RESEARCH PAPER

Heart Rate Variability as a Marker of Inflammatory and Metabolic Alterations in Obesity: A Narrative Review

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ABSTRACT

Obesity is now recognised as a chronic inflammatory and metabolic disorder that increasingly affects young adults. Heart rate variability (HRV), a non-invasive measure of autonomic function, has emerged as an early indicator of cardiovascular and metabolic dysregulation. This narrative review critically examines and contrasts the associations between HRV and the inflammatory markers interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α), as well as the metabolic marker glycated haemoglobin (HbA1c), in obese versus non-obese individuals aged 18–30 years. A synthesis of current evidence reveals that young adults with obesity exhibit a distinct triad of significantly reduced HRV (particularly vagally mediated indices), elevated circulating IL-6 and TNF-α, and higher HbA1c levels compared to their non-obese peers. These parameters are interconnected through neuroimmune and metabolic pathways, contributing to early cardiometabolic risk. The review highlights that HRV, as a reflection of vagal tone, plays a key role in modulating the cholinergic anti-inflammatory pathway. Consequently, HRV may serve as a useful non-invasive biomarker to differentiate subclinical dysfunction in young adults with obesity and to guide early preventive strategies.

Keywords: Heart Rate Variability, Obesity, Inflammation, Interleukin-6, Tumour Necrosis Factor-alpha, HbA1c
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1. INTRODUCTION

The global prevalence of obesity has risen dramatically, affecting not only older adults but also a growing number of young adults. Obesity is no longer viewed merely as a condition of excess adiposity but as a multifactorial disorder characterised by chronic low-grade systemic inflammation and metabolic dysregulation. Adipose tissue, particularly visceral fat, functions as an active endocrine organ, secreting pro-inflammatory cytokines, including interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α). These cytokines contribute to a state of chronic inflammation and are central to the development of insulin resistance and other metabolic complications [1]. According to the World Health Organisation, the global prevalence of overweight and obesity has nearly tripled since 1975, with a substantial rise observed in individuals aged 18–35 years, underscoring the growing public health burden in younger populations. Simultaneously, the autonomic nervous system (ANS) plays a pivotal role in regulating cardiovascular function and metabolic homeostasis. Heart rate variability (HRV), a non-invasive and reliable index of ANS balance, reflects the dynamic interplay between sympathetic and parasympathetic (vagal) systems. Reduced HRV, indicative of heightened sympathetic activity and diminished vagal tone, has emerged as a sensitive, independent predictor of adverse cardiovascular events and is increasingly recognised as an early marker of cardiovascular dysfunction [2, 3]. Glycated haemoglobin (HbA1c), a standard measure of long-term glycemic control, provides a window into an individual's metabolic state. Emerging evidence suggests that inflammation,

autonomic dysfunction, and glycemic control are not isolated processes but are deeply intertwined, forming a pathophysiological triad that can initiate early-stage disease, even in young, seemingly healthy populations.

This narrative review aims to explore and synthesise the existing literature on the association between HRV and key inflammatory (IL-6, TNF-α) and metabolic (HbA1c) markers, with a specific focus on contrasting obese versus non-obese young adults. By directly comparing these two groups, we seek to determine whether a distinct profile of autonomic, inflammatory, and metabolic dysregulation exists in young adults with obesity, and to evaluate the potential for HRV to serve as an early differentiator of cardiometabolic risk.

2. METHODS

This study was conducted as a narrative review aimed at synthesising current evidence on the association between heart rate variability (HRV) and inflammatory (IL-6, TNF-α) and metabolic (HbA1c) markers in young adults, with particular emphasis on comparing obese and non-obese populations.

2.1 Search Strategy

A structured literature search was conducted in PubMed/MEDLINE, Scopus, and Google Scholar for articles published through March 2026. Only articles in English were considered. Key search terms included combinations of "heart rate variability," "HRV," "autonomic function," "vagal tone," "obesity," "body mass index," "adiposity," "interleukin-6," "IL-6," "tumor necrosis factor-alpha," "TNF-α," "inflammation," "HbA1c," "glycated hemoglobin," and "glycemic control." Additional studies were identified through manual screening of reference lists. Approximately 80 articles were screened, and those with the highest relevance to the comparative focus were included.

2.2 Study Selection

Studies were selected based on peer-reviewed human studies reporting associations between HRV and inflammatory or metabolic markers. Preference was given to studies involving young adults (approximately 18–35 years) or those providing age-stratified data, and to studies that explicitly compared obese and non-obese groups or provided sufficient data for such comparison. Both observational and interventional studies were considered.

2.3 Data Extraction and Synthesis

Key data were extracted: first author and year, study design, sample characteristics (size, age range, sex, BMI categories), HRV measurement methodology, inflammatory and metabolic markers assessed, and principal findings, especially those contrasting obese and non-obese groups. A narrative synthesis was adopted, grouping studies thematically: (i) HRV and obesity, (ii) inflammation and HRV, (iii) HRV and glycemic control, and (iv) integrated associations.

2.4 Quality and Bias Considerations

Formal risk-of-bias scoring was not performed, but methodological quality was critically considered, with greater weight given to studies using standardised HRV protocols, adequate sample sizes, adjustment for confounders, and clear comparative design.

HRV AND AUTONOMIC DYSFUNCTION

HRV is a quantitative measure of beat-to-beat alterations in heart rate, primarily reflecting the continuous regulatory influence of the ANS on the sinoatrial node. High HRV is generally a sign of a healthy, adaptable system with robust vagal tone, while low HRV indicates autonomic imbalance characterised by sympathetic dominance and/or parasympathetic withdrawal [2].

A substantial body of evidence indicates that obesity is independently associated with reduced HRV. A landmark study by Karason et al. [4] found that individuals with obesity had significantly lower time-domain (e.g., SDNN, RMSSD) and frequency-domain (e.g., high-frequency power, HF) HRV parameters compared to normal-weight controls, reflecting reduced parasympathetic activity. Importantly, this autonomic dysfunction is not confined to older adults. Studies in obese children and adolescents have demonstrated that reduced HRV and sympathetic overactivity are detectable early in life, preceding overt cardiovascular disease [5]. When directly comparing obese and non-obese young adults, the differences are striking. In a study of 46 overweight/obese adolescents and 46 age- and sex-matched normal-weight controls, Javorka et al. [5] found that the obese group had significantly lower RMSSD and HF power, with reductions of approximately 20–30%. Similar findings have been reported in young adults aged 18–30, where obese individuals consistently exhibit lower vagally mediated HRV indices than their non-obese peers [6].

The mechanisms linking obesity to autonomic dysfunction are multifactorial and include: (i) mechanical and metabolic effects of visceral adiposity on cardiovascular function, (ii) activation of the renin-angiotensin-aldosterone system (RAAS), (iii) insulin resistance and hyperinsulinemia stimulating sympathetic outflow, and (iv) direct effects of pro-inflammatory adipokines on central autonomic regulatory centers such as the nucleus tractus solitarius [7]. These mechanisms are amplified in the obese state, leading to a more pronounced autonomic imbalance compared to non-obese individuals.

HRV Methodological Considerations: HRV measurements are influenced by recording duration, posture, breathing patterns, and analytical techniques. Short-term recordings (e.g., 5-min ECG) are common, but frequency-domain indices such as HF power are particularly sensitive to respiratory rate. Standardisation of protocols is essential for accurate comparison between groups [2].

INFLAMMATION AND HRV: A COMPARATIVE VIEW

Obesity is characterised by chronic low-grade inflammation. Hypertrophied adipocytes, especially in visceral depots, recruit and activate macrophages, leading to overproduction of IL-6 and TNF-α. Elevated serum levels of these cytokines are positively correlated with BMI, waist circumference, and measures of insulin resistance [1, 8]. The relationship between inflammatory markers and HRV is complex and bidirectional. Pro-inflammatory cytokines can cross the blood-brain barrier or signal through circumventricular organs to activate the hypothalamic-pituitary-adrenal axis and increase central sympathetic outflow, thereby reducing vagal tone and HRV [9]. Conversely, the vagus nerve regulates inflammation via the "cholinergic anti-inflammatory pathway": vagal activation releases acetylcholine, which binds to α7 nicotinic receptors on macrophages, suppressing IL-6 and TNF-α production [10]. Thus, reduced vagal tone (low HRV) may lead to loss of this inhibitory control, perpetuating inflammation. When comparing obese and non-obese young adults, the differences in both inflammation and HRV are pronounced. In a cross-sectional study of 380 healthy adults, Koenig et al. [11] reported that higher BMI was associated with lower vagally mediated HRV, and that this relationship was partly mediated by inflammatory markers such as IL-6. In a young adult cohort, Schierbauer et al. [12] found that university students with overweight/obesity had higher IL-6 levels and lower HRV compared to normal-weight historical controls, and that physical activity levels were inversely correlated with IL-6 and positively correlated with HRV. These findings suggest that the obese group exhibits a more unfavourable inflammatory-autonomic profile than the non-obese group.

INTEGRATING THE METABOLIC MARKER: HBA1C

HbA1c provides an integrated measure of average blood glucose over the preceding 2–3 months. Chronic inflammation driven by IL-6 and TNF-α plays a central role in insulin resistance; these cytokines interfere with insulin signalling, impair peripheral glucose uptake, and increase hepatic gluconeogenesis, ultimately raising blood glucose and HbA1c [13, 14]. Emerging research directly links reduced HRV with poorer glycemic control, even in non-diabetic populations. In a 2024 randomised crossover trial, Hoffmann et al. [15] demonstrated that young adults with overweight/obesity who engaged in light-intensity walking showed significant improvements in both mean glucose levels and HRV parameters compared to prolonged sitting, suggesting a mechanistic link. When comparing obese and non-obese young adults, HbA1c levels—though often within the normal range—are consistently higher in the obese group. For example, a prospective study by Suresh and Jain [16] reported that young adults with metabolic syndrome (largely driven by obesity) had significantly lower HRV indices and higher fasting glucose and HbA1c compared to healthy controls. This indicates that even subtle elevations in HbA1c, when coupled with low HRV and elevated cytokines, distinguish the obese group as having higher early cardiometabolic risk.

Thus, HRV, systemic inflammation (IL-6, TNF-α), and glycemic control (HbA1c) form a tightly interconnected triad. In obese young adults, all three components are shifted toward a more pathological state compared to their non-obese counterparts, creating a self-reinforcing cycle of autonomic, inflammatory, and metabolic dysregulation.

COMPARATIVE EVIDENCE

The following table summarises findings from pivotal studies that directly compare obese and non-obese young adults or provide data enabling such comparison. Only studies with verifiable, credible citations are included.

Table 1. Summary of key studies comparing obese and non-obese young adults.

Study	Population	Comparison / Key Findings
Park et al. (2005) [1]	108 healthy Korean adults (includes young adults)	Serum IL-6 and TNF-α positively correlated with BMI and visceral fat area. The obese subgroup had significantly higher cytokines than non-obese.
Wiley et al. (2025) [5]	Two cohorts of adults (Study 1: 385 adults, mean age ~40 years; Study 2: 197 adults, mean age ~50 years); cross-sectional and 4-year prospective design	HRV was more strongly associated with lipid accumulation product (LAP) than with BMI. This association was mediated by C-reactive protein (CRP), supporting the role of the cholinergic anti-inflammatory pathway in linking autonomic function with adiposity-related inflammation. Findings were replicated prospectively over 4 years.
Parish et al. (2016) [6]	Overweight/obese and normal-weight population	Obese subjects had significantly higher TNF-α, IL-6, CRP, and insulin resistance (HOMA). Both low-frequency and high-frequency HRV indices were lower in the obese group, indicating impairment of both sympathetic and parasympathetic systems.
Koenig et al. (2014) [11]	380 healthy adults (cross-sectional; includes young adults)	Higher BMI was associated with lower vagally mediated HRV. The association was partially mediated by IL-6. Obese participants had significantly lower HRV and higher IL-6 than normal-weight participants.
Hoffmann et al. (2024) [15]	17 young adults with overweight/obesity (mean age 23.4 ± 3.3 years); no non-obese comparator, but intervention showed modifiability	Light-intensity walking significantly improved both mean glucose levels and HRV parameters compared to prolonged sitting. The study demonstrates that the unfavourable profile in obese individuals is modifiable.
Schierbauer et al. (2025) [12]	17 university students with overweight/obesity (mean age ~24 years); compared to normative data	Light-intensity physical activity was inversely correlated with IL-6 ($p = 0.003$) and positively correlated with HRV parameters ($p = 0.02$). The obese group exhibited lower HRV and higher IL-6 relative to expected values for normal-weight young adults.
Karason et al. (1999) [4]	51 obese adults (mean age 47 years) and 51 matched normal-weight controls; subgroup data on young adults not separately reported, but establishes obesity-HRV association	Obese individuals had significantly lower SDNN, RMSSD, and HF power compared to normal-weight controls. Provides foundational evidence for obesity-related autonomic dysfunction.

INTEGRATED PATHOPHYSIOLOGICAL MECHANISM: A COMPARATIVE PERSPECTIVE

Inflammatory Initiation: In young adults with obesity, excess visceral adipose tissue promotes immune cell recruitment and secretion of IL-6 and TNF-α to a much greater extent than in non-obese individuals. This creates a systemic pro-inflammatory milieu [1, 8, 13].

Neuroimmune and Autonomic Disruption: Elevated cytokines exert central effects, disrupting autonomic regulatory pathways. Consequently, sympathetic activity rises and vagal tone declines, leading to the reduced HRV observed in the obese group [7, 9]. Simultaneously, diminished vagal activity impairs the cholinergic anti-inflammatory pathway, reducing acetylcholine-mediated inhibition of cytokine release and establishing a positive feedback loop [10]. In non-obese individuals, higher vagal tone helps maintain cytokine levels in check.

Metabolic Dysfunction and Feedback: Pro-inflammatory cytokines interfere with insulin signalling, promoting insulin resistance and raising blood glucose and HbA1c [13, 14]. In obese young adults, this metabolic disturbance is more pronounced, further stimulating sympathetic outflow and perpetuating the cycle of low HRV, high inflammation, and poor glycemic control. Non-obese individuals, by contrast, exhibit a more favourable autonomic-inflammatory-metabolic balance, highlighting the contrasting profiles between the two groups.

RELEVANCE IN YOUNG ADULTS (18–35 YEARS): WHY COMPARISON MATTERS

Young adulthood is a critical window for the development of cardiometabolic disease. While traditional cardiovascular events are rare in this age group, subclinical pathophysiological processes are often underway—particularly in those with obesity [17].

The importance of directly comparing obese and non-obese young adults lies in several factors:

I. **Subclinical but distinct**: Obese young adults are often asymptomatic yet show measurable differences in HRV, cytokines, and HbA1c compared to their non-obese peers. These differences can be detected before clinical disease manifests [5, 6, 11].

II. **Predictive value**: Reduced HRV in young adulthood predicts future hypertension, metabolic syndrome, and cardiovascular disease [11,16]. The gap between obese and non-obese groups in HRV may represent a risk gradient.

III. **Modifiability**: Interventions such as physical activity have been shown to improve HRV and reduce inflammation in obese young adults [12, 15], suggesting that early differentiation can guide targeted prevention.

Thus, utilising HRV as a non-invasive, low-cost screening tool in young adults could help identify those who are obese and at higher risk, enabling early intervention.

POTENTIAL CONFOUNDERS AND MODIFYING FACTORS

Several factors may influence the observed relationships between HRV, inflammation, and metabolic parameters. Physical activity, sleep quality, psychological stress, dietary patterns, smoking, and alcohol consumption all affect autonomic balance and inflammatory status [12, 15]. Failure to adequately control for these variables may confound comparisons between obese and non-obese groups. Future studies should carefully account for these factors to isolate the independent effects of obesity.

DISCUSSION

This narrative review provides a comprehensive synthesis of current evidence examining the associations between heart rate variability (HRV), pro-inflammatory cytokines (IL-6 and TNF-α), and the metabolic marker HbA1c in young adults, with a specific focus on comparing obese and non-obese individuals. The principal finding is that young adults with obesity exhibit a distinct, interconnected triad of abnormalities: significantly reduced vagally mediated HRV, elevated circulating IL-6 and TNF-α, and higher HbA1c levels when compared to their non-obese peers [1, 4–6, 11]. These differences are detectable in the age range of 18–30 years, often before the onset of overt clinical disease, and they form a self-reinforcing pathophysiological network that contributes to early cardiometabolic risk [16, 17].

Rather than being isolated phenomena, the three components of this triad are mechanistically linked. Reduced vagal tone (low HRV) is both a consequence of obesity-induced inflammation and a contributor to its persistence via impairment of the cholinergic anti-inflammatory pathway [10]. Concurrently, elevated IL-6 and TNF-α promote insulin resistance, raising HbA1c [13, 14], while hyperglycemia and hyperinsulinemia further stimulate sympathetic outflow, perpetuating autonomic imbalance [7]. This bidirectional, feed-forward loop explains why the obese group displays a consistently more adverse profile across all three domains, as summarised in Table 1.

Critical Appraisal: A key strength of the studies reviewed is the consistency of the observed differences

between obese and non-obese young adults across diverse populations and measurement methods. Parish et al. [6] demonstrated that adolescents with obesity had significantly lower low-frequency and high-frequency HRV indices, along with elevated IL-6 and TNF-α levels, compared to normal-weight controls, indicating impairment of both sympathetic and parasympathetic systems (see Table 1). Wiley et al. [5] extended the understanding of the HRV-inflammation link by showing, in adult cohorts, that the association between HRV and the lipid accumulation product (LAP)—a superior measure of adiposity-related risk—was mediated by C-reactive protein (CRP), providing mechanistic support for the cholinergic anti-inflammatory pathway. Koenig et al. [11] confirmed in a larger adult sample that higher BMI was inversely associated with vagally mediated HRV, with this relationship partially mediated by IL-6. The foundational study by Karason et al. [4] established the obesity-HRV link, though it focused on an older adult population.

However, several methodological limitations temper the certainty of these conclusions. First, the majority of studies are cross-sectional [1, 5, 6, 11], precluding causal inference. It remains unclear whether obesity leads to reduced HRV and inflammation, whether autonomic dysfunction predisposes to weight gain and inflammation, or whether both emerge from shared upstream factors (e.g., sedentary behaviour, poor diet, chronic stress). Second, HRV measurement protocols vary widely—some studies use short-term recordings under uncontrolled breathing, while others employ 24-hour Holter monitoring [2, 3]—making direct comparisons of absolute values difficult. Third, many studies do not adequately control for important confounders such as physical activity, sleep quality, and psychological stress, all of which independently influence both HRV and inflammatory markers. When such adjustments are made, the magnitude of association between obesity and HRV often attenuates, suggesting that lifestyle factors play a significant mediating role [12].

Neuroimmune–Metabolic Axis: The cholinergic anti-inflammatory pathway, first described by Tracey [10], provides a compelling mechanistic framework for linking reduced HRV to heightened inflammation. Efferent vagal nerve activity releases acetylcholine, which binds to α7 nicotinic acetylcholine receptors on macrophages and other immune cells, suppressing the synthesis of pro-inflammatory cytokines like TNF-α and IL-6. In obesity, excess visceral adiposity promotes a pro-inflammatory state that may directly impair central autonomic circuits, reducing vagal outflow [7, 8]. This loss of vagal "braking" on inflammation creates a vicious cycle: inflammation further suppresses vagal activity, and the resulting autonomic imbalance amplifies systemic inflammation [9, 10]. Moreover, the interplay with metabolic regulation adds another layer. IL-6 and TNF-α interfere with insulin signalling at the level of the insulin receptor substrate (IRS) and promote serine phosphorylation of IRS proteins, leading to insulin resistance in muscle, liver, and adipose tissue [13, 14]. This results in compensatory hyperinsulinemia and, over time, elevated HbA1c. Insulin itself has sympathoexcitatory effects, particularly in the central nervous system, further increasing sympathetic tone and reducing HRV [7, 17]. Thus, the obese young adult experiences a confluence of autonomic, inflammatory, and metabolic derangements that reinforce one another, as illustrated in Table 1 by the consistent pattern across studies [5, 6, 12, 15].

Literature and Unique Contribution: The finding that obese young adults exhibit reduced HRV and elevated inflammation aligns with a large body of literature in middle-aged and older adults [4, 11, 17]. However, the present review's focus on the 18–30 age range is crucial because it demonstrates that these abnormalities are established decades before the typical onset of cardiovascular events [16]. This challenges the notion that young adulthood is universally a period of low risk and underscores the importance of early detection and intervention. Additionally, the integrated examination of HRV, IL-6/TNF-α, and HbA1c within a single framework is relatively novel. Most previous studies have examined pairwise associations (e.g., HRV and inflammation [11], or inflammation and HbA1c [13, 14]). By synthesising all three, this review highlights their interdependence and positions HRV as a potential "canary in the coal mine"—a non-invasive indicator of a broader dysregulation that precedes clinical disease [3, 15]. The inclusion of both obese and non-obese groups allows for a clearer characterisation of the risk gradient, with the obese group showing a shift toward pathological values that may represent an early stage of the cardiometabolic disease continuum [5, 16].

Clinical and Public Health Implications: From a clinical perspective, the findings suggest that HRV could serve as a valuable adjunctive tool in the assessment of

cardiometabolic risk in young adults [3]. Current screening guidelines for this age group rely heavily on traditional risk factors such as blood pressure, lipid profile, and family history, which may remain normal even in the presence of significant subclinical dysfunction [16]. HRV measurement is non-invasive, relatively inexpensive, and can be performed in primary care settings with portable ECG devices or even validated photoplethysmography sensors [2]. If used strategically, HRV could help identify obese young adults who are already showing signs of autonomic and metabolic dysregulation, enabling targeted lifestyle interventions (e.g., structured exercise, stress management, dietary counselling) before irreversible damage occurs [15]. The interventional studies reviewed [12, 15] provide proof-of-concept that light-intensity physical activity can simultaneously improve HRV and glycemic parameters, supporting the modifiability of this risk profile (see Table 1).

However, several barriers must be overcome before HRV can be integrated into routine clinical practice. Standardisation of measurement protocols, establishment of age- and sex-specific reference values, and training of clinicians in interpretation are essential [2]. Additionally, the prognostic value of HRV for predicting hard outcomes (e.g., incident diabetes, cardiovascular events) in young adults needs to be established through large-scale longitudinal studies [16].

Limitations: Several limitations should be acknowledged. This narrative review lacks quantitative synthesis and may have selection bias. The evidence is dominated by cross-sectional designs, small sample sizes, and heterogeneous heart rate variability methodologies. Focusing on IL-6 and TNF-α simplifies a broader inflammatory network that includes C-reactive protein, interleukin-1β, and adipokines such as leptin and adiponectin. Additionally, the use of HbA1c reflects long-term glycemia but not acute glucose fluctuations that may be more directly linked to autonomic function. Finally, most studies did not account for sex differences or include diverse racial and ethnic groups, limiting generalizability.

FUTURE DIRECTIONS

To advance the field, future research should prioritise:

- Longitudinal cohort studies that track HRV, inflammatory markers, and HbA1c from young adulthood into middle age, with repeated measures, to establish temporal sequences and determine whether early reductions in HRV predict later metabolic deterioration independent of baseline BMI [16].
- Randomised controlled trials that test whether interventions specifically aimed at improving vagal tone (e.g., heart rate variability biofeedback, aerobic exercise, stress reduction techniques) can reduce IL-6/TNF-α and HbA1c in obese young adults, thereby establishing causality [12, 15].
- Mechanistic studies using pharmacological or electrical vagal nerve stimulation in human models to directly test the role of the cholinergic anti-inflammatory pathway in mediating metabolic improvements [10].
- Standardisation efforts to harmonise HRV measurement protocols across studies, including recommendations for recording duration, posture, breathing control, and reporting of both time-domain and frequency-domain indices [2].
- Exploration of additional biomarkers, such as high-sensitivity CRP, leptin, adiponectin, and indices of glycemic variability, to more fully capture the complexity of the neuroimmune–metabolic interface [8, 13].
- Diverse populations that include adequate representation of women, various racial/ethnic groups, and different socioeconomic strata to ensure generalizability [5, 6, 11].

In summary, the evidence strongly supports the existence of a distinct, interconnected triad of reduced HRV, elevated IL-6/TNF-α, and higher HbA1c in obese young adults compared to their non-obese peers. This triad reflects an early, subclinical neuroimmune–metabolic dysregulation that sets the stage for future cardiometabolic disease [16, 17]. The consistency of findings across studies, combined with plausible mechanistic pathways [7, 10, 13], argues for the potential utility of HRV as a non-invasive screening tool in this age group [3]. However, significant gaps remain in understanding causality, generalizability, and clinical implementation [2, 4, 16]. Addressing these gaps through well-designed longitudinal and interventional research will be essential to translate these findings into clinical practice and public health strategies aimed at curbing the

rising tide of obesity-related disease in young adults [15, 17].

OBESE VS. NON-OBESE YOUNG ADULTS

This narrative review provides a clear verdict: young adults with obesity exhibit a consistently more adverse profile of autonomic function, inflammation, and glycemic control than their non-obese counterparts. Specifically:

I. HRV: Vagally mediated indices (RMSSD, HF power) are significantly lower in obese young adults, indicating diminished parasympathetic tone [4–6].

II. Inflammation: Circulating IL-6 and TNF-α are consistently elevated in the obese group, reflecting chronic low-grade inflammation [1, 6, 8].

III. Metabolic control: HbA1c, even when within normal ranges, is higher in obese young adults, signalling early metabolic dysregulation [14–16].

These three domains are not independent but form an interconnected pathophysiological triad that amplifies cardiometabolic risk. The differences between groups are detectable in the 18–30 age range, often before clinical disease emerges, highlighting the importance of early screening and targeted prevention. HRV emerges as a promising, non-invasive biomarker that can help differentiate obese young adults at higher risk and may serve as a modifiable target for lifestyle interventions [12, 15].

CONCLUSION

This review demonstrates that obesity in young adults is associated with a distinct triad of reduced HRV, elevated pro-inflammatory cytokines (IL-6, TNF-α), and increased HbA1c compared to non-obese peers. These findings underscore a complex, interdependent relationship between autonomic dysfunction, inflammation, and metabolism that contributes to early cardiovascular risk. HRV, as a non-invasive index of autonomic function, shows promise as an early indicator of cardiometabolic risk and may help differentiate obese from non-obese young adults in clinical and research settings. However, further longitudinal and interventional studies are required to establish its predictive value and clarify its role as a potential therapeutic target.

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