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THE DAWN PHENOMENON AND GLYCEMIC VARIABILITY MEASURED WITH CONTINUOUS GLUCOSE MONITORING

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ABSTRACT

The dawn phenomenon (DP), defined as an early-morning rise in glucose unrelated to preceding nocturnal hypoglycemia, represents a significant yet often underrecognized contributor to fasting hyperglycemia and overall glycemic instability in individuals with diabetes. The expansion of continuous glucose monitoring (CGM) technologies over the past decade has enabled more precise characterization of nocturnal glucose patterns, offering new insights into the prevalence, magnitude, and clinical implications of DP across diverse glycemic states. This review synthesizes original observational, retrospective, and prospective studies published between 2010 and 2025 that used CGM to evaluate DP in type 1 diabetes, type 2 diabetes, impaired glucose tolerance, and non-diabetic populations. Data extraction focused on DP definitions, nocturnal glucose trajectories, glycemic variability metrics, associations with HbA1c and time-in-range, and emerging evidence linking severe DP to adverse clinical outcomes.

Findings indicate that DP is highly prevalent in both type 1 and type 2 diabetes, with magnitude varying widely depending on residual β-cell function, insulin sensitivity, and methodological differences in CGM-based definitions. DP correlates with increased total glucose exposure and greater glycemic variability, and may influence long-term metabolic risk. Understanding DP within the context of CGM-derived metrics is essential for optimizing individualized therapeutic strategies and improving morning glycemic control. Further standardized research is required to unify definitions and clarify the prognostic significance of DP.

KEYWORDS

Dawn Phenomenon, Continuous Glucose Monitoring (CGM), Glycemic Variability, Type 2 Diabetes, Type 1 Diabetes, Morning Hyperglycemia

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Introduction

The dawn phenomenon is a well-recognized physiological and clinical occurrence in individuals with diabetes, characterized by a spontaneous increase in blood glucose concentrations during the early morning hours, typically between 2:00 a.m. and 8:00 a.m., independent of preceding nocturnal hypoglycemia. First described in the early 1980s, DP has been historically observed in both type 1 diabetes (T1D) and type 2 diabetes (T2D), but its precise prevalence, magnitude, and clinical implications have remained variably reported due to limitations in measurement techniques and heterogeneous definitions across studies. The clinical significance of DP lies in its contribution to fasting hyperglycemia, increased total daily glucose exposure, and overall glycemic variability, which in turn can complicate diabetes management and increase the risk of long-term microvascular and macrovascular complications.

Mechanically, DP is attributed to the interaction of circadian hormonal fluctuations, especially the nocturnal increase in growth hormone, catecholamines, cortisol and glucose production in the liver, combined with reduced nocturnal insulin sensitivity.

In individuals with diabetes, the absence or insufficiency of endogenous insulin secretion exacerbates these physiological changes, resulting in elevated pre-breakfast glucose levels. The magnitude of DP is influenced by multiple factors including diabetes type, duration of disease, residual β-cell function, insulin therapy regimen, and lifestyle factors such as meal timing and physical activity.

The introduction of continuous glucose monitoring has revolutionized the understanding of DP. Unlike traditional self-monitoring of blood glucose (SMBG) or single fasting glucose measurements, CGM provides high-resolution, real-time glucose data across the nocturnal period, allowing precise quantification of the glucose rise associated with DP. CGM also enables the assessment of glycemic variability indices such as

mean amplitude of glycemic excursions (MAGE), coefficient of variation (CV), standard deviation (SD), and time-in-range (TIR), offering a comprehensive picture of the impact of DP on overall glucose homeostasis.

Despite the growing use of CGM, research on DP remains inconsistent due to variations in study design, DP definitions, population characteristics, and monitoring protocols. Some studies define DP as a fixed threshold rise in glucose (e.g., ≥20 mg/dL from nadir), while others use percentage increases or mathematical modeling of CGM data. Additionally, DP has been studied across heterogeneous populations, including individuals with T1D, T2D, impaired glucose tolerance (IGT), and even normoglycemic controls, leading to divergent prevalence estimates ranging from 20% to over 50% depending on the cohort and methodology.

Clinically, the presence of DP can complicate diabetes management strategies. In T1D, early-morning hyperglycemia may require adjustments in basal insulin dosing or timing, while in T2D, it may necessitate tailored oral hypoglycemic therapy or lifestyle interventions. Moreover, DP contributes to post-breakfast glucose excursions, which can exacerbate daily glycemic variability and negatively impact HbA1c despite apparent overall glycemic control. Emerging evidence also suggests that severe DP may correlate with long-term adverse outcomes, including increased all-cause mortality and cardiovascular risk, highlighting its importance beyond a mere laboratory phenomenon.

Given these considerations, there is a critical need for a comprehensive synthesis of the literature examining the dawn phenomenon using CGM. Such a review can clarify prevalence, magnitude, and clinical implications, compare findings across T1D and T2D populations, and provide insights into methodological challenges and opportunities for standardization. This review focuses on original observational, retrospective, and prospective studies published in the past 10–15 years, evaluating DP as measured by CGM, its impact on glycemic variability, associations with HbA1c and other glycemic metrics, and potential clinical consequences.

The objectives of this review are fourfold. First, to summarize the prevalence and magnitude of the dawn phenomenon across different populations and glycemic states. Second, to evaluate the association of DP with glycemic control indices such as HbA1c, TIR, and daily glucose exposure. Third, to assess the impact of DP on glycemic variability as measured by CGM-derived metrics, including MAGE, CV, and SD. Fourth, to discuss clinical implications, limitations of existing research, and potential directions for future investigation, including the need for standardized definitions and CGM-based assessment protocols. By systematically examining the current evidence, this review aims to provide a nuanced understanding of DP and its role in modern diabetes management.

In conclusion, the dawn phenomenon is a clinically relevant contributor to early-morning hyperglycemia and daily glucose fluctuations, particularly in individuals with diabetes. The advent of CGM has enabled more accurate detection and characterization of DP, revealing its substantial impact on glycemic variability and treatment considerations. However, methodological heterogeneity and inconsistent definitions have hindered consensus on its prevalence, clinical significance, and management strategies. A comprehensive synthesis of CGM-based studies is therefore essential to inform clinical practice, guide individualized therapeutic interventions, and highlight areas for future research aimed at mitigating the metabolic consequences of DP.

Methodology

This review focuses on original research studies examining the dawn phenomenon and its impact on glycemic variability using continuous glucose monitoring in individuals with type 1 diabetes, type 2 diabetes, impaired glucose tolerance (IGT), and normoglycemic populations. The review includes studies published over the past 15 years to capture the evolution of CGM technologies and contemporary research methodologies.

The definition of DP varied across studies. Some studies defined DP as an absolute glucose rise from nocturnal nadir to pre-breakfast values (e.g., ≥10–20 mg/dL), while others used relative or percentage-based thresholds or applied statistical modeling to CGM data. This heterogeneity is acknowledged as a methodological limitation, but it also highlights the flexibility of CGM to capture nuanced glucose patterns that may not be evident with traditional SMBG.

CGM allows for comprehensive evaluation of glucose fluctuations using metrics such as:

- **Mean Amplitude of Glycemic Excursions (MAGE):** quantifies the average magnitude of significant glucose swings.
- **Coefficient of Variation (CV):** standard deviation of glucose divided by mean glucose, expressed as a percentage.
- **Continuous Overall Net Glycemic Action (CONGA):** measures intraday glucose variability over defined time intervals.

- **Time-in-Range (TIR), Time Above Range (TAR), Time Below Range (TBR):** proportions of time glucose remains within, above, or below target thresholds.

These metrics were extracted from included studies to evaluate the contribution of DP to overall glycemic variability and to compare differences between populations and treatment strategies.

Given the heterogeneity of study designs, populations, and DP definitions, a qualitative synthesis approach was employed rather than a formal meta-analysis. Key findings were grouped thematically into prevalence and magnitude of DP, CGM-based characterization, association with glycemic control, impact on glycemic variability indices, and long-term clinical consequences. Comparisons were made between T1D and T2D populations where appropriate.

Several limitations inherent to the reviewed studies are noted. First, the diversity of DP definitions limits direct comparability between studies. Second, CGM monitoring durations varied (commonly 3–14 days), which may influence detection rates of DP episodes. Third, differences in CGM devices, calibration protocols, and sampling intervals could affect glucose measurements and derived variability indices. Finally, demographic differences, including age, ethnicity, duration of diabetes, and concomitant therapies, introduce variability in observed outcomes.

Despite these limitations, the selected studies provide robust insights into the prevalence, characteristics, and clinical impact of DP as captured by modern CGM systems.

Results

Prevalence and Magnitude of the Dawn Phenomenon Across Populations

The dawn phenomenon has been widely studied in individuals with type 1 diabetes, type 2 diabetes, and, to a lesser extent, in populations with impaired glucose tolerance and non-diabetic controls. The use of continuous glucose monitoring over the past 10–15 years has provided a high-resolution view of nocturnal glucose dynamics, allowing for precise estimation of both the prevalence and magnitude of DP across heterogeneous populations.

In T2D, prevalence estimates of DP vary considerably depending on study population, definition, and CGM methodology. Li et al. (2020) reported a prevalence of approximately 54% among adults with T2D, using a threshold rise of ≥20 mg/dL from nocturnal nadir to pre-breakfast glucose. Similarly, Monnier et al. (2013) observed that 40–60% of patients with T2D experienced a clinically significant DP, with mean glucose rises ranging from 15 to 40 mg/dL. Notably, the magnitude of DP in T2D has been correlated with residual β-cell function and hepatic glucose production, indicating that patients with lower endogenous insulin secretion tend to experience more pronounced early-morning hyperglycemia. Additionally, Roman et al. (2016) demonstrated that DP contributes disproportionately to overall glycemic variability in T2D, particularly in individuals with suboptimal basal insulin coverage or inadequate oral hypoglycemic therapy.

In T1D populations, DP is also highly prevalent, although the absolute glucose rise is often larger due to the complete absence of endogenous insulin. Yoshida et al. (2013) evaluated Japanese patients with T1D using CGM and reported that 42% exhibited a pre-breakfast glucose increase of ≥20 mg/dL. Similarly, Monnier et al. (2015) emphasized that in T1D, DP not only elevates fasting glucose but also significantly contributes to post-breakfast hyperglycemia, highlighting its dual impact on both fasting and postprandial glycemic control. Factors influencing DP magnitude in T1D include residual C-peptide secretion, overnight basal insulin dosing, and nocturnal physical activity.

In populations with impaired glucose tolerance or normoglycemia, DP has been less frequently studied, but emerging evidence suggests it is not exclusive to overt diabetes. Xia et al. (2025) reported mild yet measurable DP in individuals with prediabetes, with mean pre-breakfast glucose rises of 10–15 mg/dL. Although these rises are lower than those observed in T1D or T2D, they may nonetheless contribute to cumulative daily glucose exposure and long-term metabolic risk if persistent over time. These findings suggest that DP exists along a glycemic continuum, reflecting underlying physiological variability amplified by impaired insulin secretion or insulin resistance.

Magnitude of DP also varies depending on methodological factors. Studies using CGM devices with high sampling frequency (e.g., every 5 minutes) tend to report slightly higher DP prevalence and more precise estimates of glucose rise compared with devices with 15-minute intervals or short-duration monitoring. The duration of CGM wear is another critical factor; monitoring for at least 5–7 consecutive nights is generally recommended to capture representative nocturnal glucose trends, while shorter monitoring periods may underestimate true prevalence. Moreover, differences in DP definitions—absolute rise versus relative percentage rise, or modeling-based thresholds—introduce variability in reported results across studies.

Longitudinal studies highlight that DP may persist over time and can fluctuate in magnitude within individuals. Cai et al. (2024) demonstrated that patients with consistently severe DP experienced higher cumulative glucose exposure over months, suggesting that repeated nocturnal hyperglycemia may contribute to sustained HbA1c elevation and increased risk of microvascular complications. These findings reinforce the clinical relevance of identifying DP in routine care and tailoring therapeutic interventions accordingly.

In summary, DP is a prevalent and clinically significant phenomenon across the spectrum of diabetes. In T2D, prevalence ranges from approximately 40% to 60%, with average glucose rises of 15–40 mg/dL, while in T1D, similar prevalence is reported but with larger absolute glucose increases due to the absence of endogenous insulin. Emerging evidence indicates that DP may also occur in prediabetic populations, though with lower magnitude. Methodological variations, including CGM device type, monitoring duration, and DP definition, influence reported prevalence and magnitude. Collectively, these findings underscore the need for standardized measurement protocols to accurately characterize DP and inform individualized diabetes management strategies.

CGM-Based Characterization of the Dawn Phenomenon

Continuous glucose monitoring has fundamentally transformed the study of the dawn phenomenon by providing high-resolution, longitudinal glucose data that capture nocturnal fluctuations and early-morning hyperglycemia. Unlike traditional self-monitoring of blood glucose, which relies on discrete pre-breakfast measurements, CGM enables continuous assessment of interstitial glucose levels throughout the night, offering precise characterization of both the magnitude and temporal dynamics of DP.

One of the key contributions of CGM to DP research is the ability to identify nocturnal nadir glucose levels, which serve as a reference point for quantifying the pre-breakfast rise. Studies have consistently demonstrated that the DP typically manifests as a gradual increase in glucose beginning between 2:00 a.m. and 4:00 a.m., peaking near the time of awakening (Monnier et al., 2013; Li et al., 2020). The timing and slope of this rise can vary substantially between individuals and across diabetes types. In T1D, the glucose rise tends to be sharper and more pronounced, reflecting the absence of endogenous insulin secretion, whereas in T2D, the slope is often moderate, influenced by residual β-cell function and hepatic glucose output.

CGM also enables the assessment of interday variability in DP. Several studies (Roman et al., 2016; Wang et al., 2021) have reported that the magnitude and timing of DP episodes can fluctuate within the same individual across consecutive nights. Such variability is influenced by factors including meal timing, physical activity, stress, and insulin regimen. High-frequency CGM sampling, often at 5-minute intervals, allows researchers to capture subtle glucose excursions that may be missed by standard SMBG, providing a more accurate picture of nocturnal glucose dynamics.

Methodological heterogeneity exists in the definition and quantification of DP using CGM. Common approaches include:

1. **Absolute rise method:** defining DP as a fixed glucose increase from nocturnal nadir to pre-breakfast measurement (e.g., ≥20 mg/dL).
2. **Relative or percentage rise method:** defining DP based on a proportional increase from nadir (e.g., ≥10% rise).
3. **Modeling-based approaches:** applying statistical or mathematical models to detect systematic glucose increases during early-morning hours.

Each approach has advantages and limitations. Absolute thresholds are simple and clinically interpretable but may fail to capture individualized variations in baseline glucose. Relative rises account for inter-individual differences but can be affected by low nocturnal glucose values. Modeling-based approaches offer precision but are computationally intensive and less practical for routine clinical use.

CGM-derived metrics further enhance the characterization of DP. Parameters such as time-to-peak, rate of glucose rise, and area under the glucose curve (AUC) during the pre-breakfast period provide quantitative measures of DP severity. Studies have also examined the relationship between DP and overall glycemic variability, using indices like mean amplitude of glycemic excursions (MAGE), coefficient of variation (CV), and continuous overall net glycemic action (CONGA). These metrics demonstrate that DP contributes not only to fasting hyperglycemia but also to broader daily glucose fluctuations, amplifying metabolic stress and complicating glycemic management.

Additionally, CGM facilitates evaluation of DP under real-life conditions. Free-living CGM studies capture the effects of lifestyle behaviors, such as late-night meals, exercise, and variable sleep patterns, on

nocturnal glucose trajectories. This validity is crucial, as laboratory-based studies may underestimate DP prevalence or magnitude due to controlled conditions.

In summary, CGM has provided a nuanced understanding of the dawn phenomenon by enabling precise, continuous measurement of nocturnal glucose patterns. It allows identification of glucose nadirs, temporal dynamics of early-morning rises, interday variability, and contributions to overall glycemic variability. While methodological heterogeneity in DP definitions persists, CGM remains the gold standard for assessing DP in clinical and research settings, offering actionable insights for optimizing individualized diabetes management.

Association Between the Dawn Phenomenon and Glycemic Control (HbA1c, TIR, AUC)

The dawn phenomenon exerts a significant influence on glycemic control, as reflected in traditional markers such as glycated hemoglobin (HbA1c), time-in-range, and overall daily glucose exposure measured by area under the curve. Continuous glucose monitoring studies over the past decade have elucidated the magnitude of this effect, demonstrating that DP contributes not only to elevated fasting glucose but also to broader disruptions in overall glycemic stability.

HbA1c and the Dawn Phenomenon

HbA1c is a widely used measure of long-term glycemic control, reflecting mean glucose levels over approximately three months. Studies employing CGM have consistently shown that individuals experiencing pronounced DP tend to exhibit higher HbA1c levels, even when pre- and postprandial glucose readings appear otherwise controlled. Monnier et al. (2013) reported that T2D patients with DP had mean HbA1c levels approximately 0.5–0.8% higher than those without DP. This relationship is partly explained by the contribution of DP to fasting hyperglycemia, which constitutes a substantial proportion of daily glucose exposure. Roman et al. (2016) similarly demonstrated that DP magnitude correlates with HbA1c in both T1D and T2D, emphasizing the clinical importance of early-morning glucose rises in overall glycemic management.

Interestingly, the association between DP and HbA1c is influenced by diabetes type and residual β-cell function. In T1D, where endogenous insulin is absent, DP can account for a larger proportion of HbA1c variation, whereas in T2D, the contribution is more modest and often intertwined with postprandial hyperglycemia. These findings highlight that addressing DP may be particularly impactful in reducing HbA1c in insulin-dependent populations.

Time-in-Range (TIR)

Time-in-range, defined as the proportion of glucose readings within the target range (typically 70–180 mg/dL), has emerged as a clinically relevant metric complementing HbA1c. CGM studies have demonstrated that DP negatively impacts TIR by increasing the duration of hyperglycemia during early morning hours. Li et al. (2020) observed that T2D patients with pronounced DP spent significantly less time in range overnight and in the early morning compared to those without DP, despite similar daytime glucose profiles. In T1D, the effect of DP on TIR is even more pronounced, as early-morning glucose excursions often precede breakfast, leading to elevated postprandial glucose and reduced overall TIR (Yoshida et al., 2013).

Reductions in TIR due to DP have practical implications, as lower TIR is associated with increased risk of microvascular complications, cardiovascular events, and hypoglycemia in subsequent periods due to corrective insulin doses. Therefore, quantifying DP using CGM provides actionable insights for clinicians aiming to optimize basal insulin regimens or other therapeutic interventions to improve TIR.

Area Under the Curve (AUC) and Daily Glucose Exposure

The contribution of DP to total daily glucose exposure can be quantified using AUC, calculated from CGM-derived glucose profiles. Studies consistently report that DP accounts for a substantial fraction of daily AUC, particularly in T1D populations. Monnier et al. (2015) estimated that early-morning hyperglycemia associated with DP contributed up to 20–25% of total daily glucose exposure in some individuals. This persistent elevation can have metabolic consequences beyond fasting glucose, including increased oxidative stress, endothelial dysfunction, and higher glycemic variability.

Cai et al. (2024) further emphasized that repeated DP episodes over weeks and months lead to cumulative AUC increases, potentially driving sustained HbA1c elevations. Importantly, these studies underscore that traditional SMBG measurements, which may capture only a single fasting glucose value, often underestimate the total metabolic impact of DP. CGM enables precise calculation of nocturnal and early-morning glucose contributions to daily AUC, providing a more comprehensive understanding of glycemic burden.

Implications for Clinical Management

The association between DP and glycemic control metrics suggests that addressing early-morning hyperglycemia is crucial for optimizing both short- and long-term outcomes. Interventions targeting DP include adjusting basal insulin dosing, modifying bedtime snacks, implementing pre-breakfast rapid-acting insulin boluses, or employing newer therapeutic agents such as GLP-1 receptor agonists or SGLT2 inhibitors in T2D. By reducing the magnitude of DP, clinicians can improve HbA1c, increase TIR, and decrease daily glucose AUC, leading to more stable overall glycemic profiles.

In summary, CGM studies provide compelling evidence that the dawn phenomenon substantially impacts key measures of glycemic control. DP is associated with higher HbA1c, reduced TIR, and increased early-morning contribution to daily glucose exposure as measured by AUC. These effects are more pronounced in insulin-dependent populations but are also detectable in T2D and prediabetic individuals. Recognizing and quantifying DP using CGM is therefore essential for designing individualized therapeutic strategies and achieving comprehensive glycemic management.

Impact of the Dawn Phenomenon on Glycemic Variability Indices (MAGE, CONGA, CV)

Glycemic variability (GV) represents fluctuations in blood glucose levels over time, which have been increasingly recognized as an independent contributor to oxidative stress, endothelial dysfunction, and diabetes-related complications. The dawn phenomenon is a key driver of increased GV. Continuous glucose monitoring allows precise quantification of these fluctuations using indices such as mean amplitude of glycemic excursions, coefficient of variation, and continuous overall net glycemic action.

Mean Amplitude of Glycemic Excursions (MAGE)

MAGE quantifies the average magnitude of significant glucose swings, typically focusing on excursions exceeding one standard deviation of mean glucose. Studies have shown that DP contributes notably to MAGE by generating a consistent early-morning rise in glucose levels. Monnier et al. (2013) demonstrated that in T2D patients with DP, MAGE values were significantly higher compared to individuals without DP, suggesting that early-morning hyperglycemia amplifies overall glycemic fluctuations. Similarly, in T1D, Yoshida et al. (2013) reported that pre-breakfast glucose surges contributed disproportionately to total MAGE, highlighting DP as a critical determinant of intraday glucose variability.

Coefficient of Variation (CV)

CV, calculated as the standard deviation of glucose divided by mean glucose, provides a normalized measure of variability that accounts for differences in average glucose levels. High CV is associated with increased risk of hypoglycemia and adverse vascular outcomes. CGM studies indicate that DP elevates CV, particularly in insulin-treated patients. Li et al. (2020) reported that T2D patients with prominent DP exhibited higher CV values overnight and during the early-morning period, emphasizing the role of DP in destabilizing glycemic patterns even when mean glucose appears acceptable. In T1D populations, CV is further increased by the absence of endogenous insulin, leading to more pronounced excursions.

Continuous Overall Net Glycemic Action (CONGA)

CONGA assesses intraday glucose variability over defined time intervals (e.g., 1-hour or 2-hour windows), capturing rapid fluctuations that may not be apparent through MAGE or CV alone. CGM-based studies demonstrate that DP increases CONGA values during early-morning hours, reflecting consistent, predictable glucose excursions that contribute to overall variability (Roman et al., 2016; Wang et al., 2021). Notably, CONGA allows differentiation between rapid, transient spikes and sustained glucose increases, which is clinically relevant for understanding DP's contribution to metabolic stress.

Comparative Impact Across Diabetes Types

The impact of DP on GV indices varies between T1D and T2D. In T1D, where endogenous insulin is absent, early-morning glucose excursions often exceed 40–50 mg/dL, producing marked increases in MAGE, CV, and CONGA. In T2D, DP magnitude is generally lower (15–40 mg/dL), yet still significantly elevates these indices compared to non-DP individuals. Emerging evidence also suggests that in prediabetic populations, mild DP can cause subtle increases in GV indices, which may have long-term metabolic consequences if persistent.

Clinical Relevance of Glycemic Variability Induced by DP

Increased GV associated with DP has clinical implications beyond fasting hyperglycemia. High MAGE and CV are linked to oxidative stress, endothelial dysfunction, and accelerated progression of microvascular complications. Early-morning excursions also complicate treatment decisions, as corrective insulin doses intended to counteract DP may increase the risk of hypoglycemia later in the morning or day. By quantifying DP using CGM-derived GV indices, clinicians can tailor basal insulin regimens, consider pre-breakfast rapid-acting insulin, or adjust lifestyle interventions to mitigate early-morning glucose surges.

Overall, the dawn phenomenon is a significant contributor to increased glycemic variability, as measured by MAGE, CV, and CONGA. CGM studies consistently demonstrate that DP-induced early-morning hyperglycemia amplifies daily glucose fluctuations in both T1D and T2D, with potential clinical consequences for vascular risk, treatment safety, and metabolic stability. Accurate assessment of DP using CGM-derived GV indices provides actionable information for individualized diabetes management, emphasizing the importance of addressing early-morning glucose rises as part of comprehensive glycemic control strategies.

Long-Term Clinical Consequences of the Dawn Phenomenon

The dawn phenomenon, while often perceived as a transient early-morning glucose rise, may have meaningful long-term clinical implications for individuals with diabetes. Recurrent early-morning hyperglycemia contributes not only to fasting glucose elevation but also to overall glycemic burden, increased glycemic variability, and potentially accelerated progression of diabetes-related complications.

Microvascular Complications

Elevated fasting glucose and increased glycemic variability associated with DP have been linked to microvascular complications, including retinopathy, nephropathy, and neuropathy. Studies suggest that early-morning hyperglycemia contributes to endothelial dysfunction, oxidative stress, and inflammation, mechanisms central to microvascular damage (Monnier et al., 2015; Cai et al., 2024). Although causal relationships remain under investigation, CGM studies indicate that individuals with pronounced DP experience higher cumulative glucose exposure, which may exacerbate microvascular risk over time.

Macrovascular Risk

Emerging evidence also implicates DP in increased macrovascular risk. High glycemic variability and fasting hyperglycemia contribute to arterial stiffness, atherosclerosis, and impaired cardiovascular function. Roman et al. (2016) and Wang et al. (2021) noted that patients with severe DP exhibited markers of early cardiovascular risk, suggesting that persistent early-morning glucose surges could accelerate macrovascular disease progression in both T1D and T2D populations.

Impact on Overall Glycemic Control

DP complicates glycemic management by elevating HbA1c, reducing time-in-range, and increasing daily glucose exposure. Persistent DP episodes can lead to more aggressive insulin dosing, which may increase the risk of hypoglycemia later in the day, creating a cycle of glucose instability. These dynamics highlight the need for individualized treatment strategies to address DP and mitigate its long-term consequences.

In conclusion, while the dawn phenomenon is primarily recognized as a physiological glucose rise during early morning hours, its recurrent presence has important clinical ramifications. By contributing to increased glycemic variability, elevated fasting glucose, and higher cumulative daily glucose exposure, DP may exacerbate both microvascular and macrovascular complications. Recognition and targeted management of DP are therefore essential components of comprehensive diabetes care, particularly in patients with T1D and T2D who experience significant early-morning glucose excursions.

Discussion

The dawn phenomenon is a clinically significant contributor to early-morning hyperglycemia and overall glycemic instability in individuals with diabetes. This review, synthesizing evidence from original observational, retrospective, and prospective studies utilizing continuous glucose monitoring, highlights the prevalence, magnitude, and clinical implications of DP across diverse populations, including type 1 diabetes, type 2 diabetes, impaired glucose tolerance, and normoglycemic individuals. The findings emphasize the importance of understanding DP within the context of modern diabetes management, particularly given its impact on glycemic variability, time-in-range, HbA1c, and cumulative glucose exposure.

Prevalence and Magnitude of DP

The prevalence of DP varies depending on diabetes type, population characteristics, and methodological approaches. In T2D, DP is observed in approximately 40–60% of individuals, with mean glucose rises ranging from 15 to 40 mg/dL, while in T1D, prevalence is similar but absolute glucose increases are often higher due to absent endogenous insulin. Emerging evidence indicates that mild DP occurs even in prediabetic populations, suggesting a continuum of physiological changes influenced by insulin secretion and hepatic glucose output. These findings underscore that DP is not merely a laboratory phenomenon but a pervasive contributor to fasting hyperglycemia and daily glucose excursions.

CGM as a Transformative Tool

CGM has revolutionized the characterization of DP. Unlike traditional self-monitoring of blood glucose, CGM provides continuous, high-resolution glucose data that capture nocturnal nadirs, timing of glucose rises, and interday variability. CGM allows precise quantification of DP magnitude, duration, and contribution to glycemic variability indices such as MAGE, CV, and CONGA. Moreover, CGM enables real-world assessment of DP, capturing the impact of lifestyle behaviors, meal timing, and insulin regimens on early-morning glucose patterns.

Despite these advances, methodological heterogeneity in CGM studies persists. Differences in CGM devices, sampling intervals, monitoring duration, and DP definitions complicate cross-study comparisons. Some studies define DP based on absolute glucose rises, while others use relative or modeling-based approaches. Standardization of DP assessment protocols is necessary to enhance comparability and facilitate evidence-based clinical decision-making.

Association With Glycemic Control

DP significantly influences glycemic control metrics. Studies consistently demonstrate that individuals with pronounced DP exhibit higher HbA1c, lower TIR, and increased early-morning contribution to daily glucose exposure as measured by area under the curve. In T1D, DP can account for up to 20–25% of total daily glucose exposure, reflecting the substantial metabolic burden imposed by early-morning hyperglycemia. In T2D, the contribution is somewhat lower but remains clinically relevant. These associations highlight that addressing DP is essential for achieving optimal long-term glycemic control.

The impact of DP on TIR is particularly important, as reduced time-in-range is associated with increased risk of microvascular complications and hypoglycemia. By quantifying DP using CGM, clinicians can tailor insulin regimens and other therapeutic strategies to mitigate early-morning glucose rises, thereby improving TIR and reducing overall glycemic burden.

Impact on Glycemic Variability

DP contributes significantly to glycemic variability, a recognized independent risk factor for oxidative stress, endothelial dysfunction, and diabetes-related complications. CGM-derived indices such as MAGE, CV, and CONGA demonstrate that early-morning glucose surges amplify interday fluctuations. In T1D, these effects are pronounced due to the absence of endogenous insulin, whereas in T2D, residual β-cell function moderates the magnitude of excursions. Increased variability associated with DP complicates treatment, as corrective insulin dosing may inadvertently induce hypoglycemia later in the day, creating a cycle of glucose instability. Addressing DP is therefore critical not only for reducing fasting hyperglycemia but also for minimizing deleterious glycemic fluctuations.

Long-Term Clinical Consequences

Recurrent DP has meaningful long-term clinical implications. Persistent early-morning hyperglycemia contributes to microvascular complications, including retinopathy, nephropathy, and neuropathy, through mechanisms involving oxidative stress, inflammation, and endothelial dysfunction. Emerging evidence also suggests that DP may exacerbate macrovascular risk by promoting arterial stiffness and atherosclerosis. Although causal relationships require further investigation, CGM studies consistently indicate that severe DP is associated with cumulative glycemic burden, reinforcing its relevance in comprehensive diabetes management.

Clinical Management Implications

Recognition and management of DP are essential for optimizing diabetes care. Therapeutic strategies include adjusting basal insulin dosing or timing, implementing pre-breakfast rapid-acting insulin boluses, and modifying lifestyle factors such as late-night meals and physical activity. In T2D, pharmacologic agents including GLP-1 receptor agonists, SGLT2 inhibitors, and basal insulin analogs may mitigate early-morning hyperglycemia. Individualized treatment plans guided by CGM data can improve fasting glucose, reduce glycemic variability, increase TIR, and ultimately enhance patient outcomes.

Additionally, CGM facilitates ongoing monitoring and adjustment of therapy. By providing real-time feedback on nocturnal glucose trends, CGM enables proactive interventions to prevent excessive early-morning rises. Patient education regarding the dawn phenomenon and its management is equally important, as behavioral modifications, such as evening meal composition and timing, can complement pharmacologic strategies.

Limitations and Future Directions

Despite the robust evidence base, several limitations exist. First, methodological heterogeneity, including variations in DP definitions and CGM protocols, complicates comparisons across studies. Second, most studies are limited by short monitoring durations (commonly 3–14 days), which may not fully capture interday variability. Third, demographic factors such as age, sex, ethnicity, diabetes duration, and comorbidities may influence DP magnitude and were inconsistently reported in many studies. Finally, few longitudinal studies have directly examined the causal impact of DP on long-term complications, representing an important gap in knowledge.

Future research should prioritize standardization of DP definitions, longer-duration CGM monitoring, and inclusion of diverse populations. Randomized controlled trials assessing interventions targeting DP and their impact on glycemic control, variability, and clinical outcomes are needed. Additionally, integration of CGM with artificial intelligence and predictive algorithms may enable anticipatory management of DP, further optimizing individualized therapy.

In conclusion, the dawn phenomenon is a prevalent and clinically significant contributor to fasting hyperglycemia and glycemic variability. CGM-based studies have provided detailed characterization of DP, demonstrating its impact on HbA1c, TIR, daily glucose exposure, and glycemic variability indices. Persistent DP has potential long-term consequences, including microvascular and macrovascular complications. Recognition and individualized management of DP, guided by CGM, are essential for comprehensive diabetes care. Standardized research methodologies and longitudinal studies are required to refine treatment strategies, improve patient outcomes, and clarify the long-term clinical significance of this phenomenon.

Conclusions

The dawn phenomenon represents a distinct physiological event characterized by early-morning rises in blood glucose, which occurs in individuals across the glycemic spectrum, including those with type 1 diabetes, type 2 diabetes, impaired glucose tolerance, and even in normoglycemic populations. Continuous glucose monitoring has revolutionized our understanding of DP, enabling precise, real-time characterization of nocturnal glucose patterns, magnitude of early-morning surges, interday variability, and contributions to overall glycemic burden.

This review demonstrates that DP is highly prevalent, affecting 40–60% of individuals with T2D and a similar proportion in T1D, with absolute glucose rises often greater in insulin-dependent populations. Mild DP has also been observed in prediabetic individuals, suggesting that early-morning glucose excursions exist along a continuum influenced by β-cell function, insulin resistance, hepatic glucose output, and lifestyle factors. The consistent presence of DP has important implications for overall glycemic control, as it contributes to elevated

fasting glucose, reduced time-in-range, higher HbA1c, and increased daily glucose exposure measured by area under the curve.

DP also plays a central role in glycemic variability, as reflected in indices such as mean amplitude of glycemic excursions, coefficient of variation, and continuous overall net glycemic action. Elevated variability resulting from DP has been linked to oxidative stress, endothelial dysfunction, and increased risk of diabetes-related complications. These findings underscore that early-morning hyperglycemia is not merely a transient metabolic event but a clinically relevant contributor to daily and cumulative glucose fluctuations.

Clinically, addressing DP is essential for optimizing individualized diabetes management. Strategies include basal insulin adjustments, pre-breakfast rapid-acting insulin, lifestyle modifications, and, in T2D, the use of pharmacologic agents such as GLP-1 receptor agonists or SGLT2 inhibitors. CGM facilitates real-time monitoring of DP, guiding therapeutic decisions and enabling targeted interventions to improve fasting glucose, reduce glycemic variability, and increase TIR. Patient education regarding DP and its management is also crucial to achieve sustainable improvements in glycemic control.

Despite advances in understanding DP, several knowledge gaps remain. Methodological heterogeneity in definitions and CGM protocols limits cross-study comparability, and few studies have examined the direct long-term clinical consequences of DP. Future research should focus on standardizing DP assessment, evaluating interventions in diverse populations, and exploring predictive tools to anticipate and mitigate early-morning hyperglycemia.

In summary, the dawn phenomenon is a prevalent, clinically significant phenomenon with measurable impacts on glycemic control, variability, and potentially long-term complications. Recognition and individualized management, guided by CGM, are essential for comprehensive diabetes care. Addressing DP represents an important opportunity to optimize metabolic outcomes and improve the quality of life for individuals living with diabetes.

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