prevalence of dyslipidaemia was more than twice in the fast-eating group compared to the slow-eating group at lunch and dinner. Fast-eating group had a significantly higher risk of dyslipidaemia than the slow-eating group at lunch and dinner[42]. Another interesting study carried out in the Netherlands aimed to evaluate what was the association of eating speed with consumed foods and the macronutrient composition of nutritional pattern[52]. The authors found that eating speed was positively correlated with food and energy intake. In addition, eating speed was inversely correlated with energy density and positively with water content. The intake of carbohydrate, protein and fibre content was the greater the faster eating rate was, thus suggesting that an overconsumption of food may occur when subjects tend to eat fast[52]. Visceral adipose tissue is considered a key risk factor for obesity-related cardiometabolic diseases[2]. Japanese researchers aimed to evaluate the association of eating speed with fat deposition in subjects without obesity[53]. They enrolled 381 participants undergoing MRI in order to quantify visceral and subcutaneous fat areas. Eating speed was self-reported by each subject filling a questionnaire. Interestingly, the prevalence of visceral adipose tissue $\geq 100\,cm^2$ was higher in fast eaters compared to slow eaters while no difference in subcutaneous adipose tissue was detected between the two groups. Finally, they found that eating speed was positively correlated with visceral adipose tissue, while this association was not found with subcutaneous adipose tissue[53].

It is well known that among factors that influence the predisposition to develop obesity, physical activity plays an important role and it should be taken into account as confounding factors in studies carried out in subjects with obesity[54]. Interestingly, eating speed is associated with the risk of developing obesity, also after adjustment for physical activity as demonstrated in several studies[55–57]. Although a strong relationship has been highlighted in the earlier reported evidence between obesity and eating speed, the cross-sectional design of these studies does not allow drawing conclusion on casual association. More information was provided on that sense by a longitudinal study that was carried out aiming to investigate the effects of changes in lifestyle habits on changes in obesity, mostly focusing on eating speed and type 2 diabetes, using health checkup and insurance claims data that were generated from 2008 to 2013[58]. Data coming from 59 717 subjects that were diagnosed with type 2 diabetes in the study period were analysed. Also eating speed, BMI and lifestyle habits (eating dinner within 2 h of sleeping, after-dinner snacking, skipping breakfast, alcohol consumption frequency, sleep adequacy and tobacco consumption) were assessed. Slow eating was associated with a decreased risk of developing obesity; indeed, slow eaters had lower BMI and waist circumference than fast eaters thus suggesting that interventions focused on slowdown of eating speed may be a promising approach to prevent obesity[58]. Another longitudinal study was carried out to investigate the association of eating speed with obesity in 529 male workers receiving health checkups in 2000 and 2008[57]. In 2008, information on alcohol consumption, smoking status, self-reported speed of eating and exercise was collected while height and weight were assessed in both 2000 and 2008. Interestingly, fast eaters experienced a higher weight gain (1·9 kg) compared to slow eaters (0·7 kg). Although the statistical significance was detected in the 20–29 year age group, a trend was detected also in other age groups, thus suggesting that the rate of weight gain was tightly related to eating speed[57].

Also, a sex difference has been detected in eating speed[50,53,58]. Indeed, males are more commonly fast eaters than females[50,53,58] and this could partially explain also the increased risk of developing obesity found in this sex[59].

Eating speed has been also reported to be associated with metabolic syndrome[50,55,60]. Indeed, a study carried out in Japan in 8941 community residents from Soka city in Saitama prefecture, participating in a baseline survey in 2008 and followed up until 2011 aimed to investigate the association between eating speed and incidence of metabolic syndrome using Cox proportional hazards models adjusted for potential confounding variables[60]. During the 3-year follow-up, 647 subjects developed metabolic syndrome (25·0 cases/1000 person-years). In particular, the incidence rates of metabolic syndrome were higher in fast eaters (3·1 %) than slow eaters (2·3 %). Eating speed was significantly associated with waist circumference and HDL-cholesterol components of metabolic risk factors. These results suggested that targeting eating speed could be an additional tool to prevent metabolic syndrome[60]. Similar results were found in another cross-sectional study carried out in Japan in 56 865 subjects (41 820 males and 15 045 females)

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without history of CVD that attended a health checkup in 2011 investigating the association between self-reported eating rate and metabolic syndrome[55]. The main findings of this study were that eating speed was significantly and positively associated with metabolic syndrome and with abdominal obesity. The link of eating speed and metabolic syndrome and its components was attenuated after adjustment for BMI; however, the link between slow-eating speed and lower odds of hypertension and impaired glycaemia as well as the link between fast-eating speed and higher odds of dyslipidaemia remained significant also after adjustment for confounding factors[55]. Evidence on the association between eating speed and metabolic syndrome comes also from a prospective study carried out in a Japanese working population[50]. One thousand and eighteen workers without metabolic syndrome at baseline were enrolled and were assessed at baseline and after 3 years. Eating speed was self-reported and metabolic syndrome was diagnosed according to criteria joint from several international societies. The relevant results of this study were that fast eaters had an increased odds of developing metabolic syndrome compared with medium eaters thus suggesting that eating speed was associated with a risk of developing metabolic syndrome that was independent from BMI, BMI change between baseline and follow up surveys and total energy intake[50].

Beyond the association of eating speed with metabolic syndrome, it has been reported also a link with metabolic parameters[46]. A cross-sectional study carried out in 7972 Chinese adults found that eating speed was significantly associated with a high risk of hypertension and visceral obesity for both sexes[46]. In addition, eating speed was linked with a high risk of hypertriglyceridaemia and low HDL-cholesterol levels in males while it was associated with alterations of fasting plasma glucose in females[46].

Little is known regarding the role of eating speed in the context of diabetes; a study carried out in 5479 Japanese adults with HbA1c <6·5 % participating in health checks investigated the relationship between eating behaviour and poor glycaemic control[53]. A survey was conducted in 2013 and the very same subjects that participated in this survey were subsequently followed up until 2017. Poor glycaemic control was defined as HbA1c ≥ 6·5 % or increases in HbA1c of ≥0·5 % and/or taking antidiabetic medications. This study found that fast eaters, mostly if they skipped breakfast ≥3 times/week were at high risk of having poor glycaemic control and obesity[53]. Also in Japan, a study investigated the effect of eating speed on the risk of new onset of type 2 diabetes[49]. Data coming from the nation-wide annual health check programme in Japan examined 197 825 participants without diabetes in 2008, that were followed up for 3 years. Interestingly, the prevalence of fast eaters, subjects snacking after supper and/or before sleep and/or skipping breakfast was higher in the new-onset diabetes group than in the group not developing diabetes mellitus. Eating speed was confirmed to be a risk factor for the development of diabetes also after adjustment for confounding factors[49].

The earlier reported evidence suggests that eating speed could play an important role in determining the risk of

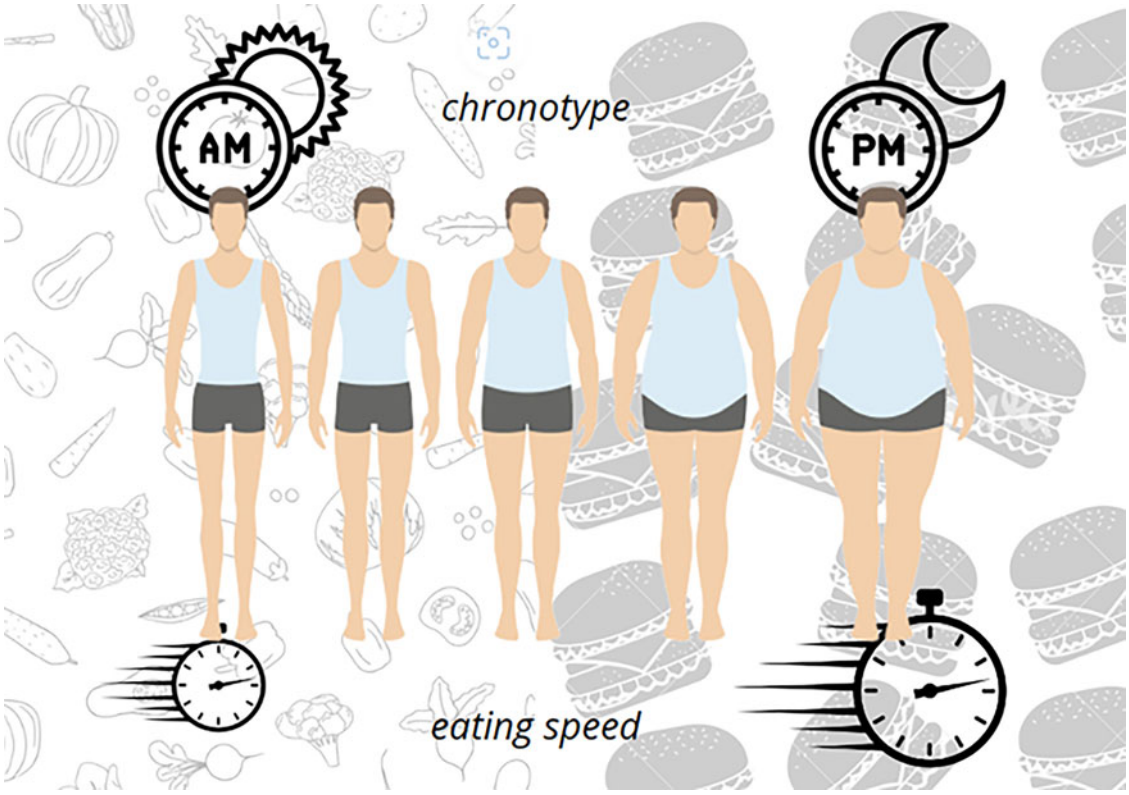

Fig. 1. Role of chronotype and eating speed in obesity.

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developing obesity and obesity-related cardiometabolic diseases. However, most of these studies were cross-sectional and further studies, mostly intervention ones, need to be carried out in order to confirm the causal association between eating speed and metabolic diseases. Another issue that should be overcome is the assessment of eating speed that being self-reported, most of the time could not be reliable. Thus, the set-up of a validated tool to assess eating speed is mandatory. If a causal role of eating speed in the context of metabolic diseases would be confirmed, it could be useful to include eating speed in the assessment of subjects with obesity in order to identify those at high risk of cardiometabolic diseases and to establish tailored intervention to slowdown eating speed.

Conclusion

Evening chronotype and fast-eating speed have been associated with an increased risk of developing obesity and obesity-related diseases (Fig.1). Thus, further intervention studies are needed to investigate if a tailored nutritional approach could be helpful to restore the right timing of the food and thus to implement the efficacy of conventional therapeutical treatment. If this would be the case, the assessment of chronotype and eating speed should be included in the baseline and follow-up assessments of subjects with obesity or at risk of developing weight excess.

Current evidence suggests a role of the evening chronotype and increased eating speed at meals in the development of obesity and obesity-related disorders. In addition, both the evening chronotype and the fast eaters have worse eating habits. The management of obesity must include the evaluation and correction of these partly genetically determined behavioral aspects in order to improve current therapies.

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Conflict of Interest

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Authorship

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