OPEN

An Exercise Intervention May Counteract the Degradation of Nerve Conduction from Age-Related Disuse

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[1]Neural Health Research Laboratory, Falk College, Syracuse University, Syracuse, NY; [2]Department of Health and Human Performance, Fairmont State University, Fairmont, WV; [3]School of Kinesiology and Recreation, Illinois State University, Normal, IL; and [4]Applied Neuromuscular Physiology Laboratory, Oklahoma State University, Stillwater, OK

ABSTRACT

SHIELDS, J. E., C. M. SMITH, S. M. REESE, M. L. DOS SANTOS, M. PARODI, and J. M. DEFREITAS. An Exercise Intervention May Counteract the Degradation of Nerve Conduction from Age-Related Disuse. *Med. Sci. Sports Exerc.*, Vol. 57, No. 10, pp. 2101–2107, 2025. The natural progression of age can result in motor neuron degeneration. Consequently, this leads to slowing of nerve conduction, denervation, and reduced motor function. Slower neural conduction can negatively alter an individual's response time, which could increase the risk of falls. Further investigation is needed to determine the potential role exercise interventions may afford in mitigating age-related nerve deterioration. **Purpose:** The purpose was twofold: first, to determine the effects of resistance training on nerve conduction velocity (NCV), and second, to determine if changes in NCV are dependent on age. We hypothesized that training would result in faster nerve conduction in both young and older adults, albeit to a lesser extent in older adults. **Methods:** Forty-eight subjects (18–84 yr) completed this study (training: $n = 14$ younger, 14 older; control: $n = 12$ younger, 8 older). Median motor NCV and maximal strength were recorded before and after 4 wk of handgrip training in both limbs. Training was conducted 3×/wk with the use of a grip training kit. **Results:** Mixed-factorial ANOVA revealed significant increases in NCV for both the young ($P < 0.001$, Cohen's $d = 0.749$) and older training groups ($P < 0.001$, Cohen's $d = 0.679$), but neither in control groups (young: $P = 0.353$, Cohen's $d = 0.326$; older: $P = 0.108$, Cohen's $d = -0.184$). **Conclusions:** The results of this study suggest that resistance training may be a viable method to counteract age-related nerve deterioration. These outcomes have the potential to improve quality of life and generate greater independence for our older populations.
Key Words: AGING, MOTOR NERVE CONDUCTION VELOCITY, NERVE DETERORIATION, RESISTANCE TRAINING

Aging is a multidimensional process characterized by physiological and morphological changes that affect one's quality of life, with declines in motor ability evident as early as the fourth decade (1,2). Evidence supports the belief that functional ability, especially after the age of 60, is substantially less, and deterioration of the peripheral nervous system (PNS) may be one of the primary causes (3–5). Unfortunately, although there is substantial sarcopenia-related research focusing on preserving muscle mass and function, the collective research into countermeasures to protect peripheral nerve function is minimal.

Deterioration of the peripheral nerves is reflected by fewer functioning motor neuron axons and/or degradation of the motor axons. These outcomes result in the slower transmission of a nerve impulse supplying a target effector muscle and can have functional consequences such as slower movements or reduced mobility, all of which may lead to a diminished quality of life (6,7). Additionally, older adults exhibit varying degrees of loss in strength and muscle mass as a result of these effects and may become more susceptible to the development of sarcopenia (e.g., age-related reduction in muscle mass) (8–10).

Commonly recognized in individuals with nerve injury or disease, declining PNS function often leads to neuromuscular dysfunction, such as neurodegenerative diseases (amyotrophic lateral sclerosis) and peripheral neuropathies (diabetic neuropathy,

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Guillain-Barré syndrome, etc.). However, age-related slowing of healthy motor nerves has been found in both humans and rodents (11–13). Previous research identifies a negative linear relationship between a slowing in nerve speed and age (≥60 yr) in those who are free of neuromuscular disease (14). Although the support for an age-related slowing of nerves exists, there is much still to be understood regarding interventions that could facilitate positive adaptations within the PNS.

Resistance training has long been prescribed to older adults as a means to long-term vitality. Individuals who remain active throughout their life have been known to have improved mobility, more independence, and greater life expectancy (15). Literature has shown that resistance training may be a counteractive modality to nerve loss in previously injured nerves (16). However, it remains unclear if resistance training can alter nerve function in healthy, noninjured adults. Moreover, resistance training has also been used in the prevention and management of sarcopenia (17,18). Although little is known about the effects of resistance training and nerve speed in healthy untrained individuals, chronically trained athletes have shown to exhibit faster nerve conduction velocity (NCV) (19). Because of the benefits resistance training provides, it is plausible to consider resistance training as a method to counteract losses in nerve function.

Although previous studies have investigated training and nerve conduction speed in adults (20–22), few studies have focused on interventions that mitigate nerve speed loss and possible adaptations training may have. Additionally, our understanding of nerve function in untrained healthy individuals is still unclear. Further investigation is needed to determine the age-related changes in nerve speed function and the potential role exercise-based interventions may afford in resisting nerve deterioration. Therefore, the purpose of this study was 1) to quantify the effects of resistance training on NCV and 2) to determine if age affects nerve plasticity in response to training. We hypothesized that resistance training would improve motor conduction velocity of the median nerve in both young and older adults in response to a handgrip resistance training program. Second, because it has been shown that older adults may have less capacity to adapt to training (23), we also hypothesized that the magnitude of adaptation was going to be significantly less in older adults.

METHODS

Participants. Forty-eight participants (mean ± SD age = 43.9 ± 24.9 yr) volunteered for this study (see Table 1 for subject characteristics). Participants completed a written informed consent and health history questionnaire before beginning the study. All participants were apparently healthy and reported having no neuromuscular disease or neurological disorders. Further, all participants reported either having no upper body resistance training within the past 6 months or limited training (≤2× per week). This study was approved by Oklahoma State University's Institutional Review Board before data collection (IRB-22-270) and was registered as a clinical trial (NCT06614556).

TABLE 1. Subject characteristics.

Group	Young Training	Young Control	Older Training	Older Control	*P* Value
Age (yr)	22.9 ± 7.3	21.1 ± 2.0	69.7 ± 7.5	69.7 ± 8.5	0.045*
Height (cm^2)	172.9 ± 9.8	167.6 ± 8.5	168.5 ± 8.1	149.3 ± 46.1	≤0.001*
Body Mass (kg)	78.8 ± 18.2	73.2 ± 23.3	68.7 ± 14.6	111.1 ± 41.8	0.003*
Sample Size (*n*)	14	12	14	8	
Males (*n*)	6	2	5	5	
Females (*n*)	8	10	9	3	

Data are presented as mean ± SD. *P* value = 2 × 2 between-group interaction.
* Significant difference at the 0.05 level.

Research design. This study consisted of four groups, first separated by age based on our target ranges (young = 18–35 yr old, and older ≥60 yr old) and then pseudorandomly assigned to either the intervention group or control group. This resulted in a young training group (YT, $n = 14$), a young control group (YC, $n = 12$), an older training group (OT, $n = 14$), and an older control group (OC, $n = 8$). Testing sessions were performed before (PRE) and after (POST) 4 wk of the study. Most participants' testing sessions (PRE and POST) were conducted at the same time of day; however, two participants' testing sessions were performed at different times of day due to availability. Motor nerve conduction and maximal handgrip strength testing were conducted during each testing session. Participants were asked to maintain normal daily activities throughout the study.

Motor nerve function assessments. Motor NCV was collected using the incremental method (24,25). This method applies low-intensity stimulation to the nerve and gradually increases stimulation intensity in small increments (5 mA) to recruit additional motor units. Using this procedure quantifies NCV by obtaining the latency of the maximal M-wave response, also referred to as the compound muscle action potential, at different segments along the median nerve and divides them by the distance between segments. The formula is shown in Figure 1.

Before electrode placement, the skin over the specified sites was shaved, abraded, and cleaned with alcohol to promote optimal signal quality. Using a clinical electrodiagnostic system (Cadwell Sierra Summit, Cadwell Industries, Inc., Kennewick, WA), motor nerve conduction was assessed for the left and right median nerve. Disposable recording electrodes (20 × 27 mm, Cadwell Industries Inc., Kennewick, WA) were placed on the belly of the flexor digitorum superficialis muscle as verified by ultrasonography. The reference and the ground electrodes were placed on the flexor carpi radialis tendon and the dorsum of hand, respectively. Maximal M-waves were obtained at the axilla region and below the elbow of each arm.

For assessment of the optimal stimulation site of the axilla region, the arm was palpated by having the participants flex their biceps. The optimal stimulation site for the elbow was located at the antecubital fossa near the biceps tendon. Participants were seated in a chair upright with their forearms in a supinated position on a table. In a cathode–anode arrangement, a single stimulus (single square wave impulse) was applied using the incremental method starting at 5 mA. Each stimulus thereafter was increased by 5 mA until no increases in M-wave amplitude were detected.

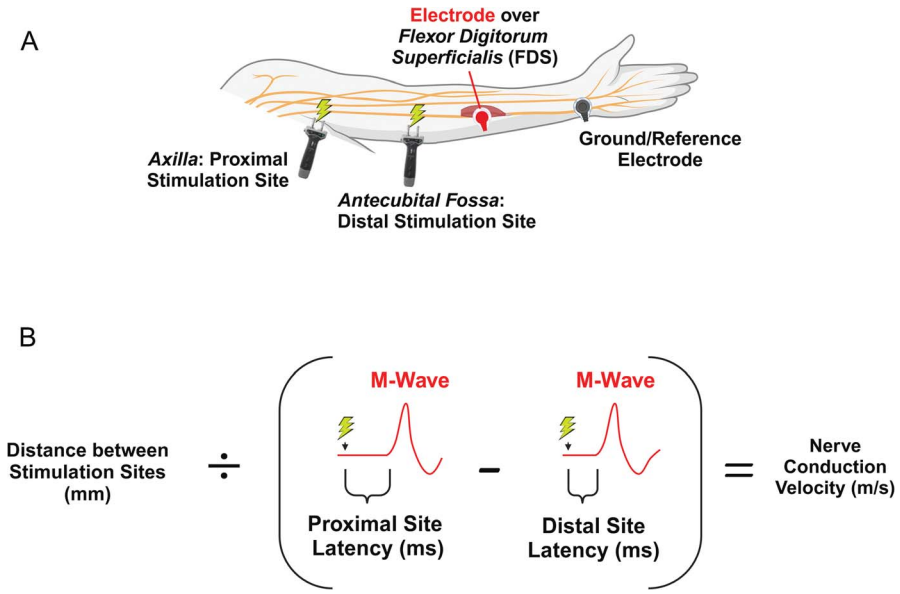

FIGURE 1—A. A visual depiction of our nerve stimulation methodology. This shows the simulation sites (axilla and antecubital fossa) and electrode placement for a nerve conduction assessment of the median nerve. B. A figure illustrating the calculation of NCV ($m \cdot s^{-1}$) from proximal latency (ms), distal latency (ms), and the distance between the stimulation sites (mm). Created in BioRender.com.

After the assessment of maximal M-wave, NCV was obtained. A temperature probe was secured to the participant's wrist to ensure consistent temperature (approximately 30°C) throughout testing (Med-Linket, Ltd., Shenzhen, Guangdong, China). A single supramaximal (e.g., 120% of maximal M-wave) stimulation was applied to each proximal and distal site (axilla and below the elbow) on both arms. The latency (ms) of each stimulation was stored and recorded for later use in the determination of NCV. Additionally, the distance (mm) from the proximal stimulation site to the distal stimulation site was verified using a standard flexible tape measure. NCV was derived by the software included in the clinical electrodiagnostic system using the equation provided in Figure 1.

Maximal voluntary contractions. Participants were asked to hold a hand dynamometer by their side and perform two maximal handgrip contractions per arm (Jamar, Sammons Preston Inc., Bolingbrook, IL). Instructions were followed based on the recommendations provided in the hydraulic hand dynamometer manual. Before the contractions, participants performed two to three warm-up contractions at half their maximal effort. Upon directions, participants were asked to raise the dynamometer to a 90° angle and contract maximally while exhaling each breath. Each contraction lasted 4–5 s with strong verbal encouragement given by the research team and 1 min of rest given between each trial. The highest value (kg) was considered the participant's maximal handgrip strength (MVC).

At-home resistance training intervention. Participants in the training groups performed handgrip resistance training on both limbs three times per week for 4 wk using a specialized handgrip kit provided to them (NONJISPT, Shenzhen, Guangdong, China). Each kit contained an adjustable hand gripper, stress ball, grip ring, grip master, and finger stretcher resistance band. Additional grip rings (10–50 lb; Refluxe, Shenzhen, Guangdong, China) and finger stretcher resistance bands (8–21 lb; Portholic, Shenzhen, Guangdong, China) were used to induce a progressive overload in resistance for 4 wk. Participants were provided with pictures and instructions for each exercise upon completion of the initial testing (PRE) session (see Supplemental Digital Content, http://links.lww.com/MSS/D250). They were asked to perform 12 training sessions (approximately 30–45 mins) over the 4 wk according to their schedule. Each participant was asked to keep a training log to assist with accountability. The PI also conducted weekly check-ins (via phone call or text) with each subject to ensure compliance to the protocol and answer any potential questions. Subjects were excluded from the study if they noted three or more missed training sessions. Upon completion of the training, participants revisited the lab for a (POST) testing session, and all previous measures were assessed. Those in the control group still received a handgrip training kit after completion of the study, along with instructions on how to conduct the training.

Statistical analyses. *A priori* power analysis (G*Power, Version 3.1.9, Universitat Kiel, Germany) was performed with a power level was set at 0.95, an alpha level set at 0.05, with an effect size of 0.8 ("large") to determine the most meaningful sample size for this study ($n = 47$). All statistical analyses were performed using SPSS Version 26 (IBM, Armonk, NY). To assess normality, the Shapiro–Wilk test and visual inspection of histograms were used. Homogeneity of variance was determined using Levene's test. Both assumptions were met. Two four-way mixed-factorial ANOVA model's (age [young vs old]–group [training vs control]–time [pre vs post]–limb [left vs right]) were conducted to determine changes in NCV and MVC strength. Partial eta squared (η_p^2) effect sizes are provided where appropriate. ANOVA's that were statistically

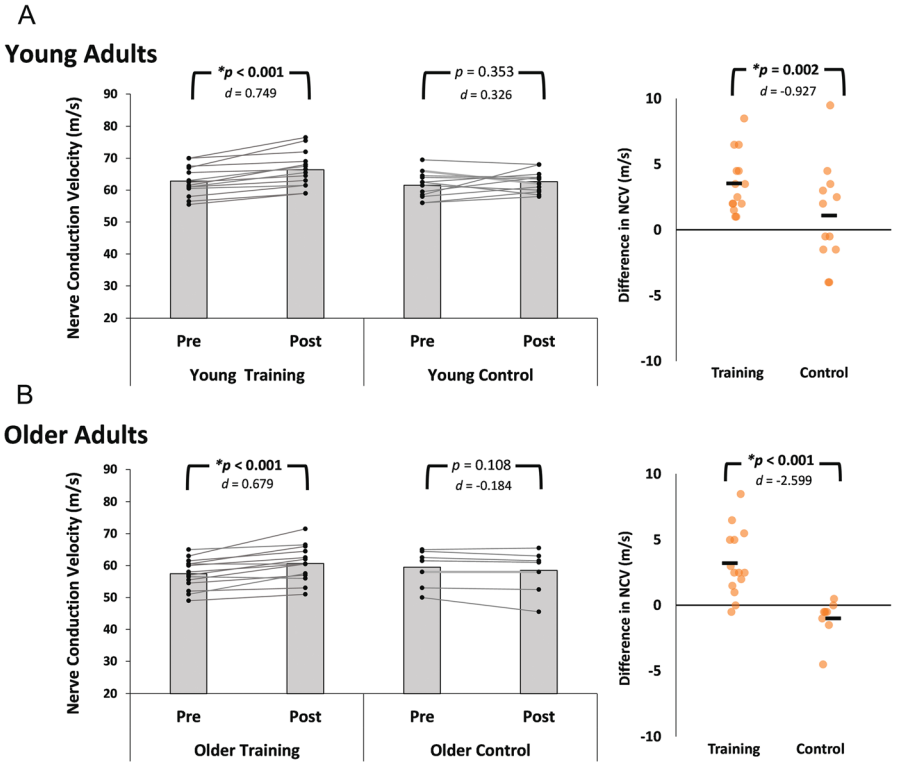

FIGURE 2—Results for NCV before and after the 4-wk training intervention for both the young (A) and the older (B) adults. The *gray bars* represent group means, and the data points are individual subjects. Significant differences ($P \leq 0.05$) are bolded and denoted (*), with Cohen's *d* effect sizes below each *P* value. The right panel depicts individual change scores in NCV.

significant were followed with *t*-tests. Cohen's *d* effect sizes were interpreted as small (0.2), medium (0.5), and large (0.8). An alpha level of 0.05 was used for all comparisons, except for the *post hoc* paired sample *t*-tests, which were Bonferroni corrected (four tests: pre vs post within each of the four groups, so $\alpha = 0.0125$).

RESULTS

The results from the mixed-factorial ANOVA showed no significant (age–group–time–limb) interaction for motor NCV ($F_{3,\ 44} = 0.006$, $P = 0.937$, $\eta_p^2 < 0.000$). However, a significant group–time interaction was identified ($F_{3,\ 44} = 26.140$, $P \leq 0.001$, $\eta_p^2 = 0.373$). None of the two- or three-way models that included age or limb as a factor were significant (*P* values ranged from 0.216 to 0.987). Subsequent *t*-tests showed that both training groups had significant changes in NCV after training (YT: $t_{13} = -5.733$, $P < 0.001$; OT: $t_{13} = -4.694$, $P < 0.001$). The effect size values based on Cohen's *d* for each of these groups were 0.749and 0.679, respectively, indicating a medium effect. Significant between-group (YT × YC) (OT × OC) changes in NCV ($t_{24} = 3.451$, $P = 0.002$; $t_{20} = 4.213$, $P < 0.001$) were also identified. Both training groups accounted for a 5.6% increase in NCV pre- to postintervention. There were no significant findings for either control groups (young: $P = 0.353$, Cohen's $d = 0.326$; older: $P = 0.108$, Cohen's $d = -0.184$). The individual subject results are shown in Figure 2.

No significant (age–group–time–limb) interaction was observed for MVC strength ($F_{3,\ 44} = 0.001$, $P = 0.970$, $\eta_p^2 < 0.000$). However, significant interactions for time–limb ($F_{3,\ 44} = 6.207$, $P = 0.018$, $\eta_p^2 = 0.120$) and group–time ($F_{3,\ 44} = 5.942$, $P = 0.019$, $\eta_p^2 = 0.119$) were found. The YT group demonstrated significant changes in MVC strength of both limbs pre- to postintervention (left: $t_{13} = -4.020$, $P = 0.001$; right: $t_{13} = -3.481$, $P = 0.004$). A small effect for both limbs was found based on Cohen's *d* (left: 0.343; right: 0.232). No other significant changes were found among limbs or groups. The individual subject results are shown in Figure 3.

DISCUSSION

This study aimed to examine the trainability of peripheral nerve function as well as its dependency on age. We found that 4 wk of handgrip resistance training increased both strength (young) and median NCV (young and older) in both forearms. The changes in strength in the young group affirm the efficacy of the resistance training intervention. Although we hypothesized that training would lead to improvements in nerve function, we did not expect the magnitude of adaptations (YT: $P < 0.001$, Cohen's $d = 0.749$; OT: $P < 0.001$, Cohen's $d = 0.679$) demonstrated in this study from such a short intervention. The 4-wk duration of the intervention was chosen more for project feasibility than it was for it representing any sort of optimal duration. As such, we expected smaller and less robust gains in which there would be more nonresponders

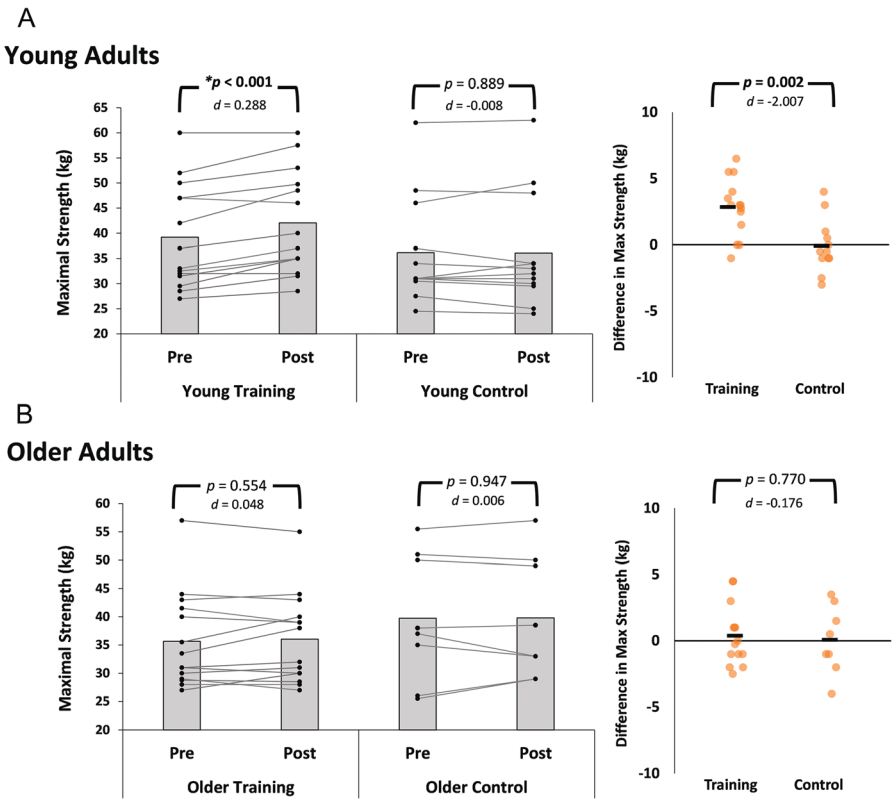

FIGURE 3—Results for maximal voluntary strength before and after the 4-wk training intervention for both the young (A) and the older (B) adults. The *gray bars* represent group means, and the data points are individual subjects. Significant differences ($P \leq 0.05$) are bolded and denoted (*), with Cohen's *d* effect sizes below each *P* value. The right panel depicts individual change scores in maximal strength.

present. The lack of significant improvements in strength (left: $P = 0.273$, Cohen's $d = 0.098$; right: $P = 0.916$, Cohen's $d = -0.010$) in the older group can likely be attributed to the intervention duration. Griffin et al. (26) showed neural adaptations (reduced motor unit firing rate variability) in older adults after 4 wk of resistance training while also not showing a significant improvement in strength. Conversely, Patten et al. (27) showed early nonsignificant neural and strength adaptations in older adults within days, with significant adaptations in 6 wk of training. However, the development that almost every participant in the 2 intervention groups showed improvement in NCV was unexpected and requires follow-up studies that examine potential underlying mechanisms to better understand this. A few studies have investigated various exercise prescriptions and their subsequent effects on conduction velocity. In a study on strength and sprint training, Sleivert et al. (20) found that conduction velocity significantly increased after 14 wk in untrained young males. However, findings on chronically trained (e.g., weightlifters and athletes) individuals show varied results (21,22). Nonetheless, these studies were only in young males and young male athletes and did not include women or older adults.

Adaptations dependency on age. Unexpectedly, older adults had just as strong of an adaptation to the training as the young adults. We anticipated that the magnitude of change would be significantly less compared with younger participants (i.e., less plasticity). For example, previous literature has found smaller responses in measures of muscle size (28,29) and muscle strength (30,31) after training in older adults. Based on these findings, we incorrectly anticipated a similar pattern in nerve function would occur.

Adaptations dependency on limb. It was also slightly surprising that limb was not a factor in any of the statistical models. We expected the dominant limb to be slightly more trained than the nondominant and, as a result, hypothesized that the dominant limb would have less gains. It is suggested (32) that the characteristics of a training paradigm, such as task complexity and subject familiarity, may influence outcomes, which could explain limb being a nonfactor in our present study.

Associated changes in strength. Our study identified changes in handgrip strength in our YT group (left: $P < 0.001$, Cohen's $d = 0.343$; right: $P = 0.004$, Cohen's $d = 0.232$), although not in other groups. One likely scenario for the outcome of our present study is that our OT group needed a longer duration to attain such changes. Although not significant, the OT group demonstrated increases in MVC strength (see Fig. 3); however, nonresponders likely accounted for the lack of a significant change. As previously referenced (26,27), there remains considerable heterogeneity in training responses among older adults. Additionally, it is unknown what degree of denervation (e.g., lack of neural input to the muscle) the OT group had at the time of this study. The denervation process is said to significantly contribute to the development of muscle weakness and frailty in older adults (33). Without

accounting for motor unit denervation, it is difficult to ascertain if a lack of neural input contributed to our findings.

Potential mechanisms. The findings shown in the present study lead to a particularly intriguing follow-up question; what are the potential mechanisms underlying this improvement in NCV? It has been well established for nearly a century that there is a strong positive relationship between the diameter of an axon and its conduction velocity (34,35). Therefore, one possible mechanism for the neurons to get faster is for them to also get larger (wider axonal diameter). There is substantial research into the training-induced hypertrophy of skeletal muscle fibers, but it is relatively unknown as to whether neurons are also capable of hypertrophy in response to training. The opposing result of nerve atrophy has been investigated in depth and does demonstrate a continued marriage between axonal size and conduction velocity (i.e., as one goes down, so does the other) (36). Therefore, although speculative, it is at least plausible that the two variables may continue to be paired for improved adaptations as well. Future studies examining resistance training interventions should include both a measure of NCV as well as a measure of axonal size (or at least an estimate, such as nerve size). Structural changes in myelin can occur with aging, resulting in thinning of the sheaths and segmental demyelination, which is believed to impair conduction (4,37). However, the degree of remyelination may be less efficient for older adults because of the severity of damage or underlying diseases. Further investigation and more precise imaging techniques should be performed to determine remyelination's potential contribution to NCV changes. Another possibility for the nerve includes potential changes in axonal excitability (38). However, because our measure of NCV requires a compound muscle action potential (M-wave), downstream mechanisms, such as increased efficiency of transmission across the neuromuscular junction or faster conduction velocity in the muscle fibers, cannot be ruled out.

Limitations and future directions. Although our study presents new and potentially impactful findings, there are some conceivable limitations to this study design: first, the level to which our participants were "untrained." Although inclusion criteria allowed participants to exercise ($\leq$2× per week), this may have contributed to our findings. Several older adults reported being active, and a few participated in racket sports, which may have influenced the changes observed. Second, it is possible that compliance may have been a factor. Although participants were asked to track their training, one can never entirely ensure participant compliance with the training program. Third, it is possible that our intervention was too short (only 4 wk), and future studies with longer intervention durations may provide stronger evidence as to the plasticity of nerve conduction. Additionally, if a future study were to perform a longer duration training program that induced significant changes to MVC, then additional analyses, such as the correlation between changes in NCV and MVC, would be possible. Last, most of the participants were female, so a gender bias may have inadvertently occurred. A more significantly powered study design would have allowed for a thorough comparison between the sexes and should be considered in the future.

More thorough investigations are needed to determine the underlying mechanisms of this study's findings. Additional research should explore the functional properties of the spinal and supraspinal systems and their contributions to peripheral nerve transmission. Moreover, it is important to identify potential interventions and optimal prescriptions that may support the neural consequences of aging.

CONCLUSIONS

These findings support our hypothesis that handgrip training improves motor conduction velocity in young and older adults. This suggests that resistance training may be a robust method to counteract NCV deficits in the short term, although more research is still needed. The results of this study could aid clinicians in exercise prescription for individuals needing to improve nerve conduction and motor function. The significance of this research line has the potential to improve the quality of life and generate greater independence for our older populations.

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REFERENCES

1. Choy NL, Brauer S, Nitz J. Changes in postural stability in women aged 20 to 80 years. *J Gerontol A Biol Sci Med Sci*. 2003;58(6):M525–30.
2. Izquierdo M, Aguado X, Gonzalez R, et al. Maximal and explosive force production capacity and balance performance in men of different ages. *Eur J Appl Physiol Occup Physiol*. 1999;79(3):260–7.
3. Carmeli E, Patish H, Coleman R. The aging hand. *J Gerontol A Biol Sci Med Sci*. 2003;58(2):M146–52.
4. Verdú E, Ceballos D, Vilches JJ, et al. Influence of aging on peripheral nerve function and regeneration. *J Peripher Nerv Syst*. 2000;5(4):191–208.
5. Lexell J. Evidence of nervous system degeneration with advancing age. *J Nutr*. 1997;127(5 Suppl):1011S–3.
6. Lange-Maia BS, Newman AB, et al. Sensorimotor peripheral nerve function and the longitudinal relationship with endurance walking in the health, aging, and body composition study. *Arch Phys Med Rehabil*. 2016;97(1):45–52.
7. Ward RE, Caserotti P, Cauley JA, et al. Mobility-related consequences of reduced lower-extremity peripheral nerve function with age: a systematic review. *Aging Dis*. 2016;7(4):466–78.
8. Metter EJ, Conwit R, Metter B, Pacheco T, Tobin J. The relationship between peripheral motor nerve conduction velocity to age-associated loss of grip strength. *Aging (Milano)*. 1998;10(6):471–8.
9. Frontera WR, Hughes VA, Fielding RA, Fiatatrone MA, Evans WJ, Roubenoff R. Aging of skeletal muscle: a 12-yr longitudinal study. *J Appl Physiol (1985)*. 2000;88(4):1321–6.
10. Naruse M, Trappe S, Trappe TA. Human skeletal muscle-specific atrophy with aging: a comprehensive review. *J Appl Physiol (1985)*. 2023;134(4):900–14.
11. Norris AH, Shock NW, Wagman IH. Age changes in the maximum conduction velocity of motor fibers of human ulnar nerves. *J Appl Physiol*. 1953;5:589–93.

12. Walsh ME, Sloane LB, Fischer KE, et al. Use of nerve conduction velocity to assess peripheral nerve health in aging mice. *J Gerontol A Biol Sci Med Sci*. 2015;70(11):1312–9.
13. Wang FC, de Pasqua V, Delwaide PJ. Age-related changes in fastest and slowest conducting axons of thenar motor units. *Muscle Nerve*. 1999;22(8):1022–9.
14. Wagman IH, Lesse H. Maximum conduction velocities of motor fibers of ulnar nerve in human subjects of various ages and sizes. *J Neurophysiol*. 1952;15(3):235–44.
15. Fragala MS, Cadore EL, Dorgo S, et al. Resistance training for older adults: position statement from the national strength and conditioning association. *J Strength Cond Res*. 2019;33(8):2019–52.
16. Eslami R, Tartibian B, Najarpour M. Effect of six weeks resistance training on nerve conduction velocity, strength, balance and walking speed in multiple sclerosis patients. *J Gorgan Univ Med Sci*. 2019; 21(3):63–8.
17. Clark BC, Clark LA, Law TD. Resistance exercise to prevent and manage sarcopenia and dynapenia. *Annu Rev Gerontol Geriatr*. 2016; 36:205–28.
18. Yarasheski K. Managing sarcopenia with progressive resistance exercise training. *J Nutr Health Aging*. 2002;6(5):349–56.
19. Sale D, McComas A, MacDougall J, et al. Neuromuscular adaptation in human thenar muscles following strength training and immobilization. *J Appl Physiol Respir Environ Exerc Physiol*. 1982;53(2):419–24.
20. Sleivert GG, Backus R, Wenger HA. The influence of a strength-sprint training sequence on multi-joint power output. *Med Sci Sports Exerc*. 1995;27(12):1655–65.
21. Sale D, Upton A, McComas A, et al. Neuromuscular function in weight-trainers. *Exp Neurol*. 1983;82(3):521–31.
22. Padkao T, Prasertsri P. Effectiveness of an upper and lower limb resistance training program on body composition, nerve conduction velocity, and cardiac autonomic nervous activity in university athletes. *J Exerc Physiol Online*. 2019;22(2):78–98.
23. Slivka D, Raue U, Hollon C, Minchev K, Trappe S. Single muscle fiber adaptations to resistance training in old (>80 yr) men: evidence for limited skeletal muscle plasticity. *Am J Physiol Regul Integr Comp Physiol*. 2008;295(1):R273–80.
24. Oh SJ. *Clinical Electromyography: Nerve Conduction Studies*. 3rd ed. Philadelphia (PA): Lippincott Williams & Wilkins; 2003. pp. 467–9.
25. Leis AA, Schenk MP. *Atlas of Nerve Conduction Studies and Electromyography*. New York (NY): Oxford University Press; 2013.
26. Griffin L, Painter PE, Wadhwa A, Spirduso WW. Motor unit firing variability and synchronization during short-term light-load training in older adults. *Exp Brain Res*. 2009;197(4):337–45.
27. Patten C, Kamen G, Rowland DM. Adaptations in maximal motor unit discharge rate to strength training in young and older adults. *Muscle Nerve*. 2001;24(4):542–50.
28. Vandeweerd J. Comparing individual muscle size and strength responses in young and older adults after prolonged resistance training [master's thesis]. University of Jyväskylä Department of Science of Sport Coaching and Fitness Testing, Faculty of Sport and Health Sciences 2021.
29. Welle S, Totterman S, Thornton C. Effect of age on muscle hypertrophy induced by resistance training. *J Gerontol*. 1996;51(6):M270–5.
30. Knight CA, Kamen G. Adaptations in muscular activation of the knee extensor muscles with strength training in young and older adults. *J Electromyogr Kinesiol*. 2001;11(6):405–12.
31. Lemmer JT, Hurlbut DE, Martel GF, et al. Age and gender responses to strength training and detraining. *Med Sci Sports Exerc*. 2000;32(8): 1505–12.
32. Farthing JP, Chilibeck PD, Binsted G. Cross-education of arm muscular strength is unidirectional in right-handed individuals. *Med Sci Sports Exerc*. 2005;37(9):1594–600.
33. Bean JF, Kiely DK, Herman S, et al. The relationship between leg power and physical performance in mobility-limited older people. *J Am Geriatr Soc*. 2002;50(3):461–7.
34. Gasser HS, Erlanger J. The role played by the sizes of the constituent fibers of a nerve trunk in determining the form of its action potential wave. *Am J Physiol*. 1927;80:522–47.
35. Hursh JB. Conduction velocity and diameter of nerve fibers. *Am J Physiol*. 1939;127(1):131–9.
36. Gillespie MJ, Stein RB. The relationship between axon diameter, myelin thickness and conduction velocity during atrophy of mammalian peripheral nerves. *Brain Res*. 1983;259(1):41–56.
37. Chapman TW, Hill RA. Myelin plasticity in adulthood and aging. *Neurosci Lett*. 2020;715:134645.
38. Kiernan MC, Bostock H, Park SB, et al. Measurement of axonal excitability: consensus guidelines. *Clin Neurophysiol*. 2020;131(1):308–23.