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Coefficient of Friction at the Fingertips in Type II Diabetics Compared to Healthy Adults

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Abstract

Clinical observations suggests that Type II Diabetes patients are more susceptible to skin changes which may be associated with reduced coefficient of friction at the fingertips. Reduced coefficient of friction may explain recent reports of fine motor dysfunction in diabetic patients. Coefficient of friction was evaluated using slip force evaluation in a cross-sectional cohort of diabetic patients and age- and sex-matched healthy controls. Covariates of tactile sensation, disease duration, glycated hemoglobin, and clinical diagnosis of peripheral neuropathy were also assessed. A significant decrease in fingertip coefficient of friction in the diabetic group was found as compared to controls. Health state covariates did not alter the strength of between-group differences in statistical analyses. This finding of between group differences for fingertip frictional properties suggests that causative factors of reported manual motor dysfunction lie in both the distal and proximal portions of the nervous system.

Keywords

tactile; hand; finger; type II diabetes; friction; sensory function

Introduction

When humans adjust their grip to prevent slipping of objects grasped by the fingertips, the component of their grip force (normal to the object surface) generated at the fingertips must be sufficient to generate a tangential frictional force that can prevent slip¹. This sufficient yet minimal grip force is known as slip force. The ratio between the tangential force and the slip force is known as the coefficient of friction (μ) a numerical value that provides a concrete method for comparing the frictional properties of the human hand^{2,3}. Reduced μ values have been reported in persons aged 60 years and older^{4–6} as well as persons with hyperhidrosis⁷. These μ changes have been associated with abnormal grip and pinch force

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production as well as reduced force modulation^{8,9}, resulting in impaired hand function during everyday tasks^{5,10–13}.

Recent studies have indicated that persons with Type II Diabetes also exhibit subtle motor function changes of the fingers^{14–16}. Patients with Type II Diabetes are known to be susceptible to skin changes including tactile impairment, vascular compromise, and dry skin. Recent data also indicates that tactile impairment is not the sole contributor to manual motor dysfunction in diabetic patients¹⁶, thus it is possible that reduced μ at the fingertips may contribute to motor dysfunction observed in diabetic patients. If a significant difference between μ values in diabetic patients versus healthy controls exists, dermal characteristics can be identified as one of the causative deficits in the disruption of manual function in Type II Diabetes beyond the normal aging process. In order to evaluate this premise, we utilized the methods and sample sizes outlined in previous studies to investigate μ values of the fingertips in a Type II Diabetes patient cohort previously evaluated by our lab. As such, the working hypothesis of the study is that Type II Diabetes patients will exhibit μ changes at the fingertips as compared to healthy age- and sex-matched controls.

Methods

Study Participants

Ten (10) individuals with Type II Diabetes and 10 healthy age- and sex-matched individuals (6 females, 4 males in each group) were recruited from a database pool created from previous studies of T2D in our lab^{14–16}. Type II Diabetes patient demographics and average healthy demographic data can be found in Table 1. The average age of the participants was 61 ± 6 years and 60 ± 6 years for the diabetic and healthy cohorts, respectively. All subjects were strongly right-handed (LQ average = +98), assessed via the Edinburgh Handedness Inventory¹⁷. Diabetic participants had a confirmed diagnosis of Type II Diabetes either with or without diagnosis of diabetic peripheral neuropathy per collaborating physician, diagnosed via clinical examination and electrodiagnostics. Healthy individuals recruited to participate in this study did not have a diagnosis of Type II Diabetes, Type I Diabetes, or pre-diabetes. Study participants were excluded if they reported a history of neurological disorders (Parkinson's disease, Stroke, Multiple Sclerosis, etc.), chemotherapy treatments, and/or hereditary or compressional neuropathies, including carpal tunnel syndrome. The protocol was reviewed and approved by the Committees established for the Protection of Human Subjects at the University of Houston. In accordance with the Declaration of Helsinki, participants provided informed consent according to the regulations established by the Committees established for the Protection of Human Subjects at the University of Houston.

Experimental Procedures

Evaluation of each participant consisted of two primary measurements: (1) tactile evaluation and (2) slip testing. Tactile evaluation was achieved via the Semmes-Weinstein Monofilament test. Monofilament testing sites include the tip of the thumb and tip of the index finger. Monofilament data were assessed as described in^{14,15}. During evaluation, participants kept their eyes closed and verbally indicated if and where they perceive

monofilament touch. The monofilament size was increased until the subject was able to detect touch a minimum of two times at the same location.

In the slip testing protocol, participants were asked to hold two identical six-component force-moment transducers fixed to an aluminum housing (Nano-25 transducers; ATI Industrial Automation, Garner, NC, USA) using solely the thumb and index finger of their dominant hand. The thumb and index fingers were aligned with each other on the transducer faces prior to the start of each trial. Sandpaper (320-grit) was attached to the contact surfaces of each sensor to increase the friction between the digits and the transducers. The device was moveable in front of each participant; additional device details can be found in ^{14,15}. A picture of the device used can be found in Figure 1. Transducer signals were amplified and multiplexed using ATI hardware prior to being routed to an analog to digital converter (via cDAQ-9174 chassis and NI-9205 input modules, National Instruments, Austin, TX, USA). A customized Labview program (National Instruments, Austin, TX, USA) was used for data acquisition and customized MATLAB (Mathworks Inc., Natick, MA, USA) programs were written for data processing. Signals were sampled at 100 Hz and low-pass filtered at 10 Hz using a 2nd order, zero-lag Butterworth filter.

In the experiment, the upper arm of each participant was vertical and the pronated forearm was horizontally oriented. Participants were asked to gradually move their index finger and thumb apart until the apparatus slipped from their hand and fell on to a padded surface, as described in ¹⁸. This procedure was carried out in order to measure minimal grip force required to prevent slipping. Each participant performed three trials of the slip test. Use of moisturizing creams was discouraged prior to the start of the data collection session. Forces generated by the index finger and the thumb were measured separately by the two sensors of the system simultaneously. Any normal force biases at the onset of the trial were detected corrected post-hoc via Matlab processing. The coefficient of friction (μ) was calculated by the equation:

$$\mu = \frac{F_t + a|T_n|}{F_n}$$

where F_t is the tangential forces applied to the transducer face, T_n is the torque about the normal force vector, F_n is the normal force at the slip threshold, $a = 0.11543/\text{mm}$, as per published values³.

Data Reporting and Statistics

All data are reported as mean $\pm$ standard deviation. The Anderson-Darling test was used to evaluate data normality. Levene's test was used to assess between group variance differences. Given the likelihood of a directional difference between groups ($\mu_{\text{Control}} > \mu_{\text{Diabetic}}$) to physiological changes of the derma, a one-sided (ie. one-tailed) paired t-test was used to evaluate simple between group differences. A mixed-model ANOVA was used to evaluate sensory detection thresholds with the factors of *Group* (diabetic vs control), *Site* (thumb vs index finger), with *Subject* as random factor. A one-way repeated measures analysis of covariance (ANCOVA) were used to investigate the individual contribution of

disease state measurements (disease duration, glycated hemoglobin, clinical diagnosis of peripheral neuropathy via indicator variable, and tactile sensation detection thresholds) on μ values.

Results

Significant impairment in tactile function was detected in the diabetic group as compared to controls (Figure 2A) (*Group*: $F_{1,28} = 37.79$, $P < .001$). Sensory detection values were consistent between the thumb and index finger (*Site*: $F_{1,28} = 0.01$, $P \sim 0.9$). Between *Group* differences were magnified when *Neuropathy Status* was included in the models (*Group*: $F_{1,27} = 45.51$, $P < .001$; *Neuropathy Status*: $F_{1,27} = 5.11$, $P < .05$).

The μ data collected in this study met normality assumptions via the Anderson-Darling test. Levene's test did not indicate significant differences in between *Group* variances. A significant difference in μ values between the two *Groups* were found ($t_{10} = 1.9$, $P < .05$) using a one-sided paired t-test (Figure 2B). Additional ANCOVAs indicated no significant difference between diabetic patients and healthy controls even when disease state covariates and tactile detection thresholds were included in the statistical models.

Discussion

Overall, the diabetic patients did show a significant difference in μ at the fingertips as compared to healthy controls. These differences were not impacted when disease state markers such as disease duration, severity (assessed via glycated hemoglobin), clinical diagnosis of peripheral neuropathy (via indicator variable), and poorer tactile sensation thresholds were accounted for in the statistical models. This suggests that dermal alterations to the skin of the fingertips with respect to frictional properties in diabetic patients may play a role in how diabetic patients interact and handle everyday objects. While the sample size used for this study is small, it is consistent with sample sizes used in evaluating μ differences between populations with known manual deficits⁴⁻⁷. The reported standard deviation values in this study are within range of those previously reported in^{4,5}. The inclusion of torque terms in our data set suggest that simple slip ratio values may not be sensitive enough to detect changes in frictional properties in patient populations with subtle dermal property changes, in contrast to a lack of reported differences in slip ratios in diabetic patients¹⁹. Further, we note that the frictional changes were not influenced by peripheral neuropathy diagnosis, as the ANCOVA models did not indicate a significant impact of neuropathy diagnosis in μ values.

Notably, participants in our diabetic and healthy control groups were between the ages of 50 and 75. This age range is one in which fingertip frictional values^{9,13} and tactile function^{10,20} are known to decline due to natural processes of aging. These changes in fingertip skin properties have been previously associated with production of excessive hand/finger forces and impaired modulation of such forces, particularly in fine motor tasks^{5,13,10-12}. Given our previous findings in further impaired fingertip dysfunction in diabetic patients in this age range (as directly compared to healthy controls), we expected to find μ changes in the diabetic group due impaired metabolic function that impacts the epidermis (egs. increased

advanced glycation end products and oxidative stress)^{21,22}. These changes in the frictional properties of the fingertips (and presumably also the volar aspect of the hand) may result in subtle yet impactful impairment in the handling and manipulation of everyday objects. Specifically, this may impair diabetic patient ability to self-monitor their capillary blood sugar, by both affecting the precision grasp need to hold and use a lancet, but also the ability of a patient to pierce the skin with the lancet. With respect to these issues, safety lancets may be the best option for self-lancing in this particular population.

In line with our expectations, the μ differences found in this study are consistent with a number of dermal changes at the fingertips associated with Type II Diabetes. In each of our previous studies, poorer tactile detection thresholds have been found in diabetic patients^{14–16}, consistent with other contemporary publications^{23,24}. Reports of reduced nerve fiber ending density^{25,26} and poor skin blood flow due to endothelial dysfunction²⁷ in diabetic patients also suggest that altered μ is entirely plausible in diabetic patients. While dermal changes are an entirely plausible source for reduced fingertip force modulation control, recent data indicate that known dermal changes such as reduced tactile function of the fingertips is not the sole source of motor dysfunction in patients living with diabetic^{16,19}. Instead, these data suggest that the causative factors of reported manual motor dysfunction lie in more proximal portions of the nervous system, supported by one of our recent studies¹⁶, particularly by between group magnification of sensory detection threshold differences due to neuropathy status.

Implicitly, this suggests that changes to dermal properties, motor unit function, corticospinal tracts, and/or cerebral cortex may all be responsible for the behavioral differences exhibited in this and previous studies^{14–16,19,24}. It is plausible that changes in motor units resulting in altered motor unit firing rates and or changes in motor unit size may contribute to motor dysfunction in the diabetic group, leading to manual motor dysfunction that we have reported on in previous manuscripts. Contributions of corticospinal and cortical changes are also plausible in this patient population due to the systemic nature of the disease. The combination of these finds have shifted the focus of our investigations to contribution of motor unit, corticospinal, and cortical dysfunction to manual motor dysfunction in diabetic patients; we are currently collecting data to evaluate the contribution of such changes to motor dysfunction in diabetic patients.

In summary, while the exact mechanisms responsible for motor dysfunction are currently unknown, the data presented in this study provide evidence add changes in fingertip frictional properties to the list of contributing factors to manual dysfunction in patients with Type II Diabetes. Results from this and future studies may be used to better understand the full scope of how self-care is impacted by systemic metabolic disease.

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Figure 1.

A photograph of the actual setup used in the study. Participants held the object (with embedded ATI transducers) between digits 1 and 2. The object was dropped onto a highly padded surface to protect the device.

Figure 2. Mean and standard error (SE) of sensation thresholds and coefficient of friction (μ) values for T2D and healthy control groups. **A:** Tactile sensation detection thresholds for the thumb and index fingers. **B:** Coefficient of friction (μ) values.

Table 1.

Clinical Characteristics of Patients and Averaged Control Information

Patient #	Age (years)	Sex	T2D Duration (months)	Glycated Hemoglobin (%)	Height (m)	Mass (kg)	Peripheral Neuropathy Diagnosis?
1	65	M	65	8.5	1.65	62.7	--
2	64	F	215	8.7	1.63	76.4	Yes
3	60	F	48	6.7	1.75	110.5	Yes
4	55	F	169	9.3	1.57	93.2	--
5	50	F	248	8.1	1.65	95.5	--
6	72	M	48	7.2	1.78	117.7	Yes
7	62	F	24	7.8	1.55	75.0	--
8	55	M	147	9	1.73	104.5	--
9	60	M	123	8.8	1.83	150.0	Yes
10	65	F	188	8.3	1.52	74.5	--
Mean $\pm$ SD	61 $\pm$ 6	--	128 $\pm$ 78	8.2 $\pm$ 0.8	1.67 $\pm$ 0.10	96.0 $\pm$ 26.0	--
Controls	60 $\pm$ 6	--	0	5.5 $\pm$ 0.2	1.73 $\pm$ 0.08	80.9 $\pm$ 12.1	--