Prevalence of Lower-Extremity Disease in the U.S. Adult Population ≥40 Years of Age With and Without Diabetes

1999–2000 National Health and Nutrition Examination Survey

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OBJECTIVE — Although lower-extremity disease (LED), which includes lower-extremity peripheral arterial disease (PAD) and peripheral neuropathy (PN), is disabling and costly, no nationally representative estimates of its prevalence exist. The aim of this study was to examine the prevalence of lower-extremity PAD, PN, and overall LED in the overall U.S. population and among those with and without diagnosed diabetes.

RESEARCH DESIGN AND METHODS — The analysis consisted of data for 2,873 men and women aged ≥40 years, including 419 with diagnosed diabetes, from the 1999–2000 National Health and Nutrition Examination Survey. The main outcome measures consisted of the prevalence of lower-extremity PAD (defined as ankle-brachial index <0.9), PN (defined as ≥1 insensate area based on monofilament testing), and of any LED (defined as either PAD, PN, or history of foot ulcer or lower-extremity amputations).

RESULTS — Of the U.S. population aged ≥40 years, 4.5% (95% CI 3.4–5.6) have lower-extremity PAD, 14.8% (12.8–16.8) have PN, and 18.7% (15.9–21.4) have any LED. Prevalence of PAD, PN, and overall LED increases steeply with age and is higher ($P < 0.05$) in non-Hispanic blacks and Mexican Americans than non-Hispanic whites. The prevalence of LEDs is approximately twice as high for individuals with diagnosed diabetes (PAD 9.5% [5.5–13.4]; PN 28.5% [22.0–35.1]; any LED 30.2% [22.1–38.3]) as the overall population.

CONCLUSIONS — LED is common in the U.S. and twice as high among individuals with diagnosed diabetes. These conditions disproportionately affect the elderly, non-Hispanic blacks, and Mexican Americans.

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Lower-extremity disease (LED), including lower-extremity peripheral arterial disease (PAD) and peripheral neuropathy (PN), is disabling and costly (1–5). PAD, which can be accompanied by intermittent claudication or rest pain, may necessitate revascularization procedures and seriously diminish health-related quality of life. Even when asymptomatic, PAD may decrease mobility and bone mineral density (1,2,4,6) and is a strong predictor of subsequent cardiovascular disease (CVD) and mortality (3,7,8). PN also erodes health-related quality of life, and when it occurs in combination with PAD, as is frequently the case with diabetes, it can lead to foot ulcers and nontraumatic amputations (1,9).

Despite the array of complications of LED, its epidemiology in the U.S. has not been well characterized. Previous prevalence estimates for PAD from the U.S. and Europe, for example, have ranged from 5–20% in general populations aged >50 years (10–18). The prevalence of PN defined by symptoms or impaired vibration sensation in populations with diabetes has ranged from 10 to 42% (19–24). Part of this variation is probably due to methodological differences, including variations in definitions, populations, and measurements. There have been no nationally representative estimates of the prevalence of PAD or PN in the overall U.S. population or those with diabetes. In addition, previous studies have not examined the combined burden of PAD and PN or the degree to which these conditions are specific to individuals with diabetes.

Because of the lack of population-based data on LEDs in the U.S. and the potential to reduce its burden through pharmacological or behavioral interventions (1,4,25–28), new measurements of these conditions were incorporated in the 1999–2000 National Health and Nutrition Examination Survey (NHANES). The objectives of the present study were to determine the prevalence of LED, including lower-extremity PAD and PN in the U.S. among both the overall population and individuals with diagnosed diabetes.

RESEARCH DESIGN AND METHODS

— The NHANES is a nationally representative survey of the U.S. civilian noninstitutionalized population conducted by the National Center for Health Statistics of the Centers for Disease

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Abbreviations: ABI, ankle-brachial index; CVD, cardiovascular disease; LED, lower-extremity disease; NHANES, National Health and Nutrition Examination Survey; PAD, peripheral arterial disease; PN, peripheral neuropathy.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Control and Prevention (29). Since 1999, NHANES staff have conducted interviews and performed physical examinations on a continuous basis. Participants are interviewed in their homes to obtain information on health history, health behaviors, and risk factors. Participants subsequently undergo the physical examination at a mobile examination center. The procedures to select the sample and conduct the interview and examination have been specified elsewhere (30). Informed consent was obtained from all participants, and the institutional review board of the National Center for Health Statistics approved the protocol.

This study was based on the initial 2 years of the continuous NHANES (1999–2000). Among the 4,274 individuals aged ≥40 years who were eligible for the survey, 3,185 (74%) were interviewed and 2,875 (67%) received the health examination. All participants aged ≥40 years who received the physical examination (n = 2,875) were invited to undergo assessment of LEDs, which included noninvasive tests for PAD and PN, as well as physical inspection for foot lesions and abnormalities. Two individuals were excluded from the entire examination because they had bilateral amputations (n = 2). Individuals with ankle-brachial index (ABI) ≥1.5 in both legs (n = 2) or in one leg (n = 4) if the ABI value for the other leg was missing were excluded, as these individuals were believed to have medial arterial calcification, which prevents accurate measurement of arterial perfusion. Data on PAD were missing on both feet for 492 individuals (17%) and data on PN were missing for 346 individuals (12%), owing to either equipment or data capture failure, limited time available to conduct the examination, participant refusal, or some other unspecified reason. An incompressible artery, absent pulse, or some other condition related to the disease may have also led to missing data, but this information was unavailable for analysis. Thus, complete information on PAD, PN, and all LED data were available on 2,375, 2,527, and 2,300 individuals, respectively, aged ≥40 years.

Assessment of PAD

PAD was assessed by determining the ABI, which is the ratio of systolic blood pressure in the ankles (posterior tibial vessels) to that in the right arm (right brachial vessel). Measurements were taken with the participant in the supine position using an 8.1-MHz Doppler probe attached to a vascular testing device (Parks Mini-Lab IV, model 3100). Trained technicians followed a standard protocol (31). For participants aged 40–59 years, two blood pressure measurements were taken at each site (right arm, both ankles), and left and right ABI were calculated as the mean of the two measurements at the right and left ankle, respectively, divided by the mean of the two brachial blood pressure measurements. For logistic reasons, participants aged ≥60 years (n = 1,605) received only one measurement at each site, and thus, a single measurement was used to calculate the right and left ABIs.

Our primary analyses defined PAD as an ABI <0.9 in either leg. The ≤0.9 cut point has been shown to have a high sensitivity and specificity in angiographic studies (4,32). We also examined the prevalence ABI <0.7, which has been defined as moderate-to-severe PAD. PAD cases were classified as symptomatic if the participant reported "yes" when asked during the home interview (before examination) whether they ever get calf pain in either leg while walking.

Assessment of PN

PN was assessed using self-reported symptoms and by testing of foot sensation with a standard monofilament (5.07-gauge Semmes-Weinstein nylon) according to a standard protocol (31). Health technicians applied pressure with the monofilament at three sites (plantar, first metatarsal head; plantar, fifth metatarsal head; and plantar, hallux) on the bottom of each foot (i.e., a total of six sites). The monofilament was applied until it buckled, then held for 1 s. A site was considered insensate if the participant incorrectly determined when the monofilament was applied to the foot on two of three applications. Impaired sensation was quantified by the total number of insensate areas for both feet (range 0–6), and PN was defined as ≥1 insensate area. Previous studies have found ≥1 insensate area to be highly predictive of ulcers and amputation and to have moderately high sensitivity (~85%) and specificity (~80%) based on vibration testing and ulcer history and prevalence (1,33–35). Participants were also asked during the interview whether in the last 3 months they had had numbness, loss of feeling, or painful sensations or tingling in their feet. PN cases were classified as symptomatic if the participant reported yes to this question.

Other assessments of LED

Participants were also asked whether they ever had an ulcer or sore on their leg or foot that took >4 weeks to heal. During the examination, health technicians also recorded the history of lower-extremity amputation. Ten individuals with a toe or foot amputation were identified. We defined any LED as a presence of either PAD (ABI <0.9), PN (≥1 insensate areas), foot ulcers, or lower-extremity amputation. We defined symptomatic LED as having either PAD or PN symptoms.

Diabetes and other measurements

A history of physician-diagnosed diabetes, hypertension, stroke, and CVD (congestive heart failure, coronary heart disease, angina, heart attack) as well as age, race/ethnicity, and smoking was assessed by questionnaire (30). Because of the limited sample, only estimates for non-Hispanic whites, non-Hispanic blacks, and Mexican Americans are reported, although data for other race/ethnicity groups are included in total estimates. Individuals were classified as never, former, and current smokers based on their responses to questions about smoking at least 100 cigarettes during their lifetime and whether they were currently smoking. Additionally, height and weight were measured with a standard protocol and used to compute BMI (kg/m^2).

Statistical analyses

Our primary analyses estimated the prevalence of the diseases of interest in the overall population and within several subgroups. All analyses were conducted using SUDAAN software to take into account the complex sampling design and to obtain estimates representative of the noninstitutionalized U.S. population aged ≥40 years (36). For each of the primary outcomes, we used the largest sample number available to estimate prevalence. For PAD and PN, this was 2,375 and 2,527 individuals, respectively. For any LED, we used the number of individuals who had all variables (PAD, PN, amputations, ulcers) present (n = 2,300). Variance estimates were calculated using the "delete one" jackknife method (37). The 95% CIs were computed using the critical value for a t dis-

Table 1—*Sociodemographic characteristics of the U.S. population aged ≥ 40 years, 1999–2000*

Characteristics	Overall sample size (*n*)	Overall weighted	No diabetes weighted	Diabetes weighted
Mean age (years)	2,873	56.9	56.3	62.2
Age (years)				
40–49	718	35.9	38.0	17.2
50–59	550	25.3	25.7	22.0
60–69	757	19.9	18.3	33.9
70–79	526	13.0	12.3	19.5
≥80	322	5.9	5.7	7.4
Sex				
Male	1,404	46.7	46.4	50.0
Female	1,469	53.3	53.6	50.0
BMI (kg/m^2)*				
<25	804	31.1	32.5	17.4
25–29	1,052	34.8	35.5	28.6
≥30	959	32.7	30.9	50.0
Race/ethnicity				
Non-Hispanic white	1,327	75.1	76.8	59.7
Non-Hispanic black	548	9.6	8.7	17.2
Mexican American	761	4.5	4.4	6.2
Smoking*				
Current	517	20.1	20.7	14.6
Former	964	32.9	32.4	37.0
Never	1,387	47.0	46.8	48.4
Hypertension*				
Yes	1,186	36.0	33.8	55.8
No	1,663	63.6	65.8	43.6
CVD*†				
Yes	376	11.5	9.8	26.4
No	2,493	88.5	90.2	73.6
Stroke*				
Yes	154	3.9	3.3	9.0
No	2,716	96.1	96.6	91.1
Diabetes*				
Yes	419	9.9	0	100
No	2,451	90.1	100	0

Data are means or percent. *Age-specific comparisons were not applicable because some of the estimates were statistically unreliable; †CVD was defined as history of congestive heart failure, coronary heart disease, angina, or heart attack.

tribution with the appropriate number of degrees of freedom for each subgroup (SDJ1REPN-1). Age-adjusted prevalence estimates were calculated using the direct method, which were age adjusted to the 2000 projected U.S. population using three age-groups: 40–59, 60–74, and ≥75 years (38). Estimates with a relative standard error >30% were considered statistically unreliable and are identified as such in tables. Statistical hypotheses were tested univariately at the 0.05 level using a *t*-statistic.

Because there were missing data for 17% of the PAD and 12% of the PN samples, we examined the characteristics of individuals with missing data. Compared with individuals with sufficient PAD data, those individuals with missing PAD data were significantly more likely to be >60 years (49 vs. 37%; $P < 0.01$), female (61 vs. 52%; $P < 0.01$), non-Hispanic black (14 vs. 9%; $P < 0.01$), and diabetic (18 vs. 9%; $P < 0.001$) but did not significantly differ with regard to CVD or current smoking status. Individuals with missing PN data did not significantly differ from those with PN data in terms of age, sex, diabetes, CVD, or current smoking status but were more likely to be non-Hispanic black (17%) than individuals with PN data (9%) ($P < 0.001$).

Because of these differences, we further examined the potential impact of nonresponse by adjusting the original sampling weights according to methods published by Lohr (39). Examination of findings using these adjusted weights led to only minor differences in point and variance estimates (0.1–0.8%); thus, in this study, we present all estimates using the original 2-year examination weights.

RESULTS — The general characteristics of the study population, weighted to be representative of the U.S. population ≥40 years of age, are shown in Table 1. The mean age of this segment of the population was estimated to be 57 years, with 9.6% non-Hispanic black and 4.5% Mexican American. In addition, by self-report, 9.9% had diabetes, 36.0% had hypertension, and 11.5% had CVD. Compared with the overall population, the diabetic population was older (mean age 62 years), consisted of more nonwhites, had higher BMI values, fewer smokers, and a higher prevalence of hypertension, CVD, and stroke.

Among U.S. adults aged ≥40 years, an estimated 4.5% had PAD (ABI <0.9) (Fig. 1), among whom almost two-thirds were asymptomatic (i.e., no calf pain with walking) and almost one-third (1.3% [95% CI 0.7–1.8]) had ABI <0.7 (data not shown). Prevalence of PN (≥1 insensate area) was 14.8%, among whom over three-fourths were asymptomatic. Almost one-half of those with PN had two or more insensate areas (6.8% [5.3–8.2]), and over one-fourth had three or more such areas (3.8% [2.8–4.9]) (data not shown). Overall, 18.7% of the U.S. population aged ≥40 years had at least one LED condition (including PAD, PN, or a history of ulcer or amputation), among whom 71% were asymptomatic.

The prevalence of PAD (9.5%), PN (28.5%), and any LED (30.2%) was approximately twice as high among individuals with diagnosed diabetes as in the overall population, whereas prevalence of ulcers (7.7%) was almost three times as high. Like the overall population, most cases of PAD (78%), PN (62%), and any LED (56%) were asymptomatic among individuals with diagnosed diabetes.

Prevalence by demographic factors

The prevalence of PAD, PN, and of any LED all increased steeply with age (Table 2). For example, although the prevalence of PAD was only 1% among individuals aged 40–49 years, it roughly doubled

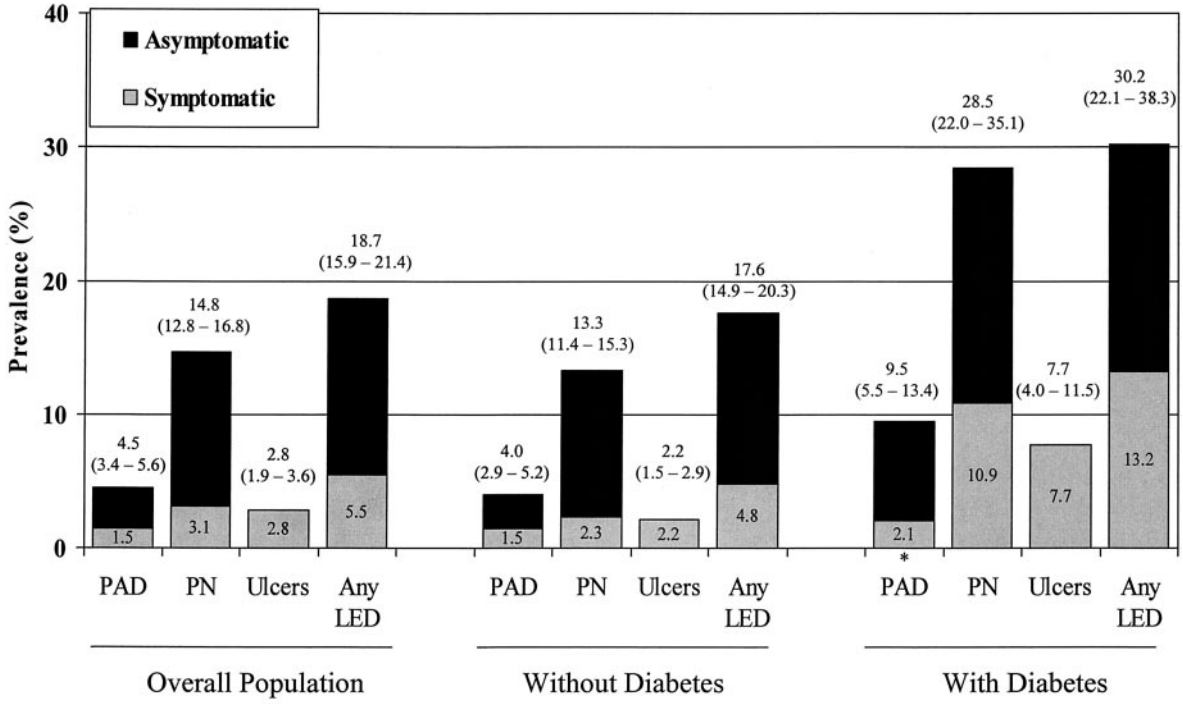

Figure 1—*Prevalence of LED among the overall, nondiabetic population, and diabetic population aged ≥40 years in the U.S., 1999–2000. *Does not meet standard of statistical reliability and precision (relative SE >30%).*

each decade, reaching 12% in those aged 70–79 years and 22% among individuals aged ≥80 years. The prevalence of PN rose from 8.1% in the 40- to 49-year age-group to 34.7% in the ≥80-years age-group. From age 50 years, the prevalence of any LED increased by roughly 10 percentage points each decade.

The age-adjusted prevalence of PAD did not significantly differ by sex, but men had a higher prevalence of PN (18.2%) and any LED (22.9%) than women (PN 12.6%; any LED 16.8%; $P < 0.05$ for each). The age-adjusted prevalence of PAD was higher among non-Hispanic blacks (9.6%; $P < 0.05$) and Mexican Americans (6.8%; $P < 0.05$) than among non-Hispanic whites (4.8%) (Table 2). Similarly, the age-adjusted prevalence of PN was higher among non-Hispanic blacks and Mexican Americans than among non-Hispanic whites. Prevalence of any LED was also lowest among non-Hispanic whites (19.0%), intermediate among Mexican Americans (23.9%; $P < 0.05$), and highest among non-Hispanic blacks (29.8%; $P < 0.05$). Differences in neuropathy and any LED between race/ethnicity tended to be greater among women than among men.

The age-adjusted prevalence of PAD did not differ appreciably between individuals with PN (6.3% [95% CI 3.0–9.6]) and those without PN (5.0% [3.8–6.2]) (data not shown). The prevalence of PN tended to be higher among those with PAD (23.1% [6.6–39.6]) than those without PAD (14.2% [12.1–16.4]), although this difference was not statistically significant (data not shown).

CONCLUSIONS — Despite the extensive toll that LED takes in terms of physical disability, amputation, mortality risk, and economic costs, no previous national prevalence estimates for either the general or diabetic population exist (1–9). We used data from the 1999–2000 NHANES, which was the first national study to use ABI and monofilament testing. We estimate that among the U.S. noninstitutionalized population aged ≥40 years, roughly 5% have lower-extremity PAD, 15% have PN, and 19% have at least one LED condition. The prevalence of these conditions is roughly twice as high among individuals with diagnosed diabetes, as ~10% have lower-extremity PAD, 29% have PN, and 30% have at least one LED condition. Consistent with previous studies, we found that most cases of PAD and PN are asymptomatic (2,3,10).

Much of the burden of both lower-extremity PAD and PN may be preventable or more effectively managed. PAD is thought to share a common atherosclerotic etiology with coronary heart disease and stroke (3,7,12,13). The efficacy of interventions to prevent PAD is not known, but surgical interventions, exercise training, smoking cessation, aspirin, and antiplatelet and lipid-lowering drugs may each be effective in managing the condition (3,4,25,26). The presence of PAD may also be an indication for broad management of CVD risk factors because of its association with stroke, myocardial infarction, and cardiovascular mortality (7,8), but studies suggest that patients with PAD are less aggressively managed than those with coronary artery disease (40).

Among people with diabetes, the progression of PN can be slowed by intensive glycemic control (27,28), and early detection and aggressive care of ulcers may reduce risk of amputation (9,41). Regular foot examinations are considered essential for individuals with diabetes, whereas the detection and management of high-risk feet among individuals without diabetes has received much less attention. Given the range of potential modifiable risk factors for LED, these national estimates provide a key baseline from which

Table 2—*Age-specific and age-adjusted prevalence of LED by sex and race/ethnicity in the U.S. population aged ≥40 years*

	PAD*			PN			Any LED		
Characteristics	Cases (*n*)	%	95% CI	Cases (*n*)	%	95% CI	Cases (*n*)	%	95% CI
n	2,375			2,527			2,300		
Age specific									
Age (years)									
40–49†	11	1.0‡	0.26–1.7	54	8.1	4.8–11.3	75	10.7	7.0–14.3
50–59	12	2.3‡	0.41–4.3	64	10.8	7.3–14.3	75	13.3	9.1–17.4
60–69	37	4.8	2.6–7.1	149	17.5§	13.8–21.1	167	23.1§	18.8–27.4
70–79	55	12.1	8.2–16.0	138	28.4§	24.3–32.5	156	37.5§	32.2–42.7
≥80	47	22.4	13.8–31.1	95	34.7§	26.9–42.5	96	44.8§	37.8–51.7
Age adjusted (to 2000 U.S. population)									
Sex									
Men†	80	4.8	3.5–6.1	283	18.2	15.1–21.4	319	22.9	19.4–26.4
Women	82	5.1	3.6–6.6	217	12.6§	10.9–14.4	250	16.8§	14.3–19.3
Race/ethnicity									
Non-Hispanic white†	81	4.8	3.6–5.9	229	14.4	12.3–16.6	274	19.0	16.3–21.7
Non-Hispanic black	43	9.6§	6.5–12.8	106	21.9§	17.9–25.9	121	29.8§	23.5–36.0
Mexican American	32	6.8§	5.4–8.2	134	19.4§	15.8–23.0	145	23.9§	21.0–26.8
Men									
Non-Hispanic white†	45	4.7	3.2–6.2	141	17.9	14.2–21.7	170	23.0	19.0–26.9
Non-Hispanic black	21	9.7§	5.0–14.4	51	22.4	16.4–28.4	57	27.8	19.8–35.7
Mexican American	13	5.3	2.6–8.0	74	21.4	17.1–25.6	77	24.9	20.2–29.5
Women									
Non-Hispanic white†	36	4.7	2.9–6.6	88	11.2	9.1–13.4	104	15.2	12.3–18.1
Non-Hispanic black	22	9.7§	5.3–14.0	55	21.2§	16.4–25.9	64	31.0§	23.3–38.8
Mexican American	19	9.2‡	1.5–16.9	60	17.7	11.2–24.2	68	23.6§	19.1–28.1

*Age-specific comparisons were not applicable because some of the estimates were statistically unreliable; †reference group for statistical analyses; ‡does not meet standard of statistical reliability and precision (relative SE >30%); §significantly different from reference group ($P < 0.05$).

to examine the impact of future prevention efforts for this disease.

Few previous studies have examined the relationship between race/ethnicity and LED. We found that non-Hispanic blacks had the highest prevalence of PAD and PN, and Mexican Americans had levels that were higher than whites but lower than blacks. In the Cardiovascular Health Study, nonwhite race was associated with twice the odds of PAD (12). Similarly, the San Antonio Heart Study reported 61% greater odds of PAD among Mexican Americans than among non-Hispanic whites, but these findings were not significant (42). In a previous national sample of individuals with diabetes, both blacks and Mexican Americans had a higher prevalence of neuropathy symptoms than whites but only among those with diabetes for <15 years (23).

Our PAD estimates are lower than those of most previous reports. For example, previous estimates using 60- to 70-year-olds have ranged from 3 to 12% (11–13,18), as compared with ~5% for this age-group in our study. Estimates similar to or lower than those in our study were observed for white women in the Study of Osteoporotic Fractures (18), women without CVD in the Cardiovascular Health Study (12), and men in the Honolulu Heart Study (3–5% among those aged 65–69 years and 6–8% among those aged 70–74 years) (15). Samples from the Netherlands, Scotland, and men in the Cardiovascular Health Study all had prevalence estimates ranging from 8 to 12% for individuals of similar age as compared with 4.5% in our study (11–13). Previous estimates among diabetic populations have been considerably higher among European studies (11,13) and in the Honolulu Heart Study (15) but comparable in India and Australia (17,43).

The lower prevalence we observed could be due to several factors. Although previous studies sampled individuals from population-based sources, there may have been varying levels of representativeness of the underlying population. It is possible that previous studies have selectively recruited individuals of worse health status, or alternatively, that the demands of the NHANES examination leads to a slightly healthier sample. It is also possible that there was a higher rate of missing data, and unmeasureable ABI values in our sample led to an underestimating prevalence. Finally, this could reflect a reduction in PAD incidence or CVD risk factors over the past decade, as most previous studies were not conducted recently. This would be consistent with the observed reductions in CVD risk factors and mortality in the U.S. (44), but no longitudinal data exist to support or refute this speculation. Unfortunately, the lack of consistency in methods of recruiting, conducting, and reporting studies of LED makes it difficult to identify the key reasons explaining discrepancies in prevalence.

Because of the wide variation in definitions, it is also difficult to compare PN prevalence estimates between populations and over time. In the San Luis Valley

Diabetes Study, prevalence based on vibration threshold testing was 28% in the diabetic population and 4% in those without diabetes (45). Similarly, a hospital-based population in the U.K. had a prevalence of 29% (22), whereas a recent population-based study in Australia found a prevalence of 10% among individuals with diabetes (17). In a recent study in Northwest England that used methods similar to our study, prevalence of insensate feet among individuals with known diabetes was 21% (35). Other studies have defined PN based on vibration threshold, temperature perception, and ankle reflexes, finding that prevalence ranged from 42 to 47% (24,46). In the 1989 National Health Interview Survey, 38% of the diabetic population and 11% of those without diabetes had symptoms of sensory neuropathy, which was defined as numbness, loss of feeling, pain, or tingling in the last 3 months. Our study is the first nationally representative study in the U.S. to examine PN based on an objective measure of sensation (monofilament testing) and, thus, should serve as an important baseline for surveillance of PN.

There are several potential limitations to our study. First, NHANES does not include individuals from nursing homes and other similar institutions. Thus, our estimates probably underestimate those of the total U.S. population. Second, within the NHANES sample, ABI measurements were not available for 17% of the sample, many of whom may have had disease. Adjustment procedures were used to decrease the potential nonresponse bias, but not all factors can be accounted for, and thus, it is more likely that we have underestimated than overestimated the true disease prevalence. Third, our prevalence definitions are based on noninvasive indicators of disease. The ABI has emerged as a measurement of choice for PAD because it is a noninvasive and inexpensive yet a more sensitive (~95%) and specific (~99%) indicator of arterial obstruction (33,34) than previously used measures, such as intermittent claudication or pulse inspection. For PN, electrophysiological and nerve conduction studies could give more accurate diagnostic data, but for epidemiological settings, monofiliament testing is still an improvement over the previously used symptom questionnaires in terms of sensitivity, specificity, and prediction of adverse outcomes (1,33,34). With regard to both PAD and PN, however, it is important to note that the choice of disease cut points are based on clinic-based, symptomatic samples of individuals for ABI (4,10) and diabetic individuals for monofilament testing (1,1,34). Thus, validation studies are still needed among broader population samples to further refine the use of these measures for epidemiological surveillance purposes. Finally, we lacked detailed information on symptoms and lacked power to examine prevalence estimates within subgroups, including individuals with undiagnosed diabetes and impaired glucose tolerance, as well as age, sex, or ethnicity subgroups among individuals with diabetes.

This study provides the first national estimates in the U.S. of the civilian, noninstitutionalized population and, thus, the most broadly representative estimates to date for the U.S. population. Because much of this burden may be preventable through existing interventions, these data should provide policy makers, clinicians, and researchers with key data for prevention and treatment efforts. This study also provides baseline data to monitor the prevalence of LED, its consequences, and the success of prevention efforts.

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