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THE INTERACTION BETWEEN SLEEP DURATION, BREAKFAST ENERGY INTAKE, AND BODY COMPOSITION (FFM, BMR, PHASE ANGLE): A NARRATIVE REVIEW

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ABSTRACT

Background: Sleep duration, chrononutrition, and meal timing are emerging determinants of metabolic health. Circadian misalignment, caused by sleep restriction or irregular eating patterns, may adversely affect body composition by promoting adiposity and reducing muscle preservation through neuroendocrine and molecular dysfunctions.
Objectives: This review synthesized research findings from the past decade (2016–2026) to evaluate how sleep duration and breakfast energy intake influence body composition parameters, specifically fat-free mass (FFM), basal metabolic rate (BMR), and cellular phase angle (PhA).
Methods: Literature search was conducted in PubMed and Google Scholar. From 306 identified papers, 12 original research studies involving diverse populations (including children, adolescents, and adults across multiple continents) were included. The review analyzed observational and experimental data focusing on sleep patterns, meal frequency, and timing as primary exposures, with body composition metrics as the main outcomes.
Results: Experimental sleep restriction (4 hours/night) significantly increased caloric intake ($P = 0.015$) and visceral fat ($P = 0.042$). Optimal sleep (6–7 hours) was associated with more favorable body composition than extreme durations ($P < 0.05$). Regarding chrononutrition, habitual breakfast skipping was positively correlated with lower appendicular skeletal muscle mass index ($\beta = 0.087$, $P = 0.031$). In large-scale observational data, night eating increased metabolic syndrome prevalence by 48% (OR: 1.48; 95% CI: 1.15–1.90), while morning eating was protective. Each one-hour delay in eating timing was associated with a 1.25-unit increase in body fat percentage ($P = 0.010$). Phase angle, reflecting cellular integrity, improved with regular breakfast consumption ($r = 0.15$) and physical activity. Notably, physical activity often moderated these relationships, acting as a stronger predictor of FFM than sleep in specific demographics.
Conclusion: Sufficient sleep and structured, early-timed meal patterns, particularly regular breakfast consumption, support lean mass preservation and reduced adiposity. Maintaining consistent sleep-wake cycles and physical activity are essential synergistic strategies to mitigate the metabolic risks associated with circadian misalignment.

Keywords: Body Composition; Sleep; Breakfast; Circadian Rhythm; Basal Metabolism

INTRODUCTION

Recent research highlights a complex link between sleep, chrononutrition, and body composition, with implications for metabolic health and obesity risk. Short or irregular sleep is associated with higher body fat, lower fat-free mass (FFM), reduced basal metabolic rate (BMR), and poorer phase angle (PhA). Experimental evidence shows that sleep restriction can increase energy intake without affecting energy expenditure, promoting weight gain and particularly abdominal fat accumulation, including visceral fat. Similarly, randomized trials consistently suggest insufficient sleep contributes to greater hunger, higher caloric and fat intake, and positive energy balance. Beyond sleep duration, meal timing and chrononutrition also appear important, as delayed eating patterns and eating jetlag have been linked to altered adiposity and appetite regulation. Together, these findings suggest that both sleep loss and circadian-related eating behaviors may contribute to unfavorable body composition and increased metabolic risk.[1-3] Skipping breakfast or consuming most daily energy later in the day is linked to increased adiposity and unfavorable body composition.[4, 5] Chronotype (morningness/eveningness) further modulates these relationships: evening types tend to have shorter sleep, skip breakfast more often, and display higher BMI and fat mass.[4] While some studies report no direct association between sleep duration and BMR

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or FFM, in specific populations, which are pediatric and geriatric [6, 7] others suggest that regular breakfast consumption supports higher FFM and PhA values.[8, 9] The timing of both sleep and meals, especially aligning food intake with circadian rhythms, emerges as a key factor influencing energy balance, body composition, and metabolic outcomes.[10, 11] This review discusses the latest research findings (from the past 10 years) related to sleep duration and breakfast energy intake influence on body composition (FFM, BMR, phase angle).

METHODS

To evaluate the impact of sleep duration and breakfast energy intake on body composition, a comprehensive review was conducted. A comprehensive electronic search was performed using the PubMed and Google Scholar databases to identify relevant peer-reviewed studies published within the last 10 years (2016–2025). The search strategy utilized targeted keywords (by combining the keywords such as "Sleeping duration", "FFM", "BMR", "PhA", and utilizing Boolean operations AND or OR in the search terms) to capture the relationships between primary exposures, specifically sleep duration and breakfast energy intake, and key metabolic outcomes, including FFM, BMR, and PhA.

The study selection process focused on original research that examined the specific interactions between these variables. The inclusion criteria prioritized studies that provided empirical data on how sleep patterns and chrononutrition influence adiposity and lean mass within adult demographics (aged 18-65). Initially, the search yielded a total of 306 papers. These records underwent a rigorous screening process to ensure relevance to the review's core objectives. Following the exclusion of non-original research and studies that did not address the specific interaction of the target variables, 12 papers were selected for final inclusion and synthesis.

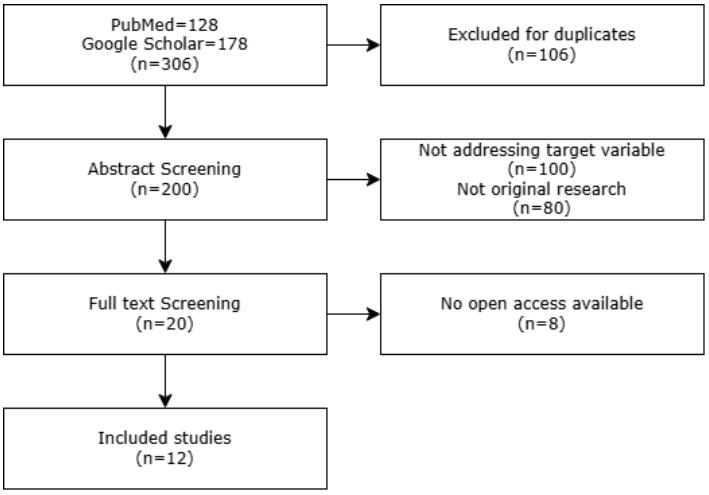


Figure 1. Overview of the search proces

RESULTS

A literature search was conducted in April 2026 across the specified electronic databases. To establish a comprehensive synthesis of the current circadian-metabolic framework, eligible studies were retrieved comprising a heterogeneous mix of observational designs, randomized controlled trials (RCTs), and randomized crossover experiments, with the details of the included studies available at **Table 1**.

Table 1. Characteristics of 12 Studies Included

Authors	Study Design	Participants	Location	Results
Yasuda et al (2018)[9]	Observational	270 students	Japan	• Habitual breakfast intake frequency was positively correlated with appendicular skeletal muscle mass index (β =.087, P =.031) and %AppFFM (β =.086, P =.045) after controlling for age, sex, living conditions, Pittsburgh Sleep Quality Index, Morningness-Eveningness Questionnaire, and International Physical Activity Questionnaire scores. • Research indicates that skipping breakfast may lead to reduced muscle mass in healthy young individuals, regardless of factors like age, sex, or physical activity.

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Table 1. Characteristics of 12 Studies Included(*Continue…)*

Authors	Study Design	Participants	Location	Results
Ha and Song (2019)[12]	Observational	14,279 Korean adults from KNHANES	South Korea	• In men only, higher meal frequency was inversely related to abdominal obesity, high blood pressure, and raised triglycerides; additionally, night eating was associated with a 48% higher prevalence of metabolic syndrome (OR: 1.48; 95% CI: 1.15–1.90). • Morning eating reduced metabolic syndrome prevalence for both men (OR: 0.73) and women (OR: 0.69), while obesity and metabolic syndrome were linked to longer fasting durations and shorter sleep. • Research on Korean adults suggests cardiometabolic risks are driven by a combination of calorie distribution, meal frequency, and sleep duration, rather than fasting duration alone.
Tan et al (2019)[13]	Observational	19,709 older adults from the Swedish EpiHeath study	Sweden	• Sleep durations of 6 to 7 hours were associated with more favorable body composition compared to extreme sleep durations ($P < 0.05$). • Both inadequate sleep (≤ 5 hours) and excessive sleep (≥ 8 hours) correlated with diminished fat-free mass and increased fat mass ($P < 0.05$).
Jansen et al (2020)[14]	Observational	458 Mexican Adolescents part of ELEMENT study	Mexico	• Adolescents consuming higher amounts of plant-based and lean proteins experienced earlier sleep timing (−0.45 h; 95% CI −0.81, −0.08), reduced phase delay during follow-up (−0.39 h; 95% CI −0.80, 0.02), and shorter weekend sleep durations (−0.5 h; 95% CI −0.88, −0.1). • While higher consumption of eggs, milk, and refined grains correlated with earlier sleep timing (−0.40 h; 95% CI −0.77, −0.04), diets high in meat and starchy foods were linked to increased social jetlag, measured by larger differences between weekday and weekend sleep schedules.
Thomas et al (2021)[10]	RCT	83 overweight and obese adults	United States of America	• Each one-hour delay in eating was associated with a 1.25 unit increase in body fat percentage (95% CI: 0.60, 1.91; FDR $P = 0.010$), while each hour delay in the meal window midpoint saw a 1.35 unit increase (95% CI: 0.51, 2.19; FDR $P = 0.029$). • Every 1-hour extension of sleep duration resulted in a 1.64 unit increase in body fat percentage (95% CI: 0.56, 2.72; FDR $P = 0.044$). • Later eating and sleep schedules were significantly correlated with reduced levels of physical activity.
Covassin et al (2022)[1]	Crossover RCT	12 healthy adults	United States of America	• Participants with restricted sleep consumed significantly more calories ($P = 0.015$), protein ($P = 0.050$), and fat ($P = 0.046$), leading to substantially higher weight growth ($P = 0.008$) despite constant energy expenditure ($P > 0.16$). • While total body fat remained stable ($P = 0.710$), sleep restriction caused a rise in total abdominal fat ($P = 0.011$), specifically increasing subcutaneous ($P = 0.047$) and visceral ($P = 0.042$) fat depots.

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Table 1. Characteristics of 12 Studies Included(*Continue…)*

Authors	Study Design	Participants	Location	Results
Goertz et al (2022)[7]	Observational	118 children	United States of America	• A later sleep midpoint is significantly linked to higher fat mass (0.33; 95% CI: 0.05, 0.60) and total body fat (0.92; 95% CI: 0.15, 1.70). • Later sleep midpoints correlate with delayed mealtimes, resulting in higher overnight energy intake (45.6; 95% CI: 19.7, 71.4) and lower morning energy intake (-44.8; 95% CI: −72.0, −17.6). • Reduced sleep duration (-0.02; 95% CI: -0.03, 0.00) and later meal timing (0.83; 95% CI: 0.24, 1.42) are predictive of higher body fat levels one year later.
Jaremkow et al (2022)[5]	Observational	281 Wroclaw Medical University students	Poland	• Physical activity shows a moderate-to-strong positive correlation (r ~ 0.4 to 0.7) with key metrics like muscle mass, bone mass, basal metabolic rate, and fat-free mass. • A negative correlation (r ~ -0.4 to -0.6) exists between activity levels and fat mass across all examined groups. • The study also linked activity to changes in height, weight, BMI, and the ratio of extracellular to intracellular water.
Alafif and Alruwaili (2023)[8]	Observational	311 students	Saudi Arabia	• Within the study population, 30.2% of students were classified as overweight or obese, with a specific BMI breakdown of 17.7% underweight, 52.1% normal weight, 20.6% overweight, and 9.6% obese. While 67.8% reported insufficient sleep, 71.7% maintained good sleep quality, and over 70% fell asleep within 30 minutes. • Dietary habits were notably poor, with only 12.5% of students consuming vegetables and 6.4% consuming fruits daily. Furthermore, only 8% of students eat breakfast regularly, compared to 62.1% for lunch and 29.9% for dinner.
Dote-Montero et al (2023)[15]	Observational	118 young adults	Spain	• Meal timing did not affect body composition ($p > 0.05$). • The eating window inversely correlated with HOMA-IR and cardiometabolic risk score in men ($R^2 = 0.348$, $\beta = -0.605$; $R^2 = 0.234$, $\beta = -0.508$; all $p \leq 0.003$). • In men, the period from midsleep to first food intake was positively correlated with HOMA-IR and cardiometabolic risk score ($R^2 = 0.212$, $\beta = 0.485$; $R^2 = 0.228$, $\beta = 0.502$; all $p = 0.003$). • These relationships persisted after controlling for covariates and multiples ($p < 0.011$).
AlMasud et al (2025)[11]	Observational	1,741 female university students	Saudi Arabia	• BMI increased significantly from the first to the third tertile for both Fat Mass Index (FMI) and Fat-Free Mass Index (FFMI) ($P < 0.001$), while the Fat Mass to Fat-Free Mass ratio (FM/FFM) decreased across tertiles ($P < 0.001$). • Significant correlations were found between physical activity (frequency and duration) and body composition (FFMI and FMI), especially when controlling age and BMI. Conversely, sleep duration and quality showed no significant link to body composition.

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Table 1. Characteristics of 12 Studies Included(*Continue…)*

Authors	Study Design	Participants	Location	Results
Sulis et al (2025)[16]	Observational	962 adults	Slovakia	• Males exhibited higher systolic blood pressure (SBP) and Fat-Free Mass Index (FFMI); specifically in men, healthy eating correlated with higher Phase Angle (PhA) (r = 0.12) and lower pulse (r = -0.16), while salt intake lowered PhA (r = -0.15). • Breakfast consumption and fewer daily meals increased PhA (r = 0.15), and while coffee/milk intake improved FFMI in women (r = 0.11), energy drink consumption was linked to reduced FFMI across both genders. • In female participants, the combination of poor sleep quality and late-night eating habits was significantly associated with an increased BMI (r = -0.11).

Sleep Duration and Body Composition

A comparative analysis of sleep duration reveals that insufficient rest acts as a potent catalyst for adiposity, though the mechanisms and population-specific impacts vary. Experimental evidence from a highly controlled in-patient study isolated the direct effects of sleep loss: restricting sleep to four hours per night for 14 days led to hyperphagia (increased intake of calories, fat, and protein) without any compensatory increase in energy expenditure, directly resulting in weight gain and a specific, hazardous accumulation of abdominal visceral and subcutaneous fat.[1] This causal link supports epidemiological findings across diverse age groups; for instance, short sleep duration is significantly associated with higher rates of obesity and lower weekly vegetable intake among university students,[8] as well as longitudinal adiposity gains in preschool-aged children.[7] Interestingly, the relationship between sleep and body composition is not strictly linear across the lifespan. Among middle-aged and older adults, both habitual short sleep (5 hours or less) and long sleep (9 hours or more) were associated with a detrimental phenotype characterized by higher fat mass and significantly lower fat-free mass, increasing the dual risks of obesity and sarcopenia.[12] Shorter sleep duration is associated with increased fat mass (FM), decreased FFM, higher BMI, and greater risk of sarcopenia across age groups.[1, 2] Both short (<6 h) and long (>8 h) sleep durations are linked to adverse body composition compared to moderate sleep durations. In children/adolescents, insufficient sleep predicts higher percent body fat longitudinally;[13] in adults/elderly populations, poor sleep quality correlates with higher FM% and lower FFM%.[14] In stark contrast, a study of young adult Saudi women found no significant associations between sleep duration or quality and advanced body composition metrics like the Fat Mass Index (FMI) or Fat-Free Mass Index (FFMI), as physical activity frequency and duration were the primary determinants of body fat and lean mass in this cohort.[11] This discrepancy suggests that in certain healthy or younger demographics, other behavioral factor, such as physical activity or diet, may override the somatic impacts of sleep duration.

The Impact of Meal Timing and Frequency on Adiposity and Lean Mass

The synthesized literature presents contrasting but complementary findings regarding how meal timing and eating windows dictate body composition, suggesting that baseline metabolic health and age strongly mediate these outcomes. In a cross-sectional study of healthy young adults, general meal timing metrics, such as the caloric midpoint and eating window, were not significantly associated with body composition parameters like fat mass or lean mass, although an earlier first food intake did improve cardiometabolic risk scores in men.[14] However, in populations already battling metabolic dysfunction, the somatic consequences of meal timing are severe. Observational data of adults with overweight or obesity revealed that a later start to the eating window and a later caloric midpoint strongly predict a higher percent body fat and reduced physical activity.[10] This adverse effect of delayed eating is corroborated by longitudinal pediatric data, which demonstrated that later meal timing and delayed sleep midpoints in preschool children led to measurable increases in percent body fat one year later.[7] Beyond fat mass, the structural timing of meals (specifically breakfast consumption) plays a protective role in preserving muscle tissue.

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Habitual breakfast skipping (eating breakfast 3 or fewer times per week) was cross-sectionally linked to a significantly lower appendicular fat-free mass and skeletal muscle mass index in healthy young adults, likely due to the prolonged negative energy and protein balances induced by fasting.[9] Supporting the benefits of early eating, morning food consumption was associated with a lower prevalence of metabolic syndrome in Korean adults, whereas night eating significantly increased metabolic risks, particularly in men.[7] Skipping breakfast or irregular breakfast consumption is associated with lower FFM/muscle mass in young adults, and increased risk of overweight/obesity,[5] poorer metabolic profiles, and less favorable PhA values.[9] Higher morning energy intake is linked to lower odds of overweight/obesity among morning chronotypes; conversely, late-night eating increases obesity risk among evening types.[12]

Circadian Alignment, Social Jetlag, and Dietary Quality

The literature robustly indicates that the timing and day-to-day regularity of sleep are intricately tied to dietary behaviors, creating a bidirectional feedback loop that impacts cellular health and weight status. Prospective cohort data showed that adolescents adhering to a healthier "Plant-Based and Lean Proteins" diet exhibited significantly earlier sleep timing and shorter weekend catch-up sleep. Conversely, diets heavy in processed "Meat and Starchy" foods were predictive of greater social jetlag (the discrepancy between weekend and weekday sleep timing).[15] This relationship is echoed in young adults, where irregular sleep routines combined with late-night eating were directly correlated with elevated Body Mass Index (BMI) and altered blood pressure in women.[16] In young children, delayed sleep midpoints disrupt nutritional pacing by shifting caloric intake away from the morning and toward the nighttime, drastically delaying the first and last meals of the day.[7] Evening chronotypes are more likely to have shorter sleep durations, skip breakfast frequently, consume more calories at night/snacks/dinner and exhibit higher BMI/FM than morning types.[4, 12] Circadian misalignment (e.g., late meal/sleep timing) independently predicts increased body fat percentage regardless of total caloric intake or macronutrient distribution.[16] Evidence for direct associations between habitual sleep duration or breakfast patterns with BMR is mixed: some studies find no significant relationship after adjusting for confounders,[6] while others suggest that time-restricted eating aligned with circadian rhythms may improve resting energy expenditure over time.[15]

Phase Angle and Moderating Role of Physical Activity

Higher PhA values are observed in individuals with regular breakfast habits and healthier eating patterns; PhA correlates positively with FFM/BMR/muscle mass parameters.[8, 9] The quality and structure of the diet further influence cellular integrity, measured via bioelectrical impedance as the PhA. A higher PhA, indicating robust cellular health, was positively associated with structured meal patterns (fewer daily meals and regular breakfast), higher fat-free mass, and adequate hydration.[5, 16] Conversely, the frequent consumption of energy drinks and high-salt foods was negatively correlated with PhA and the Fat-Free Mass Index across both sexes.[16]

Across the reviewed studies, physical activity emerges as a crucial moderator that offsets the negative impacts of poor sleep and diet on body composition. In young women, the frequency and duration of physical activity were the strongest predictors of a healthier body composition, demonstrating a significant inverse relationship with the Fat Mass Index (FMI) and a positive association with the Fat-Free Mass Index (FFMI), even after adjusting for age and BMI.[11] Similarly, frequent physical exercise was positively correlated with higher cellular Phase Angle and increased muscle mass in university students.[5] The interplay between sleep, eating, and movement is highly integrated; adults with later eating windows and delayed sleep offsets engaged in significantly fewer daily steps and lower metabolic equivalents (METs).[10] This behavioral shift indicates that late-night eating and delayed sleep schedules may exacerbate fat gain not only through direct metabolic channels, but also indirectly by impairing daily physical activity levels through altered circadian alertness and fatigue. Even in preschool children, greater day-to-day sleep regularity was independently associated with spending more time engaged in light physical activity.[7] Collectively, these findings suggest that while circadian misalignment (late sleep/eating) drives fat accumulation, maintaining regular physical activity, structured early meals, and consistent sleep schedules are essential synergistic strategies for preserving fat-free mass and mitigating adiposity.

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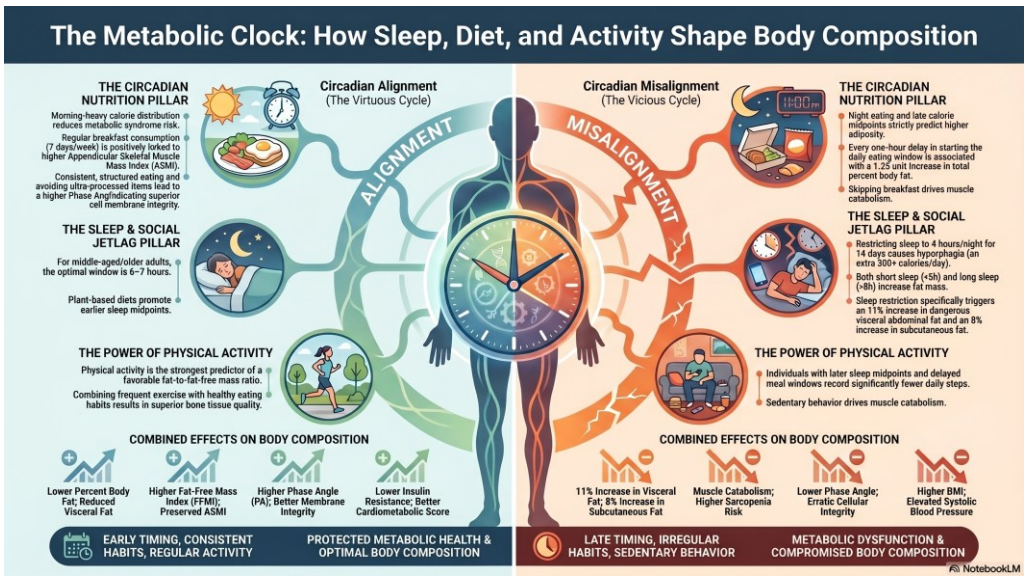


Figure 2. Overview of the synthesized 12 study sources. This figure was generated by NotebookLM.

DISCUSSIONS

The contemporary understanding of metabolic health has undergone a paradigm shift, moving beyond the simplistic "energy balance" model to a sophisticated "circadian-metabolic" framework. In this new paradigm, the timing of biological and behavioral events sleep, nutrient intake, and physical activity is recognized as a primary determinant of adiposity, muscle preservation, and cellular integrity. The endogenous circadian system, coordinated by the suprachiasmatic nucleus (SCN) and peripheral oscillators in the liver, gut, and skeletal muscle, ensures that metabolic processes are optimized for the predictable 24-hour cycle of light and dark.[17] However, the modern 24/7 society imposes chronic stressors such as sleep restriction, social jetlag, and nocturnal feeding that lead to "circadian misalignment." This state of misalignment is characterized by the desynchronization of internal biological time from external environmental and social time, triggering a cascade of neuroendocrine and molecular dysfunctions.[18]

Sleep Duration and Body Composition

Five among twelve studies included in this review, demonstrates that short or irregular sleep (especially when combined with skipping breakfast or delayed meal timing) contributes to increased adiposity (higher FM), reduced muscle mass (lower FFM), and lower phase angle values indicative of poorer cellular health. These effects are amplified among evening chronotypes who tend toward later bedtimes/wake times as well as unhealthy dietary patterns such as skipping breakfast or consuming most calories at night.[4, 13] While some cross-sectional studies report no direct association between habitual sleep duration or quality with BMR/FFM after adjusting for confounders like physical activity or BMI,[6, 7] experimental evidence suggests that aligning both sleep and meal timing with endogenous circadian rhythms can improve metabolic efficiency (even without caloric restriction) by supporting optimal substrate utilization during active periods of the day.[14]

The relationship between sleep duration and adiposity is not merely correlational; it is driven by high-impact causal mechanisms that alter both the hedonic drive for food and the molecular capacity for lipid mobilization. Experimental evidence from tightly controlled in-patient environments reveals that when sleep is restricted to four hours per night, the body enters a state of hyperphagia, where caloric intake significantly exceeds biological needs without a corresponding increase in energy expenditure.[18] At the core of sleep-induced weight gain is a neurochemical shift within the hypothalamus, the brain's primary metabolic control center. Much of what we know about these molecular mechanisms stems from experimental animal models. Chronic sleep deprivation in animal models has demonstrated that sleep loss triggers a robust upregulation of Neuropeptide Y (NPY) mRNA in the arcuate nucleus. NPY is a potent orexigenic peptide that increases appetite and favors fat storage. Simultaneously, sleep loss is associated with the downregulation of α-melanocyte stimulating hormone (α-MSH), an anorexigenic peptide that typically signals satiety.[19] In humans, this hypothalamic shift is mirrored by a

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"neurohormonal" imbalance where circulating leptin levels decrease, and ghrelin levels rise, effectively stripping away the biological brakes on overeating while intensifying the hunger signal.[18]

Beyond these homeostatic hunger signals, sleep deprivation enhances the "hedonic" drive to eat. Functional imaging studies suggest that sleep loss increases the reward value of high-calorie, palatable foods (rich in fat and sugar), making cognitive control over dietary choices significantly more difficult.[18] While the brain drives increased intake, peripheral tissues undergo molecular changes that prevent the mobilization of stored energy. Recent high-impact research identifies the suppression of the SIRT1/FOXO1/ATGL signaling pathway as a definitive mechanism for fat accumulation in visceral white adipose tissue (vWAT) during sleep deprivation.[20] Sirtuin 1 (SIRT1) is an NAD^+-dependent deacetylase that acts as a sensor of energy status and a regulator of metabolic homeostasis.[17] Under normal sleep conditions, SIRT1 deacetylates and activates the transcription factor FOXO1, which subsequently promotes the expression of Adipose Triglyceride Lipase (ATGL)—the rate-limiting enzyme for lipolysis. Sleep deprivation causes a significant reduction in the expression of SIRT1 and FOXO1 in vWAT, leading to the downregulation of ATGL. This suppression effectively inhibits the breakdown of triglycerides, promoting fat storage even when the body might otherwise require energy. Experimental evidence shows that activating this pathway with resveratrol can revitalize lipid mobilization in sleep-deprived models, underscoring SIRT1 as a potential therapeutic target for mitigating sleep-related obesity.[20]

A significant difference in the literature exists in research involving young adult Saudi women, where no significant associations were found between sleep duration and advanced body composition metrics like the Fat Mass Index (FMI) or Fat-Free Mass Index (FFMI). Rather than disproving the link between sleep duration and adiposity, this discrepancy highlights how population-specific (people aged 21-23 years old, like the majority of participants in this study) lifestyle factors can alter observed metabolic outcomes. In this cohort, the somatic impact of sleep may have been masked or overridden by high levels of physical activity and distinct dietary structures. Because physical activity frequency served as the primary predictor of body composition, the metabolic benefits of movement likely provided a compensatory buffer against the typical adipogenic effects of varied sleep. However, there were some limitations of the study such as short observation period (3 months), self-reported BMI values, gender imbalance in the number of participants (72,3% woman, 27,7% man).[11]

The Impact of Meal Timing and Frequency on Adiposity and Lean Mass

"Chrononutrition" represents the integration of nutritional science with chronobiology, focusing on the timing of energy and nutrient intake.[21] The synthesized evidence suggests that the human body is metabolically optimized for "early" feeding, and deviations from this schedule (such as breakfast skipping or late-night eating) disrupt the coordination of peripheral clocks and drive adiposity. Mechanistically, shifting toward a later caloric midpoint blunts the thermic effect of food and diminishes postprandial fat oxidation, creating a positive energy balance that accelerates fat accumulation; simultaneously, this circadian phase delay lowers morning alertness and promotes diurnal fatigue, thereby suppressing voluntary physical activity. The omission of breakfast is a widespread behavior among young adults and is strongly linked to adverse body composition, specifically the loss of skeletal muscle mass. Breakfast is critical because it terminates the overnight catabolic fast, initiating metabolic processes and providing the first stimulus for muscle protein synthesis (MPS).[22]

Research has shown that habitual breakfast skipping is associated with a lower appendicular fat-free mass and skeletal muscle mass index (SMI). The molecular mechanism involves the "mechanistic/mammalian Target Of Rapamycin" (mTOR) signaling pathway, which is the primary regulator of muscle growth.[23] MPS requires both the stimulation of mTOR through physical activity and the availability of amino acids (specifically leucine) from dietary protein. Prolonged fasting beyond the overnight period—as seen in breakfast skippers—reduces mTOR phosphorylation by approximately 50%, leading to a net catabolic state where muscle protein breakdown (MPB) exceeds synthesis. This is particularly dangerous for sedentary individuals, as it accelerates the loss of lean tissue and decreases the resting metabolic rate (RMR).[24]

Circadian Alignment, Social Jetlag, and Dietary Quality

Circadian misalignment occurs when an individual's behavioral patterns (sleep/wake, fasting/feeding) are out of phase with their internal biological clock. The most common form of this in non-shift workers is "social jetlag" (SJL), defined as the difference in the midpoint of sleep between workdays and free days. A delayed sleep midpoint directly shifts eating patterns later into the biological night, impairing glucose tolerance and lowering

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overall BMR. The 2026 literature robustly identifies SJL as a significant and independent risk factor for obesity and metabolic syndrome. Large-scale meta-analyses covering over 230,000 participants have found a consistent positive correlation between SJL and BMI, fat mass, and waist circumference. Even in individuals who obtain "adequate" total sleep duration, each hour of SJL is associated with a 33% increase in the risk of obesity.[25]

An emerging dimension of circadian research (2025–2026) is the role of the gut microbiome in mediating the metabolic effects of sleep loss and SJL. Microbial communities have their own circadian rhythms that are synchronized with the host's feeding cycles. SJL disrupts these microbial oscillations, leading to reduced production of short-chain fatty acids (SCFAs) like butyrate, which are essential for maintaining the intestinal barrier.[17]

Phase Angle and Moderating Role of Physical Activity

Phase angle is a composite measure of resistance (R) and reactance (Xc). Resistance is inversely related to total body water, while reactance represents the capacitive effect of cell membranes (their ability to store electrical charge). Therefore, a high PhA reflects superior cell membrane integrity, higher cell mass, and optimal hydration, while a low PhA is a marker of cellular death, fragmentation, and extracellular fluid overload. PhA is particularly valuable for assessing "muscle quality," which refers to the structural and qualitative characteristics of muscle tissue rather than just its quantity.[26] PhA emerges as a promising integrative biomarker: it reflects not only cell membrane integrity but also correlates positively with healthy lifestyle factors such as regular breakfast consumption/higher FFM/hydration status/physical activity, all of which are influenced by both sleep habits and meal timing patterns[8, 9]. PhA can predict body strength and agility independently of skeletal muscle mass, making it a critical tool for identifying functional vulnerability.[27]

On the other hand, Physical activity (PA) appears to be an important moderator of the negative impacts of poor sleep and diet on body composition. The research indicates a bidirectional "self-reinforcing feedback loop": regular PA improves sleep quality through the regulation of circadian rhythms and thermoregulatory effects, while sufficient sleep provides the executive functioning and energy levels necessary to sustain a consistent exercise routine.[18] High levels of physical activity have been shown to attenuate the hypothalamic-pituitary-adrenal (HPA) axis hyperactivity often seen in sleep disorders.[28] In active individuals, the negative metabolic consequences of short sleep such as elevated visceral fat are significantly reduced compared to inactive counterparts. For young women, PA was the single strongest predictor of a healthy body composition, emphasizing its role as a "metabolic safeguard".[11]

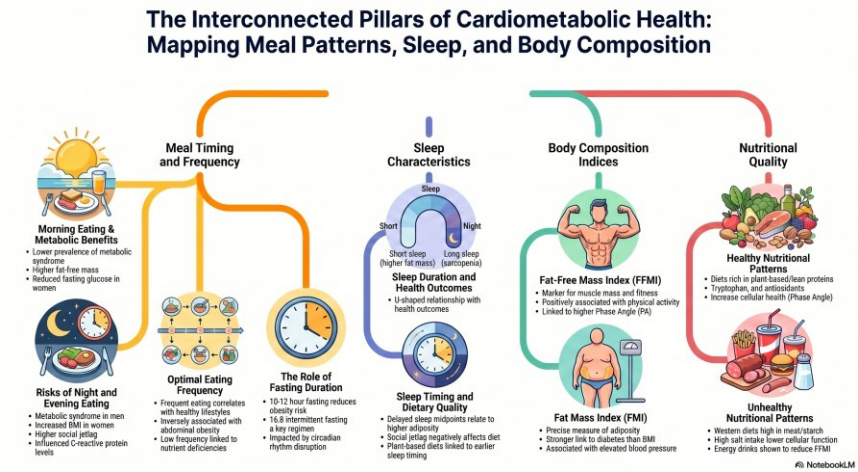


Figure 3. Summary of Meal Timing Patterns, Sleeps, and Cardiometabolic Factors on Human Health. This figure was generated by NotebookLM.

However, much of the evidence remains observational; causality is difficult to establish due to confounding factors such as physical activity levels/stress/genetic predisposition/social jetlag/work schedules/etc. There is also heterogeneity in measurement methods for both dietary/sleep variables as well as body composition indices. Future research should pursue large, well controlled trials to disentangle these complex interactions.

CONCLUSION

Sufficient nightly sleep combined with regular breakfast consumption—especially when

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timed earlier in the day supports healthier body composition (higher FFM/lower FM), better basal metabolism trajectories over time (BMR), improved phase angle values reflecting cellular health, and reduced risk for obesity/metabolic syndrome. Chronotype/circadian alignment further modulates these effects.

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