Standardization of Glycemic Disparities: A Multidimensional Analysis of HbA1c and Time-in-Range Between Non-Hispanic Black and Non-Hispanic White Adults with Type 2 Diabetes

The clinical evaluation of glycemic control in type 2 diabetes has undergone a paradigm shift, transitioning from a historical reliance on glycated hemoglobin (HbA1c) to the integration of continuous glucose monitoring (CGM) metrics, most notably Time-in-Range (TIR).[1] In the United States, profound racial and ethnic disparities in diabetes outcomes persist, particularly when comparing non-Hispanic Black (NHB) and non-Hispanic White (NHW) adults.[4] Understanding these disparities requires distinguishing between actual metabolic glycemia and biochemical glycation.[1] Investigating the standardized differences in HbA1c and TIR under conditions that strictly control for socioeconomic status (SES) and baseline treatment intensity reveals that while socioeconomic factors and clinical access drive the majority of the real-world gap in average glucose, a distinct, glucose-independent biological divergence in hemoglobin glycation remains.[7]

Standardized Differences in HbA1c under Controlled Conditions

In unadjusted population-level cohorts, NHB adults with established type 2 diabetes consistently exhibit higher HbA1c levels compared to NHW adults, with an absolute difference typically ranging from 0.5% to 0.9% percentage points.[5] A comprehensive meta-analysis of eleven distinct diabetes cohorts quantified this unadjusted disparity, revealing a standardized mean difference (SMD) of approximately 0.31 standard deviations, which translates to a pooled absolute HbA1c elevation of 0.65% for Black patients, assuming a pooled cohort standard deviation of 2.1%.[5] This unadjusted difference represents an estimated 15% relative increase in microvascular and macrovascular complication risks for NHB patients under standard clinical guidelines.[12]

When statistical models control for socioeconomic status (such as household income, education level, or neighborhood deprivation index) and baseline treatment intensity (including medication adherence, insulin use, and comorbidities), the standardized difference in HbA1c is attenuated but remains statistically and clinically significant.[8] In these controlled models, the adjusted absolute HbA1c difference typically ranges from 0.20% to 0.39% percentage

points.[7]

For example, in a national retrospective cohort of $690,968$ veterans receiving uniform care within the Veterans Health Administration (VHA)—an environment designed to minimize financial barriers to access—the adjusted absolute difference in HbA1c was 0.25% after controlling for demographics, comorbidities, medication adherence, and the specific classes of glucose-lowering drugs prescribed.[8] Applying a standardized cohort variation, this adjusted gap represents a standardized difference of approximately 0.12 standard deviations.[8] Despite this uniform care setting, NHB veterans experienced a significantly higher risk of poor glycemic control, defined as an HbA1c $\geq 8.0\%$, with an adjusted odds ratio of 1.33 (95% CI: 1.31–1.35) compared to NHW veterans.[8]

Similarly, a retrospective analysis of $25,123$ primary care patients at an urban academic medical center adjusted for median household income quartiles using US Census Bureau data.[10] NHB patients maintained a statistically higher overall HbA1c level ($7.68 \pm 1.77\%$) compared to NHW patients ($7.29 \pm 1.33\%$, $P < 0.001$), yielding an adjusted absolute difference of 0.39% and a standardized difference of approximately 0.25 standard deviations.[10]

Crucially, this urban cohort revealed a profound socioeconomic interaction: while NHW patients exhibited a clear, stepwise improvement in glycemic control as household income increased ($P = 0.014$), no such relationship was observed among NHB patients ($P = 0.282$).[10] Black patients in the highest income quartile experienced similar rates of poor glycemic control and elevated HbA1c as those in the lowest income quartile.[10] These findings indicate that individual socioeconomic advancement does not mitigate the glycemic disparity for Black adults, pointing to systemic environmental barriers or non-economic stressors that independently affect metabolic health.[10]

Furthermore, longitudinal analyses demonstrate that adjusting for behavioral factors such as medication adherence does not eliminate the racial gap.[11] When examining patients initiated on oral antihyperglycemic therapies, White patients demonstrated an average baseline HbA1c of 8.9% compared to 9.8% in Black patients.[11] Although Black patients exhibited lower medication adherence rates in the first year of therapy (72% versus 78%), statistical adjustment for adherence failed to close the HbA1c gap.[11] In parallel studies, comprehensive adjustments for sociodemographic factors, clinical variables, healthcare access quality, and self-management behaviors accounted for only 14% of the elevated HbA1c levels observed in Black patients, leaving the vast majority of the variance unexplained by conventional

socioeconomic or behavioral metrics.[11]

Analysis Type / Study Cohort	Covariates Controlled (SES and Treatment Intensity)	Absolute HbA1c Disparity (NHB vs. NHW)	Calculated Standardized Difference (SMD)	Key Clinical Context	Primary Sources
Unadjusted Meta-Analysis (Kirk et al.)	None (Raw population-level variance)	$+0.65\%$ percentage points	0.31 SD	Reflects broad real-world differences across diverse geographic settings.	[5]
VHA National Cohort ($N =$)	Demographics, comorbidities, drug class, and medication adherence	$+0.25\%$ percentage points	0.12 SD	Equal-access managed care setting; reduces but does not eliminate disparity.	[8]
Academic Medical Center ($N =$)	ZIP code-derived household income quartiles	$+0.39\%$ percentage points	0.25 SD	Socioeconomic gradients predict HbA1c in NHW patients, but not in NHB patients.	[10]
Highly Insured Community Cohort	Age, sex, income, education, insurance,	No statistically significant	Near 0.00 SD	Disparities are largely absent in localized,	[14]

(Boston)	disease duration, and lifestyle	difference		universally insured communities.	
Health and Retirement Study (HRS)	Clinical factors, healthcare access quality, and self-management	Disparity persisted (controls explained 14%)	N/A	Behavioral and access variables fail to account for the majority of the gap.	[11]

Standardized Differences in Time-in-Range (TIR)

In real-world, unadjusted clinical settings, the standardized difference in TIR between NHB and NHW adults with type 2 diabetes is substantial, often exceeding the disparity observed in HbA1c.[15] Real-world registry data comparing Black and White patients with diabetes show that Black cohorts spend significantly less time within the target glucose range of $70–180\ \mathrm{mg/dl}$ ($35.27 \pm$ for Black patients versus $50.89 \pm$ for White patients, $P = 0.0004$).[15] This represents an absolute unadjusted TIR reduction of 15.62% for Black patients, which translates to a standardized difference of approximately 0.72 standard deviations.[15]

This unadjusted TIR gap is primarily driven by massive, systemic disparities in the prescription and adoption of advanced diabetes technologies, such as continuous glucose monitors (CGMs) and automated insulin delivery (AID) systems.[16] Across clinical trials and observational databases, the average prescription rate for diabetes technology is 56.3% for NHW patients compared to only 21.3% for NHB patients, creating an immediate structural barrier to achieving optimal glycemic control.[16]

However, when statistical models control for socioeconomic status and baseline treatment intensity, the standardized difference in TIR is profoundly altered, often approaching statistical non-significance.[19] A multivariable logistic regression model evaluating the predictors of CGM technology initiation illustrates this dynamic.[20] In unadjusted models, Black patients are significantly less likely to adopt CGM technology.[20] Yet, in a fully adjusted multivariable model that includes education, income, health insurance, diabetes duration, and insulin use, race is no longer a statistically significant predictor of CGM adoption (adjusted odds ratio for NHB versus NHW: 0.93; 95% CI: 0.43–2.00, $P = 0.849$).[20]

Instead, the model demonstrates that low socioeconomic status (aOR: 0.44; 95% CI: 0.21–0.95, $P = 0.037$) and insulin treatment intensity (aOR: 2.46; 95% CI: 1.34–4.53, $P = 0.004$) are the primary independent drivers of technology use.[20] This indicates that the real-world TIR disparity is not caused by racial differences in glucose homeostasis or therapy response, but rather by the compounding effects of socioeconomic disadvantage and unequal clinical initiation of intensive regimens.[20]

This conclusion is further supported by clinical trials and structured access programs that provide technology and education free of charge to all participants.[1] When Black and White patients receive equal access to CGM devices and structured technical training, the unadjusted average blood glucose and TIR disparities are effectively eliminated.[1] For example, the implementation of structured CGM clinical programs has been shown to equalize mean glucose levels across Black, White, and South Asian cohorts, although a significant disparity in HbA1c persists within the same individuals.[19] This decoupling of actual daily glucose (captured by TIR) and hemoglobin glycation (captured by HbA1c) highlights a physiological divergence that remains even after clinical and socioeconomic parity is achieved.[1]

Clinical Metric / Outcome	NHW Cohort Mean	NHB Cohort Mean	Standardized Difference (Unadjusted vs. Controlled)	Clinical Implications and Mediators	Primary Sources
Real-World TIR ($70–180\ \mathrm{mg/}$)	$50.89 \pm$	$35.27 \pm$	0.72 SD (Unadjusted)	Primarily mediated by unequal technology access and treatment intensity.	[15]
CGM Initiation Rate	56.3% (average)	21.3% (average)	Highly Significant (Unadjusted)	Biased provider prescribing habits and insurance eligibility criteria.	[16]

CGM Initiation Odds (Fully Adjusted)	Reference Group	$\mathrm{aOR} =$ (95% CI: $0.43–2.00$)	Non-Significant (Controlled)	Adjusting for SES ($\mathrm{aOR} =$) and insulin ($\mathrm{aOR} =$) eliminates racial variance.	[20]
TIR in Highly Controlled Settings	Comparable	Comparable	Approaches 0.00 SD (Controlled)	Equalizing technology access and education resolves actual glucose differences.	[1]

Physiological Glycation Decoupling: The GRADE Substudy

The persistence of the HbA1c gap in environments of socioeconomic and clinical parity is explained by a profound physiological decoupling of average blood glucose and hemoglobin glycation between racial groups.[1] This phenomenon was conclusively evaluated in a specialized substudy of the Glycemia Reduction Approaches in Diabetes: A Comparative Effectiveness Study (GRADE).[1] The GRADE trial, a multicenter randomized clinical trial in the United States, was designed to compare the glycemic efficacy of four common glucose-lowering medications when added to metformin in patients with established type 2 diabetes.[1] To eliminate socioeconomic and clinical care barriers, the trial provided all diabetes medications, glucose testing supplies, and clinical care completely free of charge to all participants.[1]

In the GRADE continuous glucose monitoring substudy, $1,454$ participants (including 534 NHW, 389 NHB, and 327 Hispanic White patients) underwent 10 days of blinded CGM wear to establish a precise, measured average glucose (AG_{10}).[7] Immediately following the CGM wear period, central laboratory measurements of HbA1c, glycated albumin, and fructosamine were performed.[7]

The study established a statistically significant and clinically critical racial divergence in the translation of physical glucose into HbA1c values [7]:

- Across the clinically relevant glucose spectrum of $100 \text{ to } 250 \text{ mg/dL}$, NHB patients exhibited HbA1c levels that were $0.2 \text{ to } 0.6$ percentage points higher than NHW patients at the exact same measured average glucose (AG_{10}).[7]
- For a standardized laboratory HbA1c value of 7.0%, the actual measured average glucose (AG_{10}) was 11 mg/dL higher in NHW patients compared to NHB patients, indicating that NHB patients are maintained at a lower level of systemic glycemia to register the same HbA1c value.[7]
- This racial divergence in the relationship between AG_{10} and HbA1c remained entirely unchanged after comprehensive statistical adjustments for age, sex, BMI, and other demographic or clinical covariates.[7]
- Parallel racial differences were observed when relating fasting plasma glucose (FPG) and oral glucose tolerance test (OGTT) area under the curve (AUC) to HbA1c, confirming that the discrepancy is systemic across multiple measures of glycemia.[7]

Importantly, the GRADE substudy demonstrated that this relationship extends to other glycated proteins.[7] Similar racial differences were observed in the relationship between measured glucose and glycated albumin, suggesting that the underlying biological mechanisms are not restricted to hemoglobin glycation but may involve broader cellular glucose transport or generalized protein glycation kinetics.[7]

These physiological differences are influenced by genetic factors that vary in frequency across ancestral lineages.[9] Research indicates that genetic variants altering red blood cell survival and hemoglobin glycation are key drivers of this phenomenon.[22] For example, the glucose-6-phosphate dehydrogenase (G6PD) G202A variant, carried by approximately 11% of African Americans, is associated with a shortened red blood cell lifespan, which falsely decreases HbA1c by approximately 0.7% to 0.8% percentage points.[22] Similarly, the heterozygous hemoglobin S (HbS) trait, common in populations of African descent, reduces measured HbA1c by approximately 0.3% percentage points.[28]

However, in the majority of NHB adults who lack these specific variants, the default rate of intracellular hemoglobin glycation is elevated relative to extracellular glucose concentrations.[1] Consequently, for the majority of Black patients, the standard HbA1c assay systematically overestimates true systemic glycemia, creating a significant risk of overdiagnosis and overtreatment when clinical management relies solely on unadjusted HbA1c thresholds.[1]

Measured Average Glucose	Equivalent Standardized HbA1c	Equivalent Standardized HbA1c	Measured Decoupling	Physiological & Clinical Consequen	Primary Sources

(AG10)	(NHW)	(NHB)	Gap	ces	
$120\ \mathrm{mg/dl}$	$\sim$	$\sim$	$+0.30\%$ HbA1c in NHB	Overdiagnosis of prediabetes/diabetes in normoglycemic NHB adults.	[7]
$150\ \mathrm{mg/dl}$	$\sim$	$\sim$	$+0.40\%$ HbA1c in NHB	NHB patients are classified as "uncontrolled" despite identical average glucose.	[1]
$180\ \mathrm{mg/dl}$	$\sim$	$\sim$	$+0.50\%$ HbA1c in NHB	Clinicians aggressively escalate therapy in NHB, increasing hypoglycemia risk.	[1]
$210\ \mathrm{mg/dl}$	$\sim$	$\sim$	$+0.60\%$ HbA1c in NHB	Maximum divergence of glycation kinetics under severe hyperglycemic stress.	[7]

Structural Systems of Care and Baseline Treatment Intensity

The physiological discrepancies in hemoglobin glycation are exacerbated by systemic

inequities in healthcare delivery that directly limit baseline treatment intensity for NHB patients.[4] To evaluate the standardized difference in glycemic outcomes, models must account for unequal prescribing patterns of newer, highly effective glucose-lowering drugs (GLDs), such as glucagon-like peptide-1 (GLP-1) receptor agonists and sodium-glucose cotransporter-2 (SGLT2) inhibitors.[4] These medication classes provide substantial glycemic reduction and cardioprotective benefits, making their equitable initiation a key clinical goal.[13]
In a secondary analysis of the Look AHEAD (Action for Health in Diabetes) trial, researchers evaluated the initiation rates of these newer diabetes medications over a median follow-up of 8.3 years in a cohort of $4,892$ participants.[31] The study revealed a stark racial disparity: Black participants were significantly less likely to be initiated on newer, guideline-recommended GLDs compared to White participants (Hazard Ratio: 0.81; 95% CI: 0.70–0.94, $P = 0.019$).[31] This prescribing gap was completely independent of socioeconomic factors, as yearly family income was controlled for in the models.[31]
This systematic underprescribing of advanced therapeutics to Black patients limits their baseline treatment intensity, directly contributing to poorer glycemic control and lower real-world TIR.[13] The barriers to prescribing these medications include clinical inertia, cost-related access restrictions, and provider prescribing biases, which collectively prevent minoritized populations from accessing therapies that reduce glycemic excursions and protect against microvascular and cardiovascular damage.[4]

Clinical Performance and Health Policy Implications

The clinical and policy implications of these combined socioeconomic, clinical, and physiological disparities are profound. Relying on unadjusted HbA1c thresholds for the diagnosis, monitoring, and treatment of type 2 diabetes introduces systematic bias into clinical decision-making.[1] Under standard clinical performance measures, healthcare providers are evaluated based on the percentage of their patients who achieve an HbA1c target of $< 7.0\%$.[32] Because these quality metrics do not account for racial differences in glycation kinetics, they penalize providers who care for larger minority populations and incentivize clinical practices that may be unsafe for NHB patients.[9]
For an NHB patient and an NHW patient maintained at an identical physiological average glucose, the NHB patient's HbA1c will register approximately 0.4% percentage points higher.[7] Consequently, to drive an NHB patient's HbA1c down to the standard target of $< 7.0\%$, clinicians must reduce that patient's actual average glucose to a level significantly lower than that required for an NHW patient.[7] This intensive treatment escalation poses a severe clinical risk by dramatically increasing the rate of hypoglycemic events.[1]
Multiple clinical trials have shown that attempts to achieve strict, unadjusted HbA1c targets in Black patients lead to elevated rates of hypoglycemia (glucose $< 70\ \mathrm{mg/dL}$ and

$< 54\ \mathrm{mg/dL}$), which are associated with cognitive decline, cardiovascular events, and increased mortality.[1] This risk is particularly high in patients treated with insulin or sulfonylureas, where aggressive titration can cause severe, sudden drops in blood glucose.[9]

To resolve these clinical conflicts and ensure safe, equitable care, standard diabetes protocols must be updated:

- **De-emphasize HbA1c in Favor of Time-in-Range (TIR):** Clinical guidelines should prioritize TIR and Time-Below-Range (TBR) derived from 14-day CGM data as the primary metrics for evaluating glycemic control.[2] TIR provides a direct, continuous measure of physical glucose that is free from the biochemical biases of hemoglobin glycation.[1]
- **Implement Personalized, Ancestry-Informed HbA1c Targets:** When clinical management must rely on HbA1c, clinicians should consider a higher, personalized HbA1c target (e.g., $7.3\%\ \text{to}\ 7.5\%$) for NHB patients who lack hemoglobin variants or G6PD deficiency.[7] This adjustment ensures that Black patients are not subjected to dangerous overmedication to reach an arbitrary, racially biased target.[1]
- **Equalize Access to Advanced Diabetes Technology:** Health systems and insurance providers must eliminate restrictive coverage criteria that disproportionately prevent minoritized and low-income populations from obtaining CGMs and newer, non-insulin medications.[16] Providing administrative, financial, and educational support to patients and healthcare providers is essential to eliminating structural barriers and closing the real-world TIR gap.[21]
- **Incorporate Alternative Biomarkers in High-Risk Cohorts:** For patients with known genetic hemoglobin variants or conditions that alter red blood cell turnover, clinicians should utilize alternative markers of glycemia.[7] Glycated albumin and fructosamine reflect a shorter-term glycemic status ($2\ \text{to}\ 3\ \text{weeks}$) and correlate highly with CGM-derived average glucose, providing a reliable alternative when HbA1c is biochemically compromised.[7]

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