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The Insulin-Only Bionic Pancreas Improves Glycemic Control in Non-Hispanic White and Minority Adults and Children With Type 1 Diabetes

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Bionic pancreas improved glycemic control in both non-Hispanic White and minority individuals

Racial/ethnic minority individuals with type 1 diabetes have worse diabetes outcomes than non-Hispanic White individuals

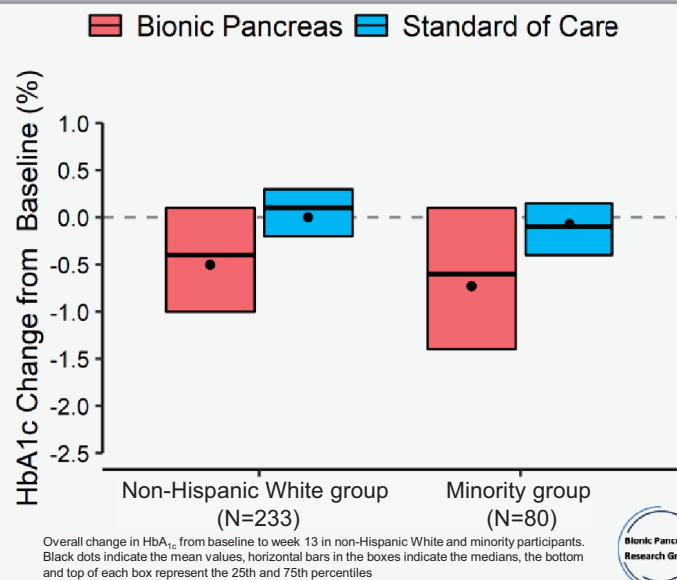


Subanalysis of the Bionic Pancreas Pivotal Trial compared glycemic control in non-Hispanic White and minority individuals using bionic pancreas vs. standard of care*



N = 240 non-Hispanic White individuals
N = 84 Minority individuals (African American, Hispanic, other)
161 adults/163 children

*Real-time continuous glucose monitoring plus injections, pump, or closed-loop



ARTICLE HIGHLIGHTS

- Racial/ethnic minority individuals with type 1 diabetes have worse health outcomes than non-Hispanic White individuals.
- The bionic pancreas has potential to improve glycemic control in minority individuals compared with standard care.
- Use of bionic pancreas at 13 weeks showed improved glycemic control in African American and Hispanic minority participants.
- The bionic pancreas may provide minorities with type 1 diabetes with improved outcomes compared with current insulin-delivery technologies.



The Insulin-Only Bionic Pancreas Improves Glycemic Control in Non-Hispanic White and Minority Adults and Children With Type 1 Diabetes

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Diabetes Care 2023;46:1185–1190 | <https://doi.org/10.2337/dc22-1478>

OBJECTIVE

We evaluated the performance of the iLet bionic pancreas (BP) in non-Hispanic White individuals (here referred to as “Whites”) and in Black, Hispanic, and other individuals (here collectively referred to as “Minorities”).

RESEARCH DESIGN AND METHODS

A multicenter, randomized controlled trial evaluated glycemic management with the BP versus standard of care (SC) in 161 adult and 165 pediatric participants with type 1 diabetes over 13 weeks.

RESULTS

In Whites ($n = 240$), the mean baseline-adjusted difference in 13-week HbA_{1c} between the BP and SC groups was -0.45% (95% CI -0.61 to -0.29 [-4.9 mmol/mol; -6.6 to -3.1]; $P < 0.001$), while this difference among Minorities ($n = 84$) was -0.53% (-0.83 to -0.24 [-6.0 mmol/mol; -9.2 to -2.8]; $P < 0.001$). In Whites, the mean baseline-adjusted difference in time in range between the BP and SC groups was 10% (95% CI 7–12; $P < 0.001$) and in Minorities was 14% (10–18; $P < 0.001$).

CONCLUSIONS

The BP improves glycemic control in both Whites and Minorities and offers promise in decreasing health care disparities.

There are striking racial/ethnic disparities in care and outcomes for people with type 1 diabetes, with worse metabolic control, higher rates of diabetes complications and ketoacidosis, and much lower use of technology among racial/ethnic groups (1–8). The reasons for these differences in technology use are largely due to socioeconomic status (SES); however, even after adjusting for SES, disparities persist for Black children, possibly due to limited access to care and system mistrust, implicit bias (such as caregivers’ perception of costs and providers’ perception of users’ competence), and structural racism (8–12). Novel therapeutic approaches are needed to diminish disparities in ways that do not increase burden, but racial and ethnic minority groups are largely underrepresented in clinical trials and clinical use of modern technologies (11,13–16). This subanalysis tested the hypothesis that White and minority individuals would have similarly improved glycemic control using the iLet bionic pancreas (BP) compared with standard care (SC).

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*A listing of the Bionic Pancreas Research Group is provided in the supplementary material online.

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RESEARCH DESIGN AND METHODS

This is a subanalysis of the Insulin-only Bionic Pancreas Pivotal Trial, a randomized controlled trial comparing the iLet (Beta Bionics) BP to SC (real-time continuous glucose monitoring [CGM] plus continuation of the method of insulin delivery used at baseline, including hybrid closed-loop [HCL] systems) in both children and adults with type 1 diabetes at 16 centers in the U.S. Participant baseline data were collected using the Dexcom G6 CGM; for those not using a Dexcom G6, 2 weeks of blinded baseline glucose data were collected before randomization. The results for the full cohort and for adults and children have been previously reported (17–19). The BP automatically delivers insulin (and in some configurations, glucagon) as needed based on CGM-sensor glucose. It is initialized using only the body weight and does not require carbohydrate counting. All insulin given, including for meals announced by the user as “Breakfast,” “Lunch,” or “Dinner,” and “Usual For Me,” “More,” or “Less,” is determined autonomously by the BP algorithms and cannot be modified by the user or health care provider. The algorithms continually adapt basal, correction, and meal-announcement insulin doses to meet the individual’s needs.

Matching the racial/ethnic diversity of people in the U.S. with type 1 diabetes was a priority during recruitment. The self-reported race/ethnicity of participants included non-Hispanic White (here referred to as “Whites”) and African American, Hispanic (White and non-White), Asian, Native Hawaiian/Pacific Islander, American Indian/Alaska Native, and more than one race (referred to collectively as “Minorities”). Insurance was classified as private, Medicare/Medicaid, other government insurance, or no coverage. The diabetes management method used at baseline was categorized as multiple daily injections (MDI), with or without CGM, pump, with or without CGM, a predictive low-glucose suspend system, or an HCL system.

The outcomes were the HbA_{1c} level at 13 weeks and the percentage of sensor glucose time in range of 70–180 mg/dL (TIR).

Statistical methods followed those reported for the primary trial analyses (19). Treatment groups were compared using a linear mixed-effects regression model adjusting for the baseline value

of the metric, age at randomization, and clinical site (random effect). The differences in the treatment effect by race were tested by adding a treatment-by-race interaction to the model. The treatment effect by SES factors was tested analogously. All CIs and *P* values are two sided. No adjustments for multiple comparisons were performed, and results should be considered exploratory. Analyses were performed with SAS 9.4 software (SAS Institute).

RESULTS

The trial randomized 326 participants from January to July 2021 (Supplementary Fig. 1), between 6 and 79 years old, with baseline HbA_{1c} from 5.5% to 13.1% (37 to 120 mmol/mol). Baseline characteristics are summarized in Table 1. The overall self-identified race and ethnicity of the participants (Supplementary Table 1) was 74% White (*n* = 240), 10% Black (*n* = 32), 10% Hispanic (*n* = 34), 3% more than one race (*n* = 11), 2% Asian (*n* = 5), <1%

Table 1—Participant characteristics by race and treatment arm

	BP		SC	
	White <i>n</i> = 157	Minority <i>n</i> = 60	White <i>n</i> = 83	Minority <i>n</i> = 24
Age at enrollment, years	29 ± 19	22 ± 17	31 ± 20	16 ± 11
Age ≥18 years	85 (54)	22 (37)	47 (57)	7 (29)
Female sex	73 (46)	34 (57)	30 (36)	11 (46)
Education†				
<Bachelor degree	41 (26)	31 (53)	26 (33)	11 (46)
Bachelor degree	62 (40)	14 (24)	30 (38)	9 (38)
Graduate/professional degree	53 (34)	13 (22)	24 (30)	4 (17)
Annual household income‡				
<\$25,000	3 (2)	3 (5)	0 (0)	2 (8)
\$25,000–<\$35,000	4 (3)	3 (5)	5 (6)	1 (4)
\$35,000–<\$50,000	6 (4)	5 (8)	1 (1)	3 (13)
\$50,000–<\$75,000	16 (10)	10 (17)	6 (7)	3 (13)
\$75,000–<\$100,000	22 (14)	5 (8)	13 (16)	3 (13)
\$100,000–<\$200,000	57 (36)	18 (30)	24 (29)	5 (21)
≥\$200,000	38 (24)	9 (15)	18 (22)	5 (21)
Insurance§				
Private	141 (90)	41 (68)	68 (82)	17 (71)
Medicare/Medicaid	11 (7)	14 (23)	8 (10)	6 (25)
Other government	4 (3)	4 (7)	5 (6)	1 (4)
No coverage	0 (0)	0 (0)	1 (1)	0 (0)
Baseline technology use				
MDI + no CGM	14 (9)	6 (10)	3 (4)	3 (13)
MDI + CGM	31 (20)	19 (32)	21 (25)	12 (50)
Pump + no CGM	3 (2)	2 (3)	4 (5)	0 (0)
Pump + CGM	47 (30)	19 (32)	24 (29)	3 (13)
Pump + CGM + PLGS	6 (4)	3 (5)	3 (4)	2 (8)
Pump + CGM + HCL	56 (36)	11 (18)	28 (34)	4 (17)
Baseline HbA _{1c} , %	7.7 ± 1.1	8.3 ± 1.5	7.6 ± 1.2	7.9 ± 0.9
Baseline HbA _{1c} , mmol/mol	61 ± 12.0	67 ± 16.4	60 ± 13.1	63 ± 9.8
<8.0% (<64 mmol/mol)	94 (60)	28 (47)	53 (65)	13 (54)
8.0%–<9.0% (64–<75 mmol/mol)	44 (28)	12 (20)	20 (24)	7 (29)
≥9.0% (≥75 mmol/mol)	19 (12)	20 (33)	9 (11)	4 (17)
Baseline TIR, %	53 ± 17	47 ± 21	52 ± 21	45 ± 16
≥70%	30 (19)	8 (13)	22 (27)	3 (13)

PLGS, predictive low-glucose suspend. †For pediatric participants, the highest education level of parent or guardian was reported. ‡Data on income were not provided by 27 White participants and 9 Minority participants. §All insurance data are self-reported. Two White participants and one Minority participant did not provide data on insurance. Six White participants and two Minority participants who indicated having private insurance also had government-funded insurance (Medicare/Medicaid/other government). ||One White participant was missing a baseline HbA_{1c} measurement.

American Indian/Alaska Native ($n = 2$), and <1% not reported ($n = 2$). The two participants with unknown race and ethnicity were excluded from this analysis. Minorities had higher Medicare/Medicaid use compared with Whites (24% vs. 8%), less HCL use (18% vs. 35%), and a higher number of participants with $HbA_{1c} \geq 9\%$ (29% vs. 12%).

A similar treatment effect of BP on change of HbA_{1c} from baseline to 13 weeks, adjusting for baseline HbA_{1c} , was observed in both Whites and Minorities (P value for interaction = 0.57). In Whites, HbA_{1c} decreased from $7.7 \pm 1.1\%$ (61 ± 12 mmol/mol) to $7.2 \pm 0.7\%$ (56 ± 7 mmol/mol) in the BP group and stayed the same, from $7.6 \pm 1.2\%$ to $7.6 \pm 1.0\%$ in the SC group (mean adjusted difference [MAD] -0.45% , 95% CI -0.61 to -0.29 [-4.9 mmol/mol, -6.6 to -3.1], $P < 0.001$) (Table 2 and Fig. 2). In Minorities, HbA_{1c} decreased from $8.3 \pm 1.5\%$ (67 ± 17 mmol/mol) to $7.4 \pm 0.6\%$ ($58 \pm$

7 mmol/mol) in the BP group and was unchanged at $7.9 \pm 0.9\%$ (63 ± 10 mmol/mol) in the SC group (MAD -0.53% , -0.83 to -0.24 [-6.0 mmol/mol, -9.2 to -2.8], $P < 0.001$). Greater changes in HbA_{1c} from baseline were noted with higher baseline HbA_{1c} levels, as shown in Table 2, Figs. 1 and 2, and Supplementary Fig. 2. Of participants with baseline $HbA_{1c} \geq 8\%$, Whites' HbA_{1c} decreased from $8.8 \pm 0.8\%$ to $7.6 \pm 0.7\%$ in the BP group and from $8.8 \pm 0.9\%$ to $8.5 \pm 1.0\%$ in the SC group (MAD -0.95% , -1.26 to -0.63 , $P < 0.001$), while Minorities' HbA_{1c} decreased from $9.4 \pm 1.2\%$ to $7.8 \pm 0.5\%$ in the BP group and from $8.8 \pm 0.4\%$ to $8.7 \pm 0.7\%$ in the SC group (MAD -1.04% , -1.53% to -0.56% , $P < 0.001$).

As with HbA_{1c} , the treatment effect of BP on TIR comparing 2 weeks of baseline to 13 weeks on the BP was similar in Whites and Minorities, adjusting for baseline TIR (P value for interaction = 0.11). TIR in Whites increased

from $53 \pm 17\%$ to $65 \pm 10\%$ in the BP group and increased from $52 \pm 21\%$ to $56 \pm 17\%$ in the SC group (MAD 10%, 95% CI 7–12, $P < 0.001$). TIR in Minorities increased from $47 \pm 21\%$ to $63 \pm 9\%$ in the BP group versus $45 \pm 16\%$ to $47 \pm 14\%$ in the SC group (MAD 14%, 10–18, $P < 0.001$).

In participants <18 years of age (Supplementary Table 2 and Supplementary Fig. 3), Whites' mean HbA_{1c} decreased from $8.1 \pm 1.2\%$ to $7.5 \pm 0.7\%$ in the BP group and did not change, from $7.8 \pm 1.2\%$ to $7.8 \pm 1.1\%$, at 13 weeks in the SC group (MAD -0.49% , 95% CI -0.77 to -0.21 , $P < 0.001$). Minorities' mean HbA_{1c} decreased from $8.2 \pm 1.2\%$ to $7.5 \pm 0.6\%$ at 13 weeks in the BP group and increased from $7.8 \pm 1.0\%$ to $7.9 \pm 0.9\%$ in the SC group (MAD -0.41% , -0.81 to -0.01 , $P = 0.05$).

Among those ≥ 18 years of age (Supplementary Table 2), Whites' mean HbA_{1c} decreased from $7.4 \pm 0.8\%$ to $7.0 \pm 0.6\%$ in

Table 2—Glycemic outcomes by treatment group, race, and baseline HbA_{1c}

	Baseline				Follow-up (at or over 13 weeks)				Adjusted difference BP minus SC (95% CI) [P value]*
	BP		SC		BP		SC		
	No.	Mean \pm SD	No.	Mean \pm SD	No.	Mean \pm SD	No.	Mean \pm SD	
HbA_{1c}, % (mmol/mol)									
Overall									
White	157	7.7 ± 1.1 (61 ± 12)	82	7.6 ± 1.2 (60 ± 13)	153	7.2 ± 0.7 (56 ± 7)	81	7.6 ± 1.0 (59 ± 11)	-0.45 ($-0.61, -0.29$) [<0.001]
Minority	60	8.3 ± 1.5 (67 ± 17)	24	7.9 ± 0.9 (63 ± 10)	57	7.4 ± 0.6 (58 ± 7)	23	7.9 ± 0.9 (63 ± 10)	-0.53 ($-0.83, -0.24$) [<0.001]
Baseline $HbA_{1c} < 8.0\%$ (<64 mmol/mol)									
White	94	7.0 ± 0.6 (53 ± 6)	53	7.0 ± 0.7 (53 ± 8)	91	7.0 ± 0.6 (53 ± 6)	53	7.1 ± 0.7 (54 ± 8)	-0.17 ($-0.32, -0.02$) [0.03]
Minority	28	7.0 ± 0.5 (53 ± 6)	13	7.2 ± 0.5 (55 ± 6)	27	7.1 ± 0.5 (54 ± 6)	12	7.2 ± 0.5 (56 ± 5)	0.04 ($-0.27, 0.34$) [0.82]
Baseline $HbA_{1c} \geq 8.0\%$ (≥ 64 mmol/mol)									
White	63	8.8 ± 0.8 (72 ± 8)	29	8.8 ± 0.9 (73 ± 10)	62	7.6 ± 0.7 (60 ± 7)	27	8.5 ± 1.0 (69 ± 11)	-0.95 ($-1.26, -0.63$) [<0.001]
Minority	32	9.4 ± 1.2 (79 ± 13)	11	8.8 ± 0.4 (73 ± 5)	30	7.8 ± 0.5 (61 ± 6)	11	8.7 ± 0.7 (71 ± 7)	-1.04 ($-1.53, -0.56$) [<0.001]
TIR 70–180 mg/dL, %									
Overall									
White	157	53 ± 17	83	52 ± 21	156	65 ± 10	83	56 ± 17	10 (7, 12) [<0.001]
Minority	60	47 ± 21	24	45 ± 16	60	63 ± 9	24	47 ± 14	14 (10, 18) [<0.001]
Baseline $HbA_{1c} < 8.0\%$ (<64 mmol/mol)									
White	94	62 ± 14	53	63 ± 15	93	68 ± 8	53	65 ± 13	5 (2, 7) [<0.001]
Minority	28	64 ± 13	13	55 ± 12	28	68 ± 9	13	56 ± 12	7 (2, 12) [0.007]
Baseline $HbA_{1c} \geq 8.0\%$ (≥ 64 mmol/mol)									
White	63	39 ± 13	29	32 ± 14	63	61 ± 9	29	40 ± 13	19 (15, 23) [<0.001]
Minority	32	33 ± 16	11	34 ± 12	32	59 ± 6	11	38 ± 10	21 (15, 28) [<0.001]

*Estimates, CIs, and P values calculated from a repeated-measures linear mixed-effects model adjusting for age at randomization as a fixed effect and clinical site as a random effect.

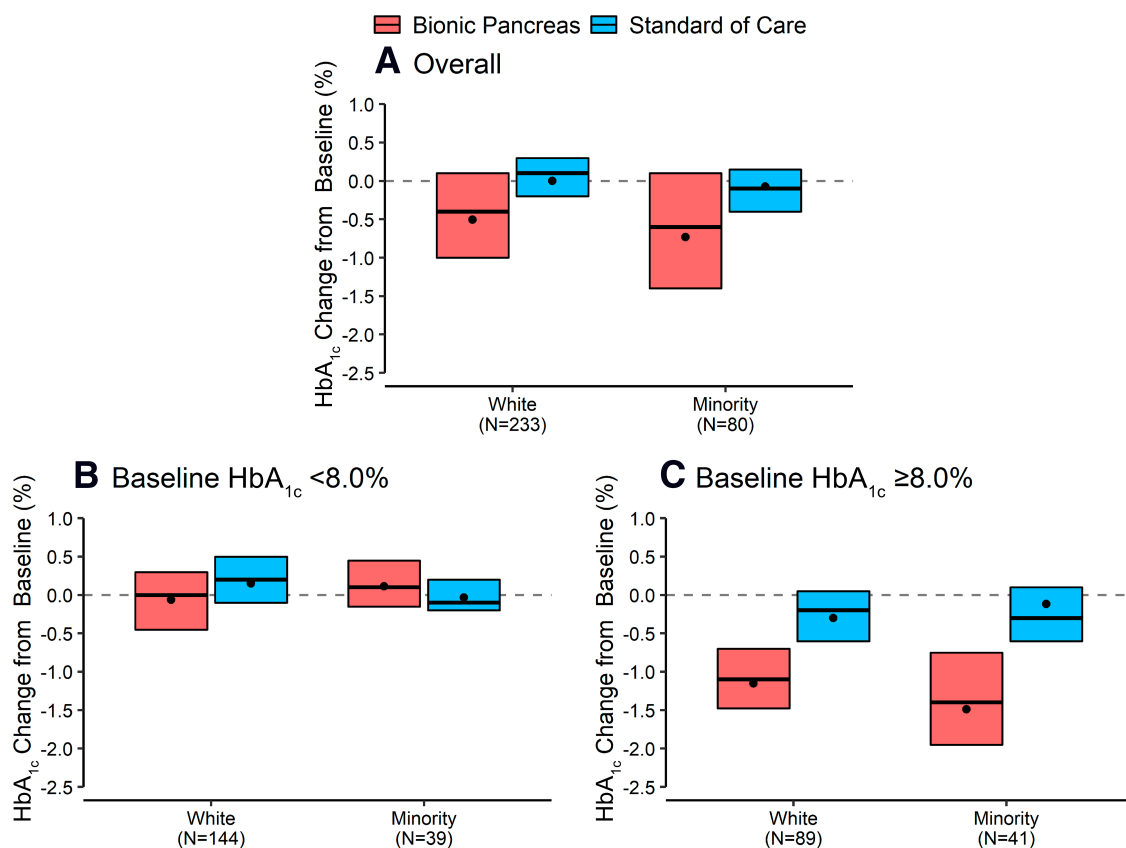


Figure 1—HbA_{1c} change from baseline by race/ethnicity and treatment group. *A*: Overall change in HbA_{1c} from baseline to week 13 in White and Minority participants. *B*: Change in HbA_{1c} from baseline to week 13 in White and Minority participants with baseline HbA_{1c} <8%. *C*: Change in HbA_{1c} from baseline to week 13 in White and Minority participants with baseline HbA_{1c} ≥8%. Black dots indicate the mean values, horizontal bars in the boxes indicate the medians, and the bottom and top of each box represent the 25th and 75th percentiles, respectively.

the BP group and decreased from $7.5 \pm 1.2\%$ to $7.4 \pm 0.9\%$ in the SC group (MAD -0.42% , 95% CI -0.60 to -0.24 , $P < 0.001$). Minorities' HbA_{1c} decreased from $8.5 \pm 2.0\%$ at baseline to $7.3 \pm 0.5\%$ in BP group and decreased from $8.3 \pm 0.9\%$ to $8.1 \pm 1.0\%$ in the SC group (MAD -0.82% , -1.27 to -0.38 , $P < 0.001$).

The mean baseline HbA_{1c} was higher and the treatment effect on HbA_{1c} and TIR was greater for those without a bachelor degree, for those with an annual household income of $< \$75,000$, for those not using a CGM before the trial, and for those using MDI (Supplementary Table 3).

CONCLUSIONS

Among children in the U.S. with type 1 diabetes, the SEARCH study found that 72.6% were non-Hispanic White, 15.7% were Hispanic, 9.3% were non-Hispanic Black, and 2.4% were Asian (5). Similar patterns are present in adults. Our pediatric cohort was 65.5% non-Hispanic White, 14.5% Hispanic, 9.7% non-Hispanic Black, and 2.4%

Asian, and therefore reflective of the population of children with type 1 diabetes. Our overall cohort, while not perfectly representative, was more reflective than previous pivotal studies of HCL systems (10,13–15,20), of the general U.S. population with type 1 diabetes in terms of race/ethnicity, SES, range of HbA_{1c}, and the distribution of baseline therapies (MDI, insulin pump, and HCL), suggesting that results observed may be more generalizable.

Our analysis shows that use of the BP yielded improvements in HbA_{1c} by a similar degree among both White and Minority participants when adjusted for baseline HbA_{1c}. Additionally, the racial and ethnic disparities in mean HbA_{1c} appear to be reduced with the BP. In the SC group, baseline difference in HbA_{1c} between White and Minority participants remained unchanged at follow-up, while in the BP group, the difference in HbA_{1c} among White and Minority participants was reduced at follow-up. Thus, while the treatment effect did not differ by race when adjusting for baseline glucose, the differences in HbA_{1c} between White and Minority

participants were reduced after using the BP system, likely because those with higher baseline HbA_{1c} had greater improvements with the BP system than those with lower baseline HbA_{1c}. Likewise, those with lower SES had higher baseline HbA_{1c}, reflected in greater improvements with the BP system.

Similar improvements in glucose control with the BP relative to SC in Minority and White participants may be due to the autonomous dosing by the BP, which determines every insulin dose independently of the skill of the health care provider or the level of numeracy or technical acumen of the user. Collectively, this operational autonomy of the BP opens the practical possibility that a broad base of prescribing health care providers and users of the BP could use the device safely and effectively.

Limitations of this study included a relatively small number of Asian and American Indian/Alaska Native participants, that we did not collect data on household size to further assess income inequality, and a duration of system use that was limited to 13 weeks. Nonetheless, these data provide a basis for additional studies to more

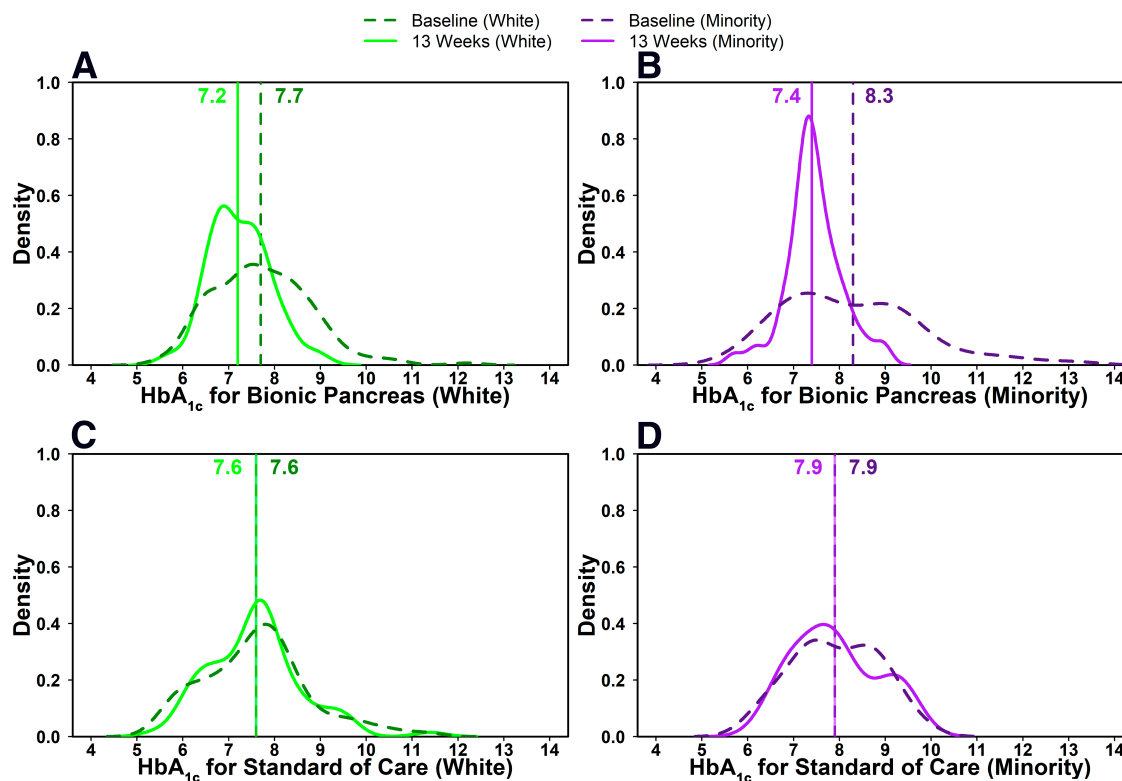


Figure 2—Distribution of HbA_{1c} at baseline and follow-up by race and treatment. HbA_{1c} data are shown at baseline and at 13 weeks for White and Minority participants in the BP groups (A and B) and SC groups (C and D). The curves represent the distribution of HbA_{1c} at baseline and week 13, with higher density values (measured from the area under the curve) representing a greater proportion of individuals with a given HbA_{1c}. The vertical lines represent the mean values, which are indicated numerically at the top of each line.

fully explore the relationship between race/ethnicity and glycemic control with automated insulin delivery systems.

Conclusion

The iLet BP improved glycemic outcomes in a racially and ethnically diverse minority cohort, with similar improvements seen in both Minority and White subpopulations, suggesting generalizability to a real-world population of people with type 1 diabetes that might be less discriminatory than current insulin-delivery technologies.

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Care. Continuous glucose monitor supplies were purchased at a discounted price from Dexcom, Inc.

Duality of Interest. This study received funding support from Beta Bionics, Inc. S.J.R. has issued patents and pending patents on aspects of the bionic pancreas that are assigned to Massachusetts General Hospital and licensed to Beta Bionics; has received honoraria and/or travel expenses for lectures from Novo Nordisk, Roche, and Ascensia; serves on the scientific advisory boards of Unomedical; served on scientific advisory board and had stock in Companion Medical, which was bought out by Medtronic; has received consulting fees from Beta Bionics, Inc., Novo Nordisk, Senseonics, and Flexion Therapeutics; has received grant support from Zealand Pharma, Novo Nordisk, and Beta Bionics, Inc.; and has received in-kind support in the form of technical support and/or donation of materials from Zealand Pharma, Ascensia, Senseonics, Adocia, and Tandem Diabetes. While this manuscript was under review, S.J.R. became an employee of Beta Bionics, Inc. E.R.D. has issued patents and pending patents on aspects of the bionic pancreas and is an employee, is the Executive Chair of the Board of Directors, and a shareholder of Beta Bionics, Inc. V.N.S.'s employer, University of Colorado, received research support from Novo Nordisk, Eli Lilly, Insulet, Dexcom, Tandem Diabetes Care, Jaeb Center for Health Research, the National Institutes of Health, and JDRF. V.N.S. also received honoraria through CU Medicine from Medscape and LifeScan for advisory boards and from Dexcom and Insulet for consulting and

speaking. P.C. is a former Dexcom employee and his current employer has received consulting payments on his behalf from vTv Therapeutics, Beta Bionics, Inc., Dexcom, and Diasome. R.W.B. reports that his institution has received funding on his behalf as follows: grant funding and study supplies from Tandem Diabetes Care, Beta Bionics, Inc., and Dexcom; study supplies from Medtronic, Ascensia, and Roche; consulting fees and study supplies from Eli Lilly and Novo Nordisk; and consulting fees from Insulet, Bigfoot Biomedical, vTv Therapeutics, and Diasome. N.M. has received research grant support through her institution from Novo Nordisk and grant funding and study supplies from Medtronic and LifeScan, and through subcontracts with the Jaeb coordinating center from Beta Bionics, Inc., Eli Lilly, Ascensia, and Dexcom. No other potential conflicts of interest relevant to this article were reported.

Author Contributions. L.E.C. researched and interpreted the data and wrote the manuscript. S.J.R., E.R.D., R.W.B., V.N.S., P.C., K.B., and N.M. researched data, contributed to the discussion, and reviewed and edited the manuscript. R.B. performed statistical analysis and contributed to writing and reviewing the manuscript. N.M. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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