Original Article

Time of Last Meal and its Association with Indices of Metabolic Health in Young Indian Adults

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Abstract

Background: Chrono-nutrition explores the role of meal timing, frequency, and regularity in shaping metabolic health through circadian mechanisms. Evidence indicates that glucose metabolism, insulin secretion, and appetite regulation follow circadian rhythms and are influenced by the temporal distribution of energy intake. Late or evening-skewed eating patterns and circadian misalignment, common in modern lifestyles, are associated with poorer metabolic outcomes. This study examined the association between meal timing, percent body fat, blood glucose, and insulin levels in adolescents and young adults(16-25 years old).

Methods: Of 1,313 apparently adolescents and young adults screened, complete data were available for 1,015 participants after applying predefined exclusion criteria. Body composition was assessed using bioelectrical impedance, while fasting and 2-hour post-glucose blood samples were analysed for plasma glucose, insulin, and HbA1c using standard methods. Meal timing was recorded based on the last meal consumed the previous night, and data were analysed using SPSS 27 with $p<0.05$ considered statistically significant.

Results: Among the 1,015 participants, 42.6% were males and 57.4% females, with 41.2% aged 16–18 years and 58.8% aged 19–25 years. The mean time of the last meal was 20.57 ± 1.10 h, ranging from 17:00 to 23:30 h. Later meal timing was associated with significantly higher percent body fat, while muscle mass was significantly greater among those who consumed their last meal earlier. Participants in the earliest meal-timing quintile had significantly lower stimulated glucose, fasting and post-glucose insulin levels, and HOMA-IR compared with later

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quintiles. Time of last meal showed positive correlations with percent body fat, glucose and insulin indices, and a negative correlation with muscle mass.

Conclusion: Late timing of the last meal was associated with higher insulin levels, increased adiposity, and poorer metabolic profiles among adolescents and young adults. These findings suggest that eating patterns misaligned with circadian rhythms may predispose metabolically healthy youth to future metabolic risk. Promoting earlier meal timing and appropriate redistribution of energy intake during waking hours may serve as a feasible strategy for metabolic health prevention.

Keywords: fasting, meal frequency, meal timing, obesity, cardiovascular health, diabetes

Introduction

Chrono nutrition investigates the health impact of three different dimensions of feeding behaviour like the regularity of meals, frequency and timing of food intake[1]. There is a complex relationship between temporal eating patterns, circadian rhythms, and metabolic health and thus the metabolic health is influenced not just by the food we eat, but also by the timing, regularity, and energy distribution in the 24 h day and across days[2,3]. Meal timings have been found to play an important role in weight loss, regain and maintenance of body weight, as well as insulin resistance and appetite[4].

Glucose metabolism follows a circadian rhythm. Typically, glucose tolerance peaks during day-light hours when humans generally consume food, whereas fasting that occurs during the dark cycle or night time, results in lowering of blood glucose levels. Glucose, insulin and cortisol levels exhibit circadian rhythms which in turn greatly influences glucose metabolism[5,6]. Consequently, when some food or a meal is consumed at a time that is not in consonance with the central sensor - the suprachiasmatic nucleus and peripheral tissue[7], glucose metabolism is affected[8-10].

Many people are required to work at night due to occupational demands. Changing lifestyles have resulted in people keeping awake for long hours and eating at late hours during the night especially in cities[11] which results in their bodies to be out of alignment with the circadian clock, which in turn can have adverse consequences on metabolism[12,13].

Insulin release produces an anorexigenic signal which decreases food intake. Perturbations in insulin secretion and sensitivity could affect hunger and food intake. Several investigators[4,7,14-17] demonstrated that when a larger proportion of energy intake was consumed in the evening, metabolic control was poorer and was associated with weight gain. Eating earlier in the evening, or consuming the main meal around midday or a larger proportion of energy intake earlier have been associated with reduced appetite, weight loss and lower risk of overweight/obesity, glycemic control, better metabolic control[16, 18-20].

In this context, we analysed the data available for meal timings in relation to their percent body fat, blood glucose and insulin levels in adolescents and young adults.

Method Materials

Study Design and Sample Selection

This was a cross –sectional study conducted on adolescents and young males and females(16-25 years of age), who were attending various academic institutions in Mumbai city. A total of 1313 young adults had volunteered to undergo screening. However, complete data was available for 1015 persons.

The inclusion criteria were: apparently healthy individuals in the age group of 16-25 years. The exclusion criteria included presence of any known chronic disease, on any specific diet plan, on prescribed medications like steroids, pregnancy and/ or lactation.

Ethical Considerations: The study was approved by the Intersystem Biomedical Ethics Committee, Mumbai, India (ISBEC version 2 dated 12th Aug, 2017) and conducted according to Good Clinical Practices and the Declaration of Helsinki. Informed written consent was taken from each participant and each guardian/parent for participants who were between 16 and 18 years of age.

Measurements

Each participant first underwent a clinical examination by a physician to assess the general health status. Body composition was measured using the TANITA body composition analyser (Model MC 780 MA).Each measurement was taken thrice and the average was calculated.

Biological Samples, Collection, Storage and Biochemical Measurements

Participants were asked to report to the laboratory after fasting overnight for at least 12 hours. Venous blood (10ml) was collected in fasting state and four mlof blood was collected 2 hrs post 75 gms glucose by a trained phlebotomist. Two ml of fasting blood sample was immediately transferred to a BD vacutainer (spray-coated K2EDTA Tubes) for complete blood count (CBC) and HbA1c, two ml of fasting and post glucose blood sample was immediately transferred to a BD vacutainer (spray-coated sodium fluoride Tubes) for estimation of plasma glucose levels. The remaining six ml of fasting blood and 2 ml of post glucose blood were transferred into plain BD vacutainer for separation of serum. The vacutainers were kept in a closed ice box, and transported to the Institute's laboratory. Fluoride and plain vacutainers were centrifuged, fluoride plasma was processed for estimation of plasma glucose levels and serum was processed for insulin levels. The remaining fasting serum was divided into aliquots and stored at -70^{0}C until further analyses.

Glucose tolerance test (fasting and 2-h post 75-g glucose administration) was conducted for all participants. Glucose was measured by the GOD POD method (Accurex Biomedical Pvt Ltd) and insulin was measured by radioimmunoassay using a Beckman Coulter Counter. Glycosylated haemoglobin was measured using Nycocard reader (Alere Technologies, Norway). Among the 1313 participants in the screening exercise prior to the intervention, data on glycosylated haemoglobin levels was available for 673 participants. Participants were said to have dysglycemia, if their fasting glucose levels were between 100-125mg/dL (5.6-6.9 mmol/L) and 2-hour post-glucose value 140-199 mg/dL (7.8-11.0 mmol/L)[21]. Hyperinsulinemia was defined as fasting hyperinsulinemia (≥ 15 mIU/ml) or glucose challenge hyperinsulinemia (≥ 80m IU/ml)[22,23].

Meal timings: Prior to collection of the blood sample, each participant was asked to write down the time of her/his last meal on the previous evening or night and they were told to follow their regular time for dinner and not alter the time.

Statistics: Descriptive data of participants are reported as means±SD and 95% confidence interval (CI) for continuous variables. Tests applied were Student's 2-tailed t-test and Pearson's Chi Square analysis. Analysis of variance and post-hoc Bonferroni were used for comparison of quintiles. The analysis was performed using STATA(14.2). A p-value <0.05 was set to determine statistically significant differences.

Results

Sample Profile: Among the 1015 participants, 42.6% (n=432) were males and 57.4% (n=583) were females wherein, 41.2% (n=418) were in age group of 16 to 18 years and the remaining 58.8% (n=597) were in the age group of 19 to 25 years. The mean for the time of eating last meal was 20.57±1.10hrs with the earliest time of eating dinner was 17.0 hrs i.e. 5.00pm and latest time of eating the meal was 23.50 hrs i.e. 11.50pm (Figure 1). Among male participants, the mean for last meal was 20.3 pm (SD 1.07, Min-Max-17.00-23.50 hrs, Median 20.50pm), whereas in female participants the mean time for last meal was 20.8 pm (SD 1.07, Min-Max: 17.00-23.5 hours, Median: 21.00pm) with no statistically significant difference. Age and sex of the participant did not show significant difference in time of last meal.

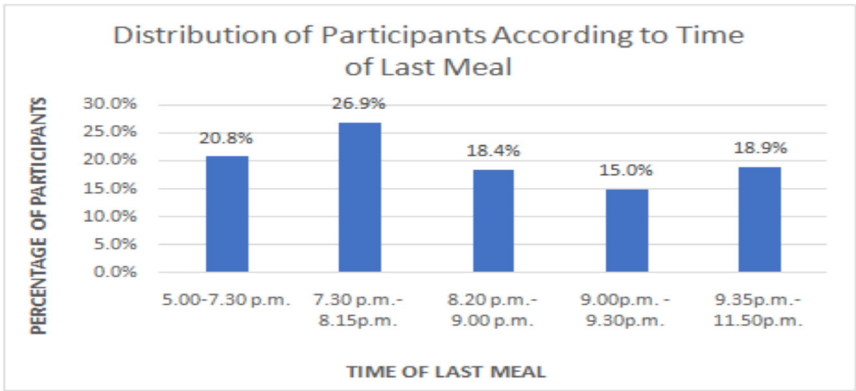

FIGURE 1: OVERALL DISTRIBUTION OF TIME OF EATING LAST MEAL IN ADOLESCENTS AND YOUNG ADULTS

Time of Last Meal, Anthropometric indices and Biochemical measurements: Significant differences were observed for percent body fat and muscle mass. Percent body fat was significantly higher in participants who ate their last meal later and conversely mean muscle mass was significantly higher in those who ate their last meal earlier as compared to those who ate dinner later. Mean muscle mass of persons in Q1 was approximately 3 kg more than those in Q3 and about 4 to 5 kg more as compared to Q4 and Q5, respectively. When Q1and Q2 were compared, mean muscle mass in Q1 was about 2 kg more than in Q2, although this difference between the two quintiles was not significant (Table 1).

Table 1: Comparison of Percent Body Fat, Between Quintiles of Time of Consumption of Last Meal

Measurement	Quintiles of Time of Last Meal Consumed					F, p
	Q1 (17.00-19.45) n=211	Q2 (19.46-20.30) n=273	Q3 (20.31-21.00) n=187	Q4 (21.01-21.30) n=152	Q5 (21.31-23.48) n=192	
% Body fat	22.0±9.2	23.2±9.5	26.5±8.6	26.2±8.0	26.7±7.40	12.639, 0.000
Visceral fat	4.3±3.4	4.0±3.4	4.2±3.3	3.5±2.7	3.9±3.1	1.622, 0.167
Muscle mass	42.2±8.8	40.3±8.4	39.0±8.7	36.9±7.9	37.9±8.7	9.525, 0.000

[1] Mean± SD

p <.0.05 was considered significant.

p values based on the ANOVAtest

Fasting and Stimulated Glucose, Insulin and HbA1c: Significant differences in the blood glucose and insulin levels were observed between the quintiles of time of consuming the last meal (Table 2). Those who ate earlier had significantly lower mean stimulated glucose levels than those who ate later, although the difference between Q1 and Q2was not significant.

Table 2: Comparison of Fasting and stimulated blood glucose and insulin, glycosylated haemoglobin between quintiles of time of Consumption of last meal

Measurement	Quintiles of Time of Last Meal Consumed					P value
	Q1 (17.00 to19.45) n=211	Q2 (19.46 to 20.30) n=273	Q3 (20.31 to 21.00) n=187	Q4 (21.01 to21.30) n=152	Q5 (21.31-23.48) n=192	
Fasting sugar	80.2±7.7	81.4±14.5	80.8±9.4	82.2±9.5	81.6±9.5	0.469
2- hour blood sugar	87.7±19.2	93.5±33.6	95.9±20.9	96.7±21.7	92.9±27.1	0.006
Fasting Insulin	7.8±4.5	8.0±4.5	8.7±4.1	8.9±5.7	9.5±8.8	0.015
2-hour insulin	62.9±51.1	72.9±63.5	73.5±47.9	83.8±66.2	73.5±57.1	0.022
HbA1c	5.4±0.5	5.4±0.7	5.5±0.5	5.5±0.5	5.4±0.5	0.626
HOMA IR	1.56±1.00	1.61±1.07	1.72±0.94	1.80±1.23	1.95±2.03	0.019

[0.0191] Mean±sd

p <.0.05 was considered significant.

p values based on the ANOVAtest

Similarly, mean 2-hour blood glucose (F=3.611, p=0.006), fasting insulin (F=2.925, p=0.020), stimulated (2-hour) insulin levels (F=3.845, p=0.004) and HOMA IR (F=2.960, p=0.019) in Q1 were significantly lower than in the higher quintiles.

In all five quintiles, a small percentage of persons had fasting glucose levels above the normal range, (Q1-1.2%, Q2-1.5%, Q3-3.1%, Q4-2.1% and Q5-2.0%), with no significant difference between the quintiles (χ^2= 2.985, p=0.394). Similarly, a small percentage of participants had higher than normal levels of blood glucose, 2 hours after consuming 75g of glucose and there was no significant difference between the quintiles (χ^2= 1.767, p=0.622) [Q1-1.5%, Q2-2.5%, Q3-3.1%, Q4-2.1% and Q5-2.3%]. Similar trends were observed for fasting insulin, with no significant difference between the quintiles (χ^2= 5.300, p=0.151), although the percentage of participants with higher-than-normal post glucose, 2-hour insulin was significantly lower in Quintile 1 (χ^2= 9.524, p=0.023) than in the higher quintiles. In Q1, 24.4% of the participants had higher stimulated insulin levels compared to 30.4% in Q2, 35.8% in Q3, 30.7% in Q4 and 30% in Q5, clearly indicating that those who consumed their last meal after 7.45 pm had higher stimulated insulin levels.

Table 3 showed that percent body fat, 2-hr blood sugar, fasting insulin, stimulated insulin and HOMA IR were significantly and positively correlated with time of last meal with later times of eating the meal associated with higher levels of these markers. Also, muscle mass showed a significant negative correlation with time of last meal (Table 3).

Table 3: Correlation Of Time of Last Meal with Body Composition and Biochemical Measurements

Measurements	Time of Last meal	
	r	P
% Body Fat	0.174**	0.000
Visceral Fat	-0.053	0.117
Muscles Mass	-0.175**	0.000
Fasting Blood sugar	0.034	0.283
2-hour blood sugar	0.068*	0.031
Fasting Insulin	0.094**	0.003
Stimulated Insulin	0.069*	0.029
HbA1c	-0.021	0.674
HOMA IR	0.086**	0.000

*Pearson's Correlation,

p <.0.05 was considered significant.

Discussion

The present study examined the association between timing of the last meal and markers related to metabolic syndrome in a large sample of adolescents and young adults. The findings demonstrate that later timing of the last meal is significantly associated with higher adiposity, reduced muscle mass, and adverse glucose–insulin dynamics, even in a relatively young population with a low prevalence of overt hyperglycaemia. These results highlight meal timing as an important behavioural determinant of early metabolic risk.

Humans are diurnal species with their food consumption and physical activity mostly occurring during the active phase of the 24-hour cycle and in line with the light-dark cycle, although the metabolic interactions can be influenced and altered by epigenetic mechanisms [24].In the present study we observed that more than 50% of the participants ate dinner only after 8.00pm among which 18.9% of the participants had their last meal after 10.00 pm.In a NHANES study involving 15,341 adults from the 2009 to 2014 cycle, the average dinner time was 6:24 p.m., with the average time of the last eating episode being 8:18 p.m[25].However, in urban cities like Mumbai, the extended commuting times, long and inflexible working hours, and a high prevalence of service-sector and shift-based employment delay the return to home, thereby postponing the main evening meal. Further, changes in lifestyle, work patterns and work timings, have led to changes in eating patterns, such that portion sizes/meals are larger than before, the selection of foods veers towards high energy, high fat and/or high sugar and sodium containing foods. It has also been observed that a greater proportion of total daily energy consumption occurs in the late evening or night [26] which could result in circadian misalignment with adverse influence on adiposity as well as (or ill-effects) insulin, glucose control and metabolic health, as well as a substantial increase in the risk of cardiovascular disease [27-32].

One of the key findings of this study is the significant increase in percent body fat with progressively later consumption of the last meal. Participants in the later quintiles (Q3–Q5) had markedly higher body fat percentages compared to those who consumed their last meal earlier (Q1–Q2). Conversely, muscle

mass showed a significant inverse association with meal timing, with individuals in the earliest quintile (by 7.00 pm) having approximately 3–5 kg higher muscle mass compared to those eating after 9:30 pm. These observations are consistent with the concept of circadian regulation of metabolism, wherein nutrient intake aligned with the biological day promotes more efficient substrate utilisation, while late-night eating favours lipogenesis and impairs fat oxidation[33]. Reduced muscle mass among late eaters may reflect prolonged postprandial inactivity, altered hormonal milieu (including suppressed growth hormone secretion), and impaired muscle protein synthesis during late evening hours[34,35]. Importantly, these associations were evident despite no significant differences in visceral fat, suggesting that overall adiposity and lean mass distribution may be more sensitive early markers of metabolic dysregulation in young populations.

While fasting glucose levels did not differ significantly across quintiles, significant differences were observed for stimulated (2-hour) glucose, fasting insulin, stimulated insulin, and HOMA-IR. Participants consuming their last meal earlier exhibited lower post-load glucose and insulin responses, indicating better insulin sensitivity. The progressive rise in fasting insulin, stimulated insulin, and HOMA-IR across later quintiles suggests compensatory hyperinsulinemia, a recognised precursor to insulin resistance and metabolic syndrome.

HbA1c reflects long-term glycaemic exposure and may remain within the normal range despite early metabolic impairments detectable through postprandial measures. This underscores the importance of stimulated glucose and insulin measurements as sensitive indicators of early metabolic risk in adolescents and young adults. Although the prevalence of impaired fasting glucose and impaired glucose tolerance was low and did not differ significantly between quintiles, a clear gradient was observed for elevated stimulated insulin levels. The significantly lower proportion of participants with hyperinsulinaemia in the earliest quintile, compared to those eating after 7:45 pm, reinforces the role of late-night eating in increasing insulin demand. Chronic exposure to such hyperinsulinaemic states may accelerate β-cell stress and progression towards metabolic syndrome over time[36].

Correlation analyses further strengthen the findings, demonstrating significant positive associations between time of last meal and percent body fat, 2-hour glucose, fasting insulin, stimulated insulin, and HOMA-IR. The negative correlation with muscle mass highlights the dual impact of late eating on both adiposity and lean tissue. Together, these findings suggest that meal timing influences multiple components of metabolic health, even in the absence of clinically overt disease.

Late-night food consumption may disrupt the synchrony between peripheral metabolic clocks and the central circadian pacemaker located in the suprachiasmatic nucleus[37]. Peripheral clocks in metabolically active tissues such as the liver, adipose tissue, pancreas, and skeletal muscle are highly responsive to feeding cues[38]. When food intake occurs late in the evening or night, these peripheral clocks may become misaligned with the central circadian rhythm, which is primarily entrained by the light–dark cycle. Such circadian misalignment has been shown to impair insulin signalling pathways, reduce glucose tolerance, and promote compensatory hyperinsulinemia[39]. Over time, this metabolic inefficiency may favour increased fat storage and reduced lipid oxidation, contributing to higher adiposity[39]. The observed association between later timing of the last meal, increased body fat, and elevated insulin resistance in the present study supports the hypothesis that meal timing plays a critical role in maintaining circadian–metabolic alignment, even in young and otherwise healthy individuals. Given the cultural trend towards late dinners, particularly in urban Indian settings, these findings have important public health implications. Encouraging earlier dinner timing may represent a simple, low-cost strategy to improve metabolic health among adolescents and young adults.

The strengths of this study include the large sample size, objective assessment of body composition, and comprehensive evaluation of glucose–insulin dynamics. However, the cross-sectional design limits causal inference. The primary objective of the study was to examine the association between meal timing, particularly the last meal

of the day, and early metabolic risk markers. Emerging evidence from chrononutrition research indicates that evening and late-night eating has a disproportionate impact on glucose metabolism, insulin sensitivity, and circadian alignment compared to other meals[40]. Therefore, the timing of the last meal was selected as a pragmatic and biologically relevant marker of eating rhythm. While overall dietary patterns are important, they were beyond the scope of the present analysis. Additionally, dietary composition, physical activity patterns, sleep duration, and chronotype were not accounted for, all of which may interact with meal timing to influence metabolic outcomes. Future longitudinal and intervention studies are needed to confirm causality and explore mechanisms.

In conclusion, later consumption of the last meal is associated with higher body fat, lower muscle mass, impaired postprandial glucose handling, and increased insulin resistance in adolescents and young adults. These findings emphasise the importance of not only what and how much is eaten, but also *when* food is consumed, in the early prevention of metabolic syndrome.

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Ethical Clearance: The study was approved by the Intersystem Biomedical Ethics Committee, Mumbai, India (ISBEC version 2 dated 12th Aug, 2017)

Declaration of conflicts of interest statement: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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