

**ORIGINAL ARTICLE**

# Peripheral Nerve Function and Lower Extremity Muscle Power in Older Men



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**Abstract**

**Objective:** To assess whether sensorimotor peripheral nerve function is associated with muscle power in community-dwelling older men.

**Design:** Longitudinal cohort study with 2.3±0.3 years of follow-up.

**Setting:** One clinical site.

**Participants:** Participants (n=372; mean age ± SD, 77.2±5.1y; 99.5% white; body mass index, 27.9±3.7kg/m<sup>2</sup>; power, 1.88±0.6W/kg) at 1 site of the Osteoporotic Fractures in Men Study (N=5994).

**Interventions:** Not applicable.

**Main Outcome Measures:** A nerve function ancillary study was performed 4.6±0.4 years after baseline. Muscle power was measured using a power rig. Peroneal motor nerve conduction amplitude, distal motor latency, and mean f-wave latency were measured. Sensory nerve function was assessed using 10-g and 1.4-g monofilaments and sural sensory nerve conduction amplitude and distal latency. Peripheral neuropathy symptoms at the leg and feet were assessed by self-report.

**Results:** After adjustments for age, height, and total body lean and fat mass, 1 SD lower motor ( $\beta = -.07, P < .05$ ) and sensory amplitude ( $\beta = -.09, P < .05$ ) and 1.4-g ( $\beta = -.11, P < .05$ ) and 10-g monofilament insensitivity ( $\beta = -.17, P < .05$ ) were associated with lower muscle power/kg. Compared with the effect of age on muscle power ( $\beta$  per year,  $-.05; P < .001$ ), this was equivalent to aging 1.4 years for motor amplitude, 1.8 years for sensory amplitude, 2.2 years for 1.4-g monofilament detection, and 3.4 years for 10-g detection. Baseline 1.4-g monofilament detection predicted a greater decline in muscle power/kg. Short-term change in nerve function was not associated with concurrent short-term change in muscle power/kg.

**Conclusions:** Worse sensory and motor nerve function were associated with lower muscle power/kg and are likely important for impaired muscle function in older men. Monofilament sensitivity was associated with a greater decline in muscle power/kg, and screening may identify an early risk for muscle function decline in late life, which has implications for disability.

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Lower extremity muscle power is an important determinant of late-life physical function.<sup>1,2</sup> Muscle power, a measure of contractile force and shortening speed, has been linked to risk of falls,<sup>3</sup> mobility loss measured by physical performance tests such as walking, chair stands, and stair climbing,<sup>4-11</sup> and self-reported functional status<sup>2,12</sup> in older adults. Compared with strength,

muscle power declines more steeply with age<sup>4,13</sup> and may be more strongly associated with certain measures of mobility.<sup>6,9,11,12</sup> Moreover, training programs designed to improve muscle power and velocity of movement may be more effective at improving physical performance than those that solely incorporate basic resistance training.<sup>14-16</sup>

Poor muscle power in late life and its unique relationship with mobility may be due, at least in part, to impairments in peripheral nerve function.<sup>17-22</sup> The components of power, force and velocity production, are likely dependent on the number and firing rate of motor units.<sup>23</sup> In addition, afferent input and impaired sensory nerve function may play an important role in muscle and physical function<sup>24-26</sup>; this is believed to occur through loss of proprioception.<sup>24,26-28</sup> Like muscle power, peripheral nerve function declines with age,<sup>29-33</sup> and has similarly been linked with physical function limitations and impairments<sup>32,34</sup> and an increased risk of falls.<sup>35-37</sup> The 1999–2000 National Health and Nutrition Examination Survey showed that 35% of adults 80 years and older had impaired nerve function measured using simple screening for reduced sensation at the foot.<sup>33</sup> Additionally, both poor motor and sensory peripheral nerve function have been related to reduced lower extremity quadriceps strength in the Health Aging and Body Composition Study; muscle power was not assessed in this study.<sup>25</sup>

Despite the independent relationships of muscle power and nerve function with mobility-related outcomes, whether peripheral nerve function loss is a determinant of muscle power decline has not been assessed. In a longitudinal cohort study of older men, we evaluated whether sensory and motor peripheral nerve function measures, commonly used in clinical evaluations and neurologic studies, are related to lower extremity muscle power cross-sectionally and longitudinally. We hypothesize that worse and declining nerve function is associated with poor and declining muscle power.

## Methods

### Study population

We used data from a nerve function ancillary study in which 372 participants had nerve function and power measured during the first visit, and 241 participants had these repeated during a second visit. The ancillary study was performed at the Monongahela Valley site 4.6±0.4 years after the 2000 to 2002 baseline visit. The second visit occurred 2.3±0.3 years later. This ancillary study was part of the Osteoporotic Fractures in Men Study, which is a cohort of community-dwelling ambulatory men (N = 5994) 65 years and older enrolled between March 2000 and April 2002 at 6 U.S. clinic sites (Birmingham, AL; Minneapolis, MN; Palo Alto, CA; Monongahela Valley near Pittsburgh, PA; Portland, OR; and San Diego, CA; n = 1005 in Pittsburgh at baseline). Eligibility for the main study included the ability to walk without assistance of another person or an aide, the ability to provide self-reported data, the ability to understand and sign an informed consent, the absence of bilateral hip replacements, the absence of a medical condition that would result in imminent death, and anticipated residence near a clinic site for the duration of the study period. The primary recruitment strategy was mailing invitations to men living in the surrounding communities of

clinic sites. Supplementary strategies included community and senior newspaper advertisements and presentations to community groups. The study protocol was approved by the University of Pittsburgh Institutional Review Board, and written informed consent was obtained from all participants before testing. Of 662 men with nerve function measured during the first visit of the ancillary study, 372 had muscle power measured and were included in the cross-sectional analyses. Reasons for muscle power not being measured included temporary equipment failure (n = 205), refusal of participants (n = 11), and inability because of participants' physical limitations (n = 74). Participants with missing cross-sectional muscle power data did not differ by age but had a slightly higher body mass index (BMI) (28.6kg/m<sup>2</sup> vs 27.9kg/m<sup>2</sup>, *P* = .03) and a higher prevalence of diabetes (27.9% vs 17.0%, *P* < .001). Of the participants included in the cross-sectional analysis, 279 returned for the second visit. The change analysis included data from 241 participants with complete nerve function and muscle power data from the first and second visits of the ancillary study. During the second visit, 1 participant refused muscle power testing, and 53 participants were unable because of physical limitations. Participants with missing data for the change analysis were older (78.8y vs 76.3y, *P* < .001) but had a similar BMI and prevalence of diabetes.

### Peripheral nerve measures

Nerve conduction was measured bilaterally on the deep peroneal motor and sural sensory nerves using an automated nerve conduction study device (NC-stat<sup>a</sup>),<sup>38</sup> which has been previously validated in healthy older adults with criterion standard nerve conduction studies (correlation coefficient >95%).<sup>39</sup> Participants' feet were warmed to at least 30°C if they were <30°C before testing. Parameters recorded from the peroneal motor nerve included the motor amplitude of the compound muscle action potential (in millivolts), measured from baseline to the negative peak of the compound muscle action potential waveform; the distal motor latency (in milliseconds), which is the time from the stimulus to the onset of motor activity; and the mean F-wave latency (in milliseconds), which is the mean value of the time from the stimulus to the onset of F-wave activity. Sensory nerve measures included the sural nerve action potential (SNAP) sensory amplitude (in microvolts), which is the difference between the negative and positive peak of the SNAP waveform, and the distal sensory latency (in milliseconds), which is the time from the stimulus to the negative peak of the SNAP. Light (1.4-g) and standard (10-g) monofilament sensitivity were defined as the ability to detect 3 of 4 touches at the dorsum of the great toes. Insensitivity was defined as the inability to detect 3 touches. The standard monofilament was performed only if the participant could not feel the light monofilament. Sensory nerve conduction was performed on the nondominant side. Motor nerve conduction and monofilament testing were performed on both sides unless technical difficulty occurred. Self-reported peripheral neuropathy symptoms occurring within the past 12 months included (1) numbness or tingling; (2) sudden stabbing, burning, pain, or aches; and (3) an open or persistent sore, or gangrene on either the feet or legs. All measures were repeated at the follow-up visit.

### Lower extremity muscle power

Muscle power was measured using a single leg press (Nottingham Leg Extensor Power Rig<sup>b</sup>).<sup>40</sup> Participants were seated with their

#### List of abbreviations:

**BMI** body mass index  
**SNAP** sural nerve action potential

arms crossed over their chest and instructed to push down on a pedal with 1 foot as hard and as fast as possible through a full range of motion. The maximum power output in watts from 5 trials was used. Both sides were tested unless the participant had a hip replacement on 1 side. The ratio of power to body weight in kilograms was chosen as the outcome of interest since it may better reflect the ability to move one's body weight when performing everyday activities.

To correspond with sensory nerve conduction measures, muscle power data on the nondominant side were used in the analysis unless prohibited by missing data, which occurred because some participants could not be tested because of physical limitations. In the case that no sensory nerve conduction data were available, muscle power data were matched to the side in which motor nerve conduction testing, monofilament testing, or both, were performed. The number of participants with discordant sides analyzed because of missing data was minimal ( $n=5$  for motor nerve conduction,  $n=1$  for monofilament testing).

### Additional covariates

All models were adjusted for age and height, measured using a stadiometer. Weight was measured with a calibrated balance beam scale but was not included as a covariate since power per kilogram of body weight was the outcome. Since 1 potential characteristic of overt neuropathy is atrophy of muscle fibers,<sup>41</sup> lean mass was included as a potential mediator of the relationship between nerve function and power. Fat mass was included because of its important metabolic and functional consequences.<sup>42,43</sup> Lean and fat mass were measured using dual-energy x-ray absorptiometry (Hologic 4500A<sup>®</sup>). To ensure reproducibility of dual-energy x-ray absorptiometry measurements, standardized measurement and quality-control procedures were used and operators were certified. More localized measures of calf muscle density, which has been positively associated with peroneal motor amplitude,<sup>44</sup> and muscle area were added in place of lean and fat mass as potential mediators in subsequent models. Muscle density (in  $\text{mg}/\text{cm}^3$ ), a measure of intermuscular fat, and muscle area (in  $\text{mm}^2$ ) were measured at 66% of the calf length using peripheral quantitative computed tomography (Stratec XCT-2000 scanner<sup>d</sup>), as previously described.<sup>45</sup> Each of the following covariates was significantly related to muscle power or one of the nerve function predictors at an alpha level of 0.1. Diabetes was defined by self-report, use of hypoglycemic medications, or having a baseline fasting glucose level  $\geq 126\text{mg}/\text{dL}$ .<sup>46</sup> Other chronic health conditions included self-reported hypertension, congestive heart failure, myocardial infarction, stroke, osteoarthritis, and hip pain. Participants self-reported if a physician or other health care provider had ever told them that they had the condition. An ankle-brachial index  $<0.9$  was used to define peripheral vascular disease, and an ankle-brachial index  $>1.3$  was used to define arterial stiffening. Cognitive function was assessed using the Teng Modified Mini-Mental State Exam.<sup>47</sup> Lifestyle factors included smoking status (past and current), alcohol consumption (drinks/wk), and physical activity, which was measured using the Physical Activity Scale for the Elderly.<sup>48</sup> Variance inflation factors were calculated to assess collinearity. No variance inflation factor exceeded 3.

### Statistical analysis

Jonckheere-Terpstra tests and generalized linear models were used to test for trends in participant characteristics across muscle power/kg tertiles. Pairwise comparisons were made between muscle

power tertiles using  $t$  tests and chi-square statistics. Multivariable linear regression was used to compare (1) each measure of baseline nerve function with baseline muscle power/kg; (2) each measure of baseline nerve function with the change in muscle power/kg; and (3) each measure of change in nerve function with the change in muscle power/kg. Separate models for each measure of nerve function were built progressively in order, starting with the measure of nerve function. Age and height were added to the first set of minimally adjusted models. Total body lean and fat mass were added to the second set of models. For the third set of models, lean and fat mass were replaced with more localized measures of calf muscle density and cross-sectional muscle area. Finally, models were adjusted for lifestyle factors, chronic health conditions, and cognition to assess independent associations.

### Results

Participant characteristics were compared across muscle power tertiles (table 1). Those in the lowest power tertile were older and shorter; had a higher BMI, greater fat mass, and lower lean mass; and were more likely to have a history of hypertension and worse Teng Modified Mini-Mental State Exam scores. Alcohol consumption frequency (mean,  $2.6 \pm 1.3$  drinks/wk), current smoking status (2.7%), and history of stroke (4.6%), congestive heart failure (7.3%), myocardial infarction (17.7%), osteoarthritis (24.2%), and hip pain (15.9%) did not differ across muscle power tertiles (data not shown). Men in the lowest tertile had lower motor and sensory amplitudes (table 2) and were less likely to have 1.4-g and 10-g monofilament sensitivity and more likely to report numbness symptoms in the legs or feet (fig 1). Distal motor latency (mean,  $4.41 \pm 0.8\text{ms}$ ), F-wave latency (mean,  $60.6 \pm 5.9\text{ms}$ ), distal sensory latency (mean,  $3.12 \pm 0.4\text{ms}$ ), and self-report of open or persistent sores on the feet or legs (2.2%) did not differ across power tertiles (data not shown).

One SD lower motor and sensory amplitude were associated with lower muscle power/kg when adjusted for age and height (table 3). While the associations between motor and sensory amplitude and power remained significant on further adjustment, fat mass (2nd models) and muscle density (3rd models) attenuated the effect sizes by  $\geq 10\%$ . Insensitivity with 10-g and 1.4-g (2nd model only) monofilaments was also associated with lower muscle power/kg but was attenuated to nonsignificant by muscle density. Fat mass was negatively associated with muscle power/kg ( $\beta = -.02$ ,  $P < .01$ ), whereas muscle density was positively associated with muscle power/kg ( $\beta = .04$ ,  $P < .001$ ).

We compared the effect sizes of significant nerve function measures (from 2nd models) with the effect size of age on muscle power in SDs of muscle power/kg (fig 2). One SD lower motor and sensory amplitude had the effect of aging 1.4 and 1.8 years, respectively, and the inability to detect 1.4-g and 10-g monofilaments had the effect of aging 2.2 and 3.4 years, respectively. Table 4 shows that the inability to detect a 1.4-g monofilament at baseline predicted a greater decline in muscle power/kg when adjusted for age and height. Results were consistent when adjusted for lean and fat mass or muscle density and area and additional covariates. Change in nerve function was not associated with a change in power (results not shown).

### Discussion

Although muscle power is known to be dependent on both the nervous and musculoskeletal systems, previous research has not

**Table 1** Characteristics of study population by muscle power (W/kg) tertiles

Characteristics	Lowest Tertile $\leq 1.60$ W/kg (n=122)	Middle Tertile $>1.60$ and $\leq 2.06$ W/kg (n=129)	Highest Tertile $>2.06$ W/kg (n=121)	P for Trend
Muscle power (W/kg)	1.27 $\pm$ 0.2* <sup>†</sup>	1.84 $\pm$ 0.1 <sup>‡</sup>	2.52 $\pm$ 0.4	<.001
Age (y)	80.0 $\pm$ 5.1* <sup>†</sup>	76.7 $\pm$ 4.9 <sup>‡</sup>	75.0 $\pm$ 4.1	<.001
Race: white	122 (100)	128 (99.2)	120 (99.2)	.38
Body composition				
Height (m)	171.5 $\pm$ 6.8 <sup>†</sup>	172.0 $\pm$ 6.7 <sup>‡</sup>	173.9 $\pm$ 6.3	.01
BMI (kg/m <sup>2</sup> )	28.1 $\pm$ 3.7 <sup>†</sup>	28.2 $\pm$ 3.6	27.3 $\pm$ 3.5	.13
Fat mass (kg)	23.2 $\pm$ 6.6 <sup>†</sup>	22.7 $\pm$ 7.1	21.3 $\pm$ 6.5	.09
Lean mass (kg)	55.6 $\pm$ 6.7 <sup>†</sup>	56.9 $\pm$ 7.5	57.5 $\pm$ 6.6	.09
Muscle density (mg/cm <sup>3</sup> )	67.9 $\pm$ 4.6* <sup>†</sup>	69.4 $\pm$ 3.7	70.4 $\pm$ 3.8	<.001
Chronic health conditions				
Diabetes	25 (20.5)	20 (15.5)	16 (13.2)	.25
ABI <0.9	46 (42.6)	41 (34.2)	36 (30.3)	.05
History of hypertension	74 (60.7) <sup>†</sup>	66 (51.2)	50 (41.3)	.003
Physical activity score (PASE)	147.4 $\pm$ 65.8	146.8 $\pm$ 64.6	162.4 $\pm$ 65.2	.11
Cognition 3MSE score	92.6 $\pm$ 6.1	94.0 $\pm$ 4.9	94.8 $\pm$ 4.4	.003

NOTE. Values are mean  $\pm$  SD, n (%), or as otherwise indicated.

Abbreviations: ABI, arm-brachial index; PASE, Physical Activity Scale for the Elderly; 3MSE, Teng Modified Mini-Mental State Exam.

\*  $P < .05$  for lowest tertile vs middle tertile.

<sup>†</sup>  $P < .05$  for lowest tertile vs highest tertile.

<sup>‡</sup>  $P < .05$  for middle tertile vs highest tertile.

evaluated peripheral nerve function measures commonly used in clinical practice and neurologic studies. Studies<sup>4,13</sup> have indicated that muscle power declines at an even faster rate with age than strength, and our findings show that the potential effects of peripheral nerve function are 1.5 to 3.5 times the effect of 1 year of age. This finding has particularly important consequences, given that neuropathy is a preventable risk factor. Establishing the relationship between muscle power and clinically relevant measures of nerve function in late life is crucial because older adults experience the highest burden of neuropathy and diminished muscle function, and both likely play key roles in the disablement pathway.<sup>33,49</sup> Our findings show that poor sensory nerve function and motor peripheral nerve function are independently associated with, and may be important risk factors for, poor muscle power in old age. Risk factors for poor muscle power are understudied in epidemiologic studies of older adults, yet poor muscle power has important consequences in late life such as impaired mobility,<sup>4-12</sup> disability,<sup>2,50</sup> and increased risk of falls.<sup>3</sup>

We found that lower amplitude, but not latency, was associated with poor muscle power. Consistent with our study findings, Strotmeyer et al<sup>25</sup> reported that peroneal motor nerve amplitude, but not conduction velocity, was related to lower extremity muscle strength.

Latency is the travel time of the response and is measured from the moment of stimulation to the appearance of the action potential. Nerve conduction velocity is typically calculated by dividing the distance between 2 stimulation sites by the difference between latencies.<sup>51</sup> Diminished amplitude may indicate axonal degeneration and motor nerve death, whereas latency or conduction velocity may be a measure of demyelination of the protective sheath surrounding the nerve.<sup>52</sup> Amplitude may decline in some individuals, while velocity, driven by the motor units that remain intact, remains normal.<sup>44,53</sup> In participants in the lowest muscle power tertile, we observed lower amplitudes but no difference in latency, compared with those in higher muscle power tertiles (see table 2). These 2 aspects of motor nerve decline may occur separately, with muscle power relating to axonal degeneration and nerve death.

Sensory nerve function measured by monofilament detection and average vibration perception threshold was associated with muscle strength in the previous study<sup>25</sup> as well. In our study, sensory amplitude was related to muscle power. While motor nerves directly innervate muscle, it is less clear how the sensory nerves are involved. Blocking afferent input in healthy individuals has led to impaired maximal voluntary contractions,<sup>24</sup> which may occur through loss of proprioceptive feedback.<sup>27</sup> Since severe

**Table 2** Nerve conduction amplitude by muscle power (W/kg) tertiles

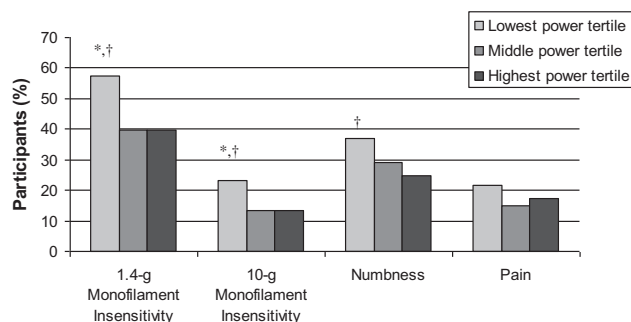
Amplitude	Lowest Tertile $\leq 1.60$ W/kg (n=122)	Middle Tertile $>1.60$ and $\leq 2.06$ W/kg (n=129)	Highest Tertile $>2.06$ W/kg (n=121)	P for Trend
Motor (mV)	2.15 $\pm$ 1.4*	2.25 $\pm$ 1.4 <sup>†</sup>	2.84 $\pm$ 1.5	<.001
Sensory ( $\mu$ V)	3.07 $\pm$ 3.2* <sup>‡</sup>	4.01 $\pm$ 3.4	4.84 $\pm$ 3.7	.002

NOTE. Values are mean  $\pm$  SD or as otherwise indicated.

\*  $P < .05$  for lowest tertile vs highest tertile.

<sup>†</sup>  $P < .05$  for middle tertile vs highest tertile.

<sup>‡</sup>  $P < .05$  for lowest tertile vs middle tertile.



**Fig 1** Monofilament insensitivity and symptoms by muscle power (W/kg) tertiles. \* $P < .05$  for lowest tertile vs middle tertile; † $P < .05$  for lowest tertile vs highest tertile.

sensory neuropathy is associated with poor ankle proprioception,<sup>54,55</sup> sensory input may be necessary to achieve proper placement, timing, and movement of the leg and foot during testing. An additional explanation could be that large-fiber neuropathies, which are the most common type of neuropathies in older adults,<sup>56</sup> may affect both sensory and motor nerves, with most deficits first presenting as sensory loss. This can progress to reduced position sense, muscle weakness and wasting, and depressed tendon reflexes.<sup>56</sup> Impaired sensory nerve function may thereby represent, or be a surrogate measure for, a more generalized loss of peripheral nerve function.

Our results suggest that insensitivity to the 1.4-g monofilament test may be an early indicator of those at future risk for muscle power decline. In contrast to the 10-g monofilament, which is generally associated with clinical disease and predictive of foot ulceration,<sup>57</sup> the 1.4-g monofilament may be a more sensitive measure, detecting sensory nerve function loss at an early stage.<sup>58,59</sup> Since risk factors for declining muscle power in older adults are understudied, this test could potentially be used to help develop prevention strategies to preserve muscle function.

Fat mass and muscle density attenuated the relationships of amplitude and monofilament insensitivity with muscle power. While data from the Health Aging and Body Composition Study show that older adults with greater fat mass had greater strength but lower muscle quality,<sup>60</sup> few have focused on the relationship between fat mass and muscle power or its additional component of velocity, which could pose an additional challenge for individuals with greater fat mass. Interestingly, diminished nerve function in older adults has been previously associated with lower muscle density, a measure of intermuscular fat, but not with cross-sectional muscle area,<sup>44</sup> which could suggest that age-related changes in nerve function lead to changes in muscle tissue structure rather than macroscopic changes in muscle mass.

A major strength of this study is the inclusion of both motor and sensory peripheral nerve function measures. We also used reproducible, sensitive, and specific measures of nerve conduction for both motor and sensory nerves.<sup>38,61</sup> Our measure of muscle power has been previously validated and is commonly used.<sup>40</sup> Models were adjusted for a number of potential confounders, including body composition, lifestyle factors, and comorbidities. Finally, we were able to examine longitudinal relationships between nerve function and muscle power change.

### Study limitations

One limitation of this study is that some of our null findings are likely attributable to the short period (2.3y) between measures. Our results may not apply to other populations such as nonwhite individuals, women, the “young-old,” and institutionalized individuals. Future studies should assess the relationship between muscle power and clinical measures of motor and sensory nerve function in a larger, more diverse population with a broader range of function.

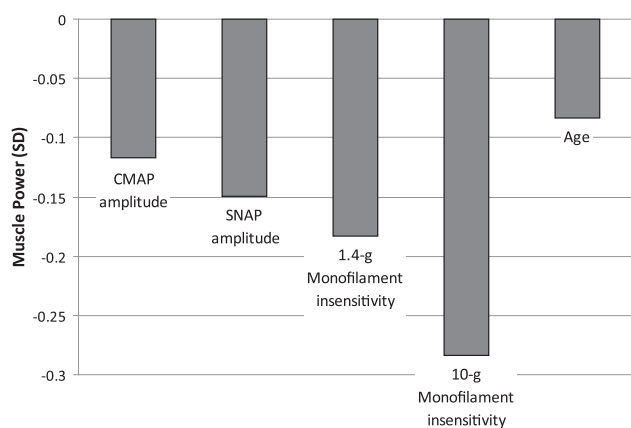
**Table 3** Separate multivariable linear regression models for each measure of nerve function and muscle power (W/kg)

Measures of nerve function	1st Models		2nd Models		3rd Models		4th Models	
	$\beta$ (SE)	$\beta$ (SE)	Attenuated by	$\beta$ (SE)	Attenuated by	$\beta$ (SE)	Attenuated by	
Motor nerve function per SD lower								
Motor amplitude	-.10* (.03)	-.07† (.03)	Fat mass	-.08† (.03)	Muscle density	-.07† (.03)	Fat mass	
Distal motor latency	-.05 (.03)	-.04 (.03)		-.04 (.03)		-.05 (.03)		
Mean F-wave latency	.05 (.03)	.05 (.03)		.05 (.04)		.06 (.04)		
Sensory nerve function per SD lower								
Sensory amplitude	-.10* (.04)	-.09† (.04)	Fat mass	-.09† (.04)	Muscle density	-.09† (.04)	Fat mass	
Distal sensory latency	-.10 (.34)	-.06 (.32)		-.21 (.34)		-.10 (.32)		
Monofilament insensitivity (yes/no)								
1.4-g	-.10 (.06)	-.11† (.05)		-.07 (.06)	Muscle density	-.12† (.03)		
10-g	-.16† (.07)	-.17† (.07)		-.12 (.07)	Muscle density	-.16† (.03)		
Neuropathic symptoms (yes/no)								
Numbness	-.10 (.06)	-.09 (.06)		-.10 (.06)		-.07 (.06)		
Pain	.01 (.07)	.04 (.07)		.07 (.07)		.04 (.07)		
Open sore	-.32 (.18)	-.36† (.18)		-.11 (.19)		-.34† (.21)		

NOTE. 1st models adjusted for age and height; 2nd models adjusted for variables in 1st models plus total body lean and fat mass; 3rd models adjusted for variables in 1st models plus muscle density and muscle cross-sectional area; 4th models adjusted for variables in 2nd models plus diabetes and hypertension.  $P > 0.1$  for arm-brachial index, cardiovascular disease, cerebrovascular disease, osteoarthritis, hip pain, smoking, alcohol use, physical activity, and cognitive function.

\*  $P < .001$ .

†  $P < .05$ .



**Fig 2** Effect size of nerve conduction amplitudes and monofilament insensitivity compared with 1 year of age. Motor and sensory amplitude per SD lower; 1.4-g and 10-g monofilament insensitivity (inability to feel 3/4 touches); age per year older; separate models adjusted for age, height, total body lean and fat mass; age adjusted for height, total body lean and fat mass. Abbreviation: CMAP, compound muscle action potential.

## Conclusions

We showed that sensory and motor nerve function are independently associated with muscle power, which is associated with poor outcomes in older adults, such as falls,<sup>3</sup> impaired mobility,<sup>4-12</sup> and disability.<sup>2,50</sup> Future work should investigate whether there is a direct relationship between poor nerve function and these poor health outcomes. Since monofilament insensitivity was predictive of greater muscle power decline, future studies should also test whether simple screening for monofilament detection may identify individuals at risk for muscle power decline. Detecting poor or declining muscle power early on could lead to more effective disability prevention and treatment efforts, such as training programs targeted at increasing muscle power.<sup>14-16</sup> Importantly, understanding risk factors in the disablement pathway, such as poor nerve function and impaired

muscle power, can help identify multiple points of intervention. Future studies should characterize the effects of known and novel risk factors of poor nerve function on muscle power.

## Suppliers

- NeuroMetrix, Inc, 62 4th Ave, Waltham, MA 02451.
- University of Nottingham, Medical Faculty Workshops, Queen's Medical Centre, Nottingham, NG7 2UH, UK.
- Hologic, Inc, 35 Crosby Dr, Bedford, MA 01730.
- Stratec Medizintechnik GmbH, Durlacher Str. 35, 75172 Pforzheim, Germany.

## Keywords

Aged; Motor neurons; Muscle weakness; Peripheral nerves; Rehabilitation; Sensory function; Sensory neurons

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**Table 4** Separate multivariable linear regression models for each measure of nerve function and decline in muscle power (W/kg)

Measures of nerve function	$\beta$ (SE)
Motor nerve function per SD lower	
Motor amplitude	-.001 (.04)
Distal motor latency	.001 (.03)
Mean F-wave latency	-.02 (.04)
Sensory nerve function per SD lower	
Sensory amplitude	.05 (.04)
Distal sensory latency	.06 (.04)
Monofilament insensitivity (yes/no)	
1.4-g	-.15* (.07)
10-g	-.11 (.09)
Neuropathic symptoms (yes/no)	
Numbness	.11 (.07)
Pain	-.13 (.09)

NOTE. Models adjusted for age and height.

\*  $P < .05$ .

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