


BRIEF REPORT

Glycaemic profiles of diverse patients with type 2 diabetes using basal insulin: MOBILE study baseline data

Anne Peters MD¹ | Nathan Cohen MS² | Peter Calhoun PhD²  |
 Katrina J. Ruedy MSPH² | Roy W. Beck MD² | Thomas W. Martens MD³ |
 Shichun Bao MD⁴ | Nelly M. Njeru BA⁵ | Stayce E. Beck PhD⁵ | David A. Price MD⁵

¹Keck School of Medicine of the University of Southern California, Los Angeles, California

²Jaeb Center for Health Research, Tampa, Florida

³International Diabetes Center, Park Nicollet Clinic, Brooklyn Center, Minnesota

⁴Vanderbilt University Medical Center, Nashville, Tennessee

⁵Dexcom, Inc., San Diego, California

Correspondence

David A. Price, Dexcom, Inc., 6340 Sequence Drive, San Diego, CA 92121, USA.
 Email: dprice@dexcom.com

Funding information

Dexcom, Inc.

Abstract

Basal insulin is often prescribed to patients with suboptimally controlled type 2 diabetes (T2D); however, its therapeutic efficacy is inadequate in many. During the MOBILE study's baseline phase, we evaluated 173 participants' continuous glucose monitoring (CGM) data (mean \pm SD age 57 ± 9 years; 50% female; HbA1c 9.1% [range 7.1%-11.6%]; 40% using sulphonylureas; 19% using NPH; reported self-monitored blood glucose [SMBG] frequency median 1.0 checks/day) who were using basal, but not prandial insulin. Blinded CGM data were recorded for 10 days prior to randomization. The mean glucose value was 208 ± 47 mg/dL and it was lowest in the early morning. Mean time in the 70-180 mg/dL range was 9.6 ± 6.1 hours/day (40% \pm 25%). Hyperglycaemia was extensive with medians of 14.7 (61%) and 5.0 (20.9%) hours/day with glucose greater than 180 and 250 mg/dL, respectively. Hypoglycaemia was infrequent (median [IQR] 0 [0, 4.3] minutes/day [0.0% {0.0%, 0.3%}]) with glucose less than 70 mg/dL. Blinded CGM highlights the limitations of infrequent SMBG in basal insulin users with T2D and allows characterization of hyperglycaemia and hypoglycaemia in basal insulin users with suboptimal control. The MOBILE study randomized phase will define the benefits of using real-time CGM compared with SMBG in this population.

KEYWORDS

basal insulin, clinical trial, continuous glucose monitoring

1 | INTRODUCTION

Despite advances in pharmacotherapy, many patients with type 2 diabetes (T2D) are suboptimally controlled, which contributes to excess morbidity and mortality for the patients and to increased costs for the healthcare system.¹ When facing elevated HbA1c levels, patients are often daunted by factors such as their own or their provider's

reluctance to change ('therapeutic inertia'), regimen complexity or cost. There are often long² and detrimental³ delays in initiating or intensifying insulin as a result.

Once-daily injections of basal insulin are often initiated to manage patients with poorly controlled fasting glucose on non-insulin therapies, and many algorithms have been developed to facilitate basal insulin titration by patients themselves or by

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited and is not used for commercial purposes.

© 2020 The Authors. *Diabetes, Obesity and Metabolism* published by John Wiley & Sons Ltd.

healthcare professionals. These algorithms uniformly adjust basal insulin based on fasting values obtained by self-monitoring of blood glucose (SMBG).⁴ The strategy of bedtime dosing confers a comparatively low risk of hypoglycaemia⁵ and is moderately effective at reducing HbA1c levels, with approximately half of participants in clinical studies achieving HbA1c levels of less than 7% or other individualized targets.⁶ Unfortunately, basal insulin regimens that rely on infrequent SMBG testing and emphasize the control of fasting glucose and avoiding night-time hypoglycaemia ignore the problems of daytime premeal and sustained postprandial hyperglycaemia, placing a ceiling effect on obtainable HbA1c reductions. Of greater concern, real-world evidence shows that only ~30% of patients with T2D who use basal insulin maintain HbA1c levels of less than 7%.⁶⁻⁹ There are many potential explanations for the discrepancy between evidence gained from clinical trials versus clinical practice. These include poor adherence to insulin, inadequate initialization and titration of insulin,¹⁰⁻¹² and concerns about hypoglycaemia.¹³

Real-time continuous glucose monitoring (rtCGM) has been shown to have both short- and long-term beneficial effects on HbA1c for patients with T2D who are not using intensive insulin therapy. A randomized study of 100 adults not using prandial insulin (half were on oral agents alone; one-third were using basal insulin) reported on the benefits of rtCGM compared with SMBG.¹⁴ After 12 weeks (four 3-week cycles, each comprising 2 weeks of CGM use and 1 week without CGM), the fall in HbA1c was significantly larger for the rtCGM group ($-1.0\% \pm 1.1\%$) than for the SMBG group ($-0.5\% \pm 0.8\%$; $P = .006$). Remarkably, the benefits were sustained 9 months later.¹⁵ More recently, the potential feasibility of a new approach to basal insulin initiation that relies in part on CGM data has been described.¹⁶ However, neither study reported the CGM profiles of study participants.

There remains a need to characterize glycaemic patterns in sub-optimally controlled basal insulin users. The MOBILE study is a randomized clinical study in patients with T2D using basal insulin with or without non-insulin therapies who have HbA1c values of 7.8%-11.5%. Prior to randomization, all participants used a blinded CGM for 10 days to record their CGM readings, but did not have access to their data. In the randomization step, participants were assigned in a 2:1 ratio to use either CGM or SMBG, respectively, as a basis for management decisions. Results of the randomized phase will be reported separately and may support a hypothesis for meaningfully different glycaemic outcomes between the two groups. Here, we report on participants' prerandomization (baseline) demographics, medication regimens and glycaemic profiles.

2 | METHODS

The MOBILE study was conducted at 22 endocrinology practices in the United States. The protocol and HIPAA (Health Insurance Portability and Accountability Act)-compliant informed consent forms were approved by institutional review boards (central commercial board for

21 sites and local boards for one site). Written informed consent was obtained from each participant. The study was registered at ClinicalTrials.gov (NCT03566693). Patients aged 30 years or older with T2D using basal insulin and with HbA1c levels of between 7.8% and 11.5% (either by a point-of-care or local laboratory test) were included, without regard to use of oral medications for diabetes or use of glucagon-like peptide-1 receptor agonists (GLP-1 RAs). Eligible patients were under the care of a primary care physician for diabetes management, reported SMBG frequencies at least three times per week, and had medication regimens that had been stable for at least 3 months. Exclusion criteria included pregnancy, advanced renal disease, conditions impacting the stability of HbA1c measurements, use of prandial insulin and use of a personal CGM device in the 3 months prior to enrolment.

Each participant wore a G6 CGM sensor paired to a receiver (Dexcom, Inc., San Diego, CA, USA) on the abdomen for 10 days. The receivers stored glucose values at 5-minute intervals, but did not display them. After download, CGM metrics were calculated using all CGM readings collected prior to randomization. Summary statistics include means with standard deviations for normally distributed data and medians with interquartile ranges for skewed data. Outcomes were reported for the entire cohort and were dichotomized by type of insulin (NPH or analogue) and GLP1-RA usage (yes or no). A hypoglycaemic event was defined as at least 15 continuous minutes with CGM readings of less than 70 mg/dL. The end of an event was defined as at least 15 continuous minutes with CGM readings of 80 mg/dL or higher (to minimize counting events when CGM readings were hovering around the threshold). Similarly, a hyperglycaemic event was defined as at least 15 continuous minutes with CGM readings of less than 250 mg/dL and the end of the event was defined as at least 15 continuous minutes with CGM readings of 240 mg/dL or less. *P*-values comparing CGM metrics by insulin usage and GLP1-RA usage were calculated using t-tests for normally distributed outcomes and Mann-Whitney U-tests for skewed outcomes. The glycaemic profile of cohorts was also characterized by plotting median (IQR) glucose values by time of day; resulting modal day plots were generated for all participants, for participants grouped by NPH versus analogue insulin usage, for participants grouped by GLP1-RA usage versus non-usage, and for participants grouped by sulphonylurea usage versus non-usage. HbA1c levels shown in the demographics table (Table S1) were obtained from a central lab. One randomized participant was excluded from all analyses after a misdiagnosis of T2D was discovered, and two randomized participants were excluded because baseline CGM data were not available.

3 | RESULTS

Data from 173 participants who enrolled from August 2018 to October 2019 were available for analysis. The population was 50% female and 53% were non-Caucasian. The mean \pm SD age of the participants was 57 ± 9 (range 33-79) years, HbA1c was $9.1\% \pm 0.9\%$ (7.1%-11.6%) and body mass index was 33.9 ± 6.6 (21.1-54.8) kg/m².

TABLE 1 CGM metrics: overall, by type of insulin, and by GLP1-RA usage

	Overall (N = 173)		Type of insulin		GLP1-RA usage		P-value*	P-value*
	235 (222, 238)	234 (221, 238)	NPH (n = 33)	Analogues ^a (n = 140)	Yes (n = 37)	No (n = 136)		
Hours of data median (quartiles)	235 (222, 238)	234 (221, 238)	234 (221, 238)	235 (222, 238)	232 (219, 238)	236 (222, 238)	NA	NA
Overall glucose control, mean (SD)								
TIR: 70-180 mg/dL, hours/day %	9.6 (6.1, 40%) (25%)	7.2 (5.0, 30%) (21%)	7.2 (5.0, 30%) (21%)	10.2 (6.2, 43%) (26%)	11.3 (6.1, 47%) (25%)	9.2 (6.0, 38%) (25%)	.01	.06
Mean glucose, mg/dL	208 (47)	231 (46)	231 (46)	202 (46)	196 (42)	211 (48)	.001	.10
Hyperglycaemia metrics, median (quartiles)								
>180 mg/dL, hours/day %	14.7 (8.8, 19.1) 61% (37%, 79%)	16.5 (13.0, 21.9) 69% (54%, 91%)	16.5 (13.0, 21.9) 69% (54%, 91%)	14.4 (8.2, 18.7) 60% (34%, 78%)	10.9 (7.5, 17.1) 45% (31%, 71%)	15.2 (9.4, 19.9) 63% (39%, 83%)	.02	.07
>250 mg/dL, hours/day %	5.0 (1.4, 9.9) 20.9% (5.9%, 41.2%)	9.2 (6.0, 13.8) 38.4% (25.1%, 57.5%)	9.2 (6.0, 13.8) 38.4% (25.1%, 57.5%)	4.1 (1.3, 8.6) 17.2% (5.4%, 35.7%)	2.7 (0.8, 6.6) 11.3% (3.4%, 27.3%)	5.7 (1.5, 10.2) 23.8% (6.2%, 42.5%)	<.001	.03
Hypoglycaemia, median (quartiles)								
<70 mg/dL, minutes/day (%)							.02	.04
0 (0%)	105 (61%)	14 (42%)	14 (42%)	91 (65%)	28 (76%)	77 (57%)		
>0-<14.4 (>0%<1%)	42 (24%)	14 (42%)	14 (42%)	28 (20%)	5 (14%)	37 (27%)		
14.4-<28.8 (1%<2%)	9 (5%)	3 (9%)	3 (9%)	6 (4%)	1 (3%)	8 (6%)		
28.8-<43.2 (2%<3%)	6 (3%)	0 (0%)	0 (0%)	6 (4%)	3 (8%)	3 (2%)		
43.2-<57.6 (3%<4%)	5 (3%)	1 (3%)	1 (3%)	4 (3%)	0 (0%)	5 (4%)		
≥57.6 (≥4%)	6 (3%)	1 (3%)	1 (3%)	5 (4%)	0 (0%)	6 (4%)		
<54 mg/dL, minutes/day (%)							.002	.08
0 (0%)	133 (77%)	18 (55%)	18 (55%)	115 (82%)	32 (86%)	101 (74%)		
>0-<14.4 (>0%<1%)	34 (20%)	14 (42%)	14 (42%)	20 (14%)	5 (14%)	29 (21%)		
14.4-<28.8 (1%<2%)	4 (2%)	1 (3%)	1 (3%)	3 (2%)	0 (0%)	4 (3%)		
28.8-<43.2 (2%<3%)	1 (<1%)	0 (0%)	0 (0%)	1 (<1%)	0 (0%)	1 (<1%)		
43.2-<57.6 (3%<4%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)		
≥57.6 (≥4%)	1 (<1%)	0 (0%)	0 (0%)	1 (<1%)	0 (0%)	1 (<1%)		
Weekly hypoglycaemic event rate ^b	0 (0, 0.7)	0.4 (0, 1.4)	0.4 (0, 1.4)	0 (0, 0.7)	0 (0, 0)	0 (0, 0.7)	.008	.05
Glycaemic variability								
CV mean (SD)	28% (7%)	31% (8%)	31% (8%)	27% (7%)	27% (6%)	28% (7%)	.02	.20

TIR, time in range (70-180 mg/dL); CV, coefficient of variation.

^aInsulin analogues included glargine (n = 110), detemir (n = 16) and degludec (n = 15). One participant was using both glargine and degludec.

^bA hypoglycaemic event was defined as at least 15 continuous minutes with CGM readings of <70 mg/dL. The end of an event was defined as at least 15 continuous minutes with CGM readings of ≥80 mg/dL.

*P-values are based on t-tests for normally distributed variables and Mann-Whitney U-tests for skewed ones.

Ninety-six subjects (55%) reported having less than a college degree and only 72 subjects (42%) carried primarily private health insurance. Basal insulin types included NPH (used by 19% of participants), glargine (64%), detemir (9%) and degludec (9%). Of the 110 glargine users, 89% used U-100 and 11% used U-300 formulations. Mean basal insulin usage was 0.47 ± 0.28 (0.09-1.78) U/kg/day, which was equivalent to 45 ± 28 (7-210) U/day. Thirty-seven subjects (21%) used a GLP1-RA and 70 subjects (40%) used a sulphonylurea. NPH insulin use was more common among sulphonylurea users compared with non-users (33% vs. 10%, respectively), and less common among GLP1-RA users compared with non-users (3% vs. 24%, respectively). The median frequency of SMBG testing was 1.0 (IQR 1-2) checks/day. Full demographic details are provided in Table S1. Educational

attainment and insurance coverage for participants according to insulin type usage, GLP1-RA usage and sulphonylurea usage are provided in Table S2.

Table 1 and Figure 1 summarize CGM-based metrics of glycaemic control during the blinded CGM use. The median (IQR) number of hours of data available was 235 (222, 238). Average glucose levels were high (208 ± 47 mg/dL), consistent with the elevated HbA1c levels used as an entry criterion, and only 9.6 ± 6.1 hours/day ($40\% \pm 25\%$) were spent in the 70-180 mg/dL target range. Hyperglycaemia was extensive, with medians of 14.7 (61%) and 5.0 (20.9%) hours/day spent with glucose levels greater than 180 and 250 mg/dL, respectively. Hyperglycaemic episodes were long-lived, with the median duration in excess of 3 hours; 49% of the population had at least one

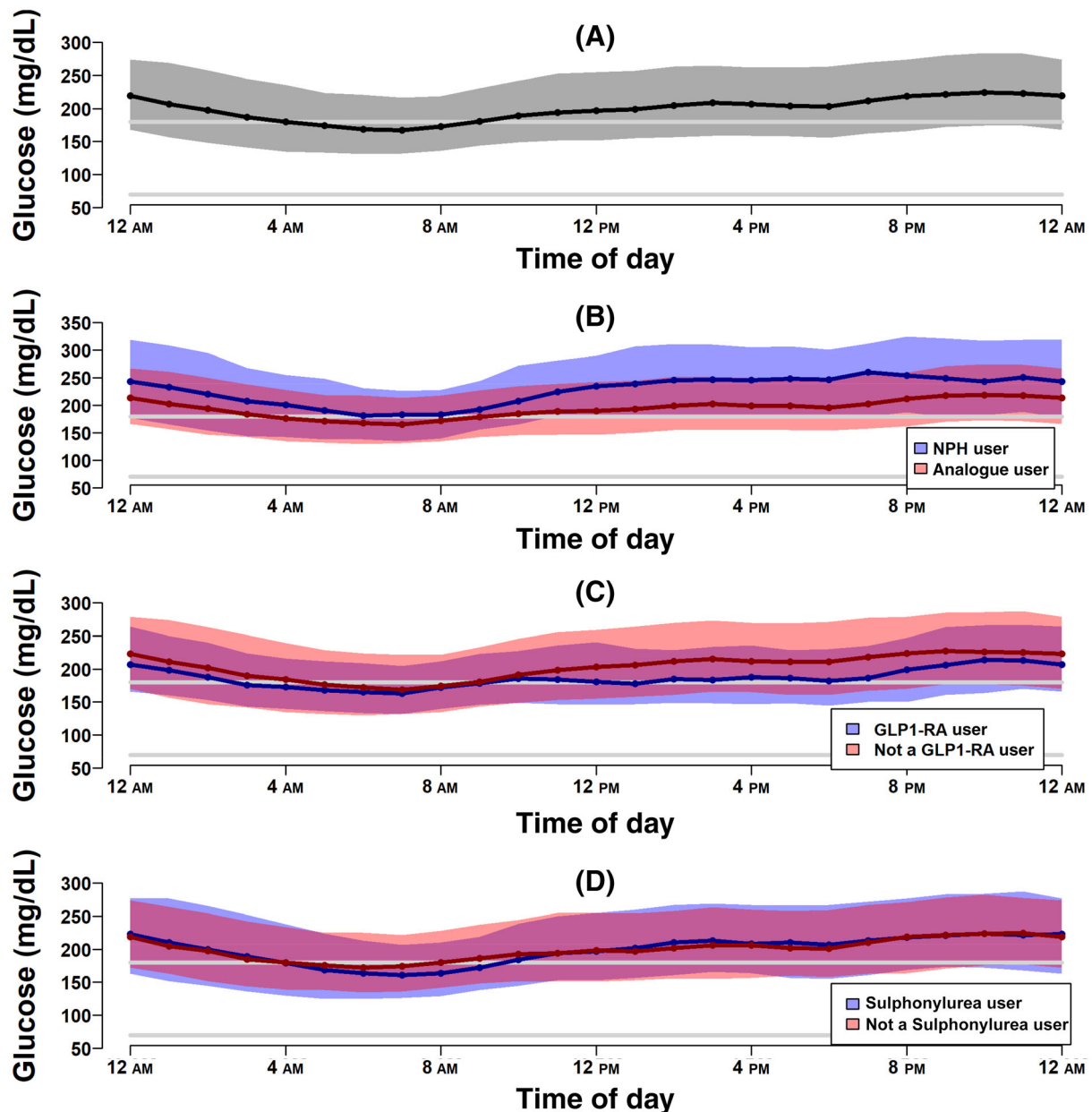


FIGURE 1 Glucose levels by time of day (main document). Dots represent median values; upper and lower bounds of the shaded areas are the 75th and 25th percentiles, respectively. A, all participants; B, groups according to type of basal insulin; C, groups according to GLP1-RA usage; D, groups according to sulphonylurea usage

episode lasting 8 hours or longer. Very few of the blinded CGM values reflected hypoglycaemia and hypoglycaemic events were infrequent. Glucose variability (coefficient of variation [CV%]) was low, indicating comparatively stable¹⁷ glucose trajectories.

As shown in Table 1, NPH users had more time with glucose levels greater than 250 mg/dL compared with analogue insulin users (median 9.2 vs. 4.1 hours/day [38.4% vs. 17.2%], respectively; $P < .001$), had a greater hypoglycaemic event rate per week (median 0.4 vs. 0, respectively; $P = .008$), and had higher glycaemic variability assessed by CV% (mean 31% vs. 27%, respectively; $P = .02$). Table 1 also shows that GLP1-RA users had lower median times spent in hyperglycaemia than participants not using GLP1-RAs (median 10.9 vs. 15.2 hours/day [45% vs. 63%] for time >180 mg/dL and 2.7 vs. 5.7 hours/day [11.3% vs. 23.8%] for time >250 mg/dL, respectively). Forty per cent of sulphonylurea users experienced a hypoglycaemic event, compared with 26% of participants not using sulphonylureas ($P = .06$; data not shown).

Figure 1 shows modal day plots for glucose levels for the entire population, for groups based on type of basal insulin, for groups based on GLP1-RA usage, and for groups based on sulphonylurea usage. Glucose levels were slightly lower at night than during the day. There was a trend towards decreasing glucose levels in the early morning hours, with increasing levels during the day. The lowest hourly median was 167 mg/dL at 07:00 AM and the highest was 225 mg/dL at 10:00 PM. At no point in the modal day did glucose values of less than 130 mg/dL make up more than 25% of the total (Figure 1A). Plots of glucose levels with particular types of basal insulin (Figure 1B) show that NPH users experienced more hyperglycaemia during the afternoon and evening hours than analogue insulin users. Lower median glucose levels were associated with GLP1-RA usage compared with GLP1-RA non-usage (Figure 1C).

4 | DISCUSSION

This is a report of blinded CGM profiles in suboptimally controlled patients with T2D using basal insulin. The participants were diverse with respect to their diabetes medication regimens, ethnic backgrounds, education levels and insurance types. Practice settings ranged from clinics for privately insured individuals to community health centres in medically underserved areas.

The data reported here have several limitations. A principal limitation is that they were gathered prior to randomization, and several confounders therefore limit our ability to comment on the relative efficacy of different therapeutic regimens. Although therapy intensification was clearly indicated for all participants, we did not investigate the reasons why primary care providers or the participants themselves failed to optimize basal insulin or intensify therapy. We do not know if participants were taught to self-titrate their basal insulin but failed to do so. The extent to which access to healthcare infrastructure, insurance coverage or economic factors contributed to regimens or outcomes is also unknown. We did not address potential differences between users of different analogue insulins (detemir, degludec, glargine) because so few

participants were in the former two groups. We did not attempt to analyse differences between groups based on the use or non-use of other non-insulin drugs such as metformin, dipeptidyl peptidase-4 inhibitors, sodium-glucose co-transporter-2 inhibitors, thiazolidinediones or the numerous possible multidrug combinations.

The data were most remarkable for the prevalence and extent of hyperglycaemia, which highlights the need for more aggressive management by the treating clinicians and the participants themselves. Regardless of whether participants used NPH or analogue insulin, time in the 70-180 mg/dL range was well below the 70% consensus goal¹⁸ but exposure to hypoglycaemia was low in both groups, discounting it as a major barrier to therapy intensification. The frequently cited fear of hypoglycaemia among physicians who are reluctant to use insulin in this context¹⁹ seems unwarranted. In addition, the data show that NPH users experienced more hypoglycaemia and higher glycaemic variability than analogue insulin users; these differences may be attributable to socioeconomic status, medication regimens or access to healthcare.

The low frequency of SMBG testing (~1 per day) underestimates the extent of hyperglycaemia in this population, especially if testing is performed before breakfast when glucose levels were seen to be at their lowest. A more complete picture of glycaemic patterns, especially of the timing and extent of hyperglycaemic excursions and the presence or absence of hypoglycaemia, is required to optimally titrate basal insulin, to provide personalized recommendations for lifestyle, and to further intensify therapy. As shown in this analysis, CGM can better characterize dysglycaemia than a single HbA1c or fasting glucose measurement. Results from the randomized phases of the MOBILE study will delineate whether decisions made by patients and their primary care physicians that are informed by RT-CGM data ultimately result in better glycaemic control than those informed by SMBG alone.

ACKNOWLEDGEMENTS

Sites and principal investigators of the MOBILE study group are as follows: University of Southern California: Anne Peters, MD; International Diabetes Center: Thomas W. Martens, MD; Vanderbilt University: Shichun Bao, MD, PhD; University of Michigan: Rodica Pop-Busui, MD, PhD; Scripps Whittier Diabetes Institute: Athena Philis-Tsimikas, MD; Emory University: Guillermo Umperrez, MD; Henry Ford Health System: Davida Kruger, MSN; Iowa Diabetes and Endocrinology Research Center: Anuj Bhargava, MD; University of North Carolina: Laura Young, MD, PhD; Northwestern University: Grazia Aleppo, MD; Washington University: Janet McGill, MD; Las Vegas Endocrinology: Quang Nguyen, DO; Amarillo Medical Specialists: William Biggs, MD; Carteret Health Care: Ian Orozco, MD; Lucas Research: K. Jean Lucas, MD; Texas Diabetes & Endocrinology: Lindsay Harrison, MD; Research Institute of Dallas: Stephen Aronoff, MD. The authors thank Tom Arant and John Welsh of Dexcom for logistical and writing support, respectively. Dexcom, Inc. provided funding and devices for the study.

CONFLICT OF INTEREST

AP reports personal fees from Abbott Diabetes Care, Boehringer Ingelheim, Eli Lilly and Company, Livongo, MannKind Corporation, Merck,

Novo Nordisk, Sanofi, and Pendulum Therapeutics; grant support from Dexcom and vTv Therapeutics; personal fees from Novo Nordisk; and other support from Mellitus Health, Inc., Omada Health, Inc., Stability Health, LLC, Pendulum Therapeutics, and Livongo outside the submitted work. NC, PC, and KJR have received grant support and donated supplies, paid to the Jaeb Center for Health Research, from Abbott Diabetes Care, Beta Bionics, and Dexcom, and grant support, paid to the Jaeb Center for Health Research, from Tandem Diabetes Care. RWB has received grant support and donated supplies, paid to the Jaeb Center for Health Research, from Abbott Diabetes Care, Ascensia Diabetes Care US, Beta Bionics, and Roche Diabetes Care, grant support, donated supplies, and consulting fees, paid to the Jaeb Center for Health Research, from Dexcom, Novo Nordisk, and Tandem Diabetes Care, grant support and consulting fees, paid to the Jaeb Center for Health Research, from Bigfoot Biomedical, and consulting fees, paid to the Jaeb Center for Health Research, from Eli Lilly and Insulet. TWM has received research support from Abbott Diabetes Care, Dexcom, Lilly, Medtronic, Novo Nordisk and Omnipod. His employer, nonprofit IDC/HealthPartners Institute, contracts for his services and he receives no direct personal income from these activities. SB has received, through her institution, research funding from Dexcom. NMN, SEB, and DAP are employees of Dexcom, Inc.

AUTHOR CONTRIBUTIONS

RWB, PC, NC, and KJR had full access to the data and take full responsibility for the integrity of the data and the accuracy of the data analysis. NMN, SEB, and DAP participated in development of the protocol, writing the manuscript, and the decision to submit the manuscript for publication. All authors participated in acquisition, analysis, or interpretation of data and in critical revisions of the manuscript for important intellectual content.

PEER REVIEW

The peer review history for this article is available at <https://publons.com/publon/10.1111/dom.14238>.

DATA AVAILABILITY STATEMENT

Data will be made available upon reasonable request.

ORCID

Peter Calhoun  <https://orcid.org/0000-0002-5325-7200>

REFERENCES

- Edelman SV, Polonsky WH. Type 2 diabetes in the real world: the elusive nature of glycemic control. *Diabetes Care*. 2017;40(11):1425-1432.
- Khunti K, Nikolajsen A, Thorsted BL, Andersen M, Davies MJ, Paul SK. Clinical inertia with regard to intensifying therapy in people with type 2 diabetes treated with basal insulin. *Diabetes Obes Metab*. 2016;18(4):401-409.
- Nichols GA, Romo-LeTourneau V, Vupputuri S, Thomas SM. Delays in anti-hyperglycaemic therapy initiation and intensification are associated with cardiovascular events, hospitalizations for heart failure and all-cause mortality. *Diabetes Obes Metab*. 2019;21(7):1551-1557.
- Strange P. Treat-to-target insulin titration algorithms when initiating long or intermediate acting insulin in type 2 diabetes. *J Diabetes Sci Technol*. 2007;1(4):540-548.
- Karl DM, Gill J, Zhou R, Riddle MC. Clinical predictors of risk of hypoglycaemia during addition and titration of insulin glargine for type 2 diabetes mellitus. *Diabetes Obes Metab*. 2013;15(7):622-628.
- Owens DR, Landgraf W, Frier BM, et al. Commencing insulin glargine 100 U/mL therapy in individuals with type 2 diabetes: determinants of achievement of HbA1c goal less than 7.0. *Diabetes Obes Metab*. 2019;21(2):321-329.
- Canivell S, Mata-Cases M, Real J, et al. Glycaemic control after treatment intensification in patients with type 2 diabetes uncontrolled on two or more non-insulin antidiabetic drugs in a real-world setting. *Diabetes Obes Metab*. 2019;21(6):1373-1380.
- Oguz A, Benroubi M, Brismar K, et al. Clinical outcomes after 24 months of insulin therapy in patients with type 2 diabetes in five countries: results from the TREAT study. *Curr Med Res Opin*. 2013;29(8):911-920.
- Meneghini LF, Mauricio D, Orsi E, et al. The diabetes unmet need with basal insulin evaluation (DUNE) study in type 2 diabetes: achieving HbA1c targets with basal insulin in a real-world setting. *Diabetes Obes Metab*. 2019;21(6):1429-1436.
- Berard L, Bonnemaire M, Mical M, Edelman S. Insights into optimal basal insulin titration in type 2 diabetes: results of a quantitative survey. *Diabetes Obes Metab*. 2018;20(2):301-308.
- Kalirai S, Stephenson J, Perez-Nieves M, et al. Primary care physician perspectives on basal insulin initiation and maintenance in patients with type 2 diabetes mellitus. *Prim Care Diabetes*. 2018;12(2):155-162.
- Khunti K, Giorgino F, Berard L, Mauricio D, Harris SB. The importance of the initial period of basal insulin titration in people with diabetes. *Diabetes Obes Metab*. 2020;22(5):722-733.
- Li P, Geng Z, Ladage VP, Wu J, Lorincz I, Doshi JA. Early hypoglycaemia and adherence after basal insulin initiation in a nationally representative sample of Medicare beneficiaries with type 2 diabetes. *Diabetes Obes Metab*. 2019;21(11):2486-2495.
- Ehrhardt NM, Chellappa M, Walker MS, Fonda SJ, Vigersky RA. The effect of real-time continuous glucose monitoring on glycemic control in patients with type 2 diabetes mellitus. *J Diabetes Sci Technol*. 2011;5(3):668-675.
- Vigersky RA, Fonda SJ, Chellappa M, Walker MS, Ehrhardt NM. Short- and long-term effects of real-time continuous glucose monitoring in patients with type 2 diabetes. *Diabetes Care*. 2012;35(1):32-38.
- Aradottir TB, Bengtsson H, Jensen ML, et al. Feasibility of a new approach to initiate insulin in type 2 diabetes. *J Diabetes Sci Technol*. 2020. <https://doi.org/10.1177/1932296819900240>.
- Monnier L, Colette C, Wojtuszczyk A, et al. Toward defining the threshold between low and high glucose variability in diabetes. *Diabetes Care*. 2017;40(7):832-838.
- Battelino T, Danne T, Bergenstal RM, et al. Clinical targets for continuous glucose monitoring data interpretation: recommendations from the international consensus on time in range. *Diabetes Care*. 2019;42(8):1593-1603.
- Ross SA, Tildesley HD, Ashkenas J. Barriers to effective insulin treatment: the persistence of poor glycemic control in type 2 diabetes. *Curr Med Res Opin*. 2011;27(Suppl 3):13-20.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

How to cite this article: Peters A, Cohen N, Calhoun P, et al. Glycaemic profiles of diverse patients with type 2 diabetes using basal insulin: MOBILE study baseline data. *Diabetes Obes Metab*. 2021;23:631-636. <https://doi.org/10.1111/dom.14238>