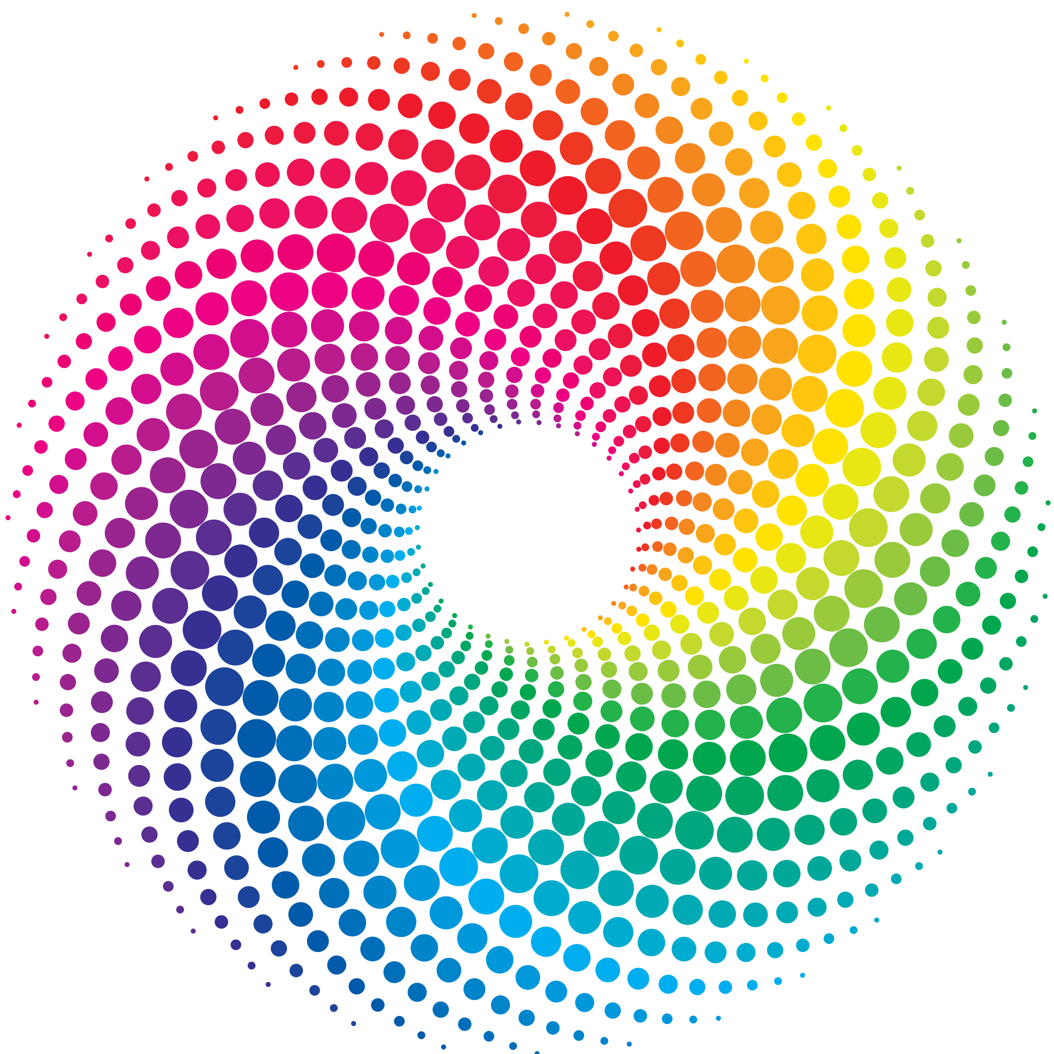
Journal of Case Management

CARE MANAGEMENT JOURNALS

The Journal of Long Term Home Health Care



SPRINGER PUBLISHING COMPANY

www.springerpub.com/cmj

Weight-Bearing Exercise and Foot Health in Native Americans

Elena Cuaderes, PhD, RN
Lise DeShea, PhD
College of Nursing, University of Oklahoma Health Sciences Center
W. Lyndon Lamb, DPM, CWS
Choctaw Nation Health Center, Talihina, Oklahoma

Diabetes contributes to sensory peripheral neuropathy, which has been linked to lower limb abnormalities that raise the risk for foot ulcers and amputations. Because amputations are a reason for pain and hospitalization in those with diabetes, it is of critical importance to gain insight about prevention of ulcer development in this population. Although the American Diabetes Association (ADA) now recommends that individuals with neuropathy can engage in moderate-intensity weight-bearing activity (WBA), they must wear appropriate footwear and inspect their feet daily. The physical forces and inflammatory processes from WBA may contribute to plantar characteristics that lead to ulcers. The purpose of this study was to compare neuropathic status and foot characteristics in Native Americans according to WBA classification. The t tests for unequal sample sizes found that exercisers had more difficulty sensing baseline temperature than nonexercisers, except at the right foot (all p values $< .05$). By dividing groups into no/low risk and high risk for ulcer, a majority showed no/low risk according to touch and vibration sense. Exercisers demonstrated higher surface skin temperature gradients at the first metatarsal head, a plantar site where wounds tend to form. The more consistently exercisers performed, the higher the plantar pressures were at the right second ($r = .24$, $p = .02$) and third metatarsal heads ($r = .26$, $p = .01$). Findings from this investigation do not refute current ADA recommendations and further intervention studies are needed that are longitudinal and measures WBA more accurately.

Keywords: diabetes; exercise; neuropathy; plantar foot

According to the Centers for Disease Control and Prevention (CDC; 2013c), diabetes mellitus (DM) continues to be a national health problem, and the most current data show that the numbers in adults have risen 3 times or more since 1980. A complication of DM is peripheral neuropathy (PN), which is thought to be a result of poorly controlled glycemia of long duration (Tesfaye & Selvarajah, 2012). Most recently, data from the Epidemiology of Diabetes Interventions and Complications Study demonstrated that current strategies used to control glycemia are not sufficient enough to slow the progression of or prevent PN (Martin, Albers, & Pop-Busui, 2014). But more seriously, an end point of neuropathy may be an infectious, nonhealing ulcer that can lead to an amputation of any part of the lower extremity. As of 2007, the age-adjusted rate was 5.7 per 1,000 for ulcer development and 6.4 per 1,000 for lower extremity neuropathy in individuals with DM (CDC, 2013a). Although the rate of nontraumatic lower extremity amputations in those with DM has steadily declined since 1988 (CDC, 2013b), it remains imperative that clinicians and scientists continue to find ways to prevent diabetic-associated neuropathic ulcers.

Basic to the prevention of diabetic neuropathy is the maintenance of a recommended serum glucose level. The American Diabetes Association (ADA; 2013) states that to control glucose, regular moderate-intensity aerobic activity for at least 150 min per week is advised. Studies have shown that walking for physical activity and health is preferred by many adults (Belza et al., 2004; Cuaderes, Lamb, Khan, & Lawrence, 2009; Cuaderes, Parker, & Burgin, 2004) and is, in fact, encouraged by the Surgeon General (U.S. Department of Health and Human Services, n.d.). However, walking may be unsafe for some individuals because of

Diabetes mellitus continues to be a national health problem, and the most current data show that the numbers in adults have risen 3 times or more since 1980.

Care Management Journals • Volume 15, Number 4 • 2014 © Springer Publishing Company
http://dx.doi.org/10.1891/1521-0987.15.4.184

complex processes that lead to changes of the lower limb tissue brought on by diabetic neuropathy. Not only can numbness of the foot from sensory neuropathy cause an individual to be unaware of tissue trauma from an unsafe walking environment but also motor neuropathy can produce atrophy of the intrinsic muscles of the foot that lead to foot deformities and limited joint mobility (Bernstein, 2003; Dinh & Veves, 2005). Furthermore, xerosis from autonomic neuropathy contributes to changes in ligaments and joints and to the formation of calluses (Freeman, 2007; Pavicic & Korting, 2006). Persons with diabetic neuropathy and peripheral arterial occlusive disease are at particular risk for problems because of the added influence of suboptimal tissue perfusion (ADA, 2003; Gibbons & Shaw, 2012; Williams, Price, & Harding, 2006). These and other changes leave the foot vulnerable to ulcer formation and infection and ultimately amputation. Through 2009, standards published by the ADA indicated that individuals with severe neuropathy should adhere to non-weight-bearing exercise. However, since 2010, moderate-intensity exercise, including the weight-bearing kind, has been encouraged as long as proper footwear is worn and individuals take the time to inspect their feet daily for signs of tissue trauma (ADA, 2013).

Among the various ethnic groups in the United States, the rate of diagnosed DM continues to be the highest among Native Americans (NAs) and Alaska Natives, after adjusting for population age differences (CDC, 2011). Knowing the impact of the amount and pattern of walking by this group of individuals with DM and neuropathy on foot characteristics may shed further light on the safety of this activity. Therefore, the purpose of this research study was to examine leisure-time, weight-bearing exercise in adult NAs according to neuropathic thresholds and plantar characteristics. More specifically, the following questions guided this study:

1. Are there differences in sensory neuropathy thresholds between exercisers and nonexercisers?
2. Are there differences in plantar pressure (PP), plantar skin hardness (PSH), plantar temperature symmetry (PTS), and plantar surface skin temperature gradient (SSTG) between exercisers and nonexercisers?
3. What are the associations between weight-bearing activity consistency (WBA[c]) and PP, PSH, PTS, and SSTG in exercisers?

REVIEW OF THE LITERATURE

Weight-Bearing Activity and Diabetic Sensory Neuropathy

Although pain is usually associated with acute neuropathy, clinical presentation of the chronic type is often insidious and typically begins as numbness starting in the toes and gradually traveling up the feet and legs in a "stocking" distribution pattern (Boulton, Malik, Arezzo, & Sosenko, 2004). Unaware of discomfort from the trauma of repetitive stress from WBA, the individual with loss of protective sensation (LOPS) from sensory neuropathy typically continues this action in the same pattern, placing the foot at risk for a diabetic foot ulcer (DFU). Nonetheless, it has been shown that increasing levels of cumulative stress do not necessarily result in plantar ulcers. In a cross-sectional study of controls and in people with DM (PWD) with and without a history of ulcers, more daily cumulative plantar forefoot stress from walking was the norm for those who were neuropathic and without a history of ulcers compared to those who had a history of ulcers (Maluf & Mueller, 2003).

There is evidence that supports the physical stress theory (Mueller & Maluf, 2002) in which significant deviations from usual WBA patterns can cause tissue injury. This was noted in a case report in which a sudden change in the walking pattern by an individual with a recently healed diabetic plantar ulcer resulted in reulceration (Armstrong et al., 2004). From more recent studies, conclusions about WBA and the risk of developing a DFU can be outlined as follows: (a) Walking did not increase the risk for DFU in those with sensory neuropathy (Lemaster, Reiber, Smith, Heagerty, & Wallace, 2003), (b) those with DM and a history of DFU may be prone to further injury at relatively lower levels of WBA (Armstrong et al., 2004; Maluf & Mueller, 2003), (c) tissue damage did not occur from the amount of cumulative walking but occurred from a rather specific pattern of this activity (Armstrong et al., 2004; Lott, Maluf, Sinacore, & Mueller, 2005), and (d) there was no difference between exercisers and nonexercisers in PPs or PSH, which are both known to occur prior to ulcer formation (Cuaderes, Lamb, et al., 2009).

> *The physical stress theory presents the idea that biological tissue responds predictably to different levels of stress in five ways—atrophy, maintenance, hypertrophy, injury, and death.*

Physical Stress Theory, Plantar Skin Hardness, and Plantar Pressures

The physical stress theory presents the idea that biological tissue responds predictably to different levels of stress in five ways—atrophy, maintenance, hypertrophy, injury, and death (Mueller & Maluf, 2002). Although the relative relationships between these levels are thought to be consistent among the general population, the absolute values of upper and lower thresholds of each response may vary between individuals. It has been proposed that if stress levels on tissue are higher or lower than routine loading, then the equal balance of tissue degeneration and production, as seen in the maintenance range, is altered. Hypertrophy (increased stress tolerance) results in tissue production that exceeds degeneration, and injury occurs when the stress exceeds the level that occurs with maintenance or hypertrophy. Death of tissue results from extreme deviations that are not adaptive.

Although the level of exposure to physical stress is a function of magnitude, time, and direction of force, tissue response is also affected by previous stress and rest experiences. Four factors that can modulate tissue response to stress are movement and alignment factors (i.e., physical activity), extrinsic factors (footwear), psychosocial factors (response to lower limb pain), and physiological factors (age, DM). Characteristics of the foot such as higher-than-normal PSH and PPs have been implicated as risk factors for developing a DFU, and both of these variables may be useful in monitoring the plantar foot subjected to WBA. Most DFUs occur at the metatarsal heads (MHs), which have been associated with pathological stiffening of the plantar tissue prior to ulcer formation (Gefen, Megido-Ravid, Azariah, Itzchak, & Arcan, 2001). Descriptive studies have generally found foot sole hardness to be the lowest in healthy individuals and the highest in PWD who have neuropathy and a history of ulceration (Charanya, Patil, Narayanamurthy, Parivalavan, & Visvanathan, 2004; Klaesner, Hastings, Zou, Lewis, & Mueller, 2002; Piaggesi et al., 1999; Thomas, Patil, Radhakrishnan, Narayanamurthy, & Parivalavan, 2003).

Neuropathy has also been generally associated with higher-than-normal dynamic PPs at the area of the MHs and hallux, the sites where ulcers are the most likely to develop (Abouaesha, van Schie, Griffths, Young, & Boulton, 2001; Frykberg et al., 1998; Lavery, Armstrong, Wunderlich, Tredwell, & Boulton, 2003; Murray, Young, Hollis, & Boulton, 1996; Pitei et al., 1999; Thomas et al., 2003; Veves, Murray, Young, & Boulton, 1992). Many factors have been found to explain high PPs, including the presence of calluses (Murray et al., 1996), decreased tissue thickness at the forefoot (Abouaesha et al., 2001), and changes in foot structure (Veves et al., 1992).

Plantar Foot Surface Skin Temperature

Knowing the surface skin temperature may be valuable in identifying an early pathological process from WBA. Some believe that it is more of the shear rather than the vertical stresses on the foot that contribute to ulcer formation (Hall, Shurr, Zimmerman, & Saltzman, 2004). Earlier investigations have demonstrated that repetitive mild-to-moderate stress on insensitive limbs can cause an inflammatory response, clinically evidenced by an increase in local skin temperatures (Beach & Thompson, 1979; Hall et al., 2004; Manley & Darby, 1980). Hyperemia is known to be an indicator of inflammation, and traditionally, sites of erythema are thought to be precursors of wound formation. However, clinicians and PWD who are neuropathic may be hindered in their ability to assess for erythema because of impaired reactive hyperemia, which has been attributable to dysfunctional unmyelinated C fibers (Bharara, Cobb, & Claremont, 2006; Richard, Lavigne, & Sotto, 2012).

METHOD

Participants

For this cross-sectional, descriptive study, participants from a large NA clinic located in southwestern United States underwent a one-time foot exam and walking trial and completed a structured questionnaire by interview. Data were collected at this clinic and protection of volunteers was facilitated through the Institutional Review Board of the University of Oklahoma Health Sciences Center and the Choctaw Nation. Volunteers were chosen conveniently based on the following inclusion criteria: diagnosis of Type 1 or 2 DM; client of the clinic; between the ages of 18 and 99 years; nonpregnant; able to read, write, and speak in English; and able to walk at least 30 ft without assistive devices. After explanation of study procedures and the consent was signed, each volunteer was interviewed for past year, leisure-time exercise habits, demographic information, and health parameters related to diabetes and foot care. They also agreed to have their bare feet examined for abnormal characteristics. If anything was judged to be highly pathological, the person was immediately referred to the podiatrist at the clinic. The last procedure was the walking trial to obtain PP measurements.

Materials and Procedure

The principal investigator (PI) conducted all interviews, examined and measured all feet, and primarily managed all walking trials. Activity category (exercisers/nonexercisers) was measured using the physical exercise questionnaire (PEQ). Developed by the PI of this study, the PEQ has been previously used to measure leisure-time activity in NAs of this study-site clinic (Cuaderes, Lamb, et al., 2009) and in NAs who attended a different health care clinic (Cuaderes et al., 2004). Reliability for items that measure frequency, duration, and intensity have yielded Cronbach's alphas of .94 and .93. Validity was demonstrated by showing that exercisers tended to be more highly self-motivated, possess higher self-efficacy, and perceive more benefits than barriers to exercise than nonexercisers (Cuaderes et al., 2004). However, this instrument has not been validated against more accurate measures of activity such as the pedometer or accelerometer. Description of this instrument and category determination has been reported in an earlier study (Cuaderes et al., 2004). WBA(c) was measured by calculating the number of months that the participant exercised consistently (most days of the week, most weeks of the month) during the past year and dividing this value by 12 to result in an index value. A higher index value indicated more consistency.

Foot temperature should be measured at a site on the dorsal surface before data collection procedures, and if less than 30° C, it should be warmed until the temperature reaches 30° C or higher.

Sensory thresholds were measured by touch (four sizes of the Semmes-Weinstein monofilament [SWM], 0.4, 4, 10, and 300 g, North Coast Medical, Inc., San Jose, CA), vibration (Bio-Thesiometer, Bio-Medical Instrument Co., Newbury, OH), and warm and cool thermal perception (NTE-2A Thermal Tester, Physitemp, Inc., Clifton, NJ). Sites tested with the SWM were the pulp of the hallux, the first, third and fifth MHs and the heel. Scoring was similar to that used by Carrington et al. (2002), but in this study, possible scores ranged from 4 to 8, with higher values indicative of less touch sense. The total foot threshold score was calculated as the average of values at all sites of each foot. SWM testing at the forefoot has been found to be only moderately reproducible (Mayfield & Sugarman, 2000) but possess high sensitivity (McGill, Molyneaux, Spencer, Heng, & Yue, 1999). Vibration threshold was assessed by using the method of limits as described by Goldberg and Lindblom (1979). The probe of the device was placed at the dorsomedial aspect of the first metatarsal bone and the pulp of the hallux, and the vibratory threshold of each left and right site was determined by calculating the average of two scores (vibration perception and vibration disappearance), yielding four scores. In a prospective multicenter trial, higher thresholds were found to be 86% sensitive, 56% specific, and possess a 32% positive predictive value for foot ulceration (Pham et al., 2000). Scoring of warm and cool perception thresholds were guided by an algorithm (Arezzo, 2006). After several placements of the probe of the thermal tester (just below the nail of the hallux of each foot for approximately two seconds) and multiple participant responses, a pattern emerged that resulted in values ranging from less than 3 to greater than 13. The lower the score, the smaller the gap between the perception and the baseline temperature. Arezzo (2006) recommends that foot temperature should be measured at a site on the dorsal surface before data collection procedures, and if less than 30° C, it should be warmed until the temperature reaches 30° C or higher. For this study, these directions were followed using a low-heat dry pad.

PSH, PP, and skin surface temperature were assessed using a handheld durometer (Model 1600, Type OO with National Institute Standards and Technology calibration certification, American Society for Testing Material 2240 standards, Rex Gauge Co., Buffalo Grove, IL), the F-Scan In-Shoe Pressure/Force Measurement Mapping System (Tekscan, Inc., Boston, MA), and an infrared dermal thermometer (Exergen DermaTemp 1001-LN, Watertown, MA), respectively. Measurements were obtained with volunteers assuming one of two positions: (a) prone position on an exam table with the leg bent at the knee and the plantar foot in a close parallel plane with the ceiling or (b) standing on one foot with body weight supported by both arms, holding onto a chair or exam table. The foot was supported by the researcher's nondominant hand, and the durometer was held with the other hand, vertically by gravity against the skin at the pulp of the hallux, first, third, and fifth MH, and heel. Each site was measured three times and a site-specific average value was calculated. Finally, a whole foot value was determined by calculating the average of scores at each site. In a previous investigation, interrater reliability of the durometer was found to be acceptable at these sites (Cuaderes, Khan, Azzarello, & Lamb, 2009). To collect data about dynamic PP, volunteers were required to wear athletic-type shoes with socks or hosiery either their own or that provided by the research study PI. A sensor was placed between the foot covering and the bottom, inside of the shoe and connected to the computer via a cable and cuff, which was placed at the ankle. Another cable from the cuff connected the sensor to the participant. On cue and after calibration procedures, the participant walked approximately 30 ft at a self-selected speed. At the completion of the procedure, each foot's data were displayed on an encrypted computer screen and saved. The highest pressures at each region, generated by the computer, were used to determine an average pressure for each foot. With accurate calibration, the F-Scan in-Shoe Pressure Mapping System measurement error has been found to be 1.3%–5.8% (Hsiao, Guan, & Weatherly, 2002).

To measure plantar temperature, the scanning probe was held 0.5–1.0 cm from the skin, one time at each site (pulp of the hallux; first, third, and fifth MH; and heel). The absolute difference between corresponding sites of each foot was calculated and designated as the site-specific SSTG value. PTS score was dichotomized as "greater than or equal to 2.2° C" or "less than 2.2° C." The dermal thermometer possesses an accuracy of ±0.1° C (Exergen Corporation, n.d.). To ascertain the extent of glycemic control of each volunteer, the most recent hemoglobin A1c (HgbA1c) value from the medical record was recorded.

RESULTS

Statistical Packages for the Social Sciences, Version 20.0 for Windows (SPSS, Inc., Cary, NC) was used for analysis of the data. For a description of the sample, see Tables 1 and 2. A power analysis indicated that a sample of 148 was sufficient to provide a power of 0.80 to detect a medium effect size of 0.50. A histogram of exercise scores revealed a bimodal distribution, with 55 nonexercisers having a mean score of 5.6 (SD = 2.4, range of 5–15) indicating

TABLE 1. Description of the Sample (N = 148)

Variable	Min	Max	*M*	*SD*	Variable	Frequency	%
Age	22.0	82.0	54.9	12.9	Female	85	57
BMI	20.1	59.7	34.5	6.9	Male	63	43
Duration of DM (years)	<0.1	41.0	10.9	9.8	Type 1	13	9
HgbA1c	4.7	14.0	7.8	1.0	Type 2	135	91

Note. BMI = body mass index; DM = diabetes mellitus; HgbA1c = hemoglobin A1c.

little or no WBA over the year and 93 exercisers having a mean score of 19.08 (SD = 1.98, range of 16–25) indicating moderate to high levels of activity in terms of frequency, duration, and intensity. The exercisers' scores were close to normal distribution (Kolmogorov-Smirnov Z = 0.80, p = .54). Of those participants who were deemed as exercisers, 96% reported that they walked, whereas 2% jogged, 1% used a stair stepper, and 1% played volleyball and basketball. Six participants were observed to have a wound dressing at particular sites on the plantar foot and one (also with a dressing) was observed to have an amputated hallux on one foot. Ten reported that they have experienced one or more ulcers from DM in the past.

To investigate the differences in sensory neuropathy thresholds between exercisers and nonexercisers, Aspin-Welch-Satterthwaite t tests for unequal sample sizes were computed to compare groups on mean touch, vibration, and warm/cool thermal thresholds. Higher means were anticipated for the nonexercisers than the exercisers, so one-tailed p values are reported. Three participants were missing an SWM score on at least one measure on the left foot, and three participants (including one with complete data on the left foot) were missing a score on at least one measure on the right foot. The data were not missing at random because of dressings or an amputation. To have complete data from the sample, a mean SWM score per foot was computed using the non-missing scores. For example, one person was missing one SWM score for the right foot, so the remaining scores—5, 5, 5, and 7—were averaged to produce a right foot touch perception threshold mean equal to 5.5. No significant differences in touch perception of both feet were found between groups (all p values > .05). No left foot vibration scores could be computed for one participant, and no right foot vibration scores could be observed for another. Like touch perception, the groups did not differ on vibration sense at any of the four sites (all p values > .05).

TABLE 2. Description of Sample—Exercise by Gender

	Male	Female	Total
Nonexerciser	27	28	55
Exerciser	36	57	93
Total	63	85	148

Because touch and vibration threshold did not differ between groups, sensory threshold scores for touch and vibration were dichotomized into groups according to the risk for foot ulcer formation. Clinically, if one cannot feel the touch of the 10-g SWM at a particular site, then there is a high risk for ulcer formation at this location (Mayfield & Sugarman, 2000; Pham et al., 2000). Therefore, all participants with total foot scores of 4, 5, or 6 were categorized as no or low risk, and all who scored 7 or 8 were deemed to be at high risk. It has been documented that a vibration score of 25–51 also places a person at risk for an ulcer (Perkins, Olaleye, Zinman, & Bril, 2001; Pham et al., 2000). Likewise, all who scored 1–24 were determined to be at no or low risk, and those who had values of 25–51 were considered high risk. Table 3 shows the number of exercisers and nonexercisers by no/low or high risk for developing a DFU. Most participants in both WBA groups were considered not to be neuropathic (88.5% touch sense and 84.5% vibration sense) in terms of having no or low risk for ulcer development.

Warm and cool thermal perception was measured for each foot, yielding four scores, one for each site; one participant had neither score for the left foot. For three of the four sites, exercisers consistently had significantly higher mean scores than nonexercisers, and differences were significant for left foot warm perception and both feet cool perception (all p values < .05; see Table 4 for details).

Aspin-Welch-Satterthwaite t tests for unequal sample sizes were used to assess the differences in PP, PSH, and SSTG between exercisers and nonexercisers. Exercisers were expected to have higher means than nonexercisers. Total PP scores were computed for each foot; one participant had no score for the left foot, and two other participants had no score for the right foot. Groups did not differ significantly on mean PP whole foot scores (all p values > .05). However, when individual sites on each foot were analyzed, it was found that exercisers possessed higher means at two locations: the right midfoot (exercisers M = 1.79, SD = 0.65; nonexercisers M = 1.61, SD = 0.51, p = .03) and the region of the left fourth and fifth toes (exercisers M = 2.41, SD = 1.51; nonexercisers M = 1.93, SD = 1.13, p = .02). One region, the left fourth MH,

TABLE 3. Description of Sample—Exercise by Ulcer Risk (Touch and Vibration Perception)

	Touch-SWM		Vibration	
	L/R	L/R	L/R	L/R
	No/low risk	High risk	No/low risk	High risk
Nonexerciser	49/50 (38%)	6/5 (31%)	44/44 (35.5%)	10/11 (45.5%)
Exerciser	81/81 (62%)	12/12 (69%)	81/79 (64.5%)	12/13 (54.5%)
Total	130/131 (88.5%)	18/17 (11.5%)	125/123 (84.5%)	22/24 (15.5%)

Note. SWM = Semmes-Weinstein Monofilaments; L = left foot; R = right foot. Percentages are an average of left and right feet.

showed a lower mean for exercisers ($M = 2.64$, $SD = 0.90$) than nonexercisers ($M = 3.04$, $SD = 1.47$), a difference that was not in the predicted direction.

PSH was measured at five locations on each foot. Seven participants were missing 1 or more of the 10 scores. Exercisers and nonexercisers did not differ significantly on any of the measures (all p values $> .05$). For PTS, all participants demonstrated less than 2.2° C difference between the 5 corresponding sites that were measured on left and right feet. However, exercisers ($M = 1.66$, $SD = 1.31$) had higher SSTG than nonexercisers ($M = 1.20$, $SD = 1.20$, $p = .02$) at the first MH. Pearson correlations were used to determine the associations between WBA(c), PSH, PP, and SSTG in exercisers. The only significant linear relationship was WBA(c) and PP and these were found at the right second MH ($r = .237$, $p = .02$) and the third MH ($r = .264$, $p = .01$).

TABLE 4. Comparison of Group Means on Warm and Cool Perception

	N	*M*	*SD*	*t*	*df*	*p* (one-tailed)
Left foot, warm						
Exercisers	93	10.0	4.6	—	—	—
Nonexercisers	54	8.6	4.5	1.8	145	.04*
Right foot, warm						
Exercisers	93	9.3	4.7	—	—	—
Nonexercisers	55	8.4	5.0	1.2	146	.12
Left foot, cool						
Exercisers	93	8.7	4.4	—	—	—
Nonexercisers	54	7.2	3.7	2.2	127.7	.01*
Left foot, cool						
Exercisers	93	9.1	4.4	—	—	—
Nonexercisers	55	7.3	3.6	2.8	132.9	.005*

Note. The Aspin-Welch-Satterthwaite t test was used because sample sizes were unequal.
*p is significant ≤ .05.

DISCUSSION

No significant differences in touch or vibration sense were found between exercise groups. However, cross tabulation of exercise groups by touch and vibration groups revealed a high number of participants who were not at high risk for DFU. One reason for this finding is the possibility, although not measured, that these people were regular visitors to the clinic and can be considered successful in self-care management of their DM to the end that the larger, myelinated fibers have not be sufficiently damaged from their disease. The current recommended standards state that individuals with diabetic neuropathy should inspect their feet daily for abnormalities (ADA, 2013), but for this study, no one reported daily inspection. However, 82% of exercisers reported that they wear either an athletic-type shoe or prescribed diabetic shoe for their activity. For all participants, 68% reported that they wear an athletic or prescription (orthopedic/diabetic) shoe for activities of daily living. It has also been documented that maintaining an HgbA1c around or less than 7% can reduce microvascular complications in nonpregnant adults (ADA, 2013); for this group, the mean was 7.8%.

Exercisers consistently had difficulty in their ability to feel warmth and coolness at the feet. Previous findings have shown that small fiber neuropathy may occur early in the course of DM, resulting in impairments of thermal sensation (McKemy, 2013; Pozzessere et al., 2002). Exercisers comprised about two-thirds of the sample; perhaps those who had findings that pointed to damage to the myelinated delta A and the unmyelinated C axons were the ones who were experiencing early onset neuropathy. Damage to these fibers is also responsible for pain in the lower limbs. Approximately 60% of the respondents in this investigation reported that they were currently taking medications for foot or leg pain. Cutoff points for risk of DFU development have not been established from thermal thresholds as measured by the NTE-2A Thermal Tester; however, it has been shown that large fiber disease is more likely to be associated with ulcer formation than small fiber neuropathy (Sosenko, Kato, Soto, & Bild, 1990). Some researchers have recommended that both vibration and thermal sense be examined to better predict the development of foot plantar wounds (Arezzo, Schaumburg, & Laudadio, 1986; Sosenko et al., 1990; Sosenko, Kato, Soto, Gadia, & Ayyar, 1988).

Because touch and vibration threshold did not differ between groups, sensory threshold scores for touch and vibration were dichotomized into groups according to the risk for foot ulcer formation.

Nonetheless, aging alone is well-known as a condition that can alter nerve function, and it has been found that pressure-induced vasodilation is impaired in healthy aging people compared to the healthy young. This blight is even worse in aging people with neuropathy (Fromy et al., 2009). In this investigation, participants had a mean age of 55 years, and older age correlated with decreased touch, vibration, and thermal perception at three sites on the foot. When age was dichotomized into two groups with the mean used as the dividing point, older age clearly differentiated those with increasing problems with vibration sense.

Groups did not differ on average PP total scores for either foot. However, they were different at the right midfoot and the region of the left fourth and fifth toes, with higher pressures at both sites for the exercisers and higher pressures for the nonexercisers at the region of the left fourth MH. Although some investigators have suggested that there is not a standard pressure threshold that if exceeded would be considered an at-risk site for the development of an ulcer, some have published a value. Peak pressures of more than 12.3 kg/cm^2 have been considered to be abnormal (Veves et al., 1992), but others have pointed out that people have been known to develop wounds at sites where pressures are considered normal (van Schie, 2005). Nevertheless, for this study, the mean total foot score for the left foot was 4.7 kg/cm^2 and for the right foot, $M = 4.6$ kg/cm^2. Only one person possessed a total foot value that was greater than 12.3 kg/cm^2. Damaged motor nerves cannot innervate muscles, and consequently, muscles will atrophy. In the foot, this results in a high arch with progression toward prominent MH and hammering of the toes with both conditions causing increased pressures at the MH and toe regions (Bus, Maas, de Lange, Michels, & Levi, 2005; Rahman et al., 2006; van Schie, 2005). It appears that both groups in this investigation may have been at some risk for developing an ulcer based on possible structural changes of the feet. It should also be kept in mind that body weight has been shown to contribute to increased pressures in children and adults (Dowling, Steele, & Baur, 2001; Mueller et al., 2003). Although the mean body mass index (BMI) was 34.6 and the mean weight for participants was 98.1 kg, there were no correlations between weight and PP and no links between BMI and PP. Increasing body weight tends to increase ground reaction forces but only mildly (van Deursen, 2004). A given PP value will decrease when the foot contact area becomes wider, and this is seen in heavier people during gait.

Poorly controlled diabetes causes nonenzymatic glycation of skin proteins, making the plantar foot prone to hyperkeratosis and callus formation (Gefen, 2003). Poor shoe fit, abnormal foot mechanics, and excessive WBA can also contribute to hyperkeratosis (Klaesner et al., 2002). And to add to this condition, diabetic autonomic neuropathy leads to xerosis, furthering the impact of stiffness of the plantar foot skin (Pavicic & Korting, 2006). In this study, WBA groups did not differ in PSH scores. In one study by Piaggesi and colleagues (1999), PWD and neuropathy possessed a mean PSH value of 51.18 at the heel, and this was significantly higher compared to PWD and no neuropathy ($M = 44.53$) and healthy controls ($M = 43.18$). In this study, the mean for the left heel was 50.24 and the mean for the right heel was 51.6. Interestingly, for this inquiry, men were different than women in PSH for all sites on the foot, except the heel, and these scores were higher. It has been found that males with Type 1 DM were 3 times more likely to have thicker plantar aponeurosis than females (Duffin, Lam, Kidd, Chan, & Donaghue, 2002).

The link between PP and PSH is worth mentioning. Thomas et al. (2003) surmises that as sensory neuropathy becomes worse, more hardness and thickening of the plantar soft tissue and fascia occur. It is thought that elasticity of the skin is reduced with stiffer tissue, resulting in decreased shock absorption, and this can be especially bothersome because the MHs and toes are primarily involved during the push-off stage of the gait cycle (van Deursen, 2004). Hsu and colleagues (2005) found impaired elastic tissue responses at the MHs of healthy older people without disease compared to younger individuals when the sites were subjected to repetitive stress. When skin is noncompliant, there is less diffusion of the skin over the contact area during contact time. With stiffer skin and no or minimal dispersal over the contact area, PP becomes higher. Although two studies found strong correlations between

Aging alone is well-known as a condition that can alter nerve function, and it has been found that pressure-induced vasodilation is impaired in healthy aging people compared to the healthy young.

> *The physical stress theory implies that significant deviations from routine exercise and the consequent imbalance between tissue degeneration and production may result in hypertrophy where tissue production exceeds degeneration.*

PP and PSH in PWD and healthy controls (Charanya et al., 2004; Thomas et al., 2003), this investigation found only one site (left heel) where PSH and PP were weakly associated. The physical stress theory implies that significant deviations from routine exercise and the consequent imbalance between tissue degeneration and production may result in hypertrophy where tissue production exceeds degeneration. Excessive tissue production results in increased collagen content, fibril diameter, and tissue thickness (Mueller & Maluf, 2002). Armstrong and colleagues (2004) found that those who deviated more from activity were the ones who ulcerated. Thus, for this study, it was thought that those who showed less consistency of exercise would be the people who would show higher PP and PSH. On the contrary, study findings were such that those who exercised on a more regular basis were the ones who tended to have the higher PP at the right second and third MH sites where ulcers most commonly form (Abouaesha et al., 2001; Frykberg et al., 1998; Lavery et al., 2003; Murray et al., 1996; Pitei et al., 1999; Thomas et al., 2003; Veves et al., 1992). Interestingly for this study, being female who consistently exercised was associated with the higher PP.

Plantar skin temperature monitoring had been deemed useful for detecting risk for ulcer development and temperature differences between corresponding sites on left and right feet of greater than 2.2° C were indication of high risk for ulcer formation (Armstrong et al., 2007; Armstrong, Lavery, Liswood, Todd, & Tredwell, 1997; Lavery et al., 2004). Although the present investigation found no one who exhibited differences of greater than 2.2° C, mean SSTG was higher in exercisers than nonexercisers at the first MH. This may be noteworthy because increased skin temperatures of the foot have been discovered in early diabetes-related neuropathy (Bharara et al., 2006) and reactive hyperemic blood flow has been found to be lower in PWD compared to healthy controls (Tur, Yosipovitch, & Bar-On, 1991). Furthermore, after skin loading, postischemic blood flow is lower than normal in PWD and neuropathy (Gibney et al., 2002). Vita and colleagues (2004) have found that as reactive hyperemia becomes more impaired, markers of inflammation increase in healthy and diseased individuals, including those with diabetes. It has been stated that higher levels of shear stress coupled with higher PP are more injurious to tissue than either one alone. However, Goldstein and Sanders (1998) found that by applying repetitive mechanical stress acutely or chronically and by shear and vertical methods to healthy pig skin, signs of inflammation were not different between groups. Other researchers, in their animal study, have discovered that the reactive hyperemic response in stressed, healthy skin was almost half that as seen in nonstressed skin (Herrman, Knapp, Donofrio, & Salcido, 1999).

LIMITATIONS AND CONCLUSIONS

This study involved a convenience sample of patients at one clinic in the southwestern United States, which could limit the external validity of the results. On the other hand, the naturally occurring groups of exercisers and nonexercisers may reflect the reality of PWD, who vary considerably in their exercise habits, foot health, access to care, psychosocial support, and so forth. As noted earlier, the sample may have consisted of participants who already were careful about monitoring the health of their feet, perhaps limiting the range of results and dampening any potential differences between exercisers and nonexercisers. The findings from this investigation are also limited by the lack of random assignment of people to groups and the lack of a manipulated independent variable; thus, causal mechanisms cannot be drawn between exercise habits and the observed differences in the two groups.

The way that exercise and its consistency were measured was clearly a limitation and self-reports of routine; leisure-time exercise has been found to have problems with validity and reliability (Prince et al., 2008). Interestingly, two previous studies with NA by this investigator found similar rates of self-reported exercisers (Cuaderes et al., 2004; Cuaderes, Lamb, et al., 2009). Future research calls for use of direct measures that provide more precision and accuracy. For instance, built-in accelerometers in smartphones for measuring physical activity have recently been described (Weiss & Lockhart, 2012). Although the PI judged all participants to be cooperative before data collection, another limitation is the way that neuropathy was measured. Quantitative sensory testing depends heavily on a cooperative participant, and scores represent, in part, a subjective response (Shy et al., 2003). Future descriptive studies need to focus on longitudinal strategies, which would reveal more about exercise, neuropathy, and foot characteristics over time. Measures of foot self-care habits over time should also expose information that would enlighten researchers about WBA and the risks for developing ulcers. Exercise intervention studies might divulge changes in plantar characteristics in correspondence with changes in consistency of the activity. However, these study results do have implications for NAs who routinely exercise, have diabetes, and show signs of lower limb neuropathy. Participants did not demonstrate a neuropathy at a level that placed them at risk for a foot wound, and the reason for this may be the fact that they are clients of a clinic that regularly provides podiatry services. It's possible that exercisers were in the early stages of neuropathy, especially because approximately two-thirds of the participants were assigned to this category and two-thirds of the sample reported taking medication for pain in the lower limb, although the nature of this pain was not explored.

Participants exhibited higher PP than healthy people (Chevalier, Hodgins, & Chockalingam, 2010), but they were not of the magnitude published by Veves et al. (1992). Given this evidence and that there may have been no large fiber neuropathy present, it can be further concluded that this sample was not at risk for developing ulcers of the feet. It is also plausible that study volunteers were experiencing some early onset neuropathy because their PSH values were the same as another sample who had neuropathy (Piaggesi et al., 1999). Although study participants did not demonstrate a high risk for ulcer formation according to the temperature magnitude as explained by researchers (Armstrong et al., 2007; Armstrong et al., 1997; Lavery et al., 2004), exercisers did have higher differences in foot temperature than nonexercisers and implications from these findings should further be explored.

In this study, most exercisers reported moderate-intensity activity and they demonstrated no differences from nonexercisers in two plantar foot characteristics, PP and PSH. Higher levels of these are thought to place a PWD at risk for ulcer formation. Although this investigation lends support for the current ADA activity recommendations (ADA, 2013), other factors such as age, gender, severity of neuropathy, BMI, and exercise pattern may need to be considered when a clinician is assessing a patient's risk for ulcer formation.

REFERENCES

Abouaesha, F., van Schie, C. H., Griffths, G. D., Young, R. J., & Boulton, A. J. (2001). Plantar tissue thickness is related to peak plantar pressure in the high-risk diabetic foot. *Diabetes Care*, *24*(7), 1270–1274.

American Diabetes Association. (2003). Peripheral arterial disease in people with diabetes. *Diabetes Care*, *26*(12), 3333–3341.

American Diabetes Association. (2013). Standards of medical care in diabetes—2013. *Diabetes Care*, *36*(Suppl. 1), S11–S66. http://dx.doi.org/10.2337/dc13-S011

Arezzo, J. C. (2006). *Manual for the assessment of thermal perception threshold (TPT)*. New York, NY: Albert Einstein College of Medicine, Yeshiva University Bronx.

Arezzo, J. C., Schaumburg, H. H., & Laudadio, C. (1986). Thermal sensitivity tester. Device for quantitative assessment of thermal sense in diabetic neuropathy. *Diabetes*, *35*(5), 590–592.

Armstrong, D. G., Holtz-Neiderer, K., Wendel, C., Mohler, M. J., Kimbriel, H. R., & Lavery, L. A. (2007). Skin temperature monitoring reduces the risk for diabetic foot ulceration in high-risk patients. *American Journal of Medicine*, *120*(12), 1042–1046.

Armstrong, D. G., Lavery, L. A., Holtz-Neiderer, K., Mohler, M. J., Wendel, C. S., Nixon, B. P., & Boulton, A. J. (2004). Variability in activity may precede diabetic foot ulceration. *Diabetes Care*, *27*(8), 1980–1984.

Armstrong, D. G., Lavery, L. A., Liswood, P. J., Todd, W. F., & Tredwell, J. A. (1997). Infrared dermal thermometry for the high-risk diabetic foot. *Physical Therapy*, *77*(2), 169–175.

Beach, R. B., & Thompson, D. E. (1979). Selected soft-tissue research: An overview from Carville. *Physical Therapy*, *59*(1), 30–33.

Belza, B., Walwick, J., Shiu-Thornton, S., Schwartz, S., Taylor, M., & LoGerfo, J. (2004). Older adult perspectives on physical activity and exercise: Voices from multiple cultures. *Preventing Chronic Disease*, *1*(4), A09.

Bernstein, R. K. (2003). Physical signs of the intrinsic minus foot. *Diabetes Care*, *26*(6), 1945–1946.

Bharara, M., Cobb, J. E., & Claremont, D. J. (2006). Thermography and thermometry in the assessment of diabetic neuropathic foot: A case for furthering the role of thermal techniques. *International Journal of Lower Extremity Wounds*, *5*(4), 250–260.

Boulton, A. J., Malik, R. A., Arezzo, J. C., & Sosenko, J. M. (2004). Diabetic somatic neuropathies. *Diabetes Care*, *27*(6), 1458–1486.

Bus, S. A., Maas, M., de Lange, A., Michels, R. P. J., & Levi, M. (2005). Elevated plantar pressures in neuropathic diabetic patients with claw/hammer toe deformity. *Journal of Biomechanics*, *38*(9), 1918–1925. http://dx.doi.org/10.1016/j.jbiomech.2004.07.034

Carrington, A. L., Shaw, J. E., Van Schie, C. H., Abbott, C. A., Vileikyte, L., & Boulton, A. (2002). Can motor nerve conduction velocity predict foot problems in diabetic subjects over a 6-year outcome period? *Diabetes Care*, *25*(11), 2010–2015.

Centers for Disease Control and Prevention. (2011). *National diabetes fact sheet: National estimates and general information on diabetes and prediabetes in the United States, 2011*. Atlanta, GA: U.S. Department of Health and Human Service, Centers for Disease Control and Prevention. Retrieved from http://www.cdc.gov/diabetes/pubs/pdf/ndfs_2011.pdf

Centers for Disease Control and Prevention. (2013a). *Age-adjusted hospital discharge rates for peripheral arterial disease (PAD), ulcer/inflammation/infection (ULCER), or neuropathy as first-listed diagnosis per 1,000 diabetic population, United States, 1988-2007. Diabetes Public Health Resource*. Retrieved from http://www.cdc.gov/diabetes/statistics/hosplea/diabetes_complications/fig2_pop.htm

Centers for Disease Control and Prevention. (2013b). *Crude and age-adjusted hospital discharge rates for nontraumatic lower extremity amputation per 1,000 diabetic population, United States, 1988-2009. Diabetes Public Health Resource*. Retrieved from http://www.cdc.gov/diabetes/statistics/lea/fig3.htm

Centers for Disease Control and Prevention. (2013c). *Number (in millions) of civilian, noninstitutionalized adults with diagnosed diabetes, United States, 1980-2011. Diabetes Public Health Resource*. Retrieved from http://www.cdc.gov/diabetes/statistics/prev/national/figadults.htm

Charanya, G., Patil, K. M., Narayanamurthy, V. B., Parivalavan, R., & Visvanathan, K. (2004). Effect of foot sole hardness, thickness and footwear on foot pressure distribution parameters in diabetic neuropathy. Proceedings of the Institution of Mechanical Engineers. *Part H: Journal of Engineering in Medicine*, *218*(6), 431–443.

Chevalier, T. L., Hodgins, H., & Chockalingam, N. (2010). Plantar pressure measurements using an in-shoe system and a pressure platform:

A comparison. *Gait & Posture*, *31*(3), 397–399. http://dx.doi.org/10.1016/j.gaitpost.2009.11.016

Cuaderes, E., Khan, M. M., Azzarello, J., & Lamb, W. L. (2009). Reliability and limitations of the durometer and PressureStat to measure plantar foot characteristics in Native Americans with diabetes. *Journal of Nursing Measurement*, *17*(1), 3–18.

Cuaderes, E., Lamb, L., Khan, M., & Lawrence, G. (2009). Weight-bearing activity and foot parameters in Native Americans with diabetes with and without foot sensation. *Journal of Cultural Diversity*, *16*(1), 10–20.

Cuaderes, E., Parker, D., & Burgin, C. (2004). Leisure time physical activity in adult Native Americans. *Southern Online Journal of Nursing Research*, *1*(5). Retrieved from http://www.resourcenter.net/images/SNRS/Files/SOJNR_articles/iss01vol05.htm

Dinh, T. L., & Veves, A. (2005). A review of the mechanisms implicated in the pathogenesis of the diabetic foot. *International Journal of Lower Extremity Wounds*, *4*(3), 154–159.

Dowling, M. A., Steele, J. R., & Baur, A. L. (2001). Does obesity influence foot structure and plantar pressure patterns in prepubescent children? *International Journal of Obesity & Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, *25*(6), 845–852.

Duffin, A. C., Lam, A., Kidd, R., Chan, A. K. F., & Donaghue, K. C. (2002). Ultrasonography of plantar soft tissues thickness in young people with diabetes. *Diabetic Medicine*, *19*(12), 1009–1013. http://dx.doi.org/10.1046/j.1464-5491.2002.00850.x

Exergen Corporation. (n.d.). *Dermatemp 1001: User's manual and reference book*. Retrieved from http://www.manualslib.com/manual/50340/Exergen-Dt-1001.html

Freeman, R. (2007). Autonomic peripheral neuropathy. *Neurologic Clinics*, *25*(1), 277–301.

Fromy, B., Sigaudo-Roussel, D., Gaubert-Dahan, M.-L., Rousseau, P., Abraham, P., Benzoni, D., . . . Saumet, J. L. (2009). Aging-associated sensory neuropathy alters pressure-induced vasodilation in humans. *Journal of Investigative Dermatology*, *130*(3), 849–855.

Frykberg, R. G., Lavery, L. A., Pham, H., Harvey, C., Harkless, L., & Veves, A. (1998). Role of neuropathy and high foot pressures in diabetic foot ulceration. *Diabetes Care*, *21*(10), 1714–1719.

Gefen, A. (2003). Plantar soft tissue loading under the medial metatarsals in the standing diabetic foot. *Medical Engineering & Physics*, *25*(6), 491–499.

Gefen, A., Megido-Ravid, M., Azariah, M., Itzchak, Y., & Arcan, M. (2001). Integration of plantar soft tissue stiffness measurements in routine MRI of the diabetic foot. *Clinical Biomechanics*, *16*(10), 921–925.

Gibbons, G. W., & Shaw, P. M. (2012). Diabetic vascular disease: Characteristics of vascular disease unique to the diabetic patient. *Seminars in Vascular Surgery*, *25*(2), 89–92.

Gibney, J., Weis, U., Turner, B., Meeking, D. R., Cansfield, J., Watts, G. F., . . . Cummings, M. H. (2002). Postischemic microcirculatory blood flow correlates negatively and independently with plasma C-reactive protein in longstanding type 1 diabetes. *Diabetes Care*, *25*(4), 802–803.

Goldberg, L. M., & Lindblom, U. (1979). Standardised method of determining vibratory perception thresholds for diagnosis and screening in neurological investigation. *Journal of Neurology, Neurosurgery & Psychiatry*, *42*(9), 793–803.

Goldstein, B., & Sanders, J. (1998). Skin response to repetitive mechanical stress: A new experimental model in pig. *Archives of Physical Medicine & Rehabilitation*, *79*(3), 265–272.

Hall, M., Shurr, D. G., Zimmerman, M. B., & Saltzman, C. L. (2004). Plantar foot surface temperatures with use of insoles. *Iowa Orthopaedic Journal*, *24*, 72–75.

Herrman, E. C., Knapp, C. F., Donofrio, J. C., & Salcido, R. (1999). Skin perfusion responses to surface pressure-induced ischemia: Implication for the developing pressure ulcer. *Journal of Rehabilitation Research & Development*, *36*(2), 109–120.

Hsiao, H., Guan, J., & Weatherly, M. (2002). Accuracy and precision of two in-shoe pressure measurement systems. *Ergonomics*, *45*(8), 537–555.

Hsu, C. C., Tsai, W. C., Chen, C. P. C., Shau, Y. W., Wang, C. L., Chen, M. J. L., & Chang, K. J. (2005). Effects of aging on the plantar soft tissue properties under the metatarsal heads at different impact velocities. *Ultrasound in Medicine & Biology*, *31*(10), 1423–1429. http://dx.doi.org/10.1016/j.ultrasmedbio.2005.05.009

Klaesner, J. W., Hastings, M. K., Zou, D., Lewis, C., & Mueller, M. J. (2002). Plantar tissue stiffness in patients with diabetes mellitus and peripheral neuropathy. *Archives of Physical Medicine & Rehabilitation*, *83*(12), 1796–1801.

Lavery, L. A., Armstrong, D. G., Wunderlich, R. P., Tredwell, J., & Boulton, A. J. M. (2003). Predictive value of foot pressure assessment as part of a population-based diabetes disease management program. *Diabetes Care*, *26*(4), 1069–1073.

Lavery, L. A., Higgins, K. R., Lanctot, D. R., Constantinides, G. P., Zamorano, R. G., Armstrong, D. G., . . . Agrawal, C. M. (2004). Home monitoring of foot skin temperatures to prevent ulceration. *Diabetes Care*, *27*(11), 2642–2647.

Lemaster, J. W., Reiber, G. E., Smith, D. G., Heagerty, P. J., & Wallace, C. (2003). Daily weight-bearing activity does not increase the risk of diabetic foot ulcers. *Medicine & Science in Sports & Exercise*, *35*(7), 1093–1099.

Lott, D. J., Maluf, K. S., Sinacore, D. R., & Mueller, M. J. (2005). Relationship between changes in activity and plantar ulcer recurrence in a patient with diabetes mellitus. *Physical Therapy*, *85*(6), 579–588.

Maluf, K. S., & Mueller, M. J. (2003). Novel Award 2002. Comparison of physical activity and cumulative plantar tissue stress among subjects with and without diabetes mellitus and a history of recurrent plantar ulcers. *Clinical Biomechanics*, *18*(7), 567–575.

Manley, M. T., & Darby, T. (1980). Repetitive mechanical stress and denervation in plantar ulcer pathogenesis in rats. *Archives of Physical Medicine & Rehabilitation*, *61*(4), 171–177.

Martin, C. L., Albers, J. W., & Pop-Busui, R. (2014). Neuropathy and related findings in the diabetes control and complications trial/epidemiology of diabetes interventions and complications study. *Diabetes Care*, *37*, 31–38. http://dx.doi.org/10.2337/dc13-2114

Mayfield, J. A., & Sugarman, J. R. (2000). The use of the Semmes-Weinstein monofilament and other threshold tests for preventing foot ulceration and amputation in persons with diabetes. *Journal of Family Practice*, *49*(11 Suppl.), S17–S29.

McGill, M., Molyneaux, L., Spencer, R., Heng, L. F., & Yue, D. K. (1999). Possible sources of discrepancies in the use of the Semmes-Weinstein monofilament. Impact on prevalence of insensate foot and workload requirements. *Diabetes Care*, *22*(4), 598–602.

McKemy, D. D. (2013). The molecular and cellular basis of cold sensation. *ACS Chemical Neuroscience*, *4*(2), 238–247.

Mueller, M. J., Hastings, M., Commean, P. K., Smith, K. E., Pilgram, T. K., Robertson, D., & Johnson, J. (2003). Forefoot structural predictors of plantar pressures during walking in people with diabetes and peripheral neuropathy. *Journal of Biomechanics*, *36*(7), 1009–1017. http://dx.doi.org/10.1016/S0021-9290(03)00078-2

Mueller, M. J., & Maluf, K. S. (2002). Tissue adaptation to physical stress: A proposed "Physical Stress Theory" to guide physical therapist practice, education, and research. *Physical Therapy*, *82*(4), 383–403.

Murray, H. J., Young, M. J., Hollis, S., & Boulton, A. J. (1996). The association between callus formation, high pressures and neuropathy in diabetic foot ulceration. *Diabetic Medicine*, *13*(11), 979–982.

Pavicic, T., & Korting, H. C. (2006). Xerosis and callus formation as a key to the diabetic foot syndrome: Dermatologic view of the problem and its management. *Journal der Deutschen Dermatologischen Gesellschaft*, *4*(11), 935–941.

Perkins, B. A., Olaleye, D., Zinman, B., & Bril, V. (2001). Simple screening tests for peripheral neuropathy in the diabetes clinic. *Diabetes Care*, *24*(2), 250–256.

Pham, H., Armstrong, D. G., Harvey, C., Harkless, L. B., Giurini, J. M., & Veves, A. (2000). Screening techniques to identify people at high risk for diabetic foot ulceration: A prospective multicenter trial. *Diabetes Care*, *23*(5), 606–611.

Piaggesi, A., Romanelli, M., Schipani, E., Campi, F., Magliaro, A., Baccetti, F., & Navalesi, R. (1999). Hardness of plantar skin in diabetic neuropathic feet. *Journal of Diabetes and its Complications*, *13*(3), 129–134.

Pitei, D. L., Lord, M., Foster, A., Wilson, S., Watkins, P. J., & Edmonds, M. E. (1999). Plantar pressures are elevated in the neuroischemic and the neuropathic diabetic foot. *Diabetes Care*, *22*(12), 1966–1970.

Pozzessere, G., Rossi, P., Gabriele, A., Cipriani, R., Morocutti, A., Di Mario, U., & Morano, S. (2002). Early detection of small-fiber neuropathy in diabetes: A laser-induced pain somatosensory-evoked potentials and pupillometric study. *Diabetes Care*, *25*(12), 2355–2358.

Prince, S., Adamo, K., Hamel, M., Hardt, J., Connor Gorber, S., & Tremblay, M. (2008). A comparison of direct versus self-report measures for assessing physical activity in adults: A systematic review. *International Journal of Behavioral Nutrition and Physical Activity*, *5*(56), 1–24.

Rahman, M. A., Aziz, Z., Rajendra Acharya, U., Ha, T. P., Kannathal, N., Ng, E. Y. K., . . . Fang, S. C. (2006). Analysis of plantar pressure in diabetic type 2 subjects with and without neuropathy. *ITBM-RBM*, *27*(2), 46–55. http://dx.doi.org/10.1016/j.rbmret.2006.03.001

Richard, J. L., Lavigne, J. P., & Sotto, A. (2012). Diabetes and foot infection: More than double trouble. *Diabetes/Metabolism Research Reviews*, *28*(Suppl. 1), 46–53.

Shy, M. E., Frohman, E. M., So, Y. T., Arezzo, J. C., Cornblath, D. R., Giuliani, M. J., . . . Weimer, L. H. (2003). Quantitative sensory testing: Report of the therapeutics and technology assessment subcommittee of the American Academy of Neurology. *Neurology*, *60*(6), 898–904.

Sosenko, J. M., Kato, M., Soto, R., & Bild, D. E. (1990). Comparison of quantitative sensory threshold measures for their association with foot ulceration in diabetic patients. *Diabetes Care*, *13*(10), 1057–1061.

Sosenko, J. M., Kato, M., Soto, R. A., Gadia, M. T., & Ayyar, D. R. (1988). Specific assessments of warm and cool sensitivities in adult diabetic patients. *Diabetes Care*, *11*(6), 481–483.

Tesfaye, S., & Selvarajah, D. (2012). Advances in the epidemiology, pathogenesis and management of diabetic peripheral neuropathy. *Diabetes/Metabolism Research Reviews*, *28*(Suppl. 1), 8–14.

Thomas, V. J., Patil, K. M., Radhakrishnan, S., Narayanamurthy, V. B., & Parivalavan, R. (2003). The role of skin hardness, thickness, and sensory loss on standing foot power in the development of plantar ulcers in patients with diabetes mellitus—A preliminary study. *The International Journal of Lower Extremity Wounds*, *2*(3), 132–139.

Tur, E., Yosipovitch, G., & Bar-On, Y. (1991). Skin reactive hyperemia in diabetic patients. A study by laser Doppler flowmetry. *Diabetes Care*, *14*(11), 958–962.

U.S. Department of Health and Human Services. (n.d.). *Walking and walkability*. Retrieved from http://www.surgeongeneral.gov/initiatives/walking/

Van Deursen, R. (2004). Mechanical loading and off-loading of the plantar surface of the diabetic foot. *Clinical Infectious Diseases*, *39*(Suppl. 2), S87–S91.

Van Schie, C. H. M. (2005). A review of the biomechanics of the diabetic foot. *The International Journal of Lower Extremity Wounds*, *4*(3), 160–170.

Veves, A., Murray, H. J., Young, M. J., & Boulton, A. J. (1992). The risk of foot ulceration in diabetic patients with high foot pressure: A prospective study. *Diabetologia*, *35*(7), 660–663.

Vita, J. A., Keaney, J. F., Larson, M. G., Keyes, M. J., Massaro, J. M., Lipinska, I. P., . . . Benjamin, E. J. (2004). Brachial artery vasodilator function and systemic inflammation in the Framingham offspring study. *Circulation*, *110*(23), 3604–3609.

Weiss, G. M., & Lockhart, J. W. (2012, July). *The impact of personalization on smartphone-based activity recognition.* Paper presented at the Association for the Advancement of Artificial Intelligence Workshop on Activity Context Representation: Techniques and Languages, Toronto, Canada.

Williams, D. T., Price, P., & Harding, G. (2006). The influence of diabetes and lower limb arterial disease on cutaneous foot perfusion. *Journal of Vascular Surgery*, *44*(4), 770–775. http://dx.doi.org/10.1016/j.jvs.2005.06.040

Elena Cuaderes, PhD, RN, has worked with older individuals with diabetes and supervised their exercise in a cardiac rehabilitation program in the past, and Dr. Cuaderes has investigated weight-bearing activity and diabetic neuropathy on plantar foot characteristics since 2005. Her work with Native Americans and leisure-time activity started with her dissertation.

Lise DeShea, PhD, is a quantitative psychologist. She has investigated emergency room utilization and return on investment in care management for Oklahoma's Medicaid agency. She serves as a consultant to nursing graduate students and faculty in addition to co-authoring her third statistics textbook.

W. Lyndon Lamb, DPM, CWS, is board-certified by the American Academy of Wound Management. He is a member of the

American Podiatric Medical Association. He has worked at the Choctaw Nation Health Center, Diabetic Wellness Center in Talihina, Oklahoma, since 2004 as a surgical podiatrist.

Acknowledgments. Research reported in this publication was supported by the National Institute on Minority Health and Health Disparities of the National Institutes of Health under Award No. P20MD000528 and with collaboration with the Choctaw Nation of Oklahoma. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health or the Choctaw Nation of Oklahoma.

Correspondence regarding this article should be directed to Elena Cuaderes, PhD, RN, University of Oklahoma Health Sciences Center, College of Nursing, 1100 N. Stonewall Ave., Oklahoma City, OK 73117. E-mail: elena-cuaderes@ouhsc.edu