Quantifying vehicle control from physiology in type 1 diabetes

Pranamesh Chakraborty[a†], Jennifer Merickel[b], Viraj Shah[c], Anuj Sharma[a], Chinmay Hegde[c], Cyrus Desouza[d], Andjela Drincic[d], Pujitha Gunaratne[e], Matthew Rizzo[b]

[a] Department of Civil, Construction, and Environmental Engineering, Iowa State University, USA
[b] Neurological Sciences, University of Nebraska Medical Center, USA
[c] Department of Electrical and Computer Engineering, Iowa State University, USA
[d] Internal Medicine, University of Nebraska Medical Center, USA
[e] Toyota Collaborative Safety Research Center, USA
[†]Corresponding Author: pranameshbesu@gmail.com

Objective: Our goal is to measure real-world effects of at-risk driver physiology on safety-critical tasks like driving by monitoring driver behavior and physiology in real-time. Drivers with type 1 diabetes (T1D) have an elevated crash risk that is linked to abnormal blood glucose, particularly hypoglycemia. We tested the hypotheses that 1) T1D drivers would have overall impaired vehicle control behavior relative to control drivers without diabetes, 2) At-risk patterns of vehicle control in T1D drivers would be linked to at-risk, in-vehicle physiology, and 3) T1D drivers would show impaired vehicle control with more recent hypoglycemia prior to driving.

Methods: Drivers (18 T1D, 14 control) were monitored continuously (4-weeks) using in-vehicle sensors (e.g., video, accelerometer, speed) and wearable continuous glucose monitors (CGMs) that measured each T1D driver's real-time blood glucose. Driver vehicle control was measured by vehicle acceleration variability (AV) across lateral (AV_Y, steering) and longitudinal (AV_X, braking/accelerating) axes in 45-second segments (N = 61,635). Average vehicle speed for each segment was modeled as a covariate of AV and mixed-effects linear regression models were used.

Results: We analyzed 3,687 drives (21,231 miles). T1D drivers had significantly higher overall $AV_{X,Y}$ compared to control drivers ($B_X = 2.5\times10^{-2}$ $B_Y = 1.6\times10^{-2}$, $p < 0.01$)—which is linked to erratic steering or swerving and harsh braking/accelerating. At-risk vehicle control patterns were particularly associated with at-risk physiology, namely hypo- and hyperglycemia (higher overall $AV_{X,Y}$). Impairments from hypoglycemia persisted for hours after hypoglycemia resolved, with drivers who had hypoglycemia within 2-3 hours of driving showing higher AV_X and AV_Y. State Department of Motor Vehicle records for the 3 years preceding the study showed that at-risk T1D drivers accounted for all crashes (N = 3) and 85% of citations (N = 13) observed.

Conclusions: Our results show that T1D driver risk can be linked to real-time patterns of at-risk driver physiology, particularly hypoglycemia, and driver risk can be detected during and prior to driving. Such naturalistic studies monitoring driver vehicle controls can inform methods for early detection of hypoglycemia-related driving risks, fitness to drive assessments, thereby helping to preserve safety in at-risk drivers with diabetes.

Keywords: diabetes mellitus, driving risk, hypoglycemia, vehicle control

INTRODUCTION

Diabetes is one of the most prevalent diseases worldwide, affecting more than 285 million people and ~6.4% of the world population in 2010 (Shaw et al. 2010). By 2030, the prevalence of diabetes is expected to rise to 439 million people and 7.7% of the world population. This presents a significant problem of patient and public safety due to

elevated vehicle crash risk in drivers with diabetes, compared to drivers without diabetes (Tregear et al. 2017). At-risk physiology in diabetes has been linked to this risk. Hypoglycemia (low blood glucose) is a primary factor impacting diabetes driver risk, particularly in insulin-dependent type 1 diabetes (T1D) (Cox et al. 2009). Although insulin is essential for diabetes patients' survival, close control over hyperglycemia (high blood glucose) can increase the risk of hypoglycemia. Driving licensing authorities of most developed countries make a distinction if a driver with diabetes is treated with insulin or not (Graveling and Frier 2015), due to an elevated risk of hypoglycemia with insulin treatment.

Severe hypoglycemia (blood glucose < 54 mg/dL) in patients with diabetes leads to the occurrence of neuroglycopenic symptoms (weakness or fatigue, confusion, warmth sensation, severe cognitive failure, coma), requiring immediate action and treatment to resolve hypoglycemia (American Diabetes Association 2019). However, impairment can also occur at less severe levels of hypoglycemia (glucose level < 70 mg/dL) (McCrimmon et al. 2012). In past studies, Deary and Zammitt (2014) and Evans et al. (2000) performed detailed analyses of cognitive tests performmed on recently hypoglycemic T1D patients to determine the timeline of cognitive function recovery post-hypoglycemia. Their analysis showed that the cognitive function of T1D patients remained impaired for approximately 75 minutes after hypoglycemia. Hypoglycemia affects attention, memory, and decision-making abilities needed for safe driving, increasing risk of crashes due to driver error. Research has shown that cognitive impairment due to hypoglycemia, even in less severe forms, can affect safe performance in complex, high-risk tasks which requires vigilance, rapid response, and hand-eye coordination—such as automobile driving. Compounding this safety risk, driver awareness of hypoglycemia varies and some drivers are unaware of hypoglycemia, which may hinder driver ability to adopt strategies to mitigate risk from hypoglycemia.

Despite these risks, driver risk from diabetes is poorly understood, particularly its relationship to real-time physiologic changes like at-risk glycemia. A critical limitation of previous literature is that driving in diabetes has been primarily assessed in controlled, simulator settings (Cox et al. 2000)—where drivers may be in different physiologic states than typical and behave differently than they would in the real-world. We overcome these limitations of prior work by applying technological advances in in-vehicle and wearable sensor-based technologies to address this problem of public health and patient in drivers with insulin-dependent T1D.

Hypotheses

We tested the hypotheses that 1) **Disease status**: T1D drivers would have impaired vehicle control behavior relative to control drivers without diabetes, 2) **In-vehicle physiology**: Impaired vehicle control would be linked to acute (in-vehicle), at-risk physiology in T1D drivers, and 3) **Prior physiology**: T1D drivers would show impaired vehicle control with more recent hypoglycemia prior to driving.

RESEARCH DESIGN AND METHODS

Data Collection

Each driver participated in the study for 4 weeks of continuous naturalistic driving data collection (all drivers) and physiology (T1D).

Participants

This study involved a total of 36 participants. Twenty had insulin-dependent T1D and 16 were control drivers who had no presence of diabetes, based on self-report or medical exam (T1D drivers, < 12% HbA1c; control drivers, < 5.7% HbA1c) (American Diabetes Association 2019). Control participants were matched to T1D participants by age (within 2 years), gender, education (within 2 years), and typical driving environment (rural vs. urban). All participants were legally licensed and active drivers who gave informed consent to the study participation according to institutional guidelines. Out of the 36 participants, two control participants were excluded after study consent due to laboratory evidence of possible diabetes. One diabetes participant was excluded due to the incompatibility of their vehicle with the study's driving instrumentation. Analyzable data were obtained from 19 T1D drivers and 14 control drivers between 21-59 years of age (μ = 33.2 years). **Table 1** shows a summary of participant demographics, driving experience, and hemoglobin A1c (Hb1Ac) levels.

All participants completed a physical examination and a full medical history at induction. Medical conditions and medications that presented a significant confounding effect on driver behavior or are known to worsen diabetes were excluded. For examples, patients with kidney failure were excluded because they are not able to process insulin and therefore have a high propensity for hypoglycemia (Sandholm et al. 2012). Excluded medical conditions included neuropathy, pulmonary disease, major psychiatric disorders, neurologic conditions, vestibular disease, sleep disorders, current substance abuse, visual field defects, and thyroid or kidney diseases. Excluded medications were narcotics, anxiolytics, anticonvulsants, sedating antihistamines, and major psychoactive medication. All participants had safe vision for driving as per the Nebraska Department of Motor Vehicle (DMV) standards (binocular, corrected or uncorrected <20/40). DM drivers had received a diagnosis of T1D, used insulin at least daily, and had self-reported at least bi-weekly hypoglycemic episodes.

Laboratory Assessments: All drivers completed a standardized self-reported demographic (e.g., age, gender, race/ethnicity, education, and socioeconomics), health (diagnosis history and medication usage), and vision assessments (corrected or uncorrected, near/far visual acuity [ETDRS OU] and contrast sensitivity [ETDRS, 2.5% OU]) at the start of the study period. Presence of diabetes and basic metabolic function were assessed in all drivers via blood labs (e.g., HbA1c, BMP [basic metabolic panel]).

Naturalistic Driving **Assessment:** Driving data were collected using in-vehicle sensor instrumentation installed in the participant's own vehicle. Driving behavior was remotely and continuously recorded from on- to off ignition in each

participant's own vehicle via "Black Box" vehicle sensor instrumentation, which collected video, accelerometer, GPS, speed, throttle, and RPM data at a frequency of 1 Hz. Each driver drove as they typically would for the entire study period (4-weeks). In addition to collected naturalistic driving data, state records of crashes and citations were collected to quantify each participant's safety in the 3 years prior to enrollment and to provide insight how driver behavior in the study linked to overall safety.

Glucose Data Collection: All DM drivers wore continuous glucose monitors (CGMs) throughout the study period. Real-world glucose levels (lows, peaks, and variability) can be obtained directly from CGMs (Klonoff, Ahn, and Drincic 2017) and linked to time synchronized driving data collected from in-vehicle sensors. All CGM devices were "blinded" so the T1D participants could not use its real-time feedback and treatment. Glucose levels were sampled by CGMs every 5 minutes. In addition to wearing the CGM, participants self-sampled their glucose with a glucometer twice daily throughout the study, which provided verification of CGM collected glucose levels. CGM data quality was determined per FDA recommendations (PMA P120005/S018 2014). Mean difference between CGM and glucometer glucose levels was 4.29% (range: 2.8% - 6.55%), well below the FDA required standard of < 25% difference between these measurements (PMA P120005/S018 2014). Glucose levels exceeding a change > 25% within a 15-minute time span were removed, removing on average 2.1% of CGM data per participant. Overall, T1D participants complied with CGM use, with only an average of 5.5% of glucose data missing per participant (range 1.7% - 10.2%). This well exceeds FDA guidelines for > 75% of data present for usable CGM data. To synchronize CGM and driving data, CGM data were first up-sampled (forward-fill interpolation) to match the frequency of driving data (from 5 minutes to 1 Hz) and merged by time-stamp. Missing CGM data was not interpolated.

Models

Vehicle Control Outcomes: Vehicle control was modeled using acceleration variability (AV) across lateral (AV_y, steering) and longitudinal (AV_x, braking/accelerating) axes in 45-second segments (Bishop et al. 2018). Vehicle acceleration has been used previously in the literature to identify individual driver patterns (Fung et al. 2017), risky driving (Kluger et al. 2014), crashes/crash severity (Stipancic et al. 2018), and driver impairments (Merickel et al. 2019). Increased AV can be linked to erratic driving, harsh braking and accelerating, poor steering control, lane variability, and swerving (Palat et al. 2019). Decreased AV has been linked to attentional impairments, reduced driver responsiveness to the environment, and may indicate failure to appropriately adjust the vehicle relative to the roadway or other on-road vehicles (Merickel et al. 2019). To calculate AV, each drive was divided into 45-seconds segments and AV was calculated using the standard deviation of lateral/longitudinal acceleration values. To permit analysis of continuous driving, driving data with speeds < 8 kph (5 mph) were removed when the car was not moving, in dense traffic, or coming to a stop. Average vehicle speed for each segment was used as a control variable in models to account for differences in AV due to speed. Vehicle control was modeled across driver covariates using mixed-effect linear regression models with a by-participant random intercept.

Driver Covariates: Driver disease status and glucose conditions were assessed and analyzed as driver covariates. To test the 1st hypothesis (**disease status**), presence of DM among drivers (i.e., T1D drivers or control drivers) was used as a categorical variable. To test the 2nd hypothesis (**in-vehicle physiology**), each T1D drivers in-vehicle glycemic state was determined by categorizing CGM data for each drive across 3 categories: hypoglycemic (< 70mg/dL), euglycemic (70 - 180 mg/dL), and hyperglycemic (> 180 mg/dL) relative to the most severe blood glucose level observed during the drive—as per ADA's standard (American Diabetes Association 2019). T1D driver in-vehicle glycemic state was assessed relative to control driver vehicle control (baseline condition). To test the 3rd hypothesis (**prior physiology**), only euglycemic T1D drivers were used in the model to objectively determine the impact of impairment due to prior hypoglycemia without confounding effects of current at-risk glycemia. In addition to each driver's in-vehicle glycemic state, the time since each driver's most recent hypoglycemic episode prior to the drive (in minutes) and the duration of that episode (in minutes) were computed. The overall severity of each prior hypoglycemic episode was also assessed. Episodes were classified as severe if glucose fell below < 54 mg/dL durring the hypoglycemic episode (American Diabetes Association 2019). Age and gender were used as control variables in each model, to account for differences in vehicle control behavior due to driver demographics.

RESULTS

Our study data was collected from a total of 848 driver days, 3,687 drives, and 34,168 km (21,231 miles) driving. Out of this, T1D drivers' behavior was reported across 1,940 drives and 16,610 km (10,321 miles). The total drive duration was divided into 61,635 segments of 45-seconds duration (28,569 segments from T1D drivers). The mean AV_X and AV_Y across all segments for all participants were found to be 0.078 and 0.075 respectively. AV_X and AV_Y were found to decrease with the increase in vehicle speed across all the three hypotheses ($p < 0.001$). However, age and gender were not found to be statistically significant in any of the hypotheses ($p > 0.05$). The detailed results specific to each of the three hypotheses are discussed next.

Disease status

T1D drivers had higher braking/accelerating and steering changes (increased AV_X and AV_Y) compared to control drivers ($B_X = 2.5\times10^{-2}$ $B_Y = 1.6\times10^{-2}$, $p < 0.01$)—which is linked to erratic steering or swerving and harsh braking/accelerating (Figure 1, Table 2). T1D drivers showed greater reduction in AV on higher speed roadways compared to the control drivers ($B_X = -5.6\times10^{-4}$, $B_Y = -3.9\times10^{-4}$, $p < 0.001$).

In-vehicle physiology

T1D drivers who were currently hypoglycemic and hyperglycemic while driving showed higher higher/accelerating (increased AV_X) ($B_{hypo} = 3.4\times10^{-2}$, $B_{hyper} = 2.5\times10^{-2}$, $p \leq 0.001$) and increased steering changes (increased AV_Y) ($B_{hypo} = 2.6\times10^{-2}$, $B_{hyper} = 1.5\times10^{-2}$, $p < 0.01$) than control drivers (

Table 3). However, euglycemic conditions resulted in reduced steering control (decreased AV_Y) only ($B_{eu} = 1.1\times10^{-2}$, $p = 0.04$) and no statistically significant reduction in braking/accelerating (AV_X) were observed ($p > 0.05$).

Prior physiology

Hypoglycemia prior to a drive resulted in persistent impairments in vehicle control behaviors, even for T1D drivers who were euglycemic at time of driving. For up to 2 hours after hypoglycemia resolved, T1D drivers showed decreased AV_Y (B_{1hour} = -1.3×10^{-3}, B_{2hour} = -1.5×10^{-4}, $p < 0.05$,

Table 4), which may link to decreased steering changes relative to driving environment dynamics (e.g., traffic). Results suggest that after a 2-hour recovery period from hypoglycemia, T1D drivers no longer showed significant changes in AV_Y ($p > 0.05$,

Table 4). Drivers showed less braking/accelerating (decreased AV_X) for up to 3 hours since hypoglycemia resolves (B_{1hour} = -1.3×10^{-3}, B_{2hour} = -1.5×10^{-4}, B_{3hour} = -1.5×10^{-4}, $p < 0.05$). Once drivers had at least 3 hours to recover from hypoglycemia, braking/accelerating behavior was no longer affected ($p > 0.05$). While impairments in vehicle control persisted after hypoglycemia resolved, they were marginal in effect size compared to the effect of current at-risk glycemia. Impairments due to prior hypoglycemia were unaffected by episode duration and severity.

Crash Risk from State Records

In addition to these analyses and observations obtained from in-vehicle sensor data, State Department of Motor Vehicle records for the 3 years preceding the study showed that at-risk T1D drivers accounted for all crashes (N = 3, 2 at fault) and 85% of citations (N = 13) observed. In concert with our results, this links elevated crash risk in this T1D cohort to at-risk physiology, particularly hypoglycemia.

DISCUSSION

Our results link disease and at-risk physiology to impaired vehicle control in T1D. T1D drivers show impaired vehicle control related to steering, accelerating, and braking variability compared to drivers without diabetes. These impairments are elevated during at-risk physiologic states. Vehicle control impairments are linked to environment, with T1D drivers showing greater differences in behavior relative to control drives during higher-speed driving. This may link to elevated crash severity due to risk on high-speed roadways. This is noteworthy in as much as crash severity is greater on high-speed roadways.

Our results are consistent with prior studies suggesting that vehicle control impairments due to hypoglycemia may persist for hours after hypoglycemia resolves (Weinger et al. 1999; Merickel et al. 2019), even in T1D drivers whose blood glucose levels are currently normal. We find that impairments due to prior hypoglycemia affect vehicle control behavior for 2-3 hours after hypoglycemia resolves, which provides an important foundation for developing objective, clinical recommendations to preserve safety in at-risk drivers with diabetes. Importantly, our results suggest that impairments from hypoglycemia may persist for longer in complex, real-world tasks like driving than based on laboratory-based cognitive examination (Evans et al. 2000; Deary and Zammitt 2014; Zammitt et al. 2008). While braking/accelerating and steering were effected by prior hypoglycemia, steering-related impairments from prior hypoglycemia persisted for longer than braking/accelerating-related impairments—suggesting that the cognitive mechanisms and associated impairments from hypoglycemia vary relative to the particular vehicle control behavior.

Admittedly, further research is needed to map the patterns of vehicle control we observe in this study to quantitative metrics of driver risk. Video validation can provide key insights linking real-world driving risks scenarios such as lane incursions, reduced response time to lead vehicle braking, etc. to cognitive impairment related to hypoglycemia. Also, it can provide greater understanding of the different scenarios and driver distractions that might lead to variability in driving in addition to hypoglycemia related cognitive impairment. While this study focused on driver control behavior under continuous driving situations, it can be extended in future to include complex driver behavior that can be observed at traffic intersections, stop signs, or congested traffic conditions. Driver risk can be also associated in future to understand its relation to the frequency of hypoglycemic episodes, T1D disease progression, and other factors impacting T1D driver risk. Such detailed analysis can be feasible by observing a larger sample of T1D patients with healthy drivers encompassing a wider variety and uniform representability of the entire population.

ACKNOWLEDGEMENTS

This study was supported by the Toyota Collaborative Safety Research Center and the University of Nebraska Medical Center's (UNMC) Mind & Brain Health Labs. We thank our research team and the Diabetes, Endocrinology, and Metabolism Division of UNMC's Internal Medicine for their help and commitment in coordinating this project.

REFERENCES

American Diabetes Association. Standards of Medical Care in Diabetes—2019 Abridged for Primary Care Providers. *Clinical diabetes: a publication of the American Diabetes Association.* 2019; 37(1):11-34. doi: 10.2337/cd18-0105

Bishop MO, Dawson J, Merickel J, Rizzo M. Reducing accelerometer data in instrumented vehicles. Paper presented at: Joint Statistical Meetings, 2018.

Cox DJ, Ford D, Gonder-Frederick L, et al. Driving Mishaps Among Individuals With Type 1 Diabetes. *Diabetes Care*. 2009;32(12):2177-2180. doi:10.2337/dc08-1510

Cox DJ, Gonder-Frederick LA, Kovatchev BP, Julian DM, Clarke WL. Progressive hypoglycemia's impact on driving simulation performance: Occurrence, awareness, and correction. *Diabetes Care*. 2000;23(2):163-170. doi:10.2337/diacare.23.2.163

Deary IJ, Zammitt NN. Symptoms of Hypoglycaemia and Effects on Mental Performance and Emotions. In: *Hypoglycaemia in Clinical Diabetes: Third Edition*. ; 2014:23-45. doi:10.1002/9781118695432.ch2

Evans ML, Pernet A, Lomas J, Jones J, Amiel SA. Delay in onset of awareness of acute hypoglycemia and of restoration of cognitive performance during recovery. *Diabetes Care*. 2000;23(7):893-897. doi:10.2337/diacare.23.7.893

Fung NC, Wallace B, Chan ADC, et al. Driver identification using vehicle acceleration and deceleration events from naturalistic driving of older drivers. Paper presented at: 2017 IEEE International Symposium on Medical Measurements and Applications (MeMeA); 7-10 May 2017, 2017. doi: 10.1109/MeMeA.2017.7985845

Graveling AJ, Frier BM. Driving and diabetes: problems, licensing restrictions and recommendations for safe driving.

Clin Diabetes Endocrinol. 2015;1(1). doi:10.1186/s40842-015-0007-3

Klonoff DC, Ahn D, Drincic A. Continuous glucose monitoring: A review of the technology and clinical use. *Diabetes Res Clin Pract*. 2017;133:178-192. doi:10.1016/j.diabres.2017.08.005

Kluger R, Smith B. Pattern matching longitudinal acceleration time series data to identify crashes in naturalistic driving data. Paper presented at: 2014 ITS World Congress Conference; 7-11 September, 2014.

McCrimmon, RJ, Ryan, CM, Frier, BM. Diabetes and cognitive dysfunction. *The Lancet*. 2012; 379(9833): 2291-2299.

Merickel J, High R, Smith L, et al. Driving Safety and Real-Time Glucose Monitoring in Insulin-Dependent Diabetes. *Int J Automot Eng*. 2019;10(1):34-40. doi:10.20485/jsaeijae.10.1_34

PMA P120005/S018. FDA Summary of safety and effectiveness data for Dexcom G4 PLATINUM continuous glucose monitoring system. Food and Drug Administration 2014.

Sandholm N, Salem RM, McKnight AJ, Brennan EP, Forsblom C, et al. (2012) New Susceptibility Loci Associated with Kidney Disease in Type 1 Diabetes. PLOS Genetics 8(9): e1002921.

Shaw JE, Sicree RA, Sicree RA, Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res Clin Pract*. 2010;87(1):4-14. doi:10.1016/j.diabres.2009.10.007

Stipancic J, Miranda-Moreno L, Saunier N. Vehicle manoeuvers as surrogate safety measures: Extracting data from the gps-enabled smartphones of regular drivers. *Accid Anal Prev*. 2018;115:160-169.

Tregear SJ, Rizzo M, Tiller M, et al. Diabetes and Motor Vehicle Crashes: A Systematic Evidence-Based Review and Meta-Analysis. In: *Fourth International Driving Symposium on Human Factors in Driver Assessment, Training and Vehicle Design*. ; 2017:343-350. doi:doi.org/10.17077/drivingassessment.1259

Weinger K, Kinsley BT, Levy CJ, et al. The perception of safe driving ability during hypoglycemia in patients with type 1 diabetes mellitus. *Am J Med*. 1999;107(3):246-253. doi:10.1016/S0002-9343(99)00221-1

Zammitt NN, Warren RE, Deary IJ, Frier BM. Delayed recovery of cognitive function following hypoglycemia in adults with type 1 diabetes effect of impaired awareness of hypoglycemia. *Diabetes*. 2008;57(3):732-736. doi:10.2337/db07-0695

Table 1. Summary of participant demographic characteristics, driving experience, and DM presence (HbA1c)

Participant Type	Age (years)	Gender	Race	Driving Experience (Years)	Hb1Ac
T1D	Range: 21-52 μ = 32.5 *SD* = 9.6	Female: *N* = 12 Male: *N* = 7	White: *N* = 19	Range: 6-36 μ = 16.1 *SD* = 9.5	Range: 6.5-11.3 μ = 7.7 *SD* = 1.06
Control	Range: 21-55 μ = 32.3 *SD* = 10.4	Female: *N* = 10 Male: *N* = 4	White: *N* = 13 Asian: *N* = 1	Range: 4-33 μ = 15.1 *SD* = 9.8	Range: 4.7-5.9 μ = 5.3 *SD* = 0.30

Table 2. Effect of disease status (T1D or control drivers) on AV_X and AV_Y

	AV_X		AV_Y	
Variable	Coef. (*B*)	*p*	Coef. (*B*)	*p*
Intercept	0.143	< 0.001	0.137	< 0.001
Speed (kph)	-7.4×10^{-4}	< 0.001	-7.0×10^{-4}	< 0.001
DM patient				
No (Control Driver, ref)	-	-	-	-
Yes (T1D)	2.5×10^{-2}	0.001	1.6×10^{-2}	0.002
Interaction Term (Speed:DM)				
Speed:No (ref)	-	-	-	-
Speed:Yes (T1D)	-3.5×10^{-4}	< 0.001	-2.4×10^{-4}	< 0.001

Table 3. Effect of current physiologic conditions on AV_X and AV_Y

	AV_X		AV_Y	
Variable	Coef. (*B*)	*p*	Coef. (*B*)	*p*
Intercept	1.43×10^{-1}	< 0.001	1.37×10^{-1}	< 0.001
Speed (mph)	-7.4×10^{-4}	< 0.001	-7.0×10^{-4}	< 0.001
Blood Glucose Level				
Control Driver (ref)	-	-	-	-
Hypoglycemic (T1D)	3.4×10^{-2}	< 0.001	2.6×10^{-2}	< 0.001
Hyperglycemic (T1D)	2.5×10^{-2}	0.001	1.5×10^{-2}	0.002
Euglycemic (T1D)	1.4×10^{-2}	0.06	1.1×10^{-2}	0.04
Interaction Term (Speed:Blood Glucose)				
Speed:Control (ref)	-	-	-	-
Speed:Hypoglycemic (T1D)	-4.9×10^{-4}	< 0.001	-3.7×10^{-4}	< 0.001
Speed:Hyperglycemic (T1D)	-2.2×10^{-4}	< 0.001	-1.9×10^{-4}	< 0.001
Speed:Euglycemic (T1D)	-3.6×10^{-4}	< 0.001	-2.5×10^{-4}	< .001

Table 4. Effect of time since hypoglycemia on AV_X and AV_Y

Max. Time since hypoglycemia	Segments (n)	Variable	AV_X		AV_Y	
			Coef. (B)	p	Coef. (B)	p
1 hour	197	Intercept	2.3×10^{-1}	< 0.001	2.4×10^{-1}	< 0.001
		Speed (mph)	-1.7×10^{-3}	< 0.001	-1.4×10^{-3}	< 0.001
		Time since hypoglycemia (mins)	-4.3×10^{-4}	0.18	-1.3×10^{-3}	0.001
2 hours	728	Intercept	1.9×10^{-1}	< 0.001	1.7×10^{-1}	< 0.001
		Speed (mph)	-1.2×10^{-3}	< 0.001	-1.1×10^{-3}	< 0.001
		Time since hypoglycemia (mins)	-1.7×10^{-4}	0.002	-1.5×10^{-4}	0.03
3 hours	1358	Intercept	1.8×10^{-1}	< 0.001	1.6×10^{-1}	< 0.001
		Speed (mph)	-1.2×10^{-3}	< 0.001	-1.0×10^{-3}	< 0.001
		Time since hypoglycemia (mins)	-5.9×10^{-5}	0.02	-4.5×10^{-5}	0.19
4 hours	2247	Intercept	1.7×10^{-1}	< 0.001	1.6×10^{-1}	< 0.001
		Speed (mph)	-1.8×10^{-3}	< 0.001	-9.7×10^{-4}	< 0.001
		Time since hypoglycemia (mins)	-1.3×10^{-6}	0.92	-1.6×10^{-6}	0.92

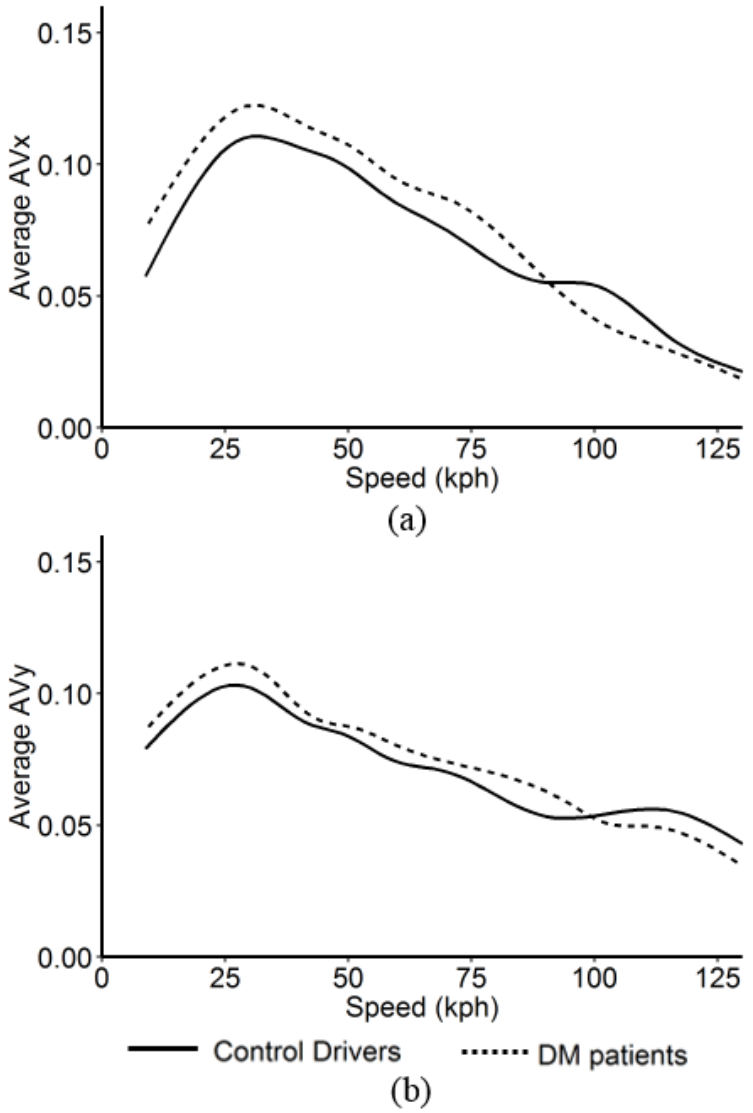

Figure 1. Variation of (a) AV_X and (b) AV_Y for DM patients and control drivers