What’s new in type 1 diabetes?

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Topics

- Research from the journal “Diabetes Care” 2016-2017
- Pathogenesis (3)
- Therapy (3)
- Complications (5)
- Education and Self-Care (4)

Pathogenesis: Gut microbiome

JAMA Pediatr. 2016;170(1):20-28. doi:10.1001/jamapediatrics.2015.2757
Published online November 9, 2015.

Original Investigation

Association of Early Exposure of Probiotics and Islet Autoimmunity in the TEDDY Study

Ulla Uusitalo, PhD; Xiang Liu, PhD; Jimin Yang, PhD, RD; Carin Andrén Aronsson, MS; Sandra Hummel, PhD; Martha Butterworth, MS; Åke Lernmark, PhD; Marian Rewers, PhD; William Hagopian, MD, PhD; Jin-Xiong She, PhD; Olli Simell, MD, PhD; Jorma Toppari, MD, PhD; Anette G. Ziegler, PhD; Beena Akolkar, PhD; Jeffrey Krischer, PhD; Jill M. Norris, PhD; Suvi M. Virtanen, MD, PhD; for the TEDDY Study Group

- Infants with high risk HLA genotypes , n=7473, were enrolled.
- USA, Finland, Germany, Sweden
- Follow up until ages 4-10
- Primary outcome = development of islet autoimmunity

Gut Immunity and Type 1 Diabetes: a Mélange of Microbes, Diet, and Host Interactions?

David Endesfelder[1] · Marion Engel[1] · Wolfgang zu Castell[2]

Curr Diab Rep (2016) 16: 60
DOI 10.1007/s11892-016-0753-3

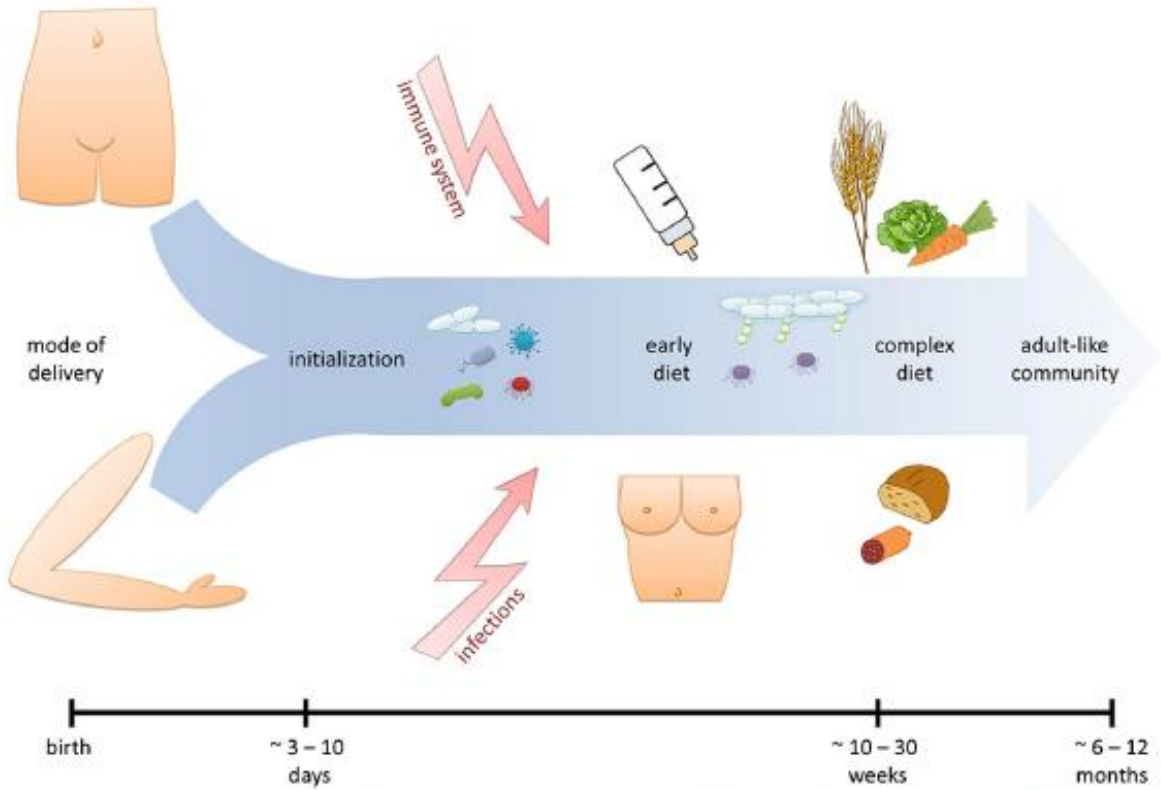
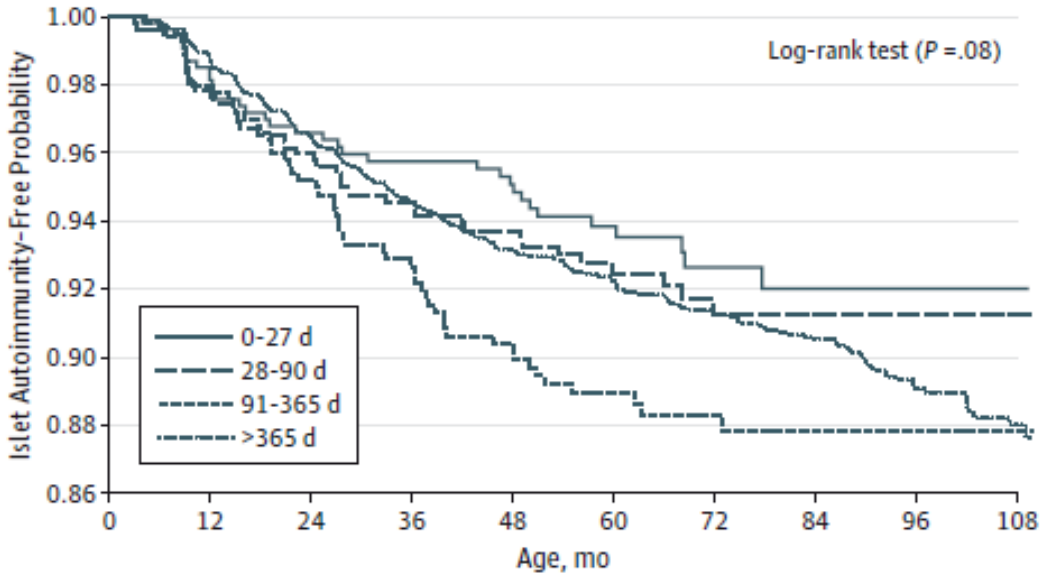


Fig. 1 Microbial waves of succession. Initial colonization occurs shortly after birth. While first colonizers of vaginally delivered babies are related to the vaginal microbiome of the mother, children delivered via C-section host gut microbial communities that are more similar to the mother's skin microbiome. In the first weeks of life, the gut microbial community is strongly influenced by breast or formula milk feeding. At the time of weaning, the variety of different glycan sources increases rapidly and the gut microbial community shifts towards and adult like community. The timing of the successional waves depends strongly on the patterns of introduction of dietary components. Here, we provide estimates which can vary strongly between individuals

Mainly in HLA DR3/4 children

Figure. Islet Autoimmunity Risk by First Probiotic Exposure Age of the Child



No. at risk										
0-27 d	540	521	478	438	412	306	184	104	48	5
28-90 d	556	537	493	455	414	318	203	111	56	5
91-365 d	538	519	466	420	389	303	202	132	72	24
>365 d	5839	5660	4972	4504	4145	3426	2521	1665	965	341

Variable	No. (%) of Infants		HR (95% CI)[a]
	Developed IA (n = 601)	Did Not Develop IA (n = 6872)	
Timing of first probiotic exposure, d			
0-27	34 (5.7)	506 (7.4)	0.66 (0.45-0.96)
28-90	41 (6.8)	515 (7.5)	0.85(0.61-1.19)
91-365	57 (9.5)	481 (7.0)	1.16 (0.86-1.57)
After 1 year or no exposure	469 (78.0)	5370 (78.1)	1 [Reference]

Pathogenesis: Antibody reversal

Diabetes Care Volume 39, September 2016

Reversion of β-Cell Autoimmunity Changes Risk of Type 1 Diabetes: TEDDY Study

Diabetes Care 2016;39:1535–1542 | DOI: 10.2337/dc16-0181

*Kendra Vehik,[1] Kristian F. Lynch,[1] Desmond A. Schatz,[2] Beena Akolkar,[3] William Hagopian,[4] Marian Rewers,[5] Jin-Xiong She,[6] Olli Simell,[7] Jorma Toppari,[7] Anette-G. Ziegler,[8] Åke Lernmark,[9] Ezio Bonifacio,[10,11] Jeffrey P. Krischer,[1] and the TEDDY Study Group**

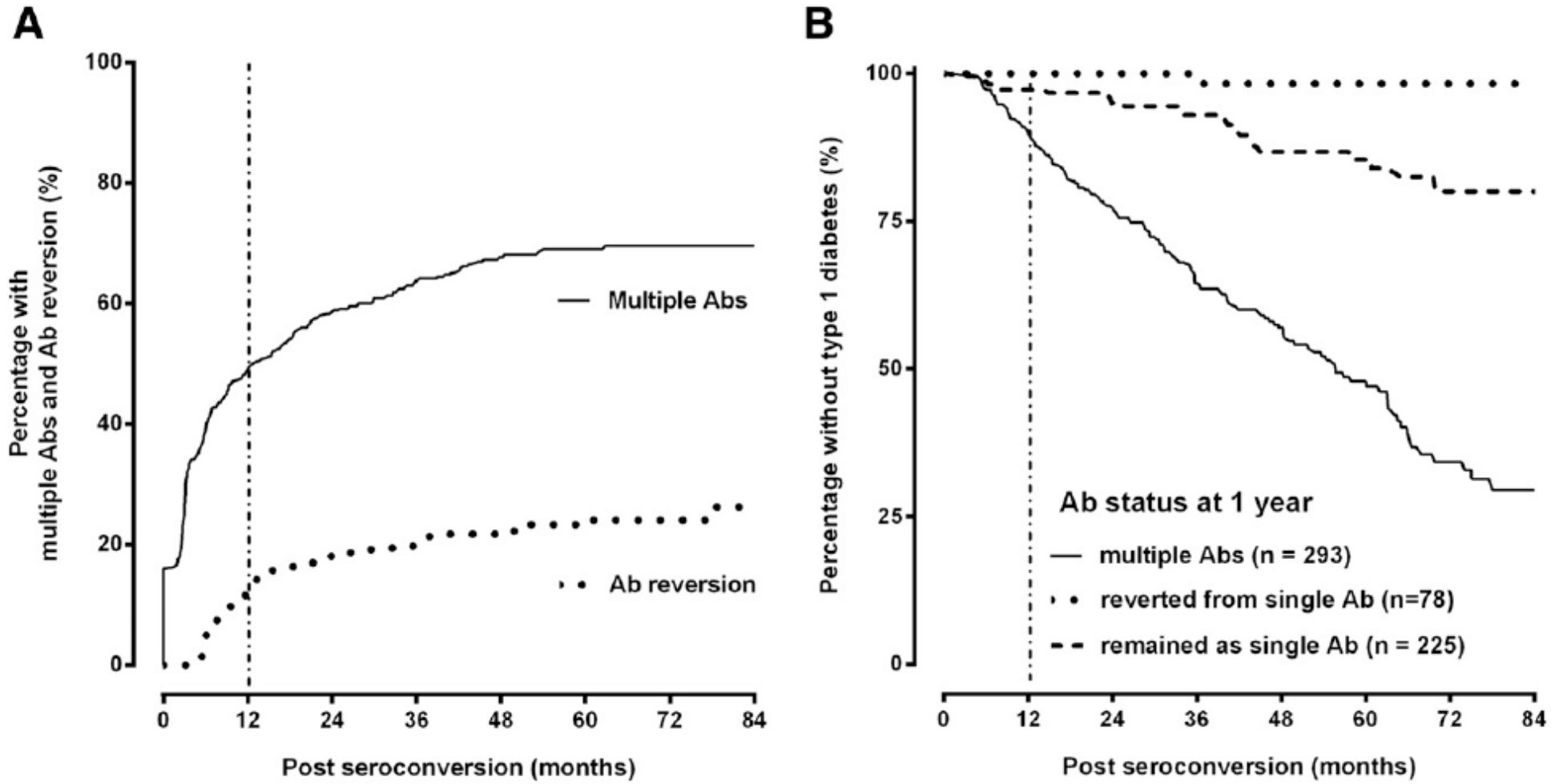


Figure 2—*A*: Cumulative incidence of development of multiple autoantibodies (Abs) after initial seroconversion and cumulative incidence of autoantibody reversion. *B*: Risk of progression to type 1 diabetes by autoantibody persistence (single and multiple) and reversion.

Reversion was relatively frequent for autoantibodies to GAD65 (19%) and insulin (29%), but was largely restricted to children who had single autoantibodies (24%) and rare in children who had developed multiple autoantibodies (<1%). Most (85%) reversion of single autoantibodies occurred within 2 years of seroconversion. Reversion was associated with HLA genotype, age, and decreasing titer. Children who reverted from single autoantibodies to autoantibody negative had, from birth, a risk for type 1 diabetes of 0.14 per 100 person-years; children who never developed autoantibodies, 0.06 per 100 person-years; and, children who remained single-autoantibody positive, 1.8 per 100 person-years.

Pathogenesis: BMI and T1D Development

Diabetes Care 2017;40:698–701 | DOI: 10.2337/dc16-2331

Excess BMI in Childhood: A Modifiable Risk Factor for Type 1 Diabetes Development?

Christine Therese Ferrara,[1] Susan Michelle Geyer,[2] Yuk-Fun Liu,[3] Carmella Evans-Molina,[4] Ingrid M. Libman,[5] Rachel Besser,[6] Dorothy J. Becker,[5] Henry Rodriguez,[2] Antoinette Moran,[7] Stephen E. Gitelman,[1] Maria J. Redondo,[8] and the Type 1 Diabetes TrialNet Study Group*

Diabetes Care 2017;40:698–701 | DOI: 10.2337/dc16-2331

1,117 Children in TrialNet Pathway to Prevention Cohort
Antibody positive relatives of T1D patients

- 1,117 Children in TrialNet Pathway to Prevention Cohort
- Antibody positive relatives of T1D patients
- ceBMI= accumulated excess BMI (≥ 85th percentile), annualized
- Higher ceBMI → increased risk of T1D, adjusting for age, #of antibody, sex
- 1 kg/m^2 increase in ceBMI → 6.3% increase RR of T1D progression
- ceBMI ≥0 → increase risk by 65%
- Threshold lower in those <12 years, and in female

Therapy: Hybrid Closed-Loop System

Day-and-Night Hybrid Closed-Loop Insulin Delivery in Adolescents With Type 1 Diabetes: A Free-Living, Randomized Clinical Trial

Diabetes Care Volume 39, July 2016

Martin Tauschmann,[1,2] Janet M. Allen,[1,2] Malgorzata E. Wilinska,[1,2] Hood Thabit,[1] Zoë Stewart,[1] Peiyao Cheng,[3] Craig Kollman,[3] Carlo L. Acerini,[2] David B. Dunger,[1,2] and Roman Hovorka[1,2]

Diabetes Care 2016;39:1168–1174 | DOI: 10.2337/dc15-2078

Table 1—Comparison of glucose control and insulin delivery during closed-loop and control period

	Closed-loop (n = 12)	Control (n = 12)	P value
Time spent at glucose level (%)			
3.9–10.0 mmol/L*	72 (59–77)	53 (46–59)	<0.001
>10.0 mmol/L	26 (21–35)	43 (38–52)	0.005
<3.9 mmol/L	2.9 (1.8–4.8)	1.7 (0.9–5.1)	0.87
<2.8 mmol/L	0.2 (0.0–0.6)	0.1 (0.0–0.6)	0.67
AUC_{day} <3.5 mmol/L (mmol/L × min)†	6.4 (2.8–23.7)	4.3 (1.8–13.6)	0.77
Mean glucose (mmol/L)	8.7 ± 1.1	10.1 ± 1.3	0.028
Within-day SD of glucose (mmol/L)	3.5 (3.3–4.2)	4.0 (3.6–4.6)	0.21
CV of glucose within day (%)	41 (40–45)	39 (38–44)	0.36
CV of glucose between days (%)	17 (11–22)	19 (17–25)	0.80
Total daily dose (units/day)	57.3 (45.6–65.2)	56.6 (44.7–61.3)	0.55
Total bolus (units/day)	31.9 (21.2–41.0)	38.3 (26.4–41.4)	0.06
Total basal (units/day)	24.3 (22.8–28.8)	20.3 (19.1–22.1)	0.001
CV of basal insulin (%)	94 (91–103)	16 (13–26)	<0.001

Data are presented as the median (interquartile range) or mean ± SD, unless otherwise indicated. *Primary end point. †AUC_{day}, glucose AUC <3.5 mmol/L/day.

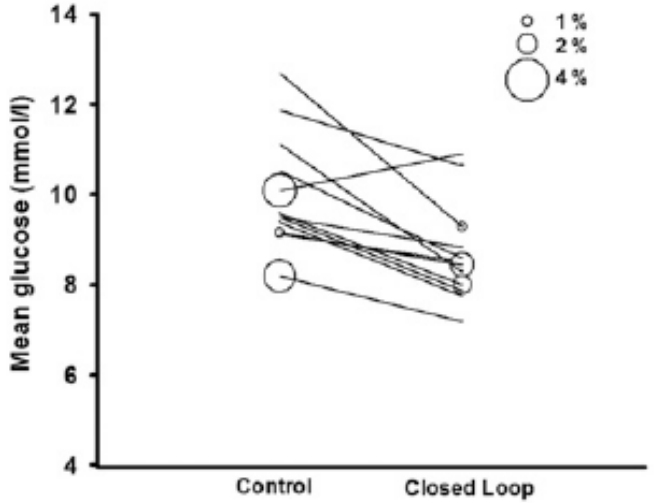
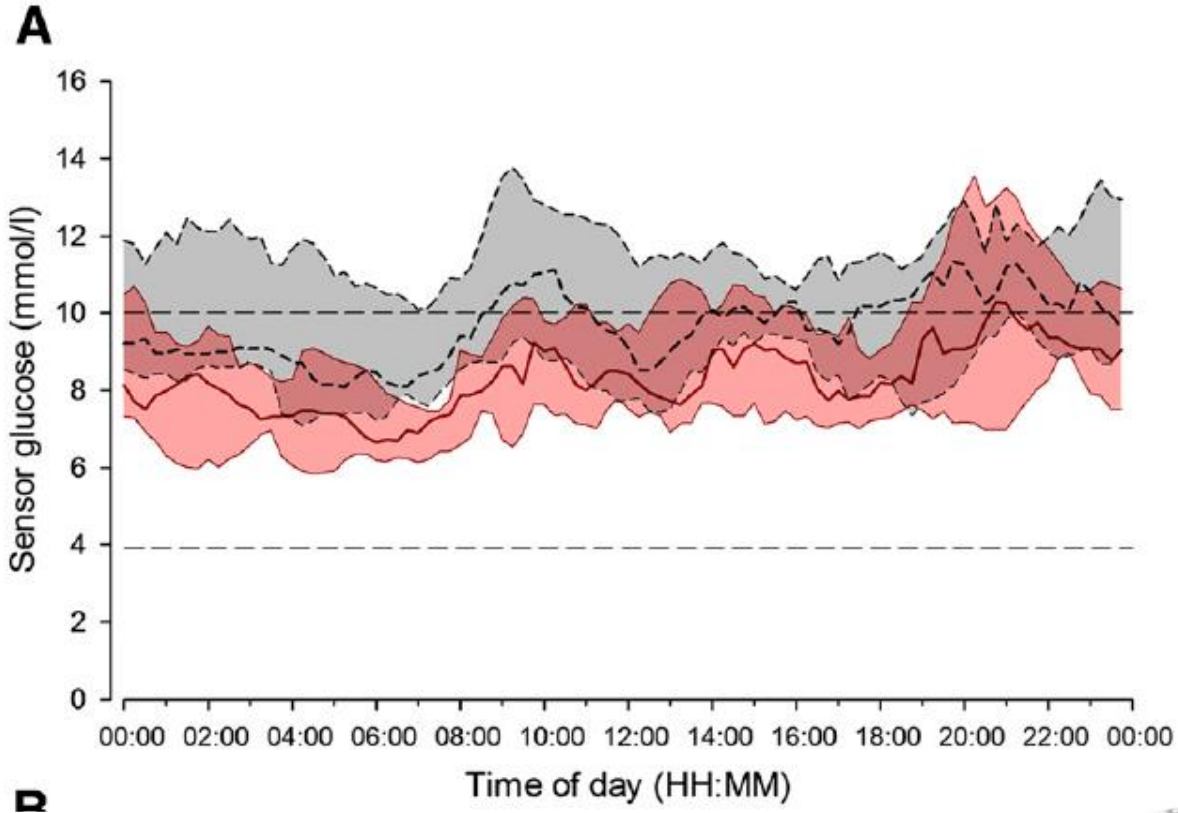
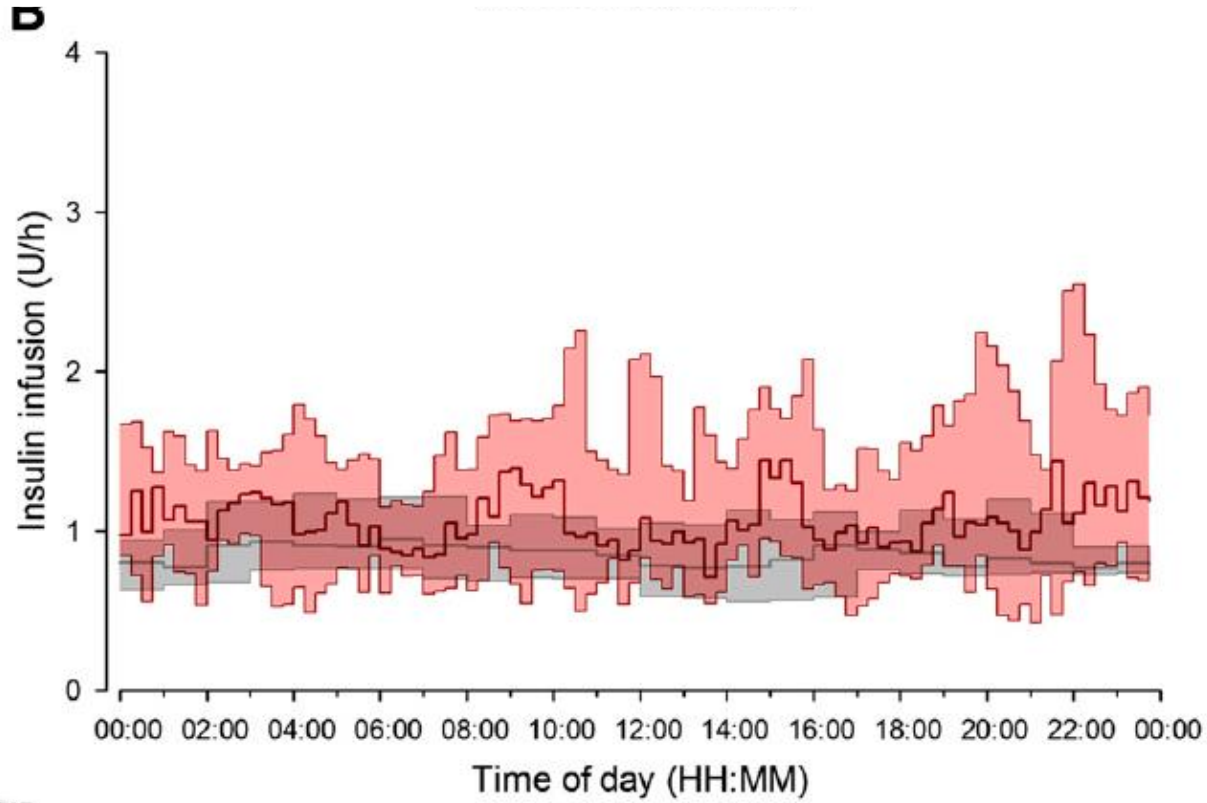


Figure 2—Individual values of mean sensor glucose levels during day-and-night closed-loop study. The size of the bubble indicates the proportion of time spent with low glucose levels <2.8 mmol/L.


A
Sensor glucose (mmol/l)
16
14
12
10
8
6
4
2
0
00:00 02:00 04:00 06:00 08:00 10:00 12:00 14:00 16:00 18:00 20:00 22:00 00:00
Time of day (HH:MM)


B
Insulin infusion (U/h)
4
3
2
1
0
00:00 02:00 04:00 06:00 08:00 10:00 12:00 14:00 16:00 18:00 20:00 22:00 00:00
Time of day (HH:MM)


5:00 PM
120
0.5 U
Act. Insulin
118
Bolus
Reminder
Notes

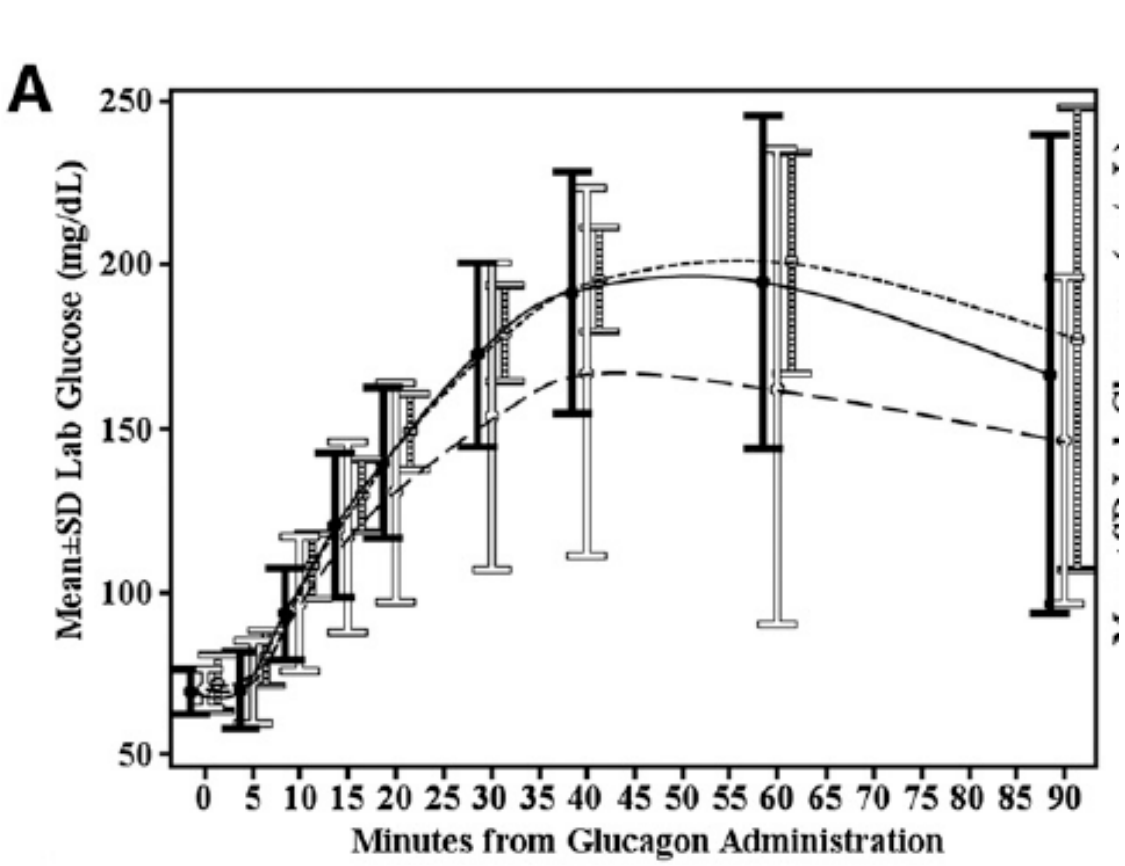
Therapy: Glucagon Nasal Powder

Diabetes Care Volume 39, April 2016

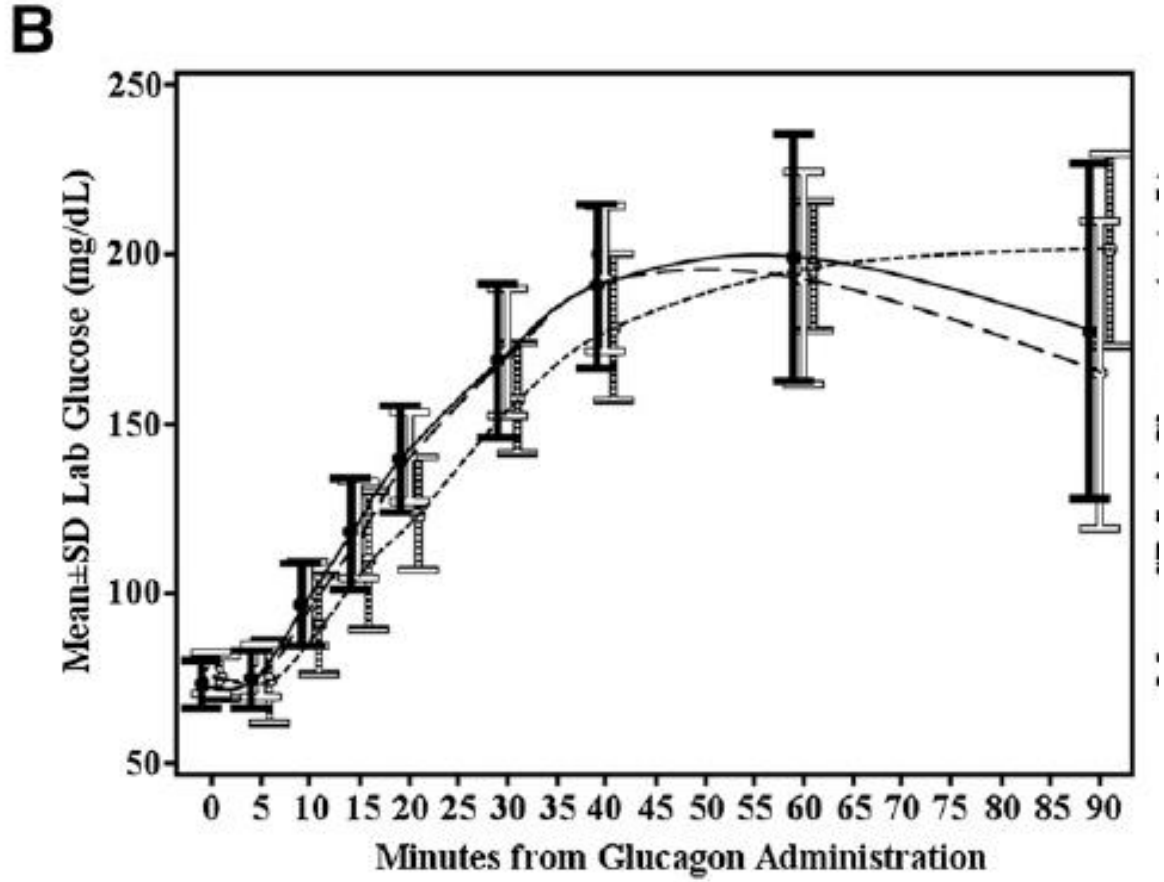
*Jennifer L. Sherr,[1] Katrina J. Ruedy,[2] Nicole C. Foster,[2] Claude A. Piché,[3] Hélène Dulude,[3] Michael R. Rickels,[4] William V. Tamborlane,[1] Kathleen E. Bethin,[5] Linda A. DiMeglio,[6] Larry A. Fox,[7] R. Paul Wadwa,[8] Desmond A. Schatz,[9] Brandon M. Nathan,[10] Santica M. Marcovina,[11] Emmanouil Rampakakis,[12] Linyan Meng,[12] and Roy W. Beck,[2] for the T1D Exchange Intranasal Glucagon Investigators**

Glucagon Nasal Powder: A Promising Alternative to Intramuscular Glucagon in Youth With Type 1 Diabetes

Diabetes Care 2016;39:555–562 | DOI: 10.2337/dc15-1606



4- <8 year old



8- <12 year old

_____ 3 mg intranasal
__ __ __ 2 mg intranasal
_ _ _ _ _ intramuscular

Therapy: Fast Acting Aspart

Fast-Acting Insulin Aspart Improves Glycemic Control in Basal-Bolus Treatment for Type 1 Diabetes: Results of a 26-Week Multicenter, Active-Controlled, Treat-to-Target, Randomized, Parallel-Group Trial (Onset 1)

https://doi.org/10.2337/dc16-1771

David Russell-Jones,[1] Bruce W. Bode,[2] Christophe De Block,[3] Edward Franek,[4] Simon R. Heller,[5] Chantal Mathieu,[6] Athena Philis-Tsimikas,[7] Ludger Rose,[8] Vincent C. Woo,[9] Anne Birk Østerskov,[10] Tina Graungaard,[10] and Richard M. Bergenstal[11]

https://doi.org/10.2337/dc16-1771

26 weeks:→Faster Aspart: Iasp: Postmeal faster aspart = 381:380:382

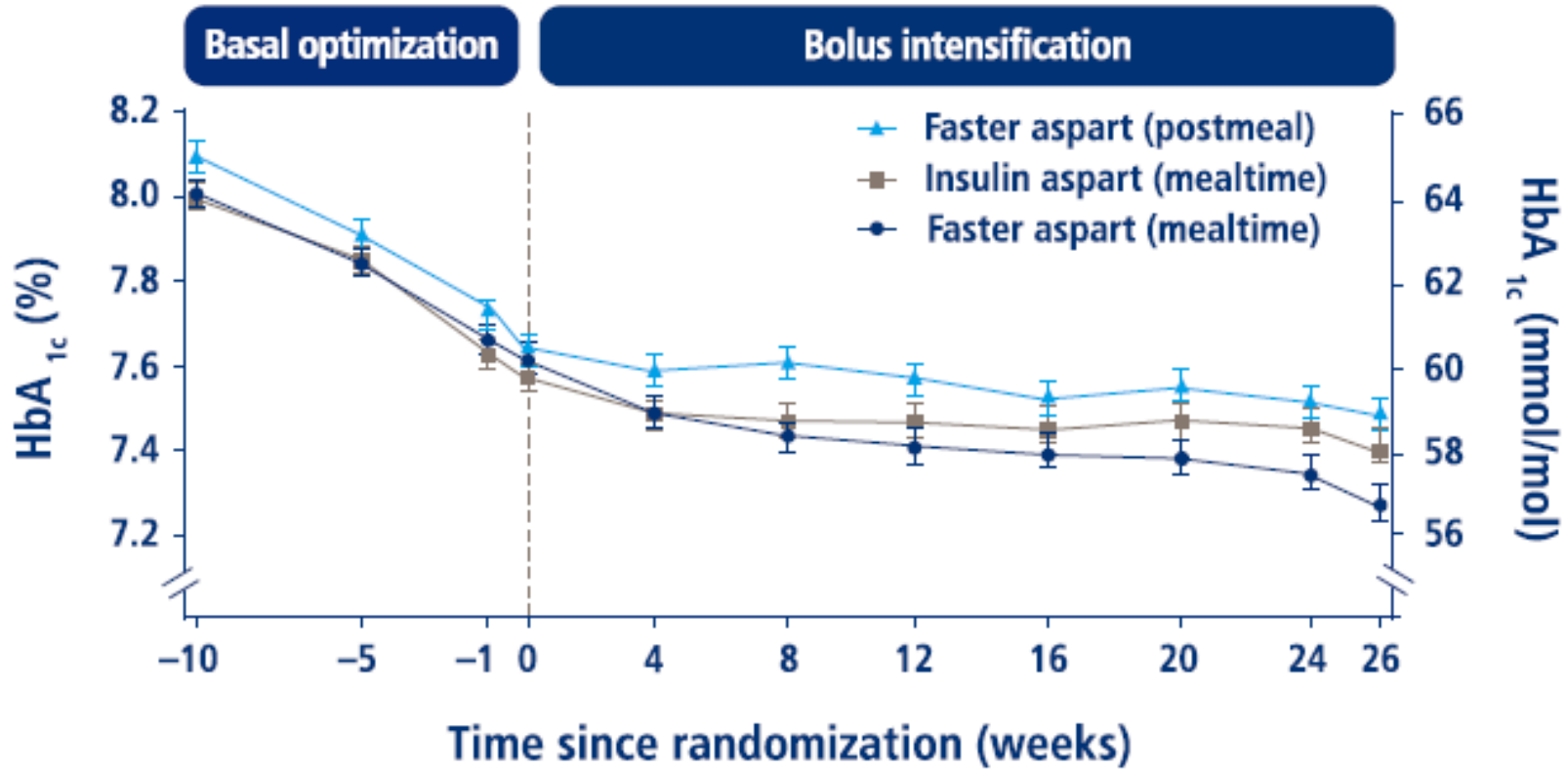
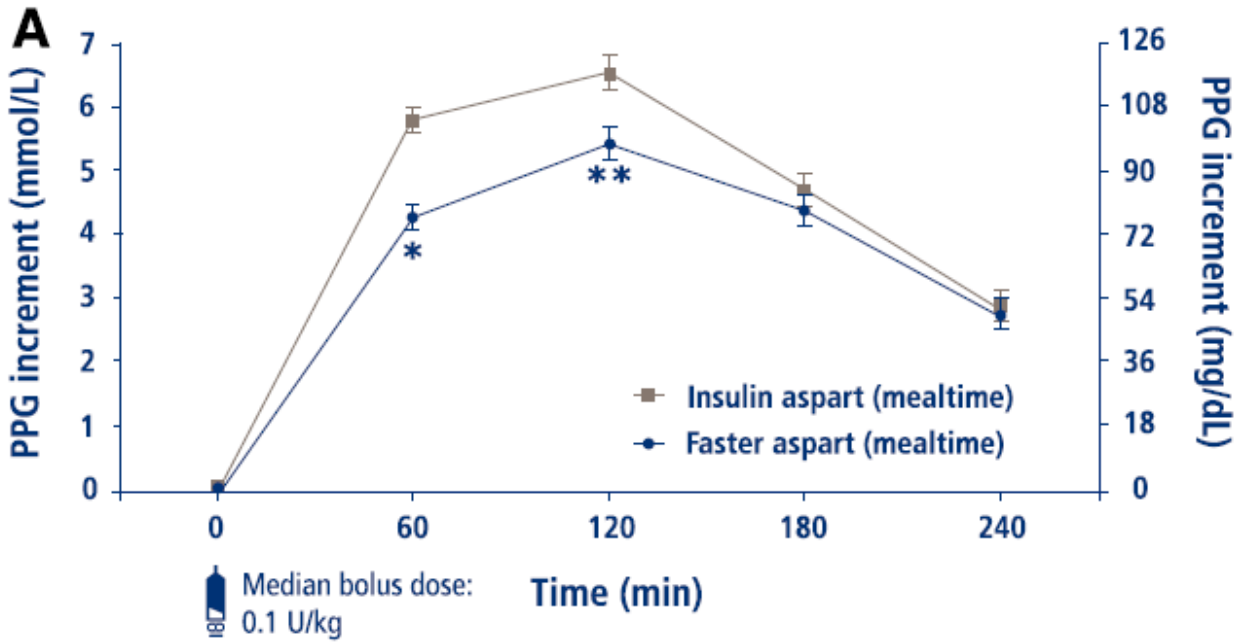
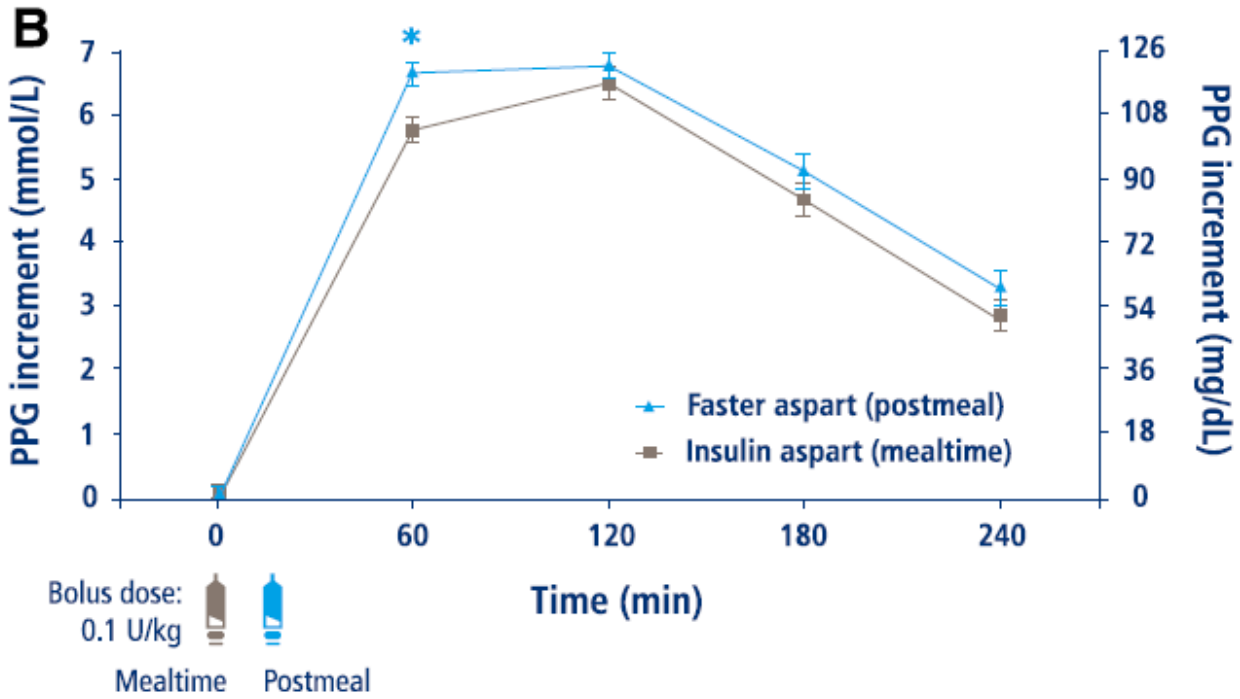


Figure 1—Mean HbA_{1c} over time. During run-in, observed mean HbA_{1c} was reduced from 8.1% (64.9 mmol/mol) to 7.6% (59.9 mmol/mol) for subjects subsequently randomized to receive postmeal faster aspart (n = 382), from 8.0% (64.0 mmol/mol) to 7.6% (59.7 mmol/mol) for subjects subsequently randomized to receive mealtime insulin aspart (n = 380), and from 8.0% (64.0 mmol/mol) to 7.6% (59.3 mmol/mol) for subjects subsequently randomized to receive mealtime faster aspart (n = 381). During the 26-week treatment period, the observed mean HbA_{1c} decreased to 7.5% (58.6 mmol/mol) with postmeal faster aspart, 7.4% (57.6 mmol/mol) with mealtime insulin aspart, and 7.3% (56.4 mmol/mol) with mealtime faster aspart. Error bars: ±SEM.





- Faster aspart improved HbA1c, non-inferior to IAsp
- Superior PPG control
- Postmeal faster aspart was non-inferior to IAsp

Complications: Hypoglycemia

Diurnal Differences in Risk of Cardiac Arrhythmias During Spontaneous Hypoglycemia in Young People With Type 1 Diabetes

Diabetes Care 2017;40:655–662 | DOI: 10.2337/dc16-2177

Diabetes Care 2017;40:655–662 | DOI: 10.2337/dc16-2177

Peter Novodvorsky,[1,2] Alan Bernjak,[1,3]
Elaine Chow,[2,4] Ahmed Iqbal,[1,2,4]
Lianne Sellors,[1,2] Scott Williams,[1,2]
Robert A. Fawdry,[1,2] Bhavin Parekh,[5]
Richard M. Jacques,[6]
Jefferson L.B. Marques,[1]
Paul J. Sheridan,[2,4] and Simon R. Heller[1,2]

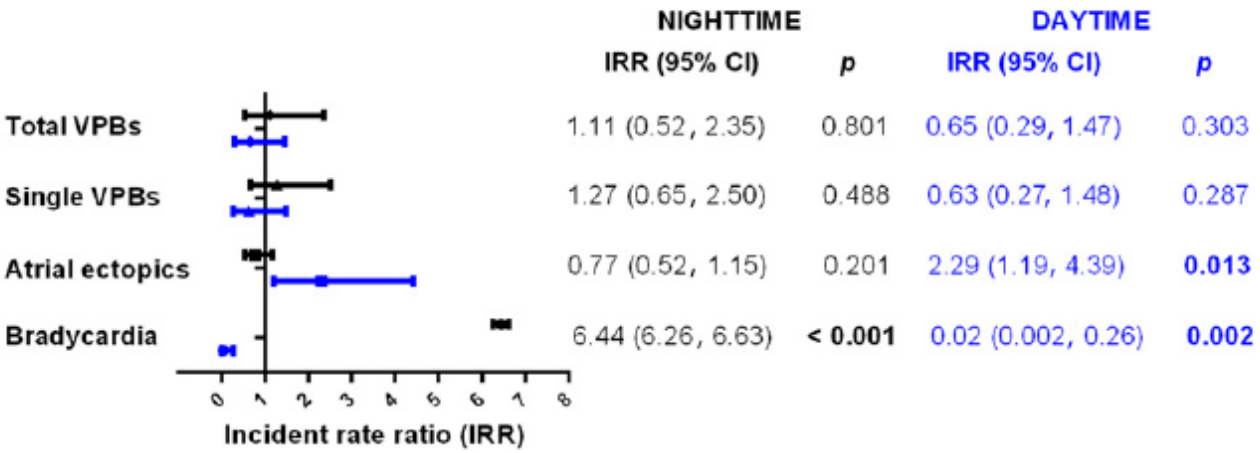


Figure 1—IRRs of distinct types of arrhythmias during hypoglycemia vs. euglycemia. Comparison between nocturnal (2300–0700 h) and daytime episodes. No complex ventricular paroxysmal beats (VPBs) were detected during nocturnal hypoglycemia (see also Table 3), and therefore no IRRs could be calculated for this type of arrhythmia. *P* values indicate significance of difference in arrhythmia rates during hypoglycemia vs. euglycemia. Values in boldface type indicate statistical significance.

- 37 T1D
- 2,395 Hr of ECG and CGM recordings
- 159 Hr of hypoglycemia
- 24% of nocturnal and 51% of daytime were symptomatic
- Nighttime duration was longer 60 vs 44 min
- During nighttime- bradycardia was more common
- Daytime- more atrial ectopy
- Hypoglycemia – both day and night- was associated with prolonged QTc and abnormal T wave

Complications: Musculoskeletal

Diabetes Care 2014;37:1863–1869 | DOI: 10.2337/dc13-2361

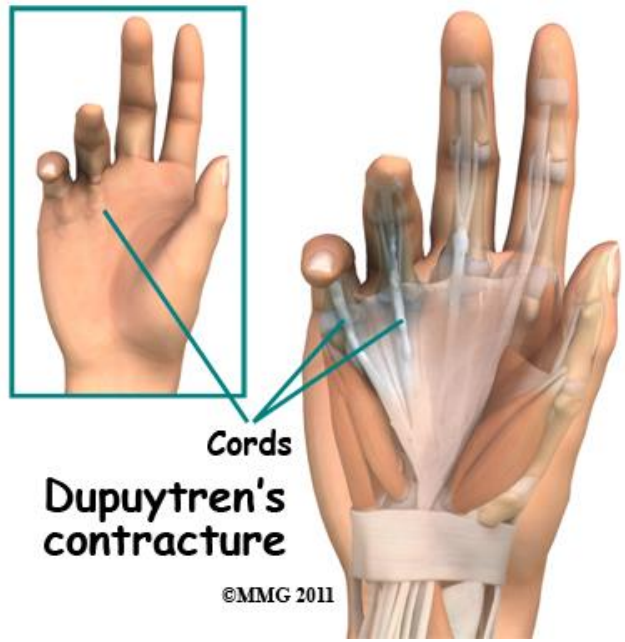
Musculoskeletal Complications in Type 1 Diabetes

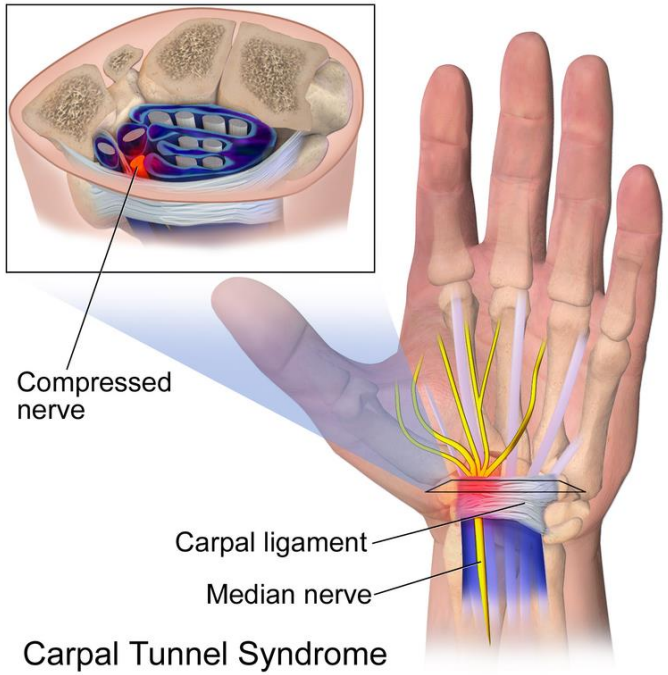
Diabetes Care 2014;37:1863–1869 | DOI: 10.2337/dc13-2361

Mary E. Larkin,[1] Annette Barnie,[2] Barbara H. Braffett,[3] Patricia A. Cleary,[3] Lisa Diminick,[3] Judy Harth,[4] Patricia Gatcomb,[5] Ellen Golden,[6] Janie Lipps,[7] Gayle Lorenzi,[8] Carol Mahony,[1] David M. Nathan,[1] and the Diabetes Control and Complications Trial/ Epidemiology of Diabetes Interventions and Complications Research Group*

1,217 Patients in EDIC cohort at 18/19 years of follow up (of 23 years)

- **<u>Cheiroarthropathy</u>**
- **<u>Periarticular thickening of skins and limited joint movement</u>**
- Adhesive capsulitis
- Carpal tunnel syndrome
- Flexor tenosynovitis
- Positive prayer sign
- Dupuytren’s contracture





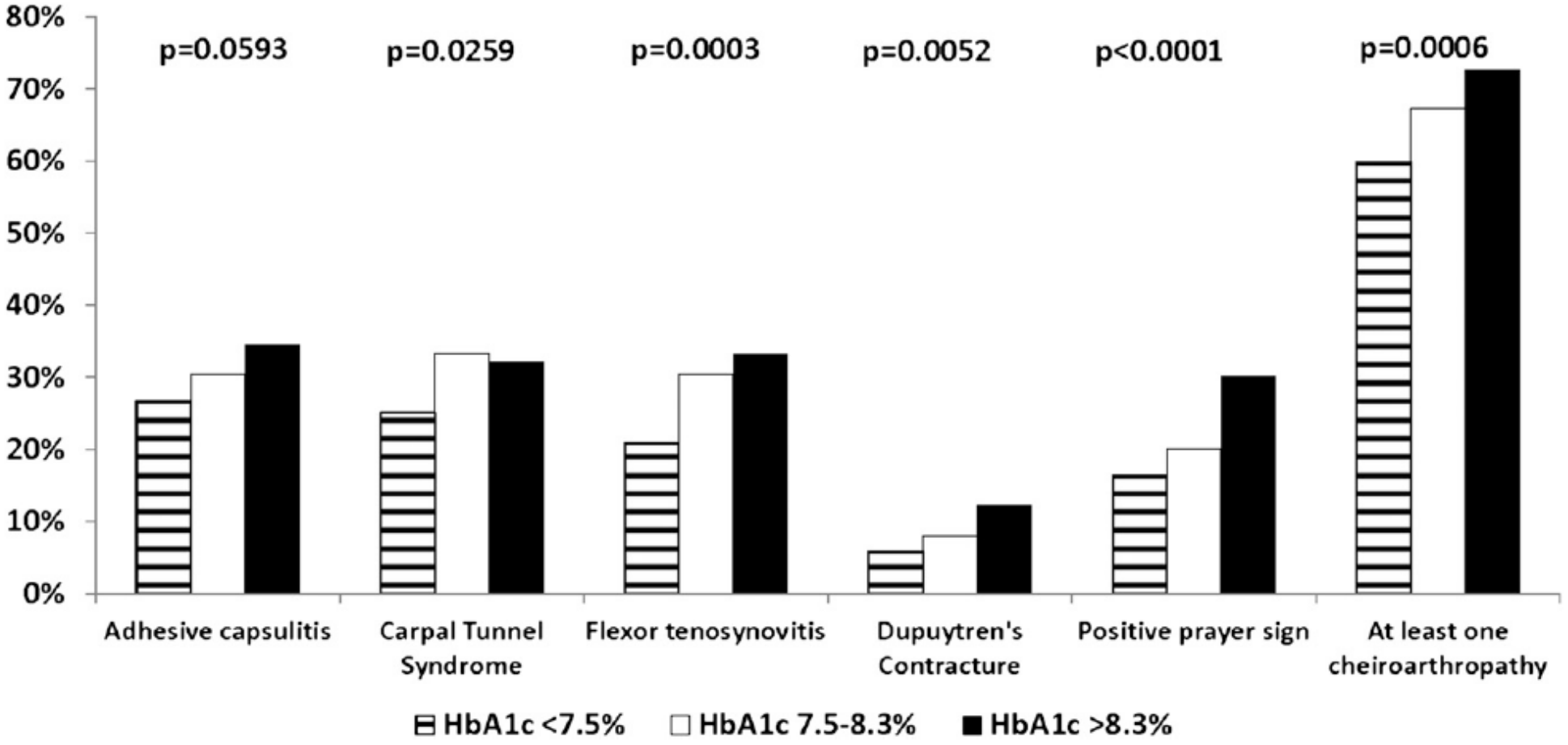


Figure 1—Association of prevalence of cheiroarthropathy by tertiles of time-weighted HbA_{1c} during the DCCT/EDIC (1983–2011). Subjects could report more than one type of cheiroarthropathy. The *P* values estimate the HbA_{1c} group differences calculated using the contingency χ^2 test for categorical variables. Twenty subjects were missing an HbA_{1c} measurement at EDIC year 18.

Cheiroarthropathy= found in 66%

Female, older age, longer DM duration, retinopathy and neuropathy

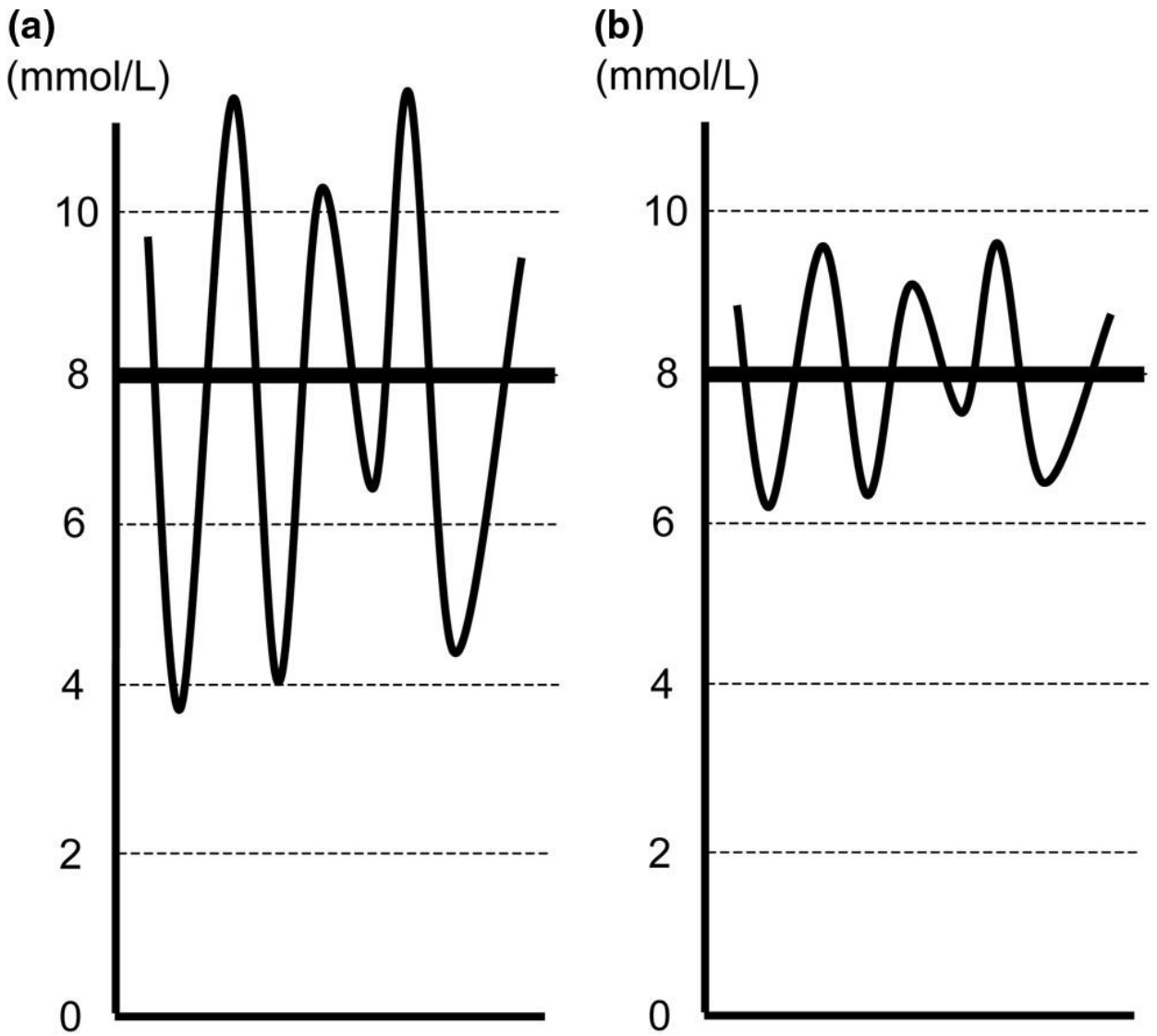
Complications: Glycemic Variability

Association of Glycemic Variability in Type 1 Diabetes With Progression of Microvascular Outcomes in the Diabetes Control and Complications Trial

DOI: 10.2337/dc16-2426

DOI: 10.2337/dc16-2426

John M. Lachin,[1] Ionut Bebu,[1] Richard M Bergenstal,[2] Rodica Pop-Busui,[3] F. John Service,[4] Bernard Zinman,[5] and David M. Nathan,[6] for the DCCT/EDIC Research Group*



- Standard deviation (SD)
- Coefficient of Variation (CV)
- MAGE
- M Value

Table 2—Association of measures of glucose variability over a mean of 6.5 years of quarterly follow-up in the DCCT*

	Adjusted for mean blood glucose*			
	Hazard ratio	95% CL	*Z* value	*P* value†
Retinopathy				
Within-day				
SD	0.937	0.834, 1.054	−1.08	0.28
MAGE	0.938	0.837, 1.050	−1.11	0.27
M-value	0.804	0.582, 1.112	−1.32	0.19
Longitudinal				
Total blood glucose variance	0.951	0.844, 1.072	−0.83	0.41
Between-day variance	0.920	0.839, 1.009	−1.76	0.08
Within-day variance	0.970	0.872, 1.080	−0.55	0.59
Mean MAGE	0.966	0.853, 1.095	−0.54	0.60
Mean M-value	0.972	0.792, 1.191	−0.28	0.79
Microalbuminuria				
Within-day				
SD	1.021	0.842, 1.238	0.21	0.84
MAGE	1.01	0.834, 1.213	0.062	0.96
M-value	0.899	0.517, 1.564	−0.38	0.71
Longitudinal				
Total blood glucose variance	1.084	0.838, 1.401	0.61	0.54
Between-day variance	1.132	0.999, 1.283	1.95	0.06
Within-day variance	0.904	0.698, 1.172	−0.76	0.45
Mean MAGE	0.812	0.621, 1.062	−1.52	0.13
Mean M-value	2.142	1.505, 3.048	4.23	<0.0001

	Odds ratio			
Cardiovascular autonomic neuropathy				
Within-day				
SD	1.098	0.952, 1.268	1.29	0.20
MAGE	1.138	0.999, 1.298	1.93	0.06
M-value	1.336	0.953, 1.874	1.68	0.10
Longitudinal				
Total blood glucose variance	1.357	1.114, 1.655	3.03	0.0025
Between-day variance	1.221	1.052, 1.416	2.63	0.0087
Within-day variance	1.132	0.946, 1.355	1.35	0.18
Mean MAGE	1.155	0.925, 1.444	1.27	0.21
Mean M-value	1.011	0.690, 1.483	0.06	0.96

Complications: Cardiovascular Outcomes

The Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications Study at 30 Years: Overview

*David M. Nathan, for the DCCT/EDIC Research Group**

Diabetes Care Volume 37, January 2014

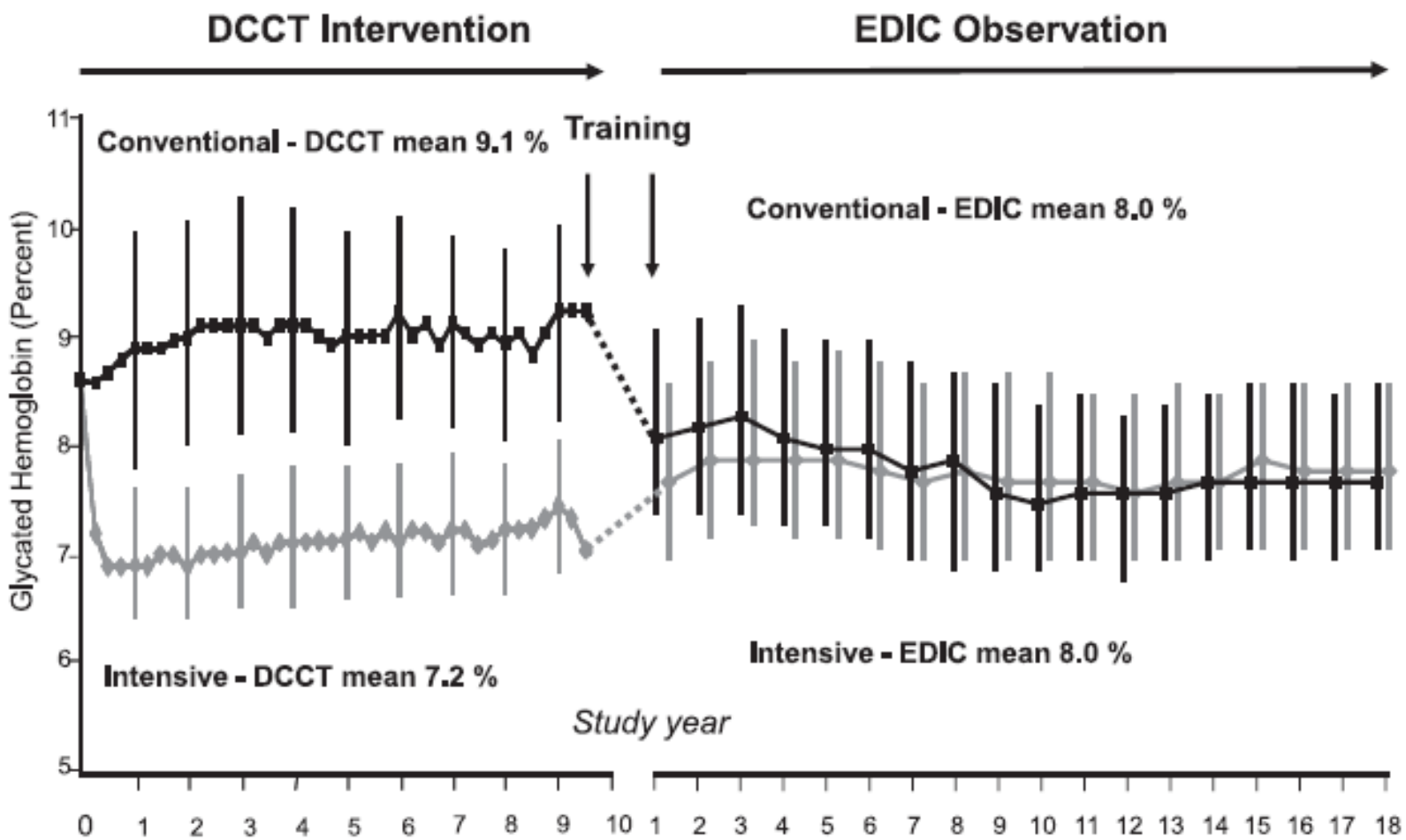


Figure 1—Median HbA_{1c} concentrations during DCCT, the "training" period between DCCT and EDIC, and EDIC. $P < 0.001$ for INT vs. CON during entire DCCT and for the first 3 years during EDIC. Reprinted and modified with permission from Nathan et al. Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications study at 30 years: advances and contributions. Diabetes 2013;62:3976–3986.

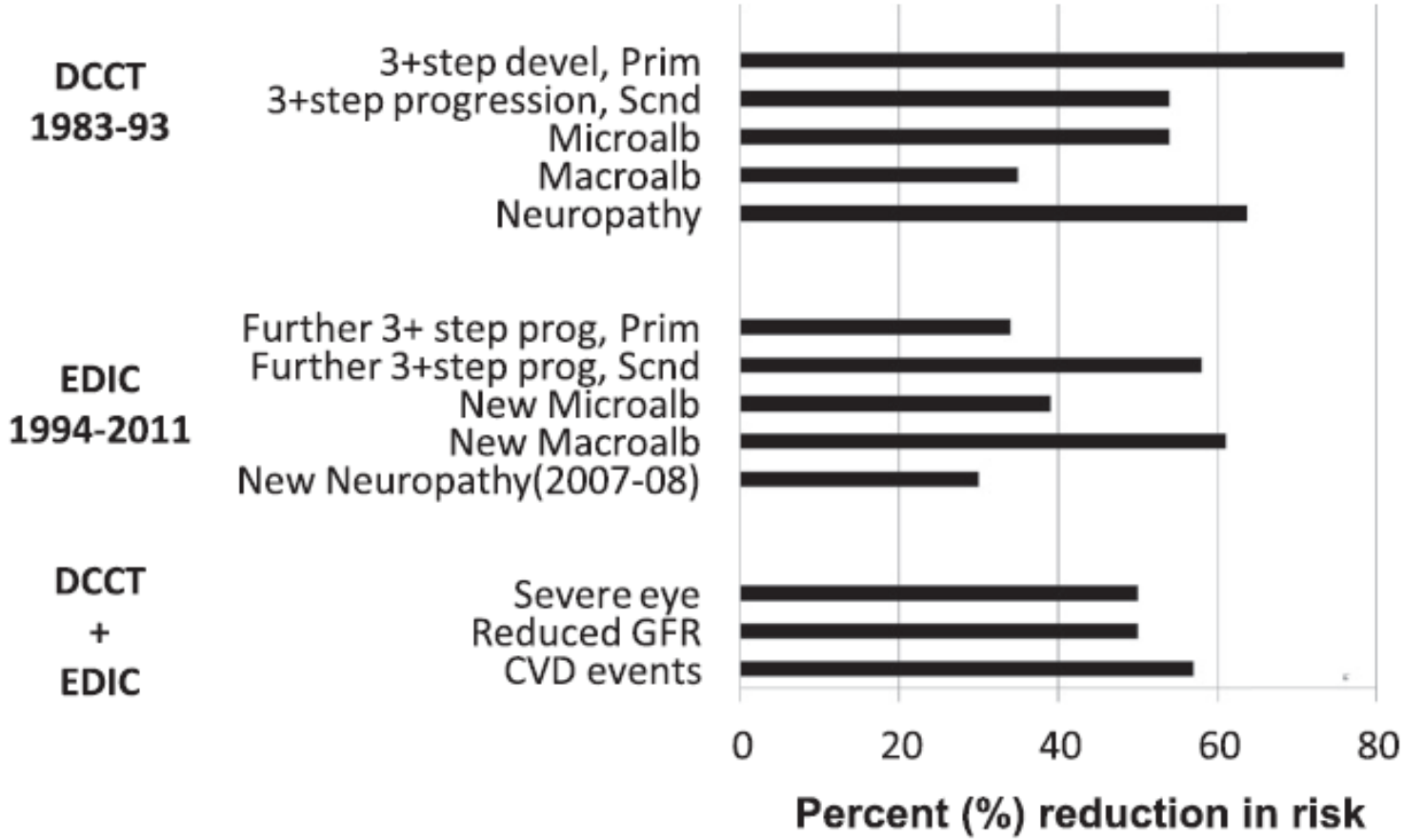


Figure 2—Summary of reduction in major complications with INT compared with CON during DCCT, EDIC, and combined study periods. 3+step devel, Prim: three-step or more development of retinopathy based on Early Treatment of Diabetic Retinopathy scale (ref. 13) in the primary prevention group. Scnd: secondary intervention group. Microalb: microalbuminuria defined as albumin excretion ≥40 mg/24 h. Macroalb: macroalbuminuria defined as albumin excretion >300 mg/24 h. Reduced GFR: estimated GFR <60 mL/min/1.73 m^2. CVD events: CVD including myocardial infarctions, stroke, and CVD death. Reprinted with permission from Nathan et al. Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications study at 30 years: advances and contributions. Diabetes 2013;62:3976–3986.

Complications: Mortality

Diabetes Care 2016;39:1378–1383 | DOI: 10.2337/dc15-2399

Mortality in Type 1 Diabetes in the DCCT/EDIC Versus the General Population

*The Diabetes Control and Complications Trial (DCCT)/Epidemiology of Diabetes Interventions and Complications (EDIC) Study Research Group**

Diabetes Care 2016;39:1378–1383 | DOI: 10.2337/dc15-2399

Table 1—DCCT/EDIC deaths and death rates by DCCT intensive versus conventional therapy group, primary versus secondary cohort, and sex, with SMRs relative to the U.S. population, along with RMRs comparing two SMRs

	Observed/expected*	Rate (95% CI)†	SMR (95% CI)‡	RMR (95% CI)§	*P*
Total (*n* = 1,441)	125/114	320 (269, 380)	1.09 (0.92, 1.30)		
Intensive (*n* = 711)	51/58	263 (200, 345)	0.88 (0.67, 1.16)	1.49 (1.04, 2.14)	0.028
Conventional (*n* = 730)	74/56	376 (301, 470)	1.31 (1.05, 1.65)		

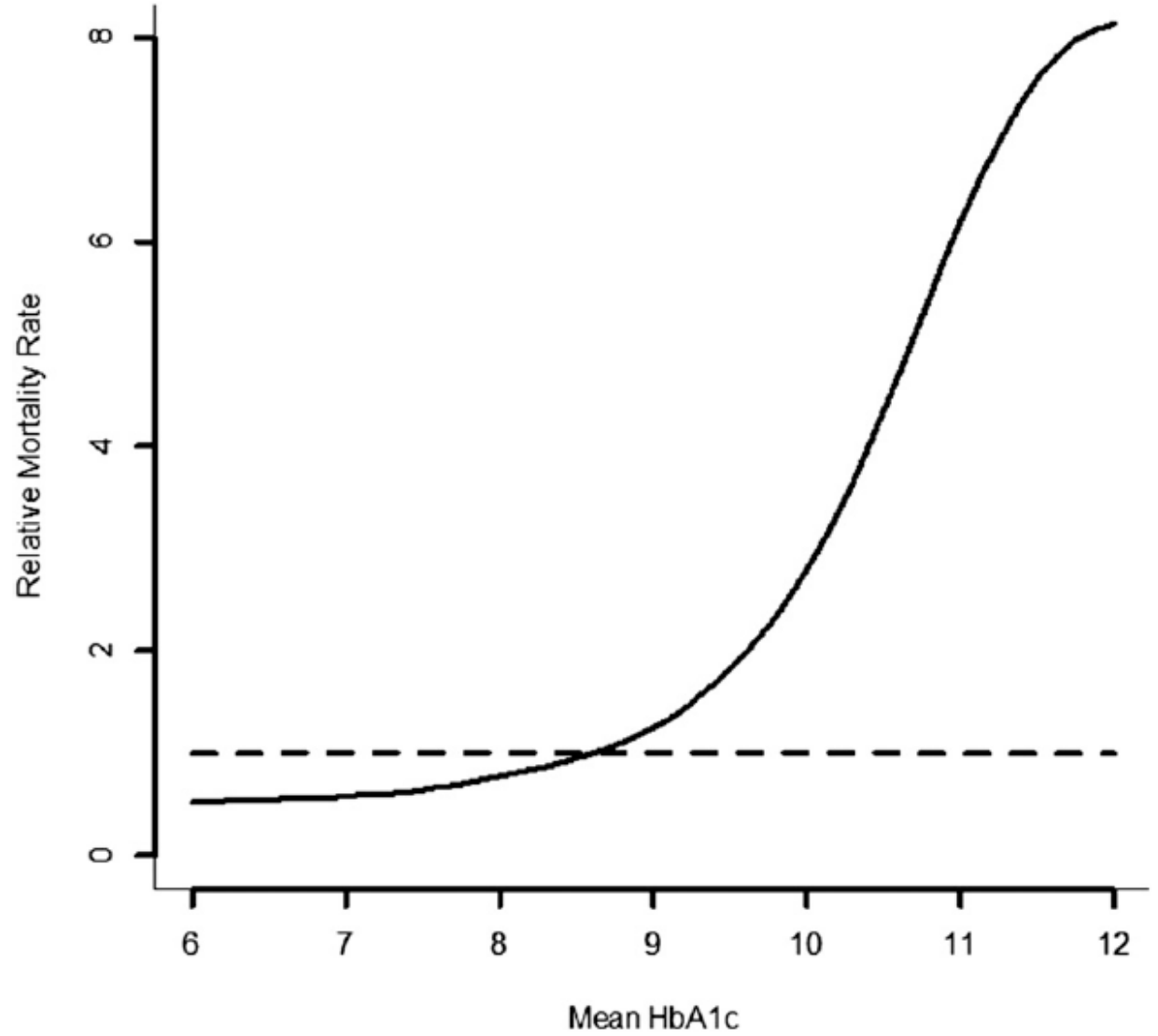


Figure 1—The RMR for the mortality in the combined DCCT/EDIC cohort relative to the age-, sex-, and race-specific risk in the general population as a function of the updated time-dependent mean HbA_{1c} during the DCCT and EDIC from a Poisson regression model. The horizontal dashed line at an RMR of 1.0 represents no difference in risk relative to the general population.

Education and Self-Care: 1000 Steps

Diabetes Care 2016;39:e108–e109 | DOI: 10.2337/dc16-0526

An Extra 1,000 Steps Per Day Relates to Improved Cardiovascular Health in Children With Type 1 Diabetes

Diabetes Care 2016;39:e108–e109 | DOI: 10.2337/dc16-0526

Jemma Anderson,[1,2]
Jennifer J. Couper,[1,2]
Christine Mpundu-Kaambwa,[3]
Lynne C. Giles,[4] Roger Gent,[5]
Brian Coppin,[6] and Alexia S. Peña[1,2]

N=90
One weekend monitoring
aIMT= aortic intima media thickness

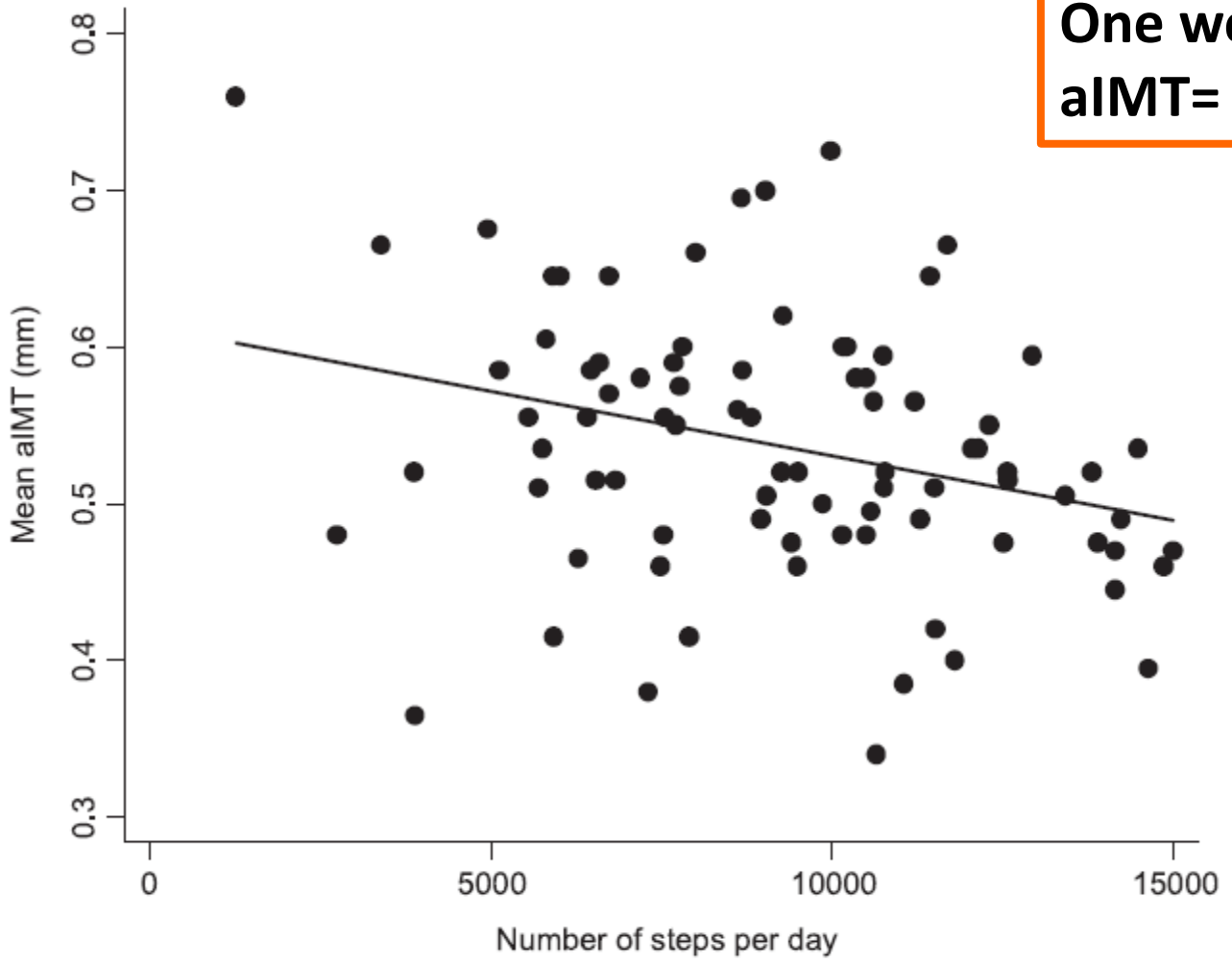


Figure 1—Mean aIMT related to average steps taken per day ($r = -0.30$, $P = 0.005$) in children with type 1 diabetes.

Education and Self-Care: GoCARB

Diabetes Care Volume 40, February 2017

Carbohydrate Estimation Supported by the GoCARB System in Individuals With Type 1 Diabetes: A Randomized Prospective Pilot Study

Diabetes Care 2017;40:e6–e7 | DOI: 10.2337/dc16-2173

Lia Bally,[1] Joachim Dehais,[2] Christos T. Nakas,[3,4] Marios Anthimopoulos,[2,5] Markus Laimer,[1] Daniel Rhyner,[2] George Rosenberg,[1] Thomas Zueger,[1] Peter Diem,[1] Stavroula Mougiakakou,[2] and Christoph Stettler[1]

- Smartphone application
- Estimated carbohydrate content from photos of plated meals
- 1 week trial

Table 1—CGM and insulin end points

	GoCARB group ($n = 20$)	Control group ($n = 20$)	*P* value
% Time hyperglycemic (>12 mmol/L)	15.0 ± 2.0	18.2 ± 2.1	0.039
% Time hypoglycemic (<3.5 mmol/L)	2.3 ± 0.8	2.6 ± 0.7	0.58
% Time in target (3.9–10 mmol/L)	65.9 ± 2.7	63.2 ± 2.8	0.19
180-min postprandial iAUC (mmol/L/min)	205.9 ± 29.3	269.9 ± 39.8	0.13
Mean glucose (mmol/L)	8.7 ± 0.3	8.9 ± 0.3	0.15
Glucose standard deviation (mmol/L)	3.0 ± 0.1	3.2 ± 0.2	0.007
Daily bolus insulin (units/24 h)	27.5 ± 2.3	30.0 ± 2.3	0.11
Number of boluses (*n*/24 h)	6.8 ± 0.4	7.3 ± 0.5	0.12
Total daily insulin (units/24 h)	47.5 ± 3.2	50.0 ± 3.2	0.14

Data presented as mean ± SEM. iAUC, incremental area under the sensor glucose curve.

Education and Self-Care: Mealtime Insulin

Optimized Mealtime Insulin Dosing for Fat and Protein in Type 1 Diabetes: Application of a Model-Based Approach to Derive Insulin Doses for Open-Loop Diabetes Management

Kirstine J. Bell,[1,2] Elena Toschi,[2,3] Garry M. Steil,[3,4] and Howard A. Wolpert[2,3]

Diabetes Care 2016;39:1631–1634 | DOI: 10.2337/dc15-2855

Diabetes Care 2016;39:1631–1634 | DOI: 10.2337/dc15-2855

50GM Carb
LFLP- 4g of fat; 9 gm protein
HFHP- 44 gm of fat, 36 gm protein

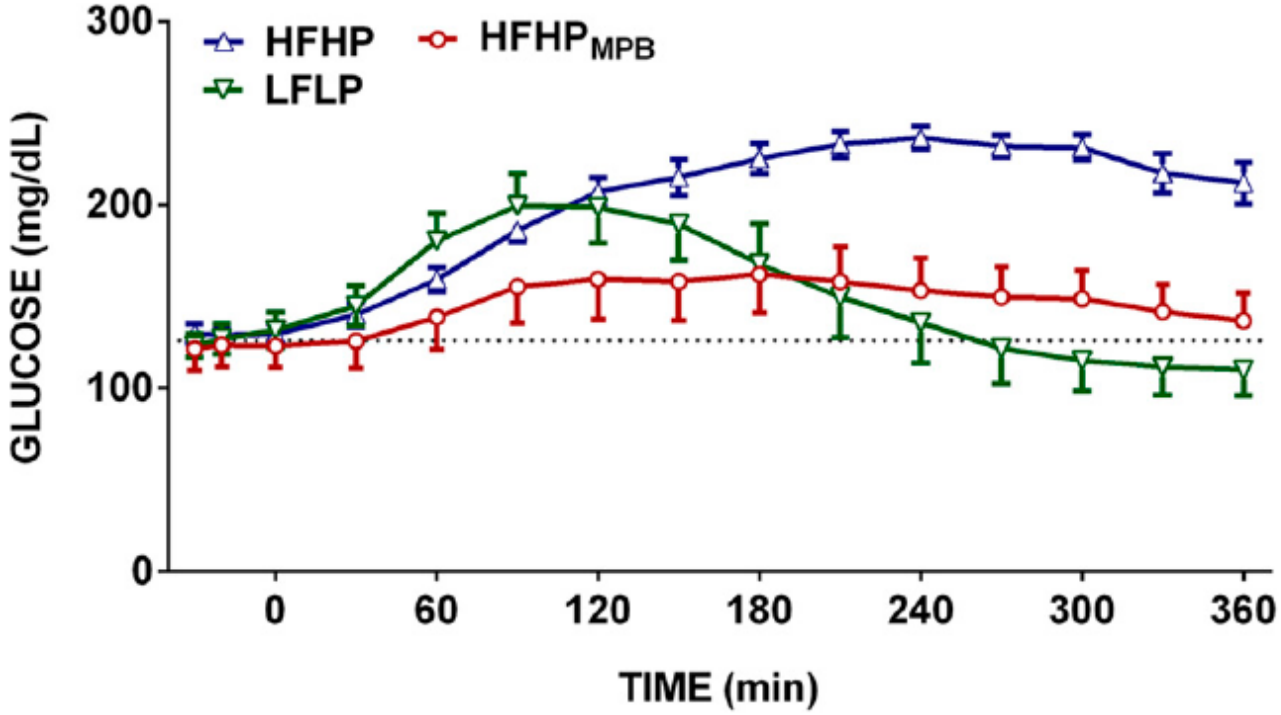


Figure 1—Postprandial plasma glucose response following LFLP and HFHP meals with identical carbohydrate content and insulin dose and an HFHP meal with optimal MPB (HFHP$_{MPB}$).

With the same insulin dose, the HFHP increased the glucose incremental area under the curve over twofold (13,320 ± 2,960 vs. 27,092 ± 1,709 mg/dL · min; *P* = 0.0013). To achieve target glucose control following the HFHP, 65% more insulin was required (range 17%–124%) with a 30%/70% split over 2.4 h.

Education and Self-Care: Health Care Transition

Health Care Transition Preparation and Experiences in a U.S. National Sample of Young Adults With Type 1 Diabetes

Diabetes Care 2017;40:317–324 | DOI: 10.2337/dc16-1729

Katharine C. Garvey,[1] Nicole C. Foster,[2] Shivani Agarwal,[3] Linda A. DiMeglio,[4] Barbara J. Anderson,[5] Sarah D. Corathers,[6] Marisa E. Desimone,[7] Ingrid M. Libman,[8] Sarah K. Lyons,[5] Anne L. Peters,[9] Jennifer K. Raymond,[10] and Lori M. Laffel[11]

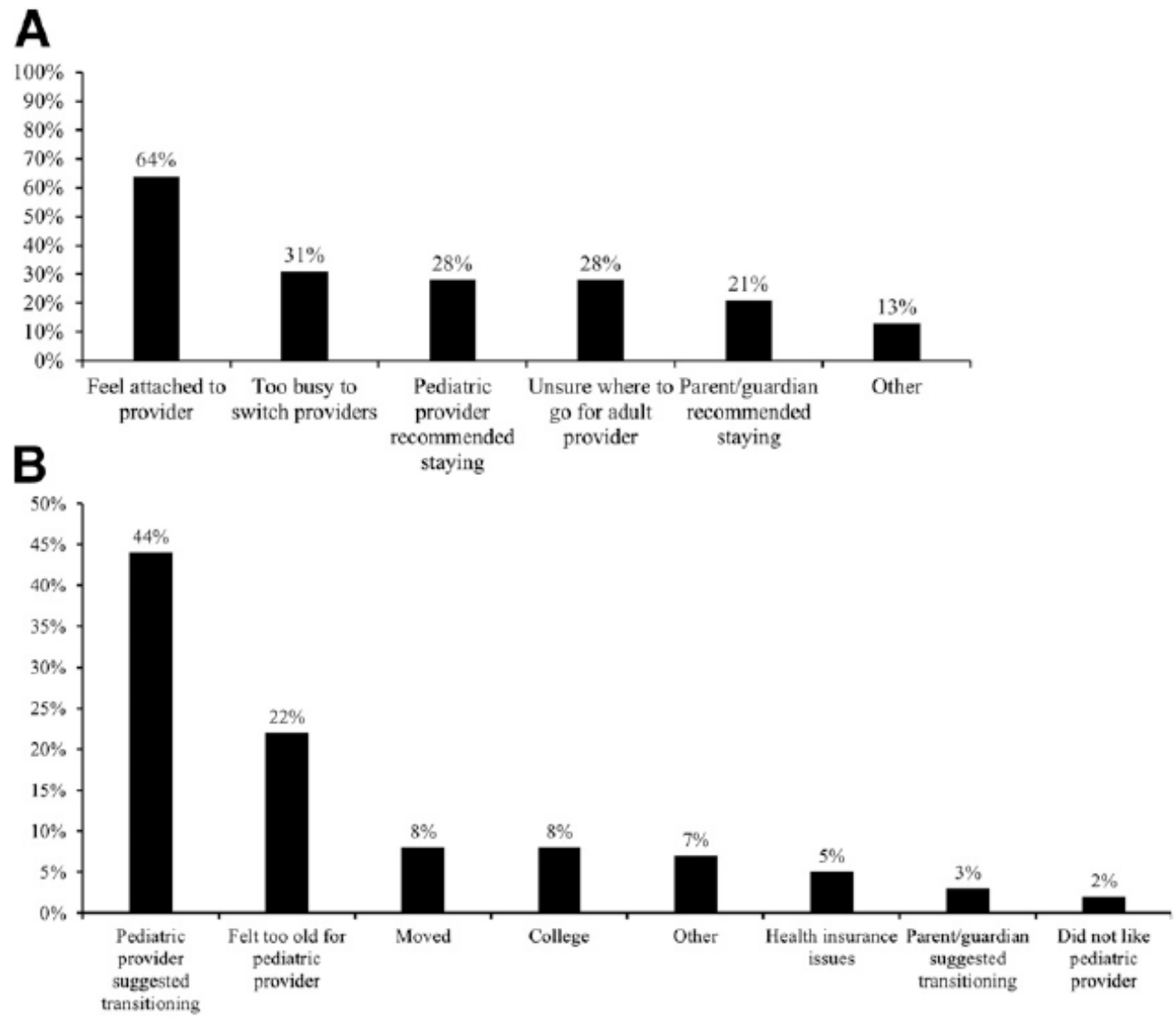
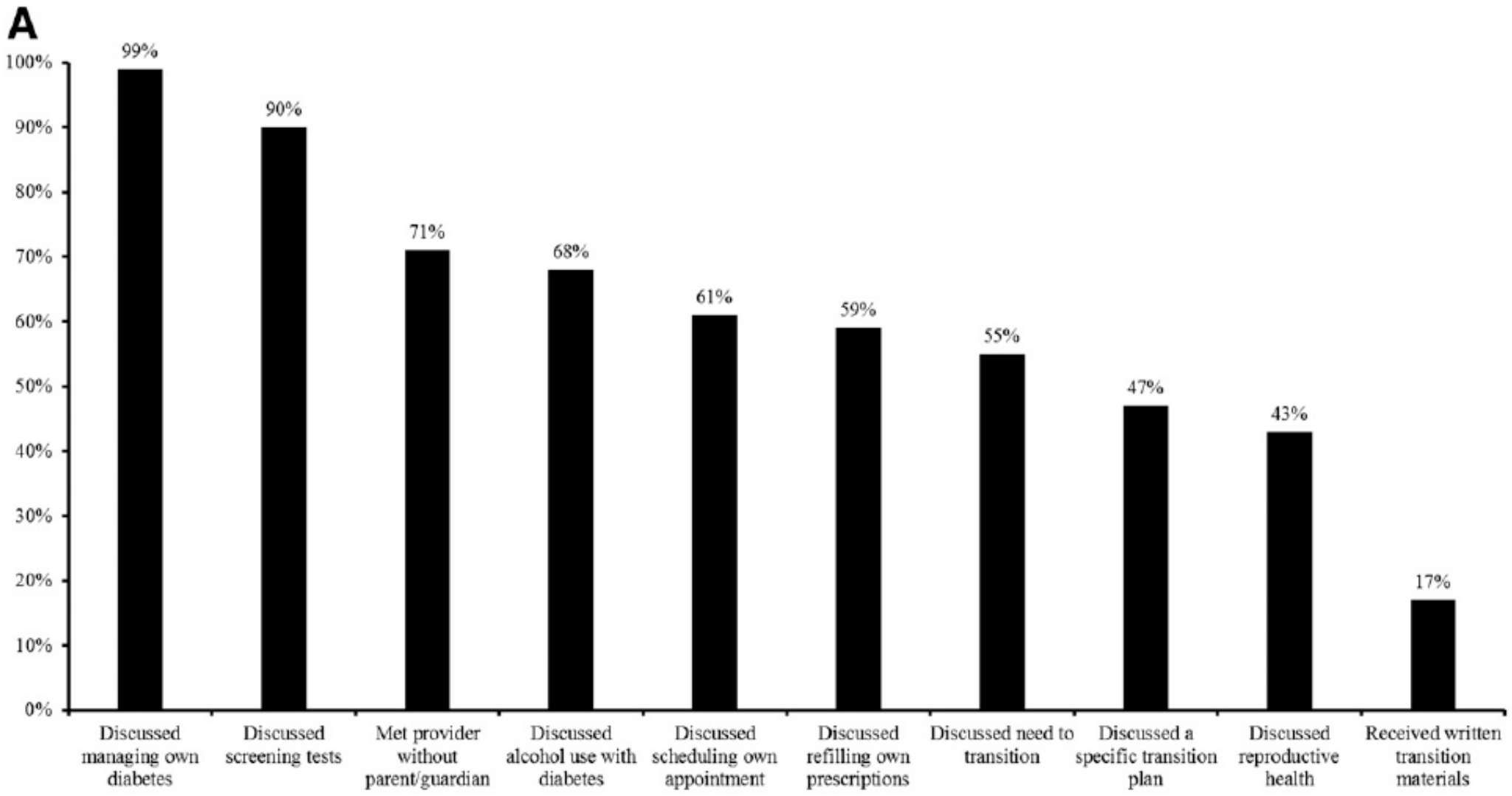


Figure 1—*A*: Reasons for remaining with the pediatric provider. *B*: Main reason for leaving the pediatric provider.

Transition preparation in those with Pediatric providers



Transition preparation in those with Adult providers

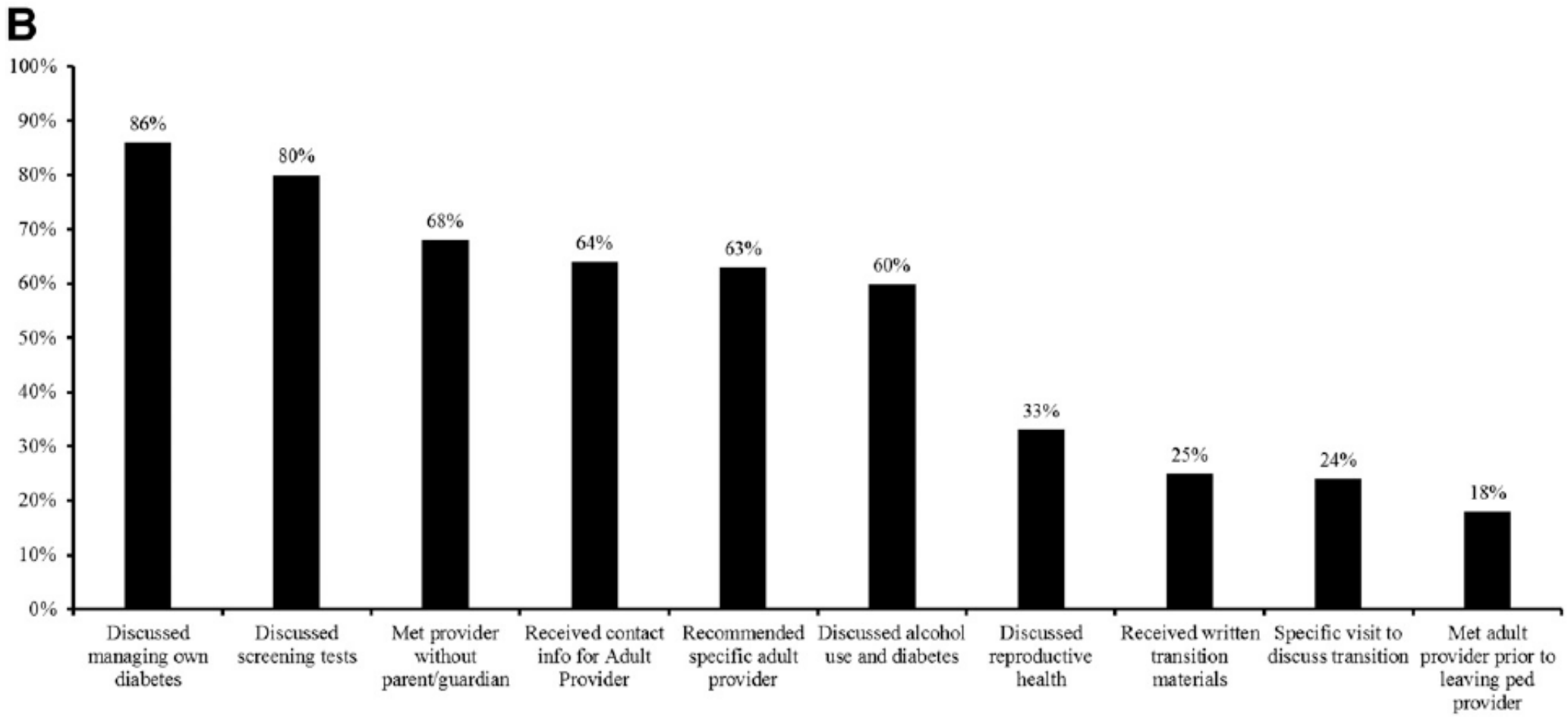


Table 2—Factors associated with a gap of >6 months in care between pediatric and adult providers

	Total *N* (%)	Gap in care >6 months *n* (%)	OR (95% CI)[a]
No. of visits to pediatric provider during 12 months before transition			
0–2 visits	101 (36)	32 (32)	3.2 (1.7, 6.1)
≥3 visits	183 (64)	25 (14)	1.0
Participant-reported preparedness to transition[b]			
Prepared	197 (66)	29 (15)	1.0
Not prepared	102 (34)	34 (33)	3.3 (1.7, 6.3)

[a]ORs were calculated from a logistic regression model including the factors of interest (number of visits to pediatric provider during the 12 months before transition and participant-reported preparedness to transition), adjusting for race/ethnicity, insurance status, and occurrence of one or more major life changes in the past year. [b]Prepared to transition was assigned for participants reporting they were mostly or completely prepared to leave their pediatric provider. Not prepared to transition was assigned for participants reporting they were completely/mostly unprepared or neutral.