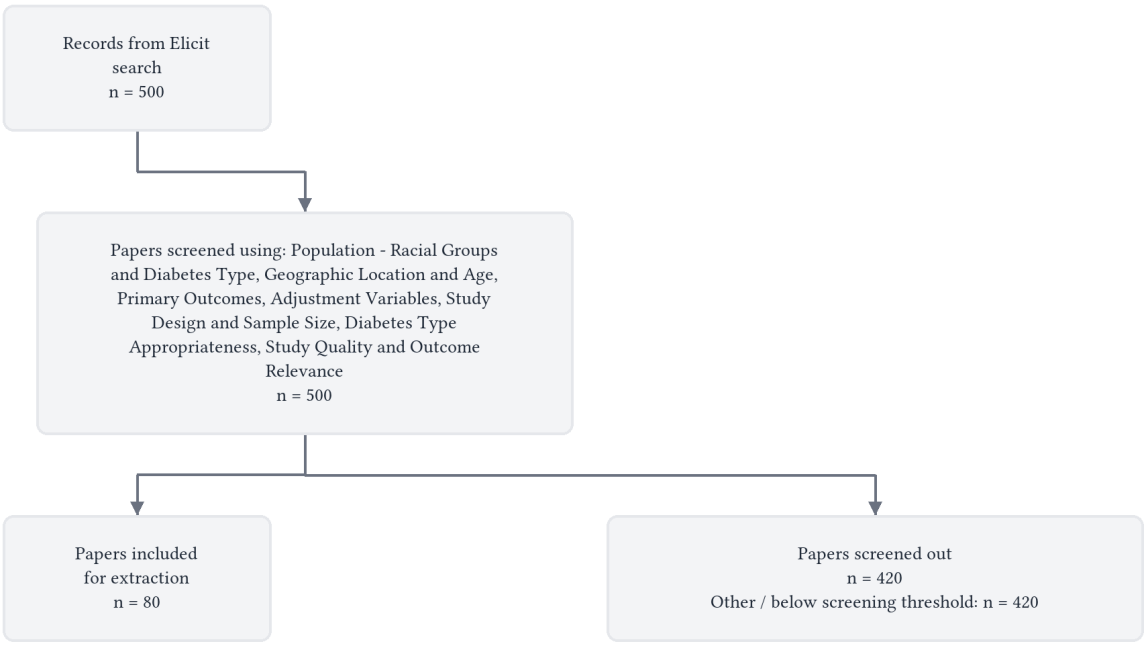
In non-Hispanic Black adults compared to non-Hispanic White adults with established type 2 diabetes in the US, what is the standardized difference in HbA1c and time-in-range (TIR) when controlling for socioeconomic status and baseline treatment intensity?

After controlling for socioeconomic status and treatment intensity, non-Hispanic Black adults with type 2 diabetes in the US have residual HbA1c levels approximately 0.2–0.4 percentage points higher than non-Hispanic White adults, though a substantial portion of this gap (approximately 0.3 percentage points) may reflect biological differences in hemoglobin glycation rather than true differences in glycemic exposure; no studies have reported time-in-range comparisons between these groups, and no standardized effect sizes (Cohen's d) have been calculated for the adjusted difference.

Abstract

Across 80 US-based studies comparing glycemic control between non-Hispanic Black (NHB) and non-Hispanic White (NHW) adults with type 2 diabetes, NHB adults consistently have higher HbA1c levels, with unadjusted differences typically ranging from 0.3% to 1.0% and a meta-analytic pooled estimate of approximately 0.65% [1]. After adjustment for socioeconomic status and treatment intensity, the disparity is attenuated but generally persists, with residual adjusted differences of approximately 0.2–0.4% in absolute HbA1c terms or odds ratios of 1.1–1.4 for uncontrolled diabetes. The largest and most comprehensively adjusted studies — including a national VA cohort of 1.14 million veterans adjusting for demographics, comorbidities, medication type, adherence, and access metrics (OR 1.11, 95% credible interval 1.09–1.14) [2], and a sequential NHANES analysis adjusting for demographic, socioeconomic, and health indicators (OR 1.38, 97.5% CI 1.04–1.83) [3] — find statistically significant residual disparities. A small number of studies conducted in structured care settings with uniform treatment protocols found that disparities can be eliminated over time [4], and one highly insured community sample found no significant disparity after comprehensive adjustment including health literacy and treatment regimen [5]. However, medication adherence, when formally tested as a mediator, did not explain the racial gap [6, 7]. Importantly, CGM-based studies indicate that HbA1c may be 0.2–0.6 percentage points higher in NHB than NHW individuals at the same average glucose level [8, 9], suggesting that a meaningful fraction of the residual HbA1c disparity reflects biological differences in hemoglobin glycation rather than true differences in glycemic exposure. No study reported time-in-range comparisons between NHB and NHW adults, and no study reported standardized effect sizes (Cohen's d) for the adjusted racial HbA1c difference, leaving the research question regarding standardized TIR differences unanswerable with current evidence.

Flow Diagram



Paper search

We performed a semantic search across over 138 million academic papers from the Elicit search engine, which includes all of Semantic Scholar and OpenAlex.

We ran this query: "In non-Hispanic Black adults compared to non-Hispanic White adults with established type 2 diabetes in the US, what is the standardized difference in HbA1c and time-in-range (TIR) when controlling for socioeconomic status and baseline treatment intensity?"

The search returned 500 total results from Elicit.

We retrieved 500 papers most relevant to the query for screening.

Screening

We screened in sources based on their abstracts that met these criteria:

- **Population - Racial Groups and Diabetes Type**: Does the study include both non-Hispanic Black and non-Hispanic White adults with established type 2 diabetes?
- **Geographic Location and Age**: Was the study conducted in the United States with participants aged 18 years or older?
- **Primary Outcomes**: Does the study report HbA1c and/or time-in-range (TIR) outcomes?

- **Adjustment Variables**: Does the study control for or report both socioeconomic status variables (income, education, insurance) and treatment intensity variables (medications, insulin use)?
- **Study Design and Sample Size**: Is this a comparative study design (observational study, RCT, systematic review, or meta-analysis) that compares outcomes between racial groups with at least 10 participants per racial group?
- **Diabetes Type Appropriateness**: Does the study focus on type 2 diabetes only (excluding type 1 diabetes, gestational diabetes, pre-diabetes, or mixed diabetes types without separate type 2 analysis)?
- **Study Quality and Outcome Relevance**: Is this study design appropriate for systematic review inclusion (excluding case reports, editorials, commentaries, conference abstracts) and does it focus on chronic glycemic control rather than solely on acute complications?

We considered all screening questions together and made a holistic judgement about whether to screen in each paper.

At abstract screening, the number of papers excluded for each primary reason was:

- **Other / below screening threshold**: n = 420

Data extraction

We asked a large language model to extract each data column below from each paper. We gave the model the extraction instructions shown below for each column.

- **Study Population**:

  Extract population characteristics to confirm study eligibility for the research question, including:

- Whether study included non-Hispanic Black adults with type 2 diabetes
- Whether study included non-Hispanic White adults with type 2 diabetes
- Sample size for each racial group
- Geographic setting (must be US)
- Age range and mean age
- Whether diabetes was 'established' (not newly diagnosed)
- Any exclusion criteria that might affect generalizability

- **Glycemic Outcomes**:

  Extract all glycemic control outcomes comparing non-Hispanic Black and non-Hispanic White adults with type 2 diabetes, including:

- HbA1c values (mean, median, or distribution) for each racial group
- Time-in-range (TIR) values for each racial group (if reported)
- How HbA1c was measured (laboratory method, point-of-care, etc.)
- How TIR was measured (CGM duration, target range definition)
- Whether outcomes represent single timepoint or averaged over time
- Any other glycemic control measures (fasting glucose, time-in-target, etc.)

- **Confounder Control**:

  Extract all variables that were controlled for or adjusted in analyses comparing glycemic outcomes between racial groups, specifically noting:

- Socioeconomic status measures (income, education, employment, insurance, neighborhood factors)
- Treatment intensity measures (medication type, insulin use, number of medications, dose intensification)

- Other demographic controls (age, sex, BMI)
- Clinical controls (diabetes duration, comorbidities, baseline HbA1c)
- Healthcare access factors (provider type, visit frequency, care setting)
- Whether adjustments were made in multivariable models vs stratified analyses

- **Racial Comparison Results**:

  Extract the statistical results comparing glycemic outcomes between non-Hispanic Black and non-Hispanic White adults, including:

- Raw mean difference in HbA1c between racial groups
- Adjusted mean difference in HbA1c after controlling for confounders
- Standardized effect size or Cohen's d (if reported)
- 95% confidence intervals for all effect estimates
- P-values for racial comparisons
- Raw and adjusted differences in TIR (if reported)
- Effect sizes in original units and standardized units
- Whether differences were clinically meaningful (>0.5% HbA1c difference)

- **Statistical Methodology**:

  Extract details about the statistical approach used to compare racial groups, including:

- Study design (cross-sectional, longitudinal cohort, clinical trial baseline data)
- Statistical methods used (t-tests, linear regression, mixed models, etc.)
- How missing data was handled
- Whether analyses accounted for complex survey design (if applicable)
- Follow-up duration (for longitudinal studies)
- Whether multiple comparisons were adjusted for
- Sample size calculations or power analysis (if reported)
- Any sensitivity analyses performed

Results

Characteristics of Included Studies

A total of 80 sources were identified and reviewed. These included primary observational studies (cross-sectional and longitudinal cohort designs), secondary analyses of national surveys, retrospective chart reviews, randomized trial substudies, systematic reviews, and meta-analyses. The vast majority were conducted in US settings, spanning community-based samples, health maintenance organizations (HMOs), Veterans Affairs (VA) systems, federally qualified health centers (FQHCs), Medicaid populations, and integrated health systems. Study populations ranged from small convenience samples (N < 100) to national cohorts exceeding 1 million veterans. Nearly all studies used HbA1c as the primary glycemic outcome; no study reported time-in-range (TIR) as a comparative metric between non-Hispanic Black (NHB) and non-Hispanic White (NHW) adults. The table below summarizes key characteristics of all included sources.

Study	Full text retrieved?	Study Type	Setting / Data Source	Approximate N	Key Glycemic Outcome	SES Controlled?	Treatment Intensity Controlled?
M. Harris et al., 1999 [10]	No	Cross-sectional (NHANES III)	US national sample [10]	1,480 [10]	HbA1c distribution by race [10]	No [10]	No [10]
Lyndsay A. Nelson et al., 2019 [11]	Yes	Cross-sectional	Primary care clinics, US [11]	444 [11]	Mean HbA1c by race [11]	Yes (income, education, insurance, housing, financial strain) [11]	Partially (number of medications, insulin use) [11]
L. Egede et al., 2010 [12]	No	Retrospective cohort	VA facility, Southeastern US [12]	8,813 [12]	Longitudinal mean HbA1c [12]	No [12]	No [12]
Alexandra Perez et al., 2016 [13]	No	Cross-sectional (NHANES 2003-2012)	US national sample [13]	Not specified [13]	HbA1c distribution by race [13]	No [13]	No [13]
B. Egan et al., 2012 [14]	No	Retrospective (EHR)	110 clinics, Southeast US [14]	22,285 [14]	HbA1c <7% by race [14]	No [14]	Partially (initial HbA1c, therapeutic inertia) [14]
Cheryl P. Lynch et al., 2014 [15]	No	Cross-sectional	Primary care, Southeastern US [15]	661 [15]	Mean HbA1c by race [15]	No [15]	No [15]
J. Andersen et al., 2020 [16]	Yes	Longitudinal cohort	Remote monitoring program, Nebraska [16]	1,279 [16]	Mean HbA1c at baseline and completion [16]	No [16]	No [16]
Kristen A Berg et al., 2024 [3]	No	Retrospective cohort (NHANES 2007-2018)	US national sample [3]	3,649 [3]	Uncontrolled T2DM (HbA1c ≥8%) [3]	Partially (demographic, socioeconomic indicators) [3]	No [3]

Study	Full text retrieved?	Study Type	Setting / Data Source	Approximate N	Key Glycemic Outcome	SES Controlled?	Treatment Intensity Controlled?
N. de Rekeneire et al., 2003 [17]	No	Cross-sectional	Community cohort, US [17]	468 [17]	Mean HbA1c by race [17]	Yes (education, income) [17]	Partially (insulin therapy) [17]
O. Akinboboye et al., 2022 [18]	Yes	Cross-sectional	Southeastern US [18]	601 [18]	Mean HbA1c by race [18]	Yes (education, income) [18]	No [18]
L. Keoki Williams et al., 2014 [19]	No	Retrospective cohort (EHR)	Health system, SE Michigan [19]	19,672 [19]	HbA1c reduction on metformin [19]	No [19]	Partially (metformin exposure) [19]
Simin Hua et al., 2024 [20]	No	Longitudinal cohort	VA national, US [20]	837,023 [20]	Early glycemic control (<7%) [20]	No [20]	No [20]
S. Goonesekera et al., 2015 [5]	Yes	Cross-sectional	Boston community sample [5]	682 [5]	Mean HbA1c by race [5]	Yes (income, education, insurance, health literacy) [5]	Yes (treatment regimens) [5]
Caroline A. Presley et al., 2023 [21]	Yes	Retrospective cohort	Alabama Medicaid [21]	43,997 person-years [21]	HbA1c <7% and <8% [21]	Partially (Medicaid eligibility) [21]	Partially (insulin use) [21]
Cheryl P. Lynch et al., 2010 [22]	No	Retrospective cohort	VA facility, US [22]	8,812 [22]	Baseline HbA1c by race [22]	Partially (employment) [22]	No [22]
David M. Nathan et al., 2024 [8]	No	Prospective substudy of RCT	36 centers, US [8]	1,454 [8]	HbA1c-glucose relationship by race [8]	No [8]	No [8]
Veronica Brady et al., 2024 [23]	No	Cross-sectional (All of Us)	US national [23]	10,016 [23]	Prescribing patterns by race [23]	Partially (sociodemographic, clinical) [23]	No [23]

Study	Full text retrieved?	Study Type	Setting / Data Source	Approximate N	Key Glycemic Outcome	SES Controlled?	Treatment Intensity Controlled?
S. Saydah et al., 2007 [24]	No	Cross-sectional (NHANES 1999-2002)	US national sample [24]	843 [24]	HbA1c <7% by race [24]	Partially (SES, obesity) [24]	Partially (diabetes treatment) [24]
Woolton Lee et al., 2020 [25]	Yes	Systematic review and meta-analysis	US studies [25]	Multiple studies [25]	OR for HbA1c control [25]	Yes (income, education, insurance) [25]	Partially (treatment type) [25]
J. Schectman et al., 2002 [26]	No	Cross-sectional	Indigent population, rural Virginia [26]	810 [26]	Adjusted mean HbA1c difference [26]	Yes (income) [26]	Yes (drug therapy intensity) [26]
Xi Tan et al., 2020 [27]	No	Retrospective cohort (linked survey-EHR)	US national [27]	4,552 [27]	HbA1c control by race [27]	Yes (education, income, insurance) [27]	No [27]
A. Adams et al., 2005 [28]	No	Longitudinal cohort	Single HMO, US [28]	2,966 [28]	Adjusted mean HbA1c difference [28]	No [28]	Yes (medication type) [28]
Ariel T. Holland et al., 2012 [29]	Yes	Cross-sectional (EHR)	Insured, Northern California [29]	15,826 [29]	Mean HbA1c by race [29]	No [29]	Partially (medication type, insulin) [29]
V. Helgeson et al., 2021 [30]	No	Longitudinal cohort	Community sample, US [30]	193 [30]	HbA1c change over 6 months [30]	No [30]	No [30]
O. K. Duru et al., 2009 [31]	No	Case-control	8 managed care plans, US [31]	764 [31]	Poor control of multiple outcomes [31]	No [31]	No [31]

Study	Full text retrieved?	Study Type	Setting / Data Source	Approximate N	Key Glycemic Outcome	SES Controlled?	Treatment Intensity Controlled?
M. Rhee et al., 2008 [4]	Yes	Observational cohort	Municipal diabetes clinic, Atlanta [4]	3,542 [4]	Mean HbA1c at presentation and follow-up [4]	No [4]	Yes (uniform treatment algorithm) [4]
R. Weinstock et al., 2011 [32]	Yes	RCT (IDEATel)	Underserved elderly, US [32]	1,665 [32]	Baseline and longitudinal HbA1c [32]	Partially (education, dual eligibility) [32]	Yes (insulin, OHA, glucose uploads) [32]
L. Hausmann et al., 2010 [33]	No	Cross-sectional (trial baseline)	US (likely) [33]	282 [33]	Mean HbA1c by race [33]	No [33]	No [33]
Ahmed Elhussein et al., 2020 [34]	No	Secondary analysis of RCT (Look AHEAD)	US national [34]	4,892 [34]	Time to newer medication initiation [34]	Yes (income, education, insurance) [34]	Yes (medication number/type) [34]
K. Hunt et al., 2013 [35]	No	Longitudinal cohort	VA national, US [35]	892,223 [35]	HbA1c-mortality association by race [35]	No [35]	Partially (medication adherence) [35]
K. J. Behan et al., 2014 [36]	Yes	Longitudinal cohort	Pensacola, Florida [36]	51 [36]	HbA1c-glucose relationship by race [36]	No [36]	No [36]
R. Walker et al., 2018 [37]	No	Cross-sectional	VA, GA/AL/SC [37]	64,022 [37]	Uncontrolled HbA1c (≥8%) by race [37]	No [37]	No [37]
O. Olukotun et al., 2020 [38]	No	Cross-sectional	Southeastern US [38]	615 [38]	Glycemic control factors by race [38]	No [38]	No [38]
D. Suh et al., 2010 [39]	No	Cross-sectional (NHANES)	US national [39]	Not specified [39]	HbA1c <7% by race [39]	No [39]	No [39]

Study	Full text retrieved?	Study Type	Setting / Data Source	Approximate N	Key Glycemic Outcome	SES Controlled?	Treatment Intensity Controlled?
C. Senteio & A. Akincigil, 2020 [40]	No	Cross-sectional (NAMCS)	US national [40]	4,106 [40]	Poor control (HbA1c >7%) [40]	Partially (insurance) [40]	No [40]
G. Fernandes et al., 2019 [41]	No	Retrospective cohort (EMR)	US national [41]	991 [41]	Clinical inertia by race [41]	No [41]	No [41]
T. Fan et al., 2006 [42]	No	Cross-sectional (NHANES)	US national [42]	Not specified [42]	HbA1c trends by ethnicity [42]	No [42]	No [42]
S. Cromer et al., 2021 [43]	No	Cross-sectional (NHANES 2005-2016)	US national [43]	723 [43]	Insulin type use by race [43]	Partially (insurance) [43]	No [43]
M. Farooq et al., 2022 [44]	No	Cross-sectional	Bronx, New York City [44]	1,218 HF patients [44]	HbA1c in HF-DM cohort [44]	Partially (median household income) [44]	No [44]
Gabriela Pacheco Sanchez et al., 2025 [45]	Yes	Cross-sectional (HANDLS + AllofUs)	Baltimore + US national [45]	40 (HANDLS); 17,339 (AllofUs) [45]	Lipid/inflammatory biomarkers by race [45]	Partially (poverty status in HANDLS) [45]	No [45]
Renée M Betancourt et al., 2013 [46]	No	Cross-sectional	U Penn Healthcare System [46]	332 [46]	Glucose control by race [46]	Partially (income) [46]	No [46]
Gabriela Pacheco Sánchez et al., 2024 [47]	Yes	Cross-sectional (HANDLS + AllofUs)	Baltimore + US national [47]	40 (HANDLS); 17,339 (AllofUs) [47]	Lipid/inflammatory features by race [47]	Partially (poverty, age, BMI) [47]	No [47]
Hadley W. Reid et al., 2020 [48]	No	Cross-sectional	34 primary care practices, US [48]	221 [48]	Mean HbA1c by race [48]	No [48]	No [48]

Study	Full text retrieved?	Study Type	Setting / Data Source	Approximate N	Key Glycemic Outcome	SES Controlled?	Treatment Intensity Controlled?
H. Fu et al., 2022 [49]	No	Retrospective cohort (EHR)	US (implied) [49]	531,995 [49]	10-year glycemic control success rate [49]	No [49]	No [49]
Brittany L. Smalls et al., 2020 [50]	Yes	Cross-sectional (NHANES 2003-2014)	US national [50]	Not specified [50]	Mean HbA1c in older adults [50]	Yes (education, income, insurance) [50]	Partially (insulin use) [50]
J. Campbell et al., 2012 [51]	Yes	Systematic review	US studies [51]	Multiple studies [51]	HbA1c differences by race [51]	Partially [51]	No [51]
Kelly J Hunt et al., 2020 [2]	Yes	Retrospective cohort	VA national, US [2]	1,140,634 [2]	Uncontrolled HbA1c (≥8%) [2]	No [2]	Yes (medication use, adherence) [2]
M. Harris et al., 1999a [52]	No	Cross-sectional (NHANES III)	US national [52]	Not specified [52]	HbA1c distribution by race [52]	No [52]	No [52]
C. Parrinello et al., 2015 [53]	Yes	Cross-sectional (ARIC Visit 5)	US multicenter [53]	1,574 with diabetes [53]	HbA1c goal attainment by race [53]	Yes (income, education, insurance) [53]	Yes (medication use) [53]
E. S. San Diego et al., 2024 [54]	No	Cross-sectional (EHR)	FQHC, San Diego [54]	35,092 [54]	Mean HbA1c by race [54]	Partially (education) [54]	No [54]
Vivienne J. Zhu et al., 2011 [55]	No	Longitudinal cohort (HIE)	Central Indiana [55]	3,976 [55]	Mean HbA1c by race [55]	Partially (insurance type) [55]	Partially (polypharmacy) [55]
A. Cunningham et al., 2020 [56]	No	Retrospective (EHR)	Large urban health system [56]	1,764 [56]	Any/persistently elevated HbA1c [56]	No [56]	No [56]
N. Mohite et al., 2016 [57]	No	Cross-sectional (NHANES 1999-2016)	US national [57]	7,521 [57]	HbA1c <8% by race [57]	Partially (sociodemographic, clinical) [57]	No [57]

Study	Full text retrieved?	Study Type	Setting / Data Source	Approximate N	Key Glycemic Outcome	SES Controlled?	Treatment Intensity Controlled?
J. Lafata et al., 2016 [6]	Yes	Longitudinal cohort	9 integrated health systems, US [6]	37,873 (HbA1c cohort) [6]	HbA1c <8% at follow-up [6]	Partially (income) [6]	Yes (adherence, intensification) [6]
L. Sears et al., 2018 [58]	No	RCT	US (implied) [58]	269 [58]	HbA1c and insulin adherence [58]	No [58]	No [58]
Frances Golden et al., 2025 [59]	No	Cohort (All of Us)	US national [59]	88,416 [59]	Composite risk factor control [59]	Yes (income, education, insurance) [59]	No [59]
C. Parrinello et al., 2015a [60]	No	Cross-sectional (ARIC)	US multicenter [60]	1,561 [60]	Risk factor control by race [60]	No [60]	No [60]
F. Sotomayor et al., 2023 [61]	No	Pilot RCT	Not specified [61]	18 [61]	HbA1c change (not by race) [61]	No [61]	No [61]
A. Adams et al., 2007 [7]	No	Longitudinal cohort	HMO, Boston [7]	2,250 [7]	Adjusted mean HbA1c over time [7]	No [7]	Partially (medication adherence) [7]
Jashalynn C. German et al., 2026 [62]	No	Retrospective cohort (EHR)	US (implied) [62]	10,345 [62]	Therapeutic inertia by race [62]	No [62]	No [62]
J. Kirk et al., 2006 [1]	Yes	Meta-analysis	US studies [1]	11 studies [1]	Pooled HbA1c difference [1]	Partially (insurance) [1]	No [1]
Nora I Zakaria et al., 2023 [63]	Yes	Cross-sectional (NHANES)	US national [63]	4,070 [63]	Poor glycemic control (HbA1c ≥7%) [63]	Yes (education, food security) [63]	No [63]

Study	Full text retrieved?	Study Type	Setting / Data Source	Approximate N	Key Glycemic Outcome	SES Controlled?	Treatment Intensity Controlled?
D. Heidemann et al., 2016 [64]	Yes	Retrospective chart review	Large urban medical center [64]	25,123 [64]	Average HbA1c by race and income [64]	Yes (income via zip code) [64]	No [64]
S. Assari et al., 2017 [65]	Yes	Cross-sectional	Midwestern urban, US [65]	112 [65]	Mean HbA1c by race and gender [65]	Yes (SES index, governmental insurance) [65]	No [65]
S. MacLeod et al., 2025 [66]	No	Cross-sectional	Western New York [66]	22,912 [66]	HbA1c <8% by race and SVI [66]	Yes (Social Vulnerability Index) [66]	No [66]
J. McWilliams et al., 2009 [67]	No	Serial cross-sectional (NHANES)	US national [67]	12,079 (age 40-85) [67]	HbA1c trends by race [67]	Yes (education, income, insurance) [67]	No [67]
M. Heisler et al., 2007 [68]	No	Cross-sectional (HRS)	US national [68]	1,233 [68]	Mean HbA1c by race [68]	Partially (SES, clinical, self-management) [68]	Partially (insulin use) [68]
S. Kamat et al., 2019 [69]	Yes	Cross-sectional (NHANES 1999-2016)	US national [69]	7,521 [69]	HbA1c <8% by race [69]	Partially (education, insurance) [69]	No [69]
F. Pasquel et al., 2025 [70]	Yes	Nonrandomized clinical trial	US multicenter [70]	305 [70]	HbA1c change with AID (not by race) [70]	No [70]	No [70]
Piaopiao Li et al., 2021 [71]	No	Trend analysis (MEPS + NHANES)	US national [71]	Not specified [71]	GLM use and HbA1c trends by race [71]	No [71]	No [71]
Jennifer G. Twombly et al., 2010 [72]	Yes	Retrospective cohort	VA, Southeastern US [72]	4,080 [72]	HbA1c at diagnosis and drug initiation [72]	No [72]	No [72]

Study	Full text retrieved?	Study Type	Setting / Data Source	Approximate N	Key Glycemic Outcome	SES Controlled?	Treatment Intensity Controlled?
Katura C Bullock et al., 2013 [73]	No	Retrospective review	US (implied) [73]	277 [73]	Insulin initiation by race [73]	No [73]	Partially (insulin therapy) [73]
Zeb I. Saeed et al., 2023 [74]	No	Retrospective cohort	Endocrinology practice [74]	Not specified [74]	HbA1c by race over pandemic [74]	Partially (socioeconomic deprivation) [74]	No [74]
S. Linde & L. Egede, 2021 [75]	Yes	Cross-sectional (Medicare)	US national (county-level) [75]	~3,124 counties [75]	A1c testing disparity [75]	Yes (household income, unemployment) [75]	No [75]
Eyiuche N Okeke et al., 2013 [76]	No	Retrospective observational	Subspecialty clinic, US [76]	3,945 [76]	HbA1c ≤7% at 1 year [76]	No [76]	No [76]
M. Marino et al., 2020 [77]	Yes	Retrospective cohort	CHCs, 10 Medicaid expansion states [77]	13,342 [77]	HbA1c pre/post ACA by race [77]	Partially (urban/rural, insurance) [77]	No [77]
A. Pérez et al., 2012 [78]	No	Cross-sectional (NHANES 2009-2010)	US national [78]	Not specified [78]	Standards of care by race [78]	Partially (education, income) [78]	Partially (insulin use, monitoring) [78]
A. Karter et al., 2023 [9]	No	Retrospective (CGM + EHR)	Kaiser Permanente N. California [9]	1,788 [9]	HbA1c-mean glucose relationship [9]	No [9]	No [9]
Y. Taylor et al., 2017 [79]	No	Longitudinal cohort (EHR)	Carolinas HealthCare System [79]	12,572 [79]	Healthcare utilization by race [79]	No [79]	No [79]
N. Pourat et al., 2022 [80]	Yes	Cross-sectional (survey)	FQHCs, US national [80]	836 [80]	Diabetes monitoring and outcomes [80]	Yes (insurance, poverty, comorbidities) [80]	Partially (insulin use) [80]

The included studies span nearly three decades of US data (NHANES III in 1988-1994 through 2022), with study

sizes ranging from 18 participants [61] to over 1.1 million veterans [2]. The majority employed cross-sectional or retrospective cohort designs [3, 10–12, 14]. A smaller number drew on randomized trial data [32, 34, 58, 70], and three were systematic reviews or meta-analyses [1, 25, 51]. Notably, only a minority of studies simultaneously controlled for comprehensive SES indicators and treatment intensity, which is central to the stated research question.

Effects

Overview of HbA1c Disparities

The table below summarizes the magnitude and direction of the Black-White HbA1c difference across studies that reported quantitative estimates. Where studies reported odds ratios for uncontrolled diabetes rather than mean HbA1c differences, these are presented separately.

Study	Raw HbA1c Difference (NHB minus NHW)	Adjusted HbA1c Difference	Adjustment Factors	P-value	Clinically Meaningful (>0.5%)?
M. Harris et al., 1999 [10]	Higher proportion with HbA1c >8% in NHB women (50%) vs others (35-38%) [10]	Not adjusted [10]	None [10]	Not reported [10]	Yes (by proportion) [10]
Lyndsay A. Nelson et al., 2019 [11]	~0.55% (8.86% vs 8.31%) [11]	~0.5% (persisted after SES adjustment) [11]	Income, education, insurance, housing, financial strain [11]	p=0.011 [11]	Yes [11]
L. Egede et al., 2010 [12]	Not reported [12]	0.54% [12]	Confounders (unspecified) [12]	p<0.001 [12]	Yes [12]
N. de Rekeneire et al., 2003 [17]	1.0% (8.4% vs 7.4%) [17]	Reduced by 27% but still significant [17]	Education, income, insulin use, CVD, cholesterol [17]	p<0.01 [17]	Yes [17]
O. Akinboboye et al., 2022 [18]	0.2% (8.0 vs 7.8) [18]	Not separately reported [18]	Education, income, diabetes duration [18]	p=0.002 [18]	No [18]
L. Keoki Williams et al., 2014 [19]	0.43% (7.81% vs 7.38%) at baseline [19]	Not adjusted [19]	Stratified by race and baseline HbA1c [19]	p<0.001 [19]	Borderline [19]

Study	Raw HbA1c Difference (NHB minus NHW)	Adjusted HbA1c Difference	Adjustment Factors	P-value	Clinically Meaningful (>0.5%)?
S. Goonesekera et al., 2015 [5]	0.5% (7.4% vs 6.9%) [5]	No significant disparity after adjustment [5]	Income, education, insurance, health literacy, treatment regimen [5]	Not significant [5]	No (after adjustment) [5]
Cheryl P. Lynch et al., 2014 [15]	0.4% (8.0 vs 7.6) [15]	Not separately reported [15]	Not specified [15]	p=0.024 [15]	No [15]
J. Andersen et al., 2020 [16]	~0.7% at baseline (8.2% vs 7.5%); ~0.5% at completion (7.54% vs 7.0%) [16]	0.23% after adjustment [16]	Age, sex, baseline HbA1c [16]	p<0.01 [16]	No (adjusted) [16]
J. Schectman et al., 2002 [26]	Not reported [26]	0.29% [26]	Income, drug therapy intensity, age, gender, encounter frequency [26]	p=0.04 [26]	No [26]
A. Adams et al., 2005 [28]	Not reported [28]	0.11% (men) to 0.30% (women) for established diabetes [28]	Age, BMI, baseline HbA1c, medications, visits [28]	p=0.0007 (women) [28]	No [28]
M. Rhee et al., 2008 [4]	0.6% at presentation (8.9% vs 8.3%); 0.4% at 1 yr; 0.1% at 2 yr [4]	0.61% at presentation; eliminated at 2 years [4]	Age, sex, BMI, diabetes duration, therapy, adherence [4]	p<0.001 (baseline); p=0.51 (2 yr) [4]	Yes at baseline; No at 2 yr [4]
R. Weinstock et al., 2011 [32]	0.56% (7.58% vs 7.02%) [32]	Not explicitly reported [32]	Education, dual eligibility, insulin, OHA, glucose uploads [32]	p<0.0001 [32]	Yes [32]
L. Hausmann et al., 2010 [33]	0.74% (8.14 vs 7.40) [33]	Persisted (β=0.18) [33]	Psychosocial factors differing by race [33]	Not reported [33]	Yes [33]
Brittany L. Smalls et al., 2020 [50]	0.4-0.5% (7.2% vs 6.8%) [50]	0.31% [50]	Education, income, insurance, insulin, BMI [50]	p=0.02 (adjusted) [50]	Borderline [50]

Study	Raw HbA1c Difference (NHB minus NHW)	Adjusted HbA1c Difference	Adjustment Factors	P-value	Clinically Meaningful (>0.5%)?
M. Heisler et al., 2007 [68]	0.85% (8.07% vs 7.22%) [68]	~14% of difference explained [68]	SES, clinical, self-management, healthcare [68]	p<0.001 [68]	Yes [68]
Vivienne J. Zhu et al., 2011 [55]	0.9% (7.4% vs 6.5%) [55]	Not reported [55]	Insurance, polypharmacy [55]	Not reported [55]	Yes [55]
J. Kirk et al., 2006 (meta-analysis) [1]	0.65% pooled [1]	Not adjusted (pooled raw) [1]	Insurance (subgroup) [1]	p<0.0001 [1]	Yes [1]
Cheryl P. Lynch et al., 2010 [22]	0.60% (7.65 vs 7.05) [22]	Not separately reported for HbA1c [22]	Employment, age, gender, marital status, comorbidities [22]	p<0.001 [22]	Yes [22]
Jennifer G. Twombly et al., 2010 [72]	0.7% at diagnosis (7.8% vs 7.1%); 0.7% at drug initiation (8.5% vs 7.8%) [72]	0.38% at diagnosis; 0.40% at drug initiation [72]	Age, BMI, medical center [72]	p<0.001 [72]	Yes (raw); Borderline (adjusted) [72]
A. Adams et al., 2007 [7]	0.9% at treatment initiation (9.8 vs 8.9) [7]	Persisted after medication adherence adjustment [7]	Medication adherence [7]	p<0.05 [7]	Yes [7]
S. Assari et al., 2017 [65]	Black men 8.63 vs White men 7.86 (0.77%); Black women 7.93 vs White women 7.09 (0.84%) [65]	SES associated with HbA1c for Black men only [65]	SES index, governmental insurance [65]	Not directly reported [65]	Yes [65]
E. S. San Diego et al., 2024 [54]	β=0.21 higher for Black vs White [54]	β=0.21 (adjusted for age, sex, education, endocrinology) [54]	Age, sex, education, provider type [54]	p<0.0001 [54]	No (based on β alone) [54]

The table below summarizes studies reporting odds ratios for uncontrolled diabetes or poor glycemic control.

Study	Outcome Definition	Unadjusted OR (NHB vs NHW)	Adjusted OR (NHB vs NHW)	95% CI	Key Adjustments
L. Egede et al., 2010 [12]	HbA1c >8%	Not reported [12]	OR 1.8 [12]	1.7-2.0 [12]	Time, confounders [12]
B. Egan et al., 2012 [14]	HbA1c <7% (White vs Black)	OR 1.59 [14]	OR 1.21 [14]	1.13-1.30 [14]	Initial HbA1c, therapeutic inertia, visit frequency [14]
Kristen A Berg et al., 2024 [3]	HbA1c ≥8%	Not reported [3]	OR 1.38 [3]	1.04-1.83 [3]	Demographic, SES, health indicators [3]
R. Walker et al., 2018 [37]	HbA1c ≥8%	OR 1.37 [37]	OR 1.07 (spatially adjusted) [37]	1.03-1.11 [37]	Demographics, comorbidities, spatial effects [37]
Woolton Lee et al., 2020 [25]	HbA1c control	Not reported [25]	OR 0.67-0.68 [25]	Significant ($p<0.05$) [25]	SES, lifestyle, health factors [25]
Kelly J Hunt et al., 2020 [2]	HbA1c ≥8%	Not reported [2]	OR 1.11 [2]	1.09-1.14 [2]	Demographics, comorbidity, medications, adherence, access [2]
S. MacLeod et al., 2025 [66]	HbA1c ≥8%	Not reported [66]	AOR 1.29 [66]	1.18-1.41 [66]	Age, sex, ethnicity, marital status, SVI [66]
A. Cunningham et al., 2020 [56]	Any elevated HbA1c	OR 1.48 [56]	Not fully adjusted [56]	Not reported [56]	Not specified [56]
A. Cunningham et al., 2020 [56]	Persistently elevated HbA1c	OR 1.75 [56]	Not fully adjusted [56]	Not reported [56]	Not specified [56]
Nora I Zakaria et al., 2023 [63]	HbA1c ≥7%	Not reported [63]	OR 1.27 [63]	1.03-1.56 [63]	Education, food security, age, sex, diabetes duration [63]
D. Suh et al., 2010 [39]	HbA1c <7% (NHB vs NHW)	Not reported [39]	OR 0.43 [39]	0.29-0.63 [39]	Treatment status [39]
C. Senteio & Akincigil, 2020 [40]	HbA1c >7%	OR 1.33 [40]	Not fully reported [40]	Not reported [40]	Insurance, comorbidities, continuity of care [40]

Study	Outcome Definition	Unadjusted OR (NHB vs NHW)	Adjusted OR (NHB vs NHW)	95% CI	Key Adjustments
J. Lafata et al., 2016 [6]	HbA1c <8%	Not reported [6]	Black less likely (p<0.01) [6]	Not reported [6]	Income, adherence, intensification, comorbidities [6]
S. Kamat et al., 2019 [69]	HbA1c <8%	Not reported [69]	OR 0.77 (Black vs White) [69]	Not reported [69]	Education, insurance, age, comorbidities [69]

Time-in-Range

No study in this review reported TIR measured via continuous glucose monitoring (CGM) as a comparative outcome between NHB and NHW adults with type 2 diabetes. Two studies (Nathan et al., 2024; Karter et al., 2023) used CGM data to evaluate the HbA1c-glucose relationship by race rather than to compare glycemic control per se [8, 9]. A pilot RCT reported TIR for a combined sample but did not stratify by race [61]. The automated insulin delivery trial by Pasquel et al. (2025) enrolled a racially diverse cohort but did not report race-stratified TIR [70]. Thus, the research question regarding standardized TIR differences cannot be addressed with the available evidence.

Magnitude of the HbA1c Disparity

Across the included studies, the unadjusted Black-White HbA1c difference ranged from approximately 0.2% to 1.0%. The meta-analysis by Kirk et al. (2006) estimated a pooled standardized effect of 0.31 (95% CI 0.25-0.39), corresponding to approximately 0.65% higher HbA1c among African Americans [1]. This estimate was robust across subgroups defined by study type, data collection method, and insurance status [1]. The systematic review by Campbell et al. (2012) similarly found HbA1c differences of 0.2 to 0.9 percentage points, with African Americans consistently exhibiting worse outcomes [51]. The meta-analysis by Lee et al. (2020) reported that Black patients had significantly lower odds of HbA1c control (OR 0.67-0.68) after adjustment for socioeconomic, lifestyle, and health factors [25].

The largest studies in terms of sample size came from the VA system. In a national cohort of over 1.1 million veterans, Hunt et al. (2020) found NHB veterans had adjusted odds of 1.11 (95% credible interval 1.09-1.14) for uncontrolled HbA1c ≥8% compared with NHW veterans, even after adjusting for demographics, comorbidity burden, medication type and adherence, and healthcare access metrics [2]. Egede et al. (2010) found a persistent adjusted mean HbA1c difference of 0.54% in a VA cohort of 8,813 veterans followed over a mean of 4.4 years [12]. Walker et al. (2018), studying 64,022 veterans, found 40.8% of NHB versus 33.4% of NHW had uncontrolled HbA1c (≥8%), with an adjusted OR of 1.07 (95% CI 1.03-1.11) after incorporating spatial effects [37].

Effect of Adjusting for Socioeconomic Status

Several studies specifically examined whether comprehensive SES adjustment attenuated the racial disparity. Nelson et al. (2019) used propensity score weighting with five SES indicators (income, education, insurance, housing status, and financial strain) and found that disparities in health literacy, numeracy, and most self-care behaviors were eliminated, but the HbA1c disparity persisted (p=0.011) [11]. Similarly, de Rekeneire et al. (2003) found that controlling for education, income, insulin therapy, cardiovascular disease, cholesterol, and healthcare quality indicators reduced the Black-White HbA1c difference by only 27% [17]. Heidemann et al. (2016) demonstrated that Black

patients had higher average HbA1c than White patients across all income quartiles, and among Black patients there was no significant relationship between income and diabetes prevalence or control [64]. MacLeod et al. (2025) found that while increasing social vulnerability worsened glycemic control across all racial groups, White patients in the highest vulnerability quartile had comparable control to Black patients in the lowest vulnerability quartile, and Black race retained an independent adjusted OR of 1.29 (95% CI 1.18-1.41) after controlling for Social Vulnerability Index [66].

Zakaria et al. (2023) specifically tested whether social, healthcare, and behavioral/health factors attenuated racial disparities in a sample of insured US adults and found that NHB individuals retained higher odds of poor glycemic control (OR 1.27, 95% CI 1.03-1.56) even among those with private insurance [63]. The disparity also persisted in the highly insured Massachusetts community sample studied by Goonesekera et al. (2015), though after comprehensive adjustment including health literacy and treatment regimen, the overall racial difference in HbA1c was no longer statistically significant [5]. Presley et al. (2023) found that in an Alabama Medicaid population, race was not associated with likelihood of meeting HbA1c targets <7% or <8% after adjusting for covariates including comorbidities and insulin use [21].

Effect of Adjusting for Treatment Intensity and Medication Adherence

Egan et al. (2012) reported that three modifiable covariates — initial HbA1c, therapeutic inertia, and visit frequency — accounted for 47.9% of variance in diabetes control and substantially reduced the independent impact of race from OR 1.59 to OR 1.21 [14]. Lafata et al. (2016), in one of the most methodologically targeted analyses, found that medication adherence was poorer in Black patients across all cardiometabolic risk factors (50.6% vs 39.7% non-adherent for HbA1c-related oral medications) but that adjusting for medication adherence and treatment intensification did not alter the pattern of racial disparities in achieving HbA1c <8% [6]. Rhee et al. (2008) found that when a uniform treatment algorithm was implemented with comparable patient adherence and provider intensification behavior, racial disparities in HbA1c were eliminated by 2 years of follow-up (p=0.51), despite a 0.6% gap at presentation [4]. Adams et al. (2007) demonstrated that even among patients with medication adherence ≥80%, Black race was associated with higher HbA1c [7]. Heisler et al. (2007) found that socioeconomic, clinical, healthcare access, and self-management measures collectively explained only ~20% of the HbA1c difference between NHB and NHW respondents [68].

Disparities in Medication Prescribing

Multiple studies documented racial disparities in access to newer glucose-lowering medications that may contribute to outcome gaps. Brady et al. (2024) found that Black patients were less likely to receive GLP-1 receptor agonist prescriptions (aOR 0.72, 95% CI 0.66-0.79), SGLT2 inhibitor prescriptions (aOR 0.86, 95% CI 0.78-0.96), and insulin prescriptions (aOR 0.81, 95% CI 0.74-0.88) compared with White patients after adjusting for sociodemographic and clinical variables [23]. Elhussein et al. (2020) found that Black race was associated with significantly lower initiation of newer diabetes medications (HR 0.81, 95% CI 0.80-0.94) independent of glycemic control [34]. German et al. (2026) found that Black patients were less likely than White patients to receive new SGLT2i prescriptions (RR 0.59, 95% CI 0.42-0.82) and GLP-1RA prescriptions (RR 0.62, 95% CI 0.49-0.79), despite having clinical indications and above-target HbA1c [62]. Cromer et al. (2021) found that African American and Hispanic respondents had lower odds of analog basal insulin use compared with NHW respondents (aOR 0.42, 95% CI 0.24-0.74 for African Americans) [43]. Li et al. (2021) documented that use of newer glucose-lowering medications increased more rapidly among NHW compared with NHB and Hispanic patients between 2005 and 2018 [71]. Fernandes et al. (2019) reported that Black patients were more likely to be intensified at higher HbA1c levels compared with White patients, suggesting clinical inertia was more pronounced [41].

The HbA1c-Glucose Relationship as a Potential Confounder

Three studies directly investigated whether the HbA1c assay itself may contribute to the observed disparity. Nathan et al. (2024), in a prospective substudy of the GRADE trial using 10-day CGM, found that HbA1c was 0.2-0.6 percentage points higher in NHB than NHW patients for the same average glucose levels (AG10 range 100-250 mg/dL), and these differences persisted after adjusting for demographic factors [8]. Karter et al. (2023), in a retrospective study of 1,788 patients with CGM data at Kaiser Permanente, found that mean HbA1c was 0.33 percentage points (95% CI 0.23-0.44) higher among African American patients relative to White patients for a given mean glucose [9]. However, within-group variance in the HbA1c-glucose relationship was substantially greater than between-group variance (65% vs 9%) [9]. In contrast, Behan et al. (2014) found no statistically significant difference in the HbA1c-glucose relationship between Black and White adults with type 2 diabetes based on approximately 42 glucose measurements per participant over 3 months (ANCOVA p=0.968 for fasting glucose, p=0.428 for postprandial glucose) [36]. Twombly et al. (2010) found that Black and White veterans had comparable glucose levels over 4-5 years before and after diagnosis despite ~0.2% higher HbA1c in Blacks, and attributed this to glucose-independent associations between race and HbA1c [72].

Synthesis

The evidence across 80 sources consistently demonstrates that NHB adults with type 2 diabetes have higher HbA1c levels than NHW adults, with unadjusted differences typically ranging from 0.3% to 1.0% and a meta-analytic pooled estimate of approximately 0.65% [1]. The central question — whether this disparity persists after controlling for both SES and treatment intensity — yields a nuanced answer that varies meaningfully by setting, adjustment approach, and follow-up duration.

Reconciling Persistent vs. Attenuated Disparities

The apparent contradiction between studies finding persistent disparities after adjustment (e.g., Nelson et al., 2019 [11]; Zakaria et al., 2023 [63]; Hunt et al., 2020 [2]) and those finding attenuation or elimination (e.g., Goonesekera et al., 2015 [5]; Rhee et al., 2008 [4]; Presley et al., 2023 [21]) can be partly reconciled by distinguishing between population-level observational studies and studies conducted within structured healthcare delivery systems.

In structured care environments where treatment algorithms are uniformly applied and adherence is monitored, disparities narrow substantially. Rhee et al. (2008) demonstrated this most clearly: in a municipal diabetes clinic using a uniform treatment algorithm, the 0.6% Black-White HbA1c gap at presentation was eliminated by 2 years of follow-up [4]. However, this study's population was 94% Black with only 218 White patients, limiting generalizability [4]. The VA system, which provides relatively uniform access to care, still showed persistent but smaller disparities: Hunt et al. (2020) found an adjusted OR of 1.11 after controlling for medication use, adherence, and access metrics in 1.14 million veterans [2], and Egede et al. (2010) found a mean difference of 0.54% in 8,813 veterans [12]. The persistence of disparities in the VA system — where financial barriers to care are minimized — suggests that access alone does not fully explain the gap.

In contrast, the highly insured Boston community sample (Goonesekera et al., 2015) found no significant racial disparity in glycemic control after comprehensive adjustment for income, education, insurance, health literacy, and treatment regimen [5]. The Alabama Medicaid cohort (Presley et al., 2023) similarly found no independent race effect [21]. These findings suggest that when comprehensive SES and treatment adjustments are made in populations with relatively uniform insurance coverage, the disparity may be substantially, though not always fully, attenuated.

The Role of Modifiable Mediators

Egan et al. (2012) identified three modifiable factors — initial HbA1c at entry to care, therapeutic inertia, and visit frequency — that together accounted for approximately 48% of variance in diabetes control and reduced the race effect from OR 1.59 to OR 1.21 [14]. This finding is particularly instructive: it suggests that roughly half of the observed disparity operates through healthcare delivery mechanisms that are amenable to intervention.

Medication adherence, despite being consistently worse among NHB patients (e.g., 50.6% vs 39.7% non-adherent in the Lafata et al. study [6]; mean PDC of 40% vs 50% in the Zhu et al. study [55]), did not explain the Black-White HbA1c gap when formally tested as a mediator. Lafata et al. (2016) explicitly demonstrated that adjusting for both adherence and treatment intensification did not alter racial disparities in achieving HbA1c <8% across nine integrated health systems [6]. Adams et al. (2007) found that even among patients with ≥80% medication adherence, Black race was associated with higher HbA1c [7]. These findings collectively suggest that the disparity is not reducible to differences in medication-taking behavior alone.

Prescribing disparities represent another modifiable mediator that most HbA1c disparity studies did not capture in their adjustments. The substantial underuse of GLP-1 receptor agonists (aOR 0.72 for Black vs White [23]) and SGLT2 inhibitors (aOR 0.86 [23]; RR 0.59 [62]) among Black patients, independent of sociodemographic and clinical variables, constitutes a pathway through which treatment intensity differences may perpetuate the HbA1c gap that is not captured by simple adjustment for insulin or oral medication use.

Biological Differences in the HbA1c Assay

A portion of the observed HbA1c disparity may reflect biological differences in hemoglobin glycation rather than true differences in glycemic exposure. Nathan et al. (2024) and Karter et al. (2023) both found that for the same average glucose, HbA1c was approximately 0.2-0.6 and 0.33 percentage points higher in NHB than NHW patients, respectively [8, 9]. If the true biological offset is approximately 0.3%, this would account for roughly half of the typical unadjusted disparity of 0.65% [1]. However, Behan et al. (2014) found no significant racial difference in the HbA1c-glucose relationship in a small sample [36], and Karter et al. (2023) emphasized that within-group variability far exceeded between-group variability [9]. Pacheco Sanchez et al. (2025) further documented racially divergent metabolic and inflammatory signatures of diabetes, with African Americans displaying Th17-related cytokine elevations rather than the classical dyslipidemia and inflammation pattern seen in White individuals [45]. These findings suggest that HbA1c may systematically overestimate glycemia in NHB patients, which has implications both for the interpretation of disparity studies and for clinical management decisions.

Context-Dependent Heterogeneity

Several population-specific patterns emerged that help explain heterogeneity across studies:

- **Sex interactions**: Adams et al. (2005) found the Black-White HbA1c gap was 0.30% in women but only 0.11% (nonsignificant) in men with established diabetes [28]. Harris et al. (1999) noted that NHB women had the highest prevalence of HbA1c >8% at 50% [10]. Parrinello et al. (2015) found that Black-White disparities in risk factor control were greater in women than men [53]. Assari et al. (2017) found that SES was associated with HbA1c for Black men but not for other race-by-gender subgroups [65].
- **Age effects**: Smalls et al. (2020) found that HbA1c disparities persisted into older age (≥65 years), with trends in mean HbA1c increasing over time for NHB and Mexican Americans but decreasing for NHW [50]. McWilliams et al. (2009) found that racial differences in disease control were smaller after age 65, coinciding with near-universal Medicare coverage [67]. Sears et al. (2018) found that insulin adherence mediated racial HbA1c

disparities only in younger adults [58].

- **Geographic variation**: Senteio & Akincigil (2020) found that the adjusted probability of poor diabetes control reached 69% for African Americans in rural areas [40]. Walker et al. (2018) demonstrated that spatial patterns of HbA1c control differed between NHB and NHW veterans, and incorporating spatial effects explained additional disparity variance [37]. Hunt et al. (2020) documented that prevalence of uncontrolled diabetes varied from 19.1% to 29.2% across VA catchment areas, with disparities largely persisting across most facilities [2].
- **Temporal trends**: Hua et al. (2024) found that NHB veterans showed improvement in early glycemic control across cohorts diagnosed from 2008-2018 [20], while Smalls et al. (2020) found worsening trends for NHB older adults across NHANES cycles [50]. Presley et al. (2023) found that later study years were associated with lower likelihood of meeting HbA1c targets in an Alabama Medicaid population [21].

Quantifying the Residual Disparity

After accounting for the most comprehensive sets of confounders available across the literature, the residual Black-White HbA1c disparity appears to be approximately 0.2-0.4% in absolute terms, or an odds ratio of approximately 1.1-1.4 for uncontrolled diabetes. The largest and most rigorously adjusted studies — Hunt et al. (2020) with 1.14 million veterans and adjustment for demographics, comorbidities, medications, adherence, and access (OR 1.11) [2]; Egan et al. (2012) with 22,285 patients adjusting for initial HbA1c, therapeutic inertia, and visit frequency (OR 1.21) [14]; and Berg et al. (2024) using NHANES with sequential adjustment for demographic, socioeconomic, and health indicators (OR 1.38) [3] — consistently find a statistically significant residual disparity. However, a meaningful fraction of this residual likely reflects biological differences in the HbA1c-glucose relationship (~0.3 percentage points) [8, 9], which would not represent a true difference in glycemic exposure.

No study in this review reported standardized effect sizes (Cohen's d) for the adjusted racial difference in HbA1c, and no study reported TIR comparisons between NHB and NHW adults with type 2 diabetes. The absence of TIR data is a notable gap, as CGM-derived metrics would bypass the potential confounding introduced by race-related differences in hemoglobin glycation and provide a more direct comparison of glycemic exposure. The small number of CGM-based studies identified focused on characterizing the HbA1c-glucose relationship itself rather than comparing glycemic control outcomes between racial groups [8, 9].

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