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Item Type	Journal article
Authors	Behraznia, Mahyar;Ditroilo, Massimiliano;Smith, Tina
Citation	Behraznia, M., Ditroilo, M. and Smith, T. (2026) Ultrasound for the assessment of muscle architecture in Parkinson's disease: a scoping review. <i>Clinical Biomechanics</i> , 132, 106733.
DOI	10.1016/j.clinbiomech.2025.106733
Publisher	Elsevier
Journal	Clinical Biomechanics
Download date	2026-05-18 16:12:39
License	https://creativecommons.org/licenses/by/4.0/
Link to Item	https://wlv.openrepository.com/handle/2436/626207



Ultrasound for the assessment of muscle architecture in Parkinson's disease: A scoping review

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ARTICLE INFO

Keywords:

Skeletal muscle
Ultrasound
Parkinson's disease
Muscle mechanics
Muscle architecture

ABSTRACT

Background: Parkinson's disease (PD) affects motor function and muscle performance, which are closely linked to muscle architecture (MA). Ultrasound (US) provides a non-invasive method to assess MA, yet its application in PD remains underexplored. This review aimed to map and synthesise existing research on US assessment of MA in individuals with PD to clarify how US imaging methodologies contribute to understanding muscle structure and function in this population.

Methods: This scoping review was conducted following established guidelines and searching these databases: Scopus, PubMed, Web of Science Core Collection, MEDLINE and CINAHL Ultimate.

Findings: Of the 913 records identified, 20 studies met the inclusion criteria. Research varied in design, US measurement methodology, and measurement and reporting approaches. The synthesis revealed that while MA at rest was largely preserved in PD with notable exceptions, muscle fascicle responsiveness during contraction was significantly impaired, potentially due to rigidity. MA differences were associated with PD clinical manifestations (i.e., bradykinesia and rigidity) and reduced functional performance. PD-related comorbidities including sarcopenia, camptocormia, and dysphagia were found to further affect MA, exacerbating muscle degradation and remodelling. Exercise was found to alter the structural characteristics of MA, suggesting beneficial adaptive potential.

Significance and interpretation: Despite consistent evidence of altered MA and muscle responsiveness in PD, methodological heterogeneity and small samples limit firm conclusions. Standardised US protocols and longitudinal studies are needed to clarify the relationship between MA, functional performance, and PD clinical features, and to evaluate the effects of exercise and rehabilitation on muscle structure and strength.

1. Introduction

Parkinson's disease (PD) is the fastest growing neurological disorder (Dorsey et al., 2018), attributed to the loss of dopaminergic neurons in the substantia nigra pars compacta (Beitz, 2014; Kalia and Lang, 2015), which disrupts the neural drive (McGregor and Nelson, 2019). This leads to characteristic motor symptoms—bradykinesia, tremor, and rigidity—that significantly impair mobility and quality of life (Jankovic, 2008). While traditionally considered a brain disorder, PD's impact extends beyond the central nervous system (CNS) to the peripheral neuromuscular system, notably affecting skeletal muscles, the final effectors of motor commands (Dalise et al., 2020; Duranti and Villa, 2024; Murphy and Lynch, 2023). As a result, PD is associated with a range of musculoskeletal complications, the most prominent being impaired

skeletal muscle health, manifesting as muscle wasting and weakness (Murphy and Lynch, 2023). Recent research (Dalise et al., 2020; Duranti and Villa, 2024; Murphy and Lynch, 2023) highlight the growing consensus that pathological changes observed in skeletal muscle in PD are not merely a consequence of inactivity or ageing, but also reflect direct, disease-specific processes. Documented muscle changes include atrophy, altered fibre composition—particularly a shift toward slow-twitch fibres—and mitochondrial dysfunction; in some cases, pathological protein aggregates have also been observed in muscle tissue (Dalise et al., 2020; Duranti and Villa, 2024; Murphy and Lynch, 2023). These findings, observed in both limb and axial muscles, support the hypothesis that muscle pathology in PD involves both impaired neural input and distinct peripheral mechanisms, which together contribute to functional decline and may even precede overt motor symptoms.

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<https://doi.org/10.1016/j.clinbiomech.2025.106733>

Received 1 August 2025; Accepted 8 December 2025

Available online 19 December 2025

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Given that skeletal muscle is a primary determinant of movement and functional capacity, its deterioration in PD has direct clinical consequences. Deficits in muscle strength, range of motion, and motor control underlie many of the characteristic functional limitations of PD, including gait disturbance, postural instability, and bradykinesia (Falvo et al., 2008; Inkster et al., 2003; Mazzoni et al., 2012; Paul et al., 2013). Importantly, muscle function often deteriorates with disease progression (Falvo et al., 2008; Stevens-Lapsley et al., 2012) but can improve with targeted rehabilitation or pharmacological treatment (Folland et al., 2011; Lima et al., 2013). Ultimately, the operational characteristics of skeletal muscle (i.e., muscular strength, range of motion, motor control relating to the force-velocity and the length-tension curves) directly influence the functional capabilities of people with PD. Many clinical interventions are therefore focused on effective strategies to optimise muscle function and performance. Therapeutic and rehabilitation interventions targeted at improving aspects of muscle function include muscle strengthening (David et al., 2011; Falvo et al., 2008; Lima et al., 2013; Paolucci et al., 2020), stretching (Duñabeitia et al., 2025), balance and postural training (Klamroth et al., 2016), treadmill training (Abbruzzese et al., 2016; Rawson et al., 2019), and automated mechanical peripheral stimulation (AMPS) (Tedeschi, 2023; Tedeschi et al., 2024). Pharmacological treatments target the CNS to modulate dopaminergic activity and basal ganglia function, in an attempt to enhance motor control and improve movement performance (Connolly and Lang, 2014), while surgical interventions aim to optimise motor function, alleviate debilitating symptoms such as tremor and rigidity, and ultimately enhance overall quality of life (Sharma et al., 2020). With so many interventions focused on the musculoskeletal system, it is important we can accurately and reliably measure changes in the morphological properties of skeletal muscle in response to those interventions, as morphological differences have been associated with improved muscle function (Blazevich et al., 2006; Lieber and Blevins, 1989; Lieber and Fridén, 2000).

Imaging modalities used to capture muscle characteristics include ultrasound (US), magnetic resonance imaging and computerized tomography (Dick and Hug, 2023). Among these techniques, US is a well-established imaging modality for assessing skeletal muscle, and has unique advantages of being radiation-free, real-time and relatively inexpensive. Historically, US has been used for muscle assessment since the 1980s, when real-time brightness-mode imaging first revealed structural changes in diseased muscle, such as atrophy and increased echo intensity due to fat and fibrous infiltration (Heckmatt et al., 1980; Pillen and van Alfen, 2011). Through the 1990s and 2000s, quantitative muscle US became an established tool in neurology, enabling the detection and grading of muscle morphology (Heckmatt et al., 1980; Heckmatt et al., 1982; Heckmatt et al., 1988; Shortland et al., 2002). Recent advances in quantitative muscle imaging, including automated and semi-automated analysis methods, have further improved the precision, reliability, and scalability of US-based assessments, enabling more detailed evaluation of muscle architecture (MA) and function (Dick and Hug, 2023; Franchi et al., 2018). As a result, US has been used to evaluate the morphological changes of muscle, specifically MA, which refers to the organisation of muscle fibres within a muscle. US offers clinicians a practical tool to monitor disease progression and treatment response by visualising muscle adaptations and distinguishing neural from muscular contributions, an approach already established in other neurological and neuromuscular disorders (Chen et al., 2015; Hannaford et al., 2025; Katzberg et al., 2016; Pillen and van Alfen, 2011).

Different morphological parameters of MA, such as fascicle length (FL), pennation angle (PA), muscle thickness (MT) and cross-sectional area (CSA), could be extracted from US to estimate muscle activity and function. Each parameter reflects distinct functional properties: increases in PA and CSA are associated with greater force-generating capacity (Blazevich et al., 2006; Lieber and Blevins, 1989; Lieber and Fridén, 2000), while longer fascicles favour higher contraction velocities and flexibility (Blazevich et al., 2006). Nevertheless, MA can be affected

by age (Strasser et al., 2013), gender (Kubo et al., 2003), inactivity (de Boer et al., 2008) and disease (Chen et al., 2018; Kirmaci et al., 2022), which all lead to decreases in MT and PA, with negative consequences for strength and contraction velocity. Since the reduced tolerance to physical activity shown in PD patients (Ellis et al., 2013; van Nimwegen et al., 2011), a drop in PA and MT due to disuse (Timmins et al., 2016), may be expected to be related to the observed atrophy. On the contrary, being physically active and undergoing resistance training has been shown to be a powerful method to improve muscle functions through favourable MA exercise-induced adaptations (Timmins et al., 2016). Existing research utilising US in the assessment of resting MA in PD has observed both similarities (Magris et al., 2024; Martignon et al., 2021; Monte et al., 2023) and differences (Masaki et al., 2022) between individuals with PD and healthy controls. Furthermore, reduced MT as measured via ultrasonography has been associated with poor functional performance in individuals with PD (Aktar et al., 2023; Masaki et al., 2023a). Moreover, muscle fascicle behaviour was found to be impaired in dynamic conditions (i.e., during explosive isometric contractions) (Magris et al., 2024; Monte et al., 2023), which was proposed to be due to muscle stiffness (rigid hypertonia), but requires further study.

Despite increasing use of US to study skeletal muscle in PD, findings remain fragmented and methods heterogeneous, limiting their translation to clinical or research practice. Importantly, it is unclear whether PD motor symptoms contribute to changes in MA. The combined effects of motor impairments and structural alterations of muscle could influence muscle fibre orientation and fascicle behaviour in ways that are not yet fully understood. This gap inhibits clinical translation and comprehensive understanding of how motor impairments and muscle structural alterations interact in PD. Addressing these issues by mapping current research, standardizing protocols, and elucidating relationships between US-derived MA parameters, clinical manifestations, and functional outcomes is essential. Such synthesis will enhance the use of US in diagnosis, monitoring, and evaluating therapeutic responses targeting muscle health in PD. Although a previous review examined US imaging of MA in neurological disorders (Chen et al., 2015), their focus was on stroke, cerebral palsy, and spinal cord injury. The authors concluded that ultrasonography is a feasible method to measure muscle-tendon architecture and it was widely used to detect morphological changes and to evaluate the functional improvement of the affected muscle after an intervention program (Chen et al., 2015). To date, no study has systematically synthesised US research specifically for assessing MA in PD. Therefore, exploring and evaluating the existing evidence of MA in PD is crucial to align current findings, along with understanding how variables of muscle measurement are being used within the PD context. In addition, by identifying US methodological inconsistencies and synthesising current approaches, this review will provide guidance for future research and contribute to the development of more standardised US assessment protocols in PD. Thus, the objective of this scoping review was to map the literature on the assessment of MA using US in PD, to highlight how the application of current US imaging techniques and approaches influences our understanding and interpretation of MA in PD. To guide this review, three research questions were developed: 1) What is the extent of research using US to assess MA in people with PD? 2) What, muscle architectural variables of PD muscle were measured using US and how? 3) What is the current state of scientific findings in MA in people with PD, and does MA correlate with functional outcome measures and PD clinical manifestations?

2. Methods

A scoping review of the literature using a well-established methodology (Levac et al., 2010) was conducted. The scoping review framework comprises identification of a research question, identification of the relevant studies, study selection, charting of data, summarising and reporting the results, and consultation. The latter step was not applied in the current study. This scoping review also followed the recommended

items in PRISMA Extension for Scoping Reviews (PRISMA-ScR) (Tricco et al., 2018).

2.1. Search strategy

The general research topic was searched across databases including Scopus (via Elsevier) and Web of Science Core Collection (via Clarivate) in July 2024. In the initial primary search, titles, keywords, and terminologies related to the scope of our study were evaluated. This process was conducted to acquire more profound knowledge and insight into the existing research, but also to pinpoint the most effective terms used in the current research to enhance our final search strategy. The final search was conducted in October 2025 using specific terms related to: 'ultrasound', 'muscle architecture' and 'Parkinson's disease' in the following databases: Scopus (via Elsevier), PubMed (via US National Library of Medicine), Web of Science Core Collection (via Clarivate), MEDLINE with Full Text (via EBSCOhost), and CINAHL Ultimate (via EBSCOhost). More details concerning search strategy and the key words used are reported in Appendix A (Supplementary Material 1, online only).

2.2. Study selection

The inclusion criteria for the studies were: (1) original research studies, (2) assessment and measurement of any characteristic of peripheral skeletal MA using US and (3) the whole population or part of the population included individuals with PD. Exclusion selection criteria comprised articles in any language other than English, not original research (e.g., book/book chapters, case report/series, commentary/opinion piece, conference paper), did not include humans In Vivo in sample, and instead includes other groups (e.g., animals, cadavers, In Vitro). Articles without available full text were also excluded. There was no restriction as to publication year, this was to facilitate an extensive search, ensuring that no articles were overlooked due to the long-standing utilisation of US. Studies that assessed MA using US, even if it was not the primary aim, were considered for this review.

Duplicate citations from the five databases and other sources were first identified and removed in Zotero (Reference Management Software, <https://www.zotero.org/>). The final set of search results were then exported and uploaded to an online screening tool, Rayyan (<https://rayyan.qcri.org/>), for screening and selection. Screening was conducted in two stages in Rayyan by the first author (MB):

1. First, the titles and abstracts were read considering the inclusion and exclusion criteria.
2. Next, the full-text of the selected articles were read in full.

Possible conflicts regarding the eligibility of the articles were discussed and solved by the author (MB) and a member of the supervisory team (MD), and if required the principal supervisor established consensus (TS).

2.3. Data charting process

During the data charting process, the relevant information was extracted into an Excel file (Microsoft Excel). During this phase, the papers that satisfied the inclusion criteria were reviewed multiple times to ensure comprehensive extraction of all relevant information regarding the scope of the study. After data extraction, tables were designed which consisted of predefined data items for extraction, which would inform understandings of what is currently known within the literature regarding US in the assessment of MA in PD. The tables were designed based on (1) general study and participant characteristics (e.g., authors name, year of publication, participant age and sex, disease severity), (2) measurement methodology (i.e., muscle of interest, reported muscle architectural variable), and approach to measurement

and reporting (e.g., US system, acquisition procedure, US settings), including clinimetric properties and reporting of additional outcomes, and (3) summary of the main finding relating to muscle variable measured.

2.4. Collating, summarising, and reporting the results

Before the analysis of the studies, a protocol was registered with the Open Science Framework (registration DOI: [10.17605/OSF.IO/2Z46M](https://doi.org/10.17605/OSF.IO/2Z46M)). Data interpretation was conducted using descriptive and numerical analysis. For studies that did not report sample mean and standard deviation, other summary metrics available (e.g., median or range) were reported. Then, all the information resulting from the analysis of the included studies were summarised by themes of the use and application of muscle imaging via US in PD, methodological approach, and current scientific research findings of MA in PD. Finally, we discuss the results and their implications for research and clinical practice.

3. Results

3.1. Data synthesis

Searching of the five databases resulted in 911 potentially eligible studies, with a further two articles identified from other sources and through citation searching. After removing duplicates, study titles and abstracts were reviewed to assess the eligibility of 534 studies. A total of 24 articles were qualified for the full-text reading stage. This last stage resulted in the identification of 20 studies as eligible in this review (Abrahin et al., 2020; Aktar et al., 2023; Calaway et al., 2025; Chen et al., 2023; Ding et al., 2024; Donizetti Verri et al., 2019; Göz et al., 2023; Lim et al., 2025; Magris et al., 2024; Martignon et al., 2021; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Monte et al., 2023; Oh et al., 2016; Şirin Ahisha et al., 2025; Smart et al., 2020; Umay et al., 2019; Yilmaz et al., 2023; Yin et al., 2021). The flowchart of the selection process is charted in Fig. 1. The earliest publication date was 2016 (Oh et al., 2016), with most articles ($n = 7$) published in 2023. The included studies used observational or experimental study designs. Studies originated from Turkey ($n = 5$), China ($n = 3$), Italy ($n = 3$), Japan ($n = 3$), Brazil ($n = 2$), Canada ($n = 1$), United States of America ($n = 1$), South Korea ($n = 1$), and Taiwan ($n = 1$).

3.2. Study and participant characteristics

Table 1 represents the general study and participant characteristics of the included articles. While there is no single common aim across all studies, many focus on understanding muscle-related changes, functional performance, and US diagnostic or evaluative techniques in PD patients. Concerning population sample, all studies included only adults. In total, 1045 adult participants were included in this review (control group of people with no diagnosis of PD; $n = 314$, patients with PD; $n = 701$). The total number of individuals with PD might be less than 701 due to unclear reporting of participants across two studies (Chen et al., 2023; Ding et al., 2024). The sample size for patients with PD ranged from 8 to 120 participants, and the sample size for the healthy control group ranged from 9 to 60 participants. Six studies reported performing an a-priori sample size calculation, ensuring adequate statistical power for their analyses (Ding et al., 2024; Göz et al., 2023; Magris et al., 2024; Martignon et al., 2021; Monte et al., 2023; Şirin Ahisha et al., 2025). Of these, three studies reported the effect size (Ding et al., 2024; Magris et al., 2024; Şirin Ahisha et al., 2025). The remaining 14 studies did not justify their sample size (Abrahin et al., 2020; Aktar et al., 2023; Calaway et al., 2025; Chen et al., 2023; Donizetti Verri et al., 2019; Lim et al., 2025; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Oh et al., 2016; Smart et al., 2020; Umay et al., 2019; Yilmaz et al., 2023; Yin et al., 2021). Several studies ($n = 4$) examined PD patients with PD phenotypes (i.e., postural instability gait difficulty (PIGD), tremor-

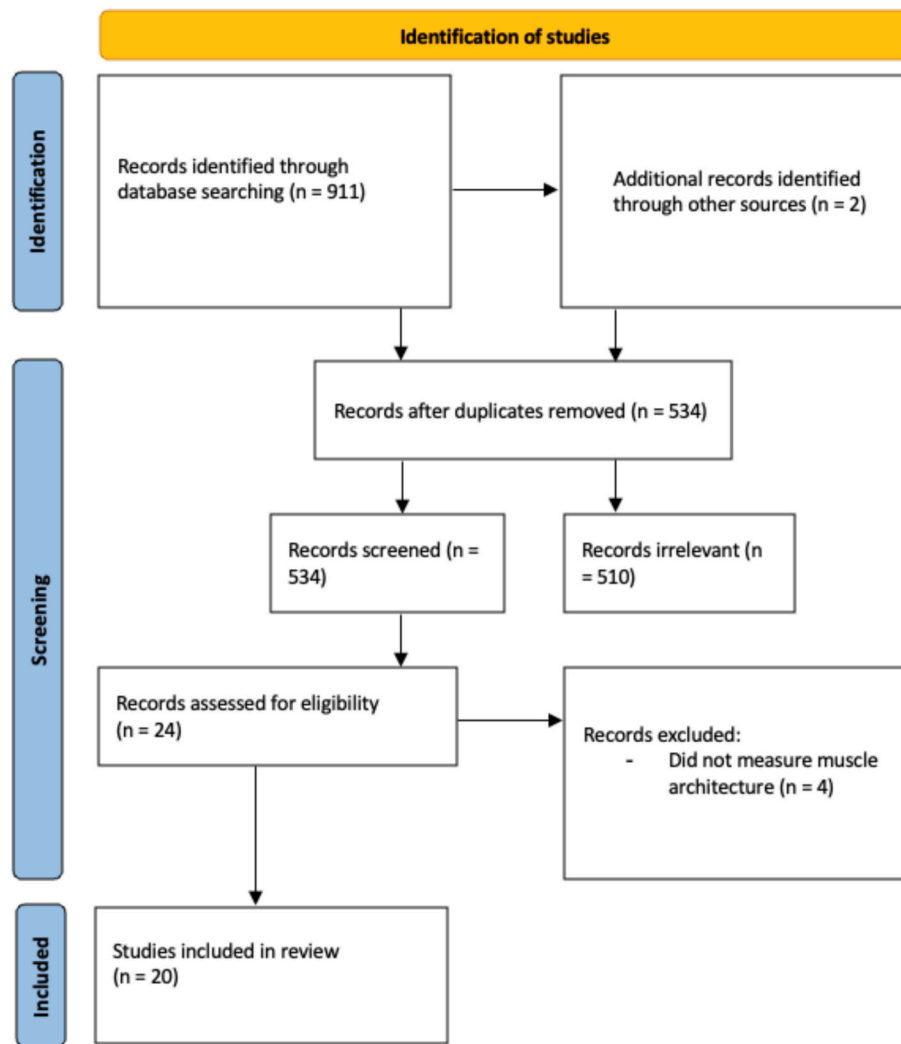


Fig. 1. PRISMA flowchart for the selection process of studies.

dominant (TD), bradykinetic/rigidity presentations) (Ding et al., 2024; Magris et al., 2024; Martignon et al., 2021; Monte et al., 2023). Of the 701 PD patients, 74 had sarcopenia (Chen et al., 2023; Lim et al., 2025), 17 had camptocormia (CC) (Yilmaz et al., 2023), 100 had dysphagia (Oh et al., 2016; Şirin Ahışha et al., 2025; Umay et al., 2019), 7 had diabetes mellitus (Şirin Ahışha et al., 2025), and 6 had coronary artery disease (Şirin Ahışha et al., 2025). The severity of PD motor symptoms was reported in 14 studies (Aktar et al., 2023; Ding et al., 2024; Göz et al., 2023; Lim et al., 2025; Magris et al., 2024; Martignon et al., 2021; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Monte et al., 2023; Şirin Ahışha et al., 2025; Smart et al., 2020; Umay et al., 2019; Yilmaz et al., 2023) using the Movement Disorder Society-Unified Parkinson's Disease Rating Scale, part III (MDS-UPDRS-III). The disease stage and progression of PD patients was reported in 17 studies (Abrahin et al., 2020; Aktar et al., 2023; Calaway et al., 2025; Ding et al., 2024; Donizetti Verri et al., 2019; Göz et al., 2023; Lim et al., 2025; Magris et al., 2024; Martignon et al., 2021; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Monte et al., 2023; Oh et al., 2016; Şirin Ahışha et al., 2025; Umay et al., 2019; Yin et al., 2021) using the Hoehn and Yahr (H&Y) scale. In the control group of people with no diagnosis of PD, 309 of these participants were classified as healthy or normal subjects. Additionally, there were four to five cases of sarcopenia in the control group of people with no diagnosis of PD (Chen et al., 2023), 6 cases of diabetes mellitus (Şirin Ahışha et al., 2025), and 4 cases of coronary artery disease (Şirin Ahışha et al., 2025).

3.3. Measurement methodology

The muscle(s) of interest and the reported muscle architectural variable(s) are detailed in Appendix A (Supplementary Material 2, on-line only). A wide range of muscles were evaluated, including upper body, abdominal, lower body, facial muscles, and the tongue. The most common muscles assessed were the vastus lateralis (VL) (Calaway et al., 2025; Magris et al., 2024; Martignon et al., 2021; Masaki et al., 2023a; Masaki et al., 2022; Monte et al., 2023), rectus femoris (RF) (Abrahin et al., 2020; Calaway et al., 2025; Lim et al., 2025; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b), and medial and lateral heads of the gastrocnemius (Ding et al., 2024; Masaki et al., 2023a; Masaki et al., 2022; Smart et al., 2020; Yin et al., 2021). The most common muscle architectural variable investigated was MT (Abrahin et al., 2020; Aktar et al., 2023; Calaway et al., 2025; Chen et al., 2023; Ding et al., 2024; Donizetti Verri et al., 2019; Göz et al., 2023; Lim et al., 2025; Magris et al., 2024; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Monte et al., 2023; Oh et al., 2016; Şirin Ahışha et al., 2025; Umay et al., 2019; Yilmaz et al., 2023; Yin et al., 2021), followed by PA (Chen et al., 2023; Ding et al., 2024; Magris et al., 2024; Martignon et al., 2021; Monte et al., 2023; Smart et al., 2020), FL (Chen et al., 2023; Ding et al., 2024; Magris et al., 2024; Monte et al., 2023; Smart et al., 2020) and CSA (Lim et al., 2025; Smart et al., 2020; Umay et al., 2019).

Table 1
Study details, location (by primary author), aim(s), and participant demographics and general characteristics of the studies.

Author & Country	Title	Study Aim(s) and Objectives	Sample Characteristics						
			n	Age (years) Mean \pm SD reported, unless mentioned otherwise	Sex	Disease Duration (years and Mean \pm SD reported, unless mentioned otherwise)	Severity of Motor Function (Assessed by UPDRS-III)	Symptoms Progression / Staging of Functional Disability (Assessed by Hoehn and Yahr scale)	Medication Details
Abrahin et al. (2020) Brazil	Muscle thickness and functional performance of patients with Parkinson's disease.	To check whether there is difference between MT values measured by US at three different points in the flexor muscles of the elbow and knee extensors of PD patients; and to check whether there is correlation between MT measures and functional performance (chair stand test and arm curl) in patients with PD.	PD: 31	PD: 64.6 \pm 10.6	N/A	4.5 \pm 2.7 years	N/A	H&Y: 1 (1–2) median (25th – 75th quartile)	N/A
Aktar et al. (2023) Turkey	The Relationship between Transversus Abdominis and Internal Oblique Thickness and Disease-Related Characteristics in Parkinson's Disease: An Ultrasound-Based Study.	To investigate whether changes in TrA and IO thickness during the ADIM were associated with clinical manifestations, core endurance, and functional mobility in patients with PD.	PD: 22	PD: 65 (median)	PD: 17M/5F	4 (median)	UPDRS-III: 24 (20.25–34.00) Median (IQR)	Modified H&Y: Stage 1: 5 Stage 2: 8 Stage 2.5: 8 Stage 3: 1	N/A
Chen et al. (2023) China	The value of ultrasound measurement of muscle thickness at different sites and shear wave elastography in Parkinson's disease with sarcopenia: a pilot study.	To elucidate the specific ultrasonic diagnostic parameters associated with PD accompanied by sarcopenia through a comparative analysis of muscle US parameters in patients with PD.	PD: 68 (12 with sarcopenia, 56 without sarcopenia) CG: 42 (4 or 5 with sarcopenia, 37 without sarcopenia)	PD: (range 51–79) CG: (range 50–78)	PD: 33M/35F CG: 16M/25F	N/A	N/A	N/A	N/A
Göz et al. (2023) Turkey	The effects of Pilates training on abdominal muscle thickness and core endurance in patients with Parkinson's disease: a single-blind controlled clinical study.	Examine the effects of Pilates training on the TrA and IO MT and core endurance in different positions in patients with PD.	PD: 23	PD Intervention Group: 65.62 \pm 8.94 PD CG: 68.20 \pm 7.69	PD Intervention Group: 11M/2F PD CG: 9M/1F	PD Intervention Group: 4.63 \pm 2.85 PD CG: 4.70 \pm 3.36	UPDRS-III: PD Intervention Group 26.61 \pm 10.47 PD CG 27.40 \pm 5.60	Modified H&Y PD Intervention Group: Stage 1: 3 Stage 2: 4 Stage 2.5: 5 Stage 3: 1 PD CG: Stage 1: 1 Stage 2: 5 Stage 2.5: 4 H&Y: 1.50 \pm 0.65	N/A
Magris et al. (2024) Italy	Characterization of the vastus lateralis torque-length, and knee extensors torque-velocity and power-velocity	To characterise the VL torque-fascicle length and knee extensors torque-angular velocity and power-angular velocity relationships in PD	PD: 11 CY: 10 CR: 9	PD: 70.5 \pm 5.28 CY: 25.1 \pm 1.73	PD: 9M/2F CY: 6M/4F CR: 8M/1F	PD: 6.1 \pm 4.36	UPDRS-III: 27.3 \pm 13.2	H&Y: 1.50 \pm 0.65	Madopar, Sinemed, Mirapixin, Oxyen, Sirio, Rantal, Ropinirol, Requir.

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Table 1 (continued)

Author & Country	Title	Study Aim(s) and Objectives	Sample Characteristics						
			n	Age (years) Mean \pm SD reported, unless mentioned otherwise	Sex	Disease Duration (years) and Mean \pm SD reported, unless mentioned otherwise)	Severity of Motor Function (Assessed by UPDRS-III)	Symptoms Progression / Staging of Functional Disability (Assessed by Hoehn and Yahr scale)	Medication Details
Masaki et al. (2022) Japan	relationships in people with Parkinson's disease.	patients and to investigate the influence of muscle geometry on muscle mechanics.		CR: 68.3 \pm 5.36					
	Comparison of the mass and amount of intramuscular non-contractile tissue and lower extremity muscles between patients with Parkinson's disease and community-dwelling older adults.	To compare the masses and amounts of intramuscular non-contractile tissue of the trunk and lower extremity muscles measured in detail using an US imaging device, sagittal spinal alignment, and mobility and balance between patients with PD and community dwelling older adults.	PD: 8 CG: 26	PD: 69.4 \pm 2.8 CG: 71.2 \pm 2.8	PD: 1M/7F CG: 6M/20F	PD: 189.5 (months)	UPDRS-III: 9.5 (12.0–4.5) Median (IQR)	H&Y: 3 (median)	N/A
	Association of sagittal spinal alignment in the standing position with the masses and amounts of intramuscular non-contractile tissue of the trunk and lower extremity in muscles in patients with Parkinson's disease.	To examine the association of sagittal spinal alignment in the standing position with the masses and amounts of intramuscular non-contractile tissue of multiple trunk and lower extremity muscles, such as the hip joint muscles.	PD: 10	PD: 69.9 \pm 7.7	PD: 0M/10F	PD: 166.0 \pm 78.4 (months)	UPDRS-III: 9.2 \pm 5.8 Range 3.0–23.0	H&Y: 2.7 \pm 0.7	N/A
Japan	Association of activities of daily living, mobility and balance ability, and symptoms of Parkinson's disease with the masses and amounts of intramuscular non-contractile tissue of the trunk and lower extremity in patients with Parkinson's disease.	To examine the association of ADL, mobility and balance ability, and PD symptoms with the masses and amounts of intramuscular non-contractile tissue of the trunk and lower extremity muscles, measured in detail using an ultrasound imaging device in patients with PD.	PD: 11	PD: 69.7 \pm 7.3	PD: 1M/10F	PD: 184.4 \pm 96.2 (months)	UPDRS-III: 9.5 \pm 5.6 Range 3.0–23.0	H&Y: 3 (median)	N/A
Monte et al. (2023) Italy	Muscle shape changes in Parkinson's disease impair function during rapid contractions.	To better understand how an impairment in structural/mechanical (peripheral) factors could explain the difficulty of PD patients to raise torque rapidly.	PD: 15 CG: 12	PD: 70 \pm 4.8 CG: 67.5 \pm 5.7	PD: 11M/4F CG: 10M/2F	N/A	UPDRS-III: 26.5 \pm 12.5	H&Y: 1.54 \pm 0.66	N/A
Martignon et al. (2021) Italy	The key role of physical activity against the neuromuscular deterioration in patients with Parkinson's disease.	To compare quadriceps maximal force and the contribution of central and peripheral components of force production during a maximal isometric task between physically active PD and healthy individuals.	PD: 10	PD: 66 \pm 7	PD: 5M/5F	5.9 \pm 4.1	UPDRS-III:	H&Y: 2.3 \pm 0.4	N/A
			CG: 10	CG: 70 \pm 6	CG: 5M/5F		25 \pm 7		

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Table 1 (continued)

Author & Country	Title	Study Aim(s) and Objectives	Sample Characteristics						
			n	Age (years) Mean \pm SD reported, unless mentioned otherwise	Sex	Disease Duration (years) and Mean \pm SD reported, unless mentioned otherwise	Severity of Motor Function (Assessed by UPDRS-III)	Symptoms Progression / Staging of Functional Disability (Assessed by Hoehn and Yahr scale)	Medication Details
Smart et al. (2020) Canada	Importance of Maximal Strength and Muscle-Tendon Mechanics for Improving Force Steadiness in Persons with Parkinson's Disease.	To ascertain the influence of MVC force and gastrocnemius-Achilles MTU behaviour on FS in patients with PD.	PD: 9	PD: 70 \pm 5	PD: 3M/6F	6 \pm 3	UPDRS-III:	N/A	Seven of the PD participants were on prescribed dosages of levodopa/carbidopa.
			CG: 9	CG: 70 \pm 7	CG: 3M/6F	12.7 \pm 6.7			
Yilmaz et al. (2023) Turkey	Characterizing Camptocormia in Parkinson's Disease Using Muscle Ultrasonography.	To explore whether muscle ultrasonography is applicable to detect muscular changes of CC in PD.	PD CC: 17 (seven aCC, 10 cCC)	PD wnCC: 71.5 \pm 9.2	PD wnCC: 10M/9F	PD: 7.8 \pm 6.3	UPDRS-III:	N/A	Levodopa equivalent daily dose (mg/d):
			PD wnCC: 19	PD-aCC: 77.6 \pm 4.5	PD-aCC: 5M/2F	PD-aCC: 10.7 \pm 8.4	PD: 23.0 \pm 9.5		
			CG: 18	PD-cCC: 72.5 \pm 6.8	PD-cCC: 6M/4F	PD-cCC: 15.5 \pm 6.8	PD-aCC: 24.7 \pm 10.6		
				CG: 75.5 \pm 6.4 PD: 66.1 \pm 3.3	CG: 11M/7F N/A	N/A	PD-cCC: 31.9 \pm 4.6		
Donizetti Verri et al. (2019) Brazil	Effects of Parkinson's disease on molar bite force, electromyographic activity and muscle thickness of the masseter, temporal and sternocleidomastoid muscles: A case-control study.	To investigate the impairment of the stomatognathic function regarding molar bite force, electromyographic activity and thickness of the craniocervical muscles in patients with PD in comparison with those in asymptomatic controls.	PD: 12	PD: 63.3 \pm 8.67	PD: 77M/43	PD: 9.27 \pm 5.13	UPDRS-III:	PD patients with stages I and III according to the H&Y scale	N/A
			CG: 12	CG: 65.8 \pm 3.0	CG: 47M/13F	14.56 \pm 9.14			
Umay et al. (2019) Turkey	Swallowing in Parkinson's disease: How is it affected?.	To assess the swallowing function in PD patients in both clinical and subclinical period of dysphagia compared to healthy subjects by using multimodal anatomic and physiologic methods.	PD: 120 (63 with dysphagia, 57 without dysphagia) CG: 60	PD: 63.3 \pm 8.67 CG: 62.97 \pm 7.79	PD: 77M/43 CG: 47M/13F	PD: 9.27 \pm 5.13	UPDRS-III: 14.56 \pm 9.14	Modified H&Y: 2.03 \pm 0.12	N/A
Ding et al. (2024) China	Shear wave elastography characteristics of the gastrocnemius muscle in postural instability gait disorder vs tremor dominant Parkinson's disease patients.	To explore the force changes of the lateral head of the gastrocnemius muscle at both resting and exercise states in patients with PIGD and TD using SWE; and measure some two-dimensional ultrasonic parameters and make a comparative analysis.	PD: 75 (prospectively) – 31 TD and 44 PIGD CG: 40	PD PIGD: 66.0 \pm 8.1 PD TD: 65.3 \pm 6.8 CG: 63.2 \pm 5.3	PD PIGD: 20M/24F PD TD: 20M/11F CG: 17M/23F	PD PIGD: 4.5 yrs. PD TD: 3.0 yrs	UPDRS-III: PD PIGD 23.2 \pm 10.2 PD TD: 19.8 \pm 12.6	PD PIGD: H&Y – 2.5 (1.50–3.0) IQR PD TD: H&Y – 2.0 (1.0–2.3) IQR	Author states that the main types of medication consumed by patients include hydrochloric acid benserazide (Madopar), pramipexole hydrochloride (Sifrol), and amantadine hydrochloride (Amantadine).
			Yin et al. (2021) China	Quantitative Evaluation of Gastrocnemius Medialis Stiffness During Passive Stretching Using Shear Wave Elastography in Patients with Parkinson's Disease: A	To investigate the feasibility of SWE as a new quantitative and objective method to evaluate muscle stiffness of the gastrocnemius medialis muscle in patients with PD during passive stretching.	PD: 28 CG: 12	PD: 63 \pm 8.5 CG: 59.3 \pm 6.4	PD: 15M/13F CG: 7M/5F	PD: 5.2 \pm 2.7

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Table 1 (continued)

Author & Country	Title	Sample Characteristics							
		Study Aim(s) and Objectives	n	Age (years) Mean \pm SD reported, unless mentioned otherwise	Sex	Disease Duration (years) and Mean \pm SD reported, unless mentioned otherwise	Severity of Motor Function (Assessed by UPDRS-III)	Symptoms Progression / Staging of Functional Disability (Assessed by Hoehn and Yahr scale)	Medication Details
Oh et al. (2016) South Korea	Prospective Preliminary Study. Assessment of Oropharyngeal Dysphagia in Patients With Parkinson Disease: Use of Ultrasonography.	To use US in healthy controls and PD patients to compare the shortest hyoid-thyroid distance, tongue thickness, and the time interval between the initiation of tongue movement and the shortest hyoid-thyroid approximation; and to demonstrate whether the above US parameters were correlated with PD severity and VFSS findings.	PD: 24 (all had dysphagia) CG: 24	PD: 71.67 \pm 5.10 CG: 66.71 \pm 12.97	PD: 24M CG: 24M	PD: 7.21 \pm 5.96	N/A	H&Y: 2 \pm 1.54	N/A
Lim et al. (2025) Taiwan	Muscle Ultrasonography as a Diagnostic Tool for Assessing Sarcopenia in Parkinson's Disease.	To evaluate the feasibility of muscle ultrasonography as a diagnostic tool for assessing sarcopenia in patients with Parkinson's disease.	PD: 85 (31 with sarcopenia, 54 without sarcopenia)	PD with sarcopenia: 73 (69–77) PD without sarcopenia: 66.5 (63–70.3) (median and interquartile range)	PD with sarcopenia: 15M/16F PD without sarcopenia: 26M/28F	N/A	PD with sarcopenia: 26.5 (16.8–33.3) PD without sarcopenia: 19.5 (11.8–26.3) (median and interquartile range)	H&Y PD with sarcopenia: 1.5 (1–2.5) PD without sarcopenia: 1 (1–2) (median and interquartile range)	Levodopa equivalent daily dose (mg/d): PD with sarcopenia: 750 (393.8–917.2) PD without sarcopenia: 568.8 (328.1–1001.6)
Calaway et al. (2025) United States of America	Velocity-Based-Training Frequency Impacts Changes in Muscle Morphology, Neuromuscular Performance, and Functional Capability in Persons With Parkinson's Disease.	To compare the impact of two different frequencies of velocity-based training—specifically, 2 versus 3 days per week—on changes in lower-limb muscle morphology (muscle thickness and echo intensity), neuromuscular performance (strength and power), and functional capacity in persons with Parkinson's disease.	PD: 47 individuals with PD were enrolled in the study, with 18 participants included in the final analysis—9 in the group training three days per week and 9 in the group training two days per week.	PD group training 2 days per week: 73.1 \pm 6.3 PD group training 3 days per week: 71.3 \pm 5.2	PD group training 2 days per week: 6M/3F PD group training 3 days per week: 6M/3F	PD group training 2 days per week: 6.3 6 5.1 PD group training 3 days per week: 5.7 6 4.1	N/A	H&Y: 1–3	Unclear. Author states that variations in PD medications used and time between administration of medications and testing or training may have affected the intervention and results.
Şirin Ahuşa et al. (2025) Turkey	Ultrasonographic assessment of dysphagia in Parkinson's disease: a controlled study.	To dynamically assess swallowing function in early- and mid-stage PD patients using ultrasonography and compare the findings with healthy controls.	PD: 30 (13 PD subjects with dysphagia, 17 PD subjects without dysphagia) CG: 30	PD: 71.50 (11) CG: 68 (7) (median and interquartile range)	PD: 15M/15F CG: 20M/10F	PD with dysphagia: 17 (36) PD without dysphagia: 12 (38)	UPDRS-III: PD with dysphagia: 26 (40) PD without	H&Y PD with dysphagia: 2 (2) PD without dysphagia: 2 (1)	Clinically stable and not in the off period, and not to be receiving any treatment other than routine antiparkinsonian medication that could

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Table 1 (continued)

Author & Country	Title	Study Aim(s) and Objectives	n	Age (years) Mean ± SD reported, unless mentioned otherwise	Sex	Disease Duration (years) and Mean ± SD reported, unless mentioned otherwise	Severity of Motor Function (Assessed by UPDRS-III)	Symptoms Progression / Staging of Functional Disability (Assessed by Hoehn and Yahr scale)	Medication Details
						Months, median (IQR)	dysphagia: 12 (10) median (IQR)	median (IQR)	affect swallowing function.

Abbreviations: aCC, acute camptocormia; ADIM, abdominal drawing-in manoeuvre; ADL, activities of daily living; CC, camptocormia; cCC, chronic camptocormia; CG, control group; CR, age-matched healthy controls; CY, young healthy controls; F, female; FS, force steadiness; H&Y, Hoehn and Yahr; IO, internal oblique; IQR, interquartile range; M, male; MT, muscle thickness; MTU, muscle-tendon unit; MVC, maximum voluntary contraction; N/A, not available; PD, Parkinson's disease; PIGD, postural instability gait disorder; SD, standard deviation; SWE, shear wave elastography; TD, tremor dominant; TRA, transversus abdominis; UPDRS, unified Parkinson's disease rating scale; US, ultrasound; VFSS, videofluoroscopic swallowing study; VL, vastus lateralis; wnCC, with no camptocormia.

3.4. Approach to measurement and reporting

A detailed summary of the US methodology used across the included studies is provided in Appendix A (Supplementary Material 2, online only). General Electric was the most frequently used US system, with 9 studies using this system. Most studies ($n = 14$) used a linear probe (Aktar et al., 2023; Calaway et al., 2025; Chen et al., 2023; Ding et al., 2024; Donizetti Verri et al., 2019; Göz et al., 2023; Lim et al., 2025; Magris et al., 2024; Martignon et al., 2021; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Monte et al., 2023; Umay et al., 2019), and three studies used a convex (curved) probe (Oh et al., 2016; Şirin Ahışha et al., 2025; Yilmaz et al., 2023). Anatomical assessment sites varied significantly between studies. With respect to probe/transducer frequency and sampling frequency, there was considerable variability in the way these parameters were reported. Some studies report the central frequency of the transducer (frequency of the US waves emitted by the transducer), while others mention sampling frequency (potentially the rate at which the US machine samples the returning echoes from the tissue and converts them into digital signals), though the exact meaning of these frequencies was not always clear. Moreover, probe positioning on the muscle of interest varied significantly across studies. The application of US transmission gel was documented in two studies (Abraham et al., 2020; Oh et al., 2016). Four studies (Abraham et al., 2020; Chen et al., 2023; Oh et al., 2016; Umay et al., 2019) reported minimal probe compression or efforts to avoid excessive pressure. Other US device settings (i.e., measurement depth, frame rate, gain) varied depending on the target muscle. All studies evaluated the muscle(s) of interest at rest ($n = 20$, see Supplementary Material 2, online only). Of these, eight studies used US to observe dynamic changes in muscle architectural parameters, with isometric contraction of lower limb muscles being the most assessed movement (Magris et al., 2024; Monte et al., 2023; Smart et al., 2020). Additionally, two studies assessed MA during the abdominal drawing-in manoeuvre (ADIM) (Aktar et al., 2023; Göz et al., 2023), while another three focused on mouth clenching and swallowing (Donizetti Verri et al., 2019; Şirin Ahışha et al., 2025; Umay et al., 2019). Three studies reported that the probe was attached to the muscle of interest (Magris et al., 2024; Monte et al., 2023; Smart et al., 2020). To prevent probe movement during dynamic assessments, the probe was secured onto the muscle of interest using a plastic strap (Magris et al., 2024) or a custom probe holder (Smart et al., 2020). The positioning of the participants' joints varied depending on the research objectives, the muscle being studied and the measurement parameter of interest (e.g. assessment of the posterior muscles via US is typically completed with the participants lying prone, while anterior muscles are assessed with participants in a supine position). For data analysis, filtering of the US data was not reported in any of the studies reviewed. Moreover, there were no reports of MA normalisation in the included studies. Finally, manual analysis of MA changes was used in nine studies (Aktar et al., 2023; Calaway et al., 2025; Lim et al., 2025; Magris et al., 2024; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Smart et al., 2020; Yilmaz et al., 2023), while one study utilised an automated tracking algorithm, UltraTrack, for the quantification of muscle architectural parameters (Monte et al., 2023), and the remaining studies using an unspecified/unclear method.

3.5. Clinimetric properties

Six of the 20 papers directly reported clinimetric properties of the muscle measurement technique(s) they employed (Abraham et al., 2020; Donizetti Verri et al., 2019; Lim et al., 2025; Oh et al., 2016; Şirin Ahışha et al., 2025; Yilmaz et al., 2023) and five papers only cited clinimetric values published previously (Aktar et al., 2023; Göz et al., 2023; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b), and the remaining nine studies did not report on the reliability or validity of the employed US measurement technique. The statistical method used to assess these clinimetric properties was the intraclass correlation

coefficient (ICC). The reliability of US assessments of MA in PD was consistently high across multiple studies. Ultrasonographic muscle architectural measurements demonstrated consistently high reliability. Intra-rater reliability was generally good to excellent, with ICCs ranging from 0.68 to 0.999 across different muscle groups, including craniofacial, limb, and swallowing muscles, and most studies reporting ICCs above 0.90, indicating excellent reproducibility when measurements were performed by the same examiner (Abrahin et al., 2020; Donizetti Verri et al., 2019; Lim et al., 2025; Oh et al., 2016; Şirin Ahışha et al., 2025). Inter-rater reliability was substantial to excellent, with ICCs between 0.75 and 0.97, reflecting consistent agreement across evaluators for both limb and paravertebral muscle assessments (Lim et al., 2025; Yilmaz et al., 2023). Together, these findings support the methodological robustness and reproducibility of ultrasonographic assessments of MA in clinical and research settings.

3.6. Reporting of additional outcomes

All twenty studies incorporated additional measures beyond muscle variable(s), reflecting the distinct aims of each study. These additional outcomes provide a more comprehensive understanding of PD and its effects. They not only expand on muscle-related findings but also illustrate how changes in MA may relate to broader PD symptomatology and progression. As PD impacts various aspects of health, including motor function, physical performance, and induces clinical manifestations, these additional outcomes offer valuable insights into the wider scope of the disease. The most common additional outcomes assessed included PD clinical manifestations ($n = 14$) (Aktar et al., 2023; Chen et al., 2023; Ding et al., 2024; Göz et al., 2023; Lim et al., 2025; Magris et al., 2024; Martignon et al., 2021; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Monte et al., 2023; Şirin Ahışha et al., 2025; Umay et al., 2019; Yin et al., 2021), PD symptom progression and staging of functional disability ($n = 12$) (Chen et al., 2023; Ding et al., 2024; Göz et al., 2023; Lim et al., 2025; Magris et al., 2024; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Monte et al., 2023; Oh et al., 2016; Şirin Ahışha et al., 2025; Umay et al., 2019), strength ($n = 8$) (Abrahin et al., 2020; Calaway et al., 2025; Chen et al., 2023; Donizetti Verri et al., 2019; Lim et al., 2025; Magris et al., 2024; Monte et al., 2023; Smart et al., 2020), physical performance, functional mobility, and capacity ($n = 7$) (Abrahin et al., 2020; Aktar et al., 2023; Calaway et al., 2025; Chen et al., 2023; Lim et al., 2025; Masaki et al., 2023a; Masaki et al., 2022) and muscle echogenicity/echo intensity (muscle quality) ($n = 5$) (Calaway et al., 2025; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Yilmaz et al., 2023).

3.7. Muscle architecture in individuals with PD

This section explores current findings on MA within individuals with PD and in comparison, to their healthy peers, during resting and contracted conditions. The main findings relating to the muscle variable(s) measured are presented comprehensively in Appendix A (Supplementary Material 3, online only).

3.7.1. Rested condition individuals with PD vs. healthy peers

A total of 13 studies examined MA in individuals with PD compared to their healthy peers in the resting condition (Chen et al., 2023; Ding et al., 2024; Donizetti Verri et al., 2019; Magris et al., 2024; Martignon et al., 2021; Masaki et al., 2022; Monte et al., 2023; Oh et al., 2016; Şirin Ahışha et al., 2025; Smart et al., 2020; Umay et al., 2019; Yilmaz et al., 2023; Yin et al., 2021). MA differences were small and inconsistent across studies. Most studies ($n = 9$) reported no significant differences in key muscle architectural parameters—MT, FL, PA, and CSA—between PD patients and healthy controls across various muscle groups (Chen et al., 2023; Magris et al., 2024; Martignon et al., 2021; Monte et al., 2023; Oh et al., 2016; Şirin Ahışha et al., 2025; Smart et al., 2020; Yilmaz et al., 2023; Yin et al., 2021). However, MT was significantly

smaller in the PD group for gluteus maximus (Masaki et al., 2022), tibialis anterior (Masaki et al., 2022), genioglossus (Umay et al., 2019), masseter (Donizetti Verri et al., 2019; Umay et al., 2019), temporalis (Umay et al., 2019), orbicularis oris (Umay et al., 2019), and sternocleidomastoid muscles (Donizetti Verri et al., 2019), yet greater MT for short head of the biceps femoris (Masaki et al., 2022) and temporalis muscles (Donizetti Verri et al., 2019). Further differences were found between PD and healthy controls when subgroups of the PD population were investigated. Ding et al. (2024) reported that PD patients with PIGD had significantly smaller gastrocnemius MT compared to healthy controls; however, the same study also noted greater gastrocnemius MT in PD patients with PIGD patients than in healthy controls, presenting contradictory findings that raises questions about the consistency of the results. Sex has also been shown to be an associated factor for specific MA parameters. When compared to healthy controls of the same sex female PD patients had reduced gastrocnemius MT, whereas male PD patients displayed greater gastrocnemius PA (Chen et al., 2023). With respect to studies investigating PD in combination with other conditions associated with the elderly, Chen et al. (2023) observed a significant reduction in gastrocnemius MT in male PD patients with sarcopenia compared to healthy controls with sarcopenia. Furthermore, Yilmaz et al. (2023) identified a significant reduction in MT of the lumbar paravertebral muscles in PD patients with chronic CC compared to healthy controls. In the study by Umay et al. (2019) healthy controls exhibited significantly thicker oral phase muscles (i.e., genioglossus, masseter, temporalis, orbicularis oris) compared to PD patients with dysphagia. Umay et al. (2019) also reported that PD patients with dysphagia had reduced CSA of the geniohyoid and anterior digastric muscles when compared to healthy controls.

3.7.2. Contracted condition individuals with PD vs. healthy peers

A total of six studies examined MA in individuals with PD compared to their healthy peers in the contracted condition (Donizetti Verri et al., 2019; Magris et al., 2024; Monte et al., 2023; Şirin Ahışha et al., 2025; Smart et al., 2020; Umay et al., 2019). The findings indicate that PD is associated with impaired muscle adaptation and responsiveness during contraction; but evidence is scarce and heterogenous. For instance, Magris et al. (2024) reported reduced VL MT and less FL shortening in the more affected limb of PD patients compared to healthy controls during maximum isometric voluntary contraction (MIVC) of the knee extensors. Similarly, Monte et al. (2023) observed smaller changes in MT and PA of the VL during knee extensor MIVCs in PD patients compared to healthy controls. However, findings are not consistent across all muscles. Smart et al. (2020) found no significant differences in medial gastrocnemius FL and PA during isometric plantar flexion between PD patients and healthy controls, however, fascicle shortening was greater in healthy controls than in PD patients. With respect to muscle function during dental clenching, Donizetti Verri et al. (2019) demonstrated a significantly greater temporal thickness and significantly thinner masseter and sternocleidomastoid muscles in PD patients compared to healthy controls. Moreover, Umay et al. (2019) reported that healthy controls exhibited significantly thicker oral phase muscles (masseter, temporalis, orbicularis oris) compared to both dysphagic and non-dysphagic PD patients in the contracted states.

3.7.3. Rested condition individuals with PD

Fifteen studies assessed MA within individuals with PD in the rested state, revealing both commonalities and distinct patterns (Abrahin et al., 2020; Aktar et al., 2023; Calaway et al., 2025; Chen et al., 2023; Ding et al., 2024; Göz et al., 2023; Lim et al., 2025; Magris et al., 2024; Masaki et al., 2023a; Masaki et al., 2023b; Monte et al., 2023; Şirin Ahışha et al., 2025; Umay et al., 2019; Yilmaz et al., 2023; Yin et al., 2021). Of these, four assessed bilateral measurements and found no significant differences in MA parameters (Chen et al., 2023; Magris et al., 2024; Monte et al., 2023; Yin et al., 2021). Furthermore, differences were found within PD patients when subgroups of the PD population were

investigated. Ding et al. (2024) reported that MT of the gastrocnemius was smaller in PD PIGD patients when compared to PD TD patients. Sarcopenia and sex have also been shown to be an associated factor for specific MA parameters within individuals with PD (Chen et al., 2023; Lim et al., 2025); for instance, Chen et al. (2023) found that PD sarcopenic patients showed reduced gastrocnemius MT compared to non-sarcopenic PD patients. With respect to sex, female PD patients with sarcopenia had smaller gastrocnemius MT compared to female PD patients without sarcopenia, whereas male PD patients with sarcopenia had smaller biceps brachii (BB) MT compared to male PD patients without sarcopenia (Chen et al., 2023). Similarly, in the Lim et al. (2025) study, the authors found that the PD sarcopenia group exhibited lower BB MT, TA MT, and TA CSA compared to the non-sarcopenia PD group. Furthermore, spinal muscle thinning was observed in PD patients with chronic CC, distinguishing them from PD patients without CC, whereas those with acute CC exhibited comparable MT with PD patients without CC (Yilmaz et al., 2023). One study assessed patterns of MT along anatomical gradients; they found that the distal sites of the BB muscle and knee extensor muscles were thinner than their medial and proximal landmarks (Abrahin et al., 2020). Meanwhile, analysis of craniofacial muscles revealed no significant differences in the relaxed genioglossus, masseter, temporalis, or orbicularis oris muscles between dysphagic and non-dysphagic PD patients (Umay et al., 2019). However, pharyngeal-phase muscles (i.e., geniohyoid and anterior digastric muscles) were significantly thinner in dysphagic PD patients compared to non-dysphagic PD patients, suggesting a localised thinning associated with swallowing dysfunction (Umay et al., 2019).

3.7.4. Contracted condition individuals with PD

In contrast to the rested state, six studies assessed MA within PD patients in the contracted state (Aktar et al., 2023; Göz et al., 2023; Magris et al., 2024; Monte et al., 2023; Şirin Ahışha et al., 2025; Umay et al., 2019). Of these studies, two studies focused on bilateral measurements in PD patients during MIVCs of the knee extensor (Magris et al., 2024; Monte et al., 2023), two studies assessed MA during the ADIM (Aktar et al., 2023; Göz et al., 2023), one study explored MA differences between PD patients with and without dysphagia during contractions of the face muscles (i.e., clenching the teeth, slight smile) (Umay et al., 2019), and one study assessed tongue thickness during swallowing of water (Şirin Ahışha et al., 2025). Magris et al. (2024) observed no differences in VL MT and PA between the more affected side and the less affected side in PD patients. However, there was a significant difference in FL between the more affected side and the less affected side; where the FL of the VL in the more affected side did not shorten as much as the less affected side during MIVCs of the knee extensors. Monte et al. (2023) reported significant differences in the change in VL PA during MIVCs, with the change in VL PA being smaller on the more affected side compared to the less affected side; there were no significant bilateral differences in the change in MT. Furthermore, in PD patients with dysphagia, the contracted state thickness of the masseter muscle was reported to be significantly reduced compared to those without dysphagia, suggesting impaired chewing function (Umay et al., 2019). Lastly, Şirin Ahışha et al. (2025) found that tongue thickness during swallowing of water is similar between PD patients with and without dysphagia.

3.7.5. Exercise-induced changes in muscle architecture in PD

Two studies examined how exercise affects MA in PD (Calaway et al., 2025; Göz et al., 2023). Göz et al. (2023) reported that six weeks of Pilates (twice weekly) significantly increased abdominal MT, particularly in the IO and TrA during rest and ADIM. Many of these MT gains were maintained at the 12-week follow-up. Calaway et al. (2025) found that 12 weeks of velocity-based resistance training increased MT in the RF and VL bilaterally. Training 2 vs. 3 days per week produced similar hypertrophic effects (Calaway et al., 2025). Overall, both studies show that targeted exercise such as Pilates and velocity-based resistance

training can increase muscle size in people with PD.

3.7.6. Correlations with PD clinical manifestations

The correlation between MA and clinical manifestations of PD was explored in four studies (Aktar et al., 2023; Magris et al., 2024; Masaki et al., 2023a; Şirin Ahışha et al., 2025). The MDS-UPDRS and H&Y have been frequently used as a scale to grade the clinical manifestations of PD and disease progression/staging. Aktar et al. (2023) found no significant correlation between MT of transversus abdominis and clinical manifestations of PD, whereas MT of internal oblique at rest and percentage change (the change in MT between at rest and during ADIM) were significantly correlated with MDS-UPDRS-I, MDS-UPDRS-II, rigidity and bradykinesia subscores of MDS-UPDRS-III. In the Masaki et al. (2023a) study, increased UPDRS-III score was related to decreased tibialis anterior MT. In the Magris et al. (2024) study there were no significant correlations between VL MT, PA, FL and UPDRS and H&Y. Similarly, Şirin Ahışha et al. (2025) reported no significant correlations between ultrasonographically assessed tongue thickness, and clinical measures of disease severity.

3.7.7. Correlations with functional performance

For the purposes of this review, “functional performance” refers to the ability to perform physical tasks and movements that are relevant to daily activities or physical assessments. This includes tasks like standing, walking, lifting, or maintaining balance which require strength, which are often used to evaluate muscle function and overall physical capacity. Several studies ($n = 6$) examined the relationship between MA and functional performance in individuals with PD (Abrahin et al., 2020; Aktar et al., 2023; Lim et al., 2025; Magris et al., 2024; Masaki et al., 2023a; Masaki et al., 2023b). The findings, however, are mixed; two studies found no significant associations (Abrahin et al., 2020; Magris et al., 2024). In contrast, other studies observed that abdominal and lumbar MT were significantly associated with core endurance, balance, gait speed, or postural alignment (Aktar et al., 2023; Masaki et al., 2023a; Masaki et al., 2023b). In the Lim et al. (2025) study, the authors further demonstrated positive correlations between lower-limb MT/CSA and handgrip strength, walking speed, balance scores, and appendicular skeletal muscle mass index, while smaller muscle size related to poorer functional scores. Overall, these findings suggest that greater MT and CSA generally correspond to better strength, gait, and balance in PD, supporting the functional relevance of US-derived muscle parameters in this population.

4. Discussion

This scoping review provides a comprehensive summary of 20 studies using the US to investigate MA in individuals with PD. Regarding the first research question, the application of US in PD research is relatively recent, with the earliest publication dating back to 2016 and seven studies published in 2023 alone. This trend highlights global advances in US technology and growing interest in understanding musculoskeletal impairments in PD. The included studies varied considerably in aims, origin, design, and sample characteristics. This heterogeneity complicates direct comparisons but illustrates the broad interest in this area of research. For the second research question, US measurement methodology (i.e., muscle of interest, reported muscle architectural variable) and approach to measurement and reporting (i.e., US system and acquisition procedure) varied significantly between studies. Only a minority of studies evaluated the clinimetric properties of the muscle measurement technique(s) they employed, highlighting the need for greater methodological standardisation. With respect to the third research question, at rest MA differences between PD patients and controls were minimal, though isolated variations (e.g., sex, PD subtype, PD-related comorbid conditions) were noted. During muscle contractions, however, PD patients exhibit impaired responsiveness highlighting neuromuscular dysfunction. Emerging evidence suggests

associations between MA and both PD manifestations (e.g., rigidity, bradykinesia) and functional performance, indicating that worsening PD symptoms may be associated with MA impairments, which in turn could contribute to poor physical performance. Furthermore, the impact of exercise on MA remains unclear. Only two intervention studies—one using Pilates-based training and one employing velocity-based resistance training—were identified, both of which demonstrated increases in MT; this highlights a critical gap in the literature regarding whether exercise interventions can modify MA and lead to improvements in strength, function, and overall performance in PD populations.

4.1. What is the extent of the research?

4.1.1. Geographic distribution and study design

The published research originated from research groups based in East Asia, Europe, North America, and South America. A notable feature of the included studies was the broad variation in research design and objectives. The majority of the included studies utilised a cross-sectional study design (Abraham et al., 2020; Aktar et al., 2023; Chen et al., 2023; Ding et al., 2024; Magris et al., 2024; Martignon et al., 2021; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b; Monte et al., 2023; Oh et al., 2016; Şirin Ahisha et al., 2025; Smart et al., 2020; Umay et al., 2019; Yilmaz et al., 2023; Yin et al., 2021) to compare variable(s) of muscle(s) (i.e., within people with PD, between people with PD and healthy controls, or between 'more affected side' and 'less affected side'). One study adopted a case-control design (Donizetti Verri et al., 2019), while another employed a prospective single-centre diagnostic design (Lim et al., 2025). In addition, one study was a single-blind controlled clinical trial (Göz et al., 2023), and another applied a randomised parallel experimental design, representing a secondary analysis of two randomised parallel trials (Calaway et al., 2025). Longitudinal research which tracks the natural progression of muscle in PD was noticeably absent. While the numerous interventions available to persons with PD admittedly make longitudinal study designs challenging and restrictive, certain questions can only be answered using repeated-measures or longitudinal methods. Therefore, efforts are needed to investigate how these study designs might be incorporated into clinical care (Vaswani et al., 2020).

4.1.2. Disease severity, functional disability and PD-related comorbidities

Regarding the severity of motor function, current research tends to include a mix of individuals with relatively high motor functioning and those with more substantial deficits. However, in terms of disease progression and functional disability, the current body of research predominantly investigates individuals with lower levels of functional disability; few studies have investigated muscle parameters in PD patients at more advanced stages of the H&Y—such as those who are still able to stand or walk unaided despite severe disability (H&Y stage 4) (Masaki et al., 2023a; Masaki et al., 2022; Oh et al., 2016), and only one study included PD patients who were wheelchair-bound or bedridden unless assisted (H&Y stage 5) (Oh et al., 2016). Investigating MA across low and higher spectrums of disease severity and disability, or both early and late phases of PD is crucial, as it can provide a comprehensive understanding of disease progression and inform targeted therapeutic interventions for all stages of the condition. This constrains the generalisability of current findings, as severe disease may entail greater muscle atrophy, echogenicity changes, and architectural disorganisation from rigidity and disuse; future research should therefore include individuals with advanced PD stages to delineate disease-stage-specific patterns of muscle change. Some PD patients also presented PD-related comorbidities such as sarcopenia, CC, and dysphagia (Chen et al., 2023; Oh et al., 2016; Umay et al., 2019; Yilmaz et al., 2023); these three conditions are common in PD (Ponsoni et al., 2023; Srivanthapoom and Hallett, 2016; Suttrup and Warnecke, 2016), and may each independently influence muscle structure and function (Laroche et al., 1995; Larsson et al., 2019; Margraf et al., 2016; McCarty and Chao, 2021). As a

result, it is difficult to disentangle muscle changes attributable to PD from those arising from secondary comorbid processes. The lack of systematic screening or adjustment for these factors may therefore confound the interpretation of US-derived muscle parameters. Future research should incorporate structured assessment of these comorbidities and consider them in study design, analysis, and reporting to improve the accuracy and interpretability of muscle-related outcomes in PD.

4.1.3. Muscle selection and assessment

The VL, RF and gastrocnemius (medial and lateral) were the most common muscles assessed; likely owing to being superficial muscles, which allows for easier measurement via US, and their known clinical relevance for PD (Allen et al., 2010) and functional importance (de Almeida et al., 2022; Nocera et al., 2010; Stevens-Lapsley et al., 2012). However, this common focus presents many issues. Most of our current understanding of PD muscle is derived from the lower-limb muscles, which may not necessarily provide a complete reflection of PD muscle. Second, several studies reported on outcomes of only the VL muscle (Magris et al., 2024; Martignon et al., 2021; Monte et al., 2023). While the VL is undoubtedly functionally important, contributing to knee extension and gait mechanics (Allen et al., 2010; de Almeida et al., 2022; Nocera et al., 2010; Stevens-Lapsley et al., 2012), muscles very rarely work in isolation; muscles and muscle groups continuously coordinate their activation and synchronise their functions to maintain posture, stability and balance of the body at rest and during dynamic movement (Kimura et al., 2021). We therefore recommend that future research should consider exploring the muscle group (e.g., quadriceps femoris complex, triceps surae complex) rather than a single muscle (e.g., VL, RF). Third, since most studies have examined lower limb muscles, the upper limb musculature remains largely unexplored; this is restrictive, since many clinically relevant PD symptoms and standard clinical assessments involve the upper limbs, including finger tapping, bradykinesia evaluation, and tremor, which is predominantly assessed in the upper extremities (Bhidayasiri and Martinez-Martin, 2017); this is a major gap that reduces the translational value of the current scientific evidence. Expanding research to include upper-limb and deeper muscles will provide a more comprehensive understanding of muscular changes in PD.

4.1.4. Anti-parkinsonian medication

A small number of studies explicitly reported that PD patients were consuming anti-parkinsonian medication(s) (Ding et al., 2024; Magris et al., 2024; Şirin Ahisha et al., 2025; Smart et al., 2020; Yilmaz et al., 2023; Yin et al., 2021), and most studies tested patients during the "ON" phase, when medication is active, and symptoms are controlled (Aktar et al., 2023; Ding et al., 2024; Göz et al., 2023; Lim et al., 2025; Magris et al., 2024; Martignon et al., 2021; Masaki et al., 2022; Masaki et al., 2023b; Monte et al., 2023; Smart et al., 2020; Umay et al., 2019; Yilmaz et al., 2023). Although anti-parkinsonian medications target dopaminergic pathways rather than peripheral muscle tissue directly, their potential influence on MA has not been examined and remains an open question. Existing literature demonstrates clear effects of dopaminergic medication on neuromuscular activation and biomechanical performance, including alterations in voluntary activation, electromyogram amplitude, and maximal torque output (Folland et al., 2011). These findings indicate that medication can substantially modify neural drive and motor behaviour. However, it is unknown whether such physiological and biomechanical changes extend to muscle mechanical properties such as fascicle behaviour, pennation dynamics, or muscle-tendon interaction, all of which are directly relevant to US-based MA assessment. Interestingly, Calaway et al. (2025) reported that variations in PD medications used and time between administration of medications and testing or training may have affected the intervention and results. Therefore, future US MA research should carefully document medication regimens and consider testing both "ON" and "OFF" states to clarify

whether and how medication status affects MA dynamics.

4.2. Ultrasound measurement techniques of muscle architecture in PD and methodological quality

This review confirms that US has emerged as a practical, accessible modality for examining MA in people with PD. However, the utility of US in PD research depends on measurement methodology (i.e., muscle of interest, architectural variables measured), and the rigour with which acquisition and reporting procedures are implemented. These considerations form the foundation for evaluating how US has been applied in the current literature and highlight the methodological issues, as outlined below. Furthermore, the published research had challenges with small sample sizes, varying PD severity and disease staging, differences in PD subtypes, medication use, and other confounding factors. Sample size is crucial, because a sample size that is smaller than necessary would have insufficient statistical power, and a statistically non-significant result could merely be because of inadequate sample size (type II or false negative error). The included studies may have lacked an adequately powered sample, making it difficult to draw strong conclusions about MA in PD patients and limiting confidence in their findings. A properly calculated sample size helps increase the reproducibility of studies and generalisability of the results.

4.2.1. Measurement parameters and influencing factors

The muscle architectural parameters assessed include MT and CSA (both indicators of muscle size and mass), FL (representing the number of sarcomeres arranged in series, influencing contraction velocity), and PA (reflecting the fibre packing strategy where shorter fibres are oriented at an angle to the line of pull). Interpretation of these parameters are highly sensitive to methodological choices; there is robust evidence that joint position, contraction state, and region of muscle interest are known to influence MA parameters (Abrahin et al., 2020; Charles et al., 2022; Coenning et al., 2024; Fukunaga et al., 1997; Herbert and Gandevia, 1995; Torres et al., 2017). Attention to these factors is important in PD; joint angle could increase muscle stiffness, bradykinesia could affect fascicle contraction dynamics (Allen et al., 2009), and tremor introduces involuntary oscillatory activity (Dirkx and Bologna, 2022), all of which could modify MA at the moment of US measurement. The current review confirms major variation in joint positioning across studies, and none used electromyography (EMG) to monitor unintended muscle activation during MA assessment (Donizetti Verri et al., 2019; Martignon et al., 2021; Monte et al., 2023; Yin et al., 2021). Careful control and transparent reporting of these factors are essential to ensure that observed differences in MA reflect true disease-related changes rather than measurement artefacts.

4.2.2. Image analysis techniques and extrapolation methods

MA was analysed mostly manually using the inherent tools of the US machine (i.e., caliper-based tools, linear measurement function) (Aktar et al., 2023; Smart et al., 2020; Yilmaz et al., 2023). Four studies employed image processing software, ImageJ, to analyse MA parameters manually (Calaway et al., 2025; Masaki et al., 2023a; Masaki et al., 2022; Masaki et al., 2023b), while one study manually analysed MA through a MATLAB custom script (Magris et al., 2024). Yet, manual analysis is laborious and subjective and requires experience (Sarto et al., 2021). Furthermore, manually analysing large image datasets is not only time consuming but also leads to reduced focus and, thus, error proneness (Ritsche et al., 2024). One study (Monte et al., 2023) used an automated tracking software, UltraTrack, to quantify muscle architectural variables; UltraTrack has been found to be repeatable, accurate, and time-efficient, offering a more objective alternative to manual analysis (Gillett et al., 2013). Auto tracking should be considered in future studies to improve reliability and validity of data processing methods. Where not possible robust definitions of manual processing methods should be reported and inter-analyst reliability conducted to

demonstrate reliability of the process. Moreover, two studies (Magris et al., 2024; Smart et al., 2020) used extrapolation when fascicles extended beyond the US field-of-view, but methodological details were limited or unclear. This lack of transparency is important, as FL estimation depends heavily on the extrapolation technique used. Common approaches include: manual linear extrapolation (MLE) (Potier et al., 2009); a trigonometric equation proposed by Blazeovich et al. (2006), providing FL estimations based on linear extrapolation of a straight line parallel to a visible fascicle portion; and a second trigonometric equation, as detailed by Finni et al. (2001), where only the nonvisible segment of the fascicle (outside the field of view) is linearly extrapolated. A study by Franchi et al. (2020) showed that while all methods are reliable, accuracy varies markedly: trigonometric equations—particularly Blazeovich et al. (2006)—tend to overestimate FL, whereas MLE shows the closest agreement with extended field-of-view imaging. Extended field-of-view US is recommended for accurate FL assessment, and if unavailable, MLE using the largest possible field-of-view is preferred, though further validation is needed (Franchi et al., 2020). Consequently, FL measurements reported in the studies included in this review may be inaccurate or non-comparable, which limits confidence in their architectural findings.

4.2.3. Probe selection and imaging conditions

In terms of the types of probes employed, it should be noted that while linear array probes were commonly used, their reported frequencies varied. Linear probes generally offer higher-resolution images, but they are limited in penetration depth due to their higher frequency range (Markowitz, 2011; Royall et al., 2011). The use of curvilinear probes, which were reported in three studies (Oh et al., 2016; Şirin Ahisha et al., 2025; Yilmaz et al., 2023), may indicate an attempt to image deeper anatomical structures, as curvilinear probes typically operate at lower frequencies, which allow for greater tissue penetration but at the cost of resolution (Markowitz, 2011; Royall et al., 2011). However, the exact reasoning behind choosing one type of probe over another was articulated in the studies reviewed. Most studies obtained three images per muscle site, which is strongly recommended because the US is an “operator dependent” method and calculating the average of three measures ensures more accurate results. Most studies measured MA in resting conditions, but some used US to examine muscle behaviour during dynamic movement. The latter approach presents several technical challenges. First, the transducer must be securely fixed to the limb, which compresses underlying structures (Cronin and Lichtwark, 2013). Second, operators must ensure that the image plane remains aligned with the fascicle direction; misalignment of the transducer may cause FL measurements to be underestimated or overestimated (Bolsterlee et al., 2016). Maintaining this alignment during dynamic movements is challenging, especially since muscle fascicle arrangement and shape change with contraction.

4.2.4. Reliability and blinding

Although not included in the results, assessor blinding was rarely reported in the included studies (Ding et al., 2024; Umay et al., 2019; Yilmaz et al., 2023). While blinding the assessor during US-based muscle parameter acquisition can be challenging, it is possible to blind the individual responsible for digitizing the US assessments to the diagnostic and intervention status and participant information. Reliability testing of the acquisition and/or digitizing of the measurement technique was reported in less than 25% of the papers, with most of the studies either not reporting it at all or citing previous studies, often involving middle-aged and elderly populations (particularly women), systematic reviews, or studies including children and adolescents with cerebral palsy. While the US is generally reliable for assessing MA in healthy populations (Kwah et al., 2013), its suitability for PD patients remains unclear. Even though the reported ICCs identified through this scoping review were good (in those studies that did report reliability), the potential for measurement error must be considered due to the unique challenges

posed by PD. Tremors, muscular stiffness, and bradykinesia in PD may complicate the assessment of MA. Future studies should address these issues by incorporating rigorous reliability and validity testing in their designs.

4.2.5. Data analysis and normalisation

For data analysis, filtering of the US data was not reported in any of the studies reviewed. Due to the inherent noise in US based muscle tracking, especially during dynamic tasks, temporal filtering (i.e., low-pass filtering) is typically expected to reduce measurement error. The absence of reported filtering methods raises concerns about the reliability of dynamic measurements. Furthermore, a significant finding is that none of the included studies in this scoping review reported normalising MA parameters. Normalisation is crucial for allowing comparison among muscles, but also reducing variability related to individual differences in body size, limb length, or muscle mass, making results more comparable across populations (Son et al., 2024; Ward et al., 2009). The broader literature has been shown to have conducted normalisation to account for individual differences in body size or morphology, for instance, FL to the length of the leg (Shortland et al., 2002) or height (Gao et al., 2011), and CSA to body mass (Bland et al., 2011). We do not propose the correct answer to normalising MA parameters, as there appears to be no consensus across the literature (both healthy and diseased populations).

4.3. Muscle architecture

The underlying mechanism of MA alterations in PD are unknown; however, in healthy muscle, MA is shaped by load-dependent processes whereby FL, PA and MT remodel in response to mechanical demand (Blazevich and Sharp, 2006). These adaptations are regulated by satellite-cell activity, IGF-1/Akt-mTOR signalling, protein turnover via the ubiquitin-proteasome and calpain systems, and mechanosensitive proteins such as titin and dystrophin (Blazevich and Sharp, 2006). In the context of PD, however, MA may be influenced not only by mechanical loading but also by the CNS directly or indirectly. Impaired motor unit recruitment, reduced neuromuscular drive, and compromised neuromuscular transmission could inhibit contraction-induced architectural adaptation; these neuromuscular deficits may also contribute to disuse-related atrophy and structural remodelling, ultimately accelerating muscle wasting, weakness and functional decline. Across studies, resting MA in PD appears largely preserved relative to healthy controls, especially in physically active PD patients (Magris et al., 2024; Martignon et al., 2021; Monte et al., 2023). Yet, structural preservation does not translate into normal neuromuscular function, as weakness and mobility impairments persist (Chen et al., 2023). Within this broader pattern of MA preservation, selective and clinically relevant atrophy is found in gait (Masaki et al., 2022) and orofacial muscles (Donizetti Verri et al., 2019; Umay et al., 2019), which may stem from disuse, altered activation, or chronic denervation, while increased thickness in compensatory muscles (e.g., biceps femoris, temporalis) reflects maladaptive recruitment. Moreover, sex-specific patterns—thinner gastrocnemius in females and greater PA in males with PD (Chen et al., 2023)—suggest that males with PD had increased PA (Chen et al., 2023). Indicating sex may influence the distribution and severity of muscle wasting in PD, potentially driven by hormonal, genetic, or activity-level differences (Atkinson et al., 2010; Roth, 2012), and warrant consideration in clinical assessments and therapeutic strategies.

The PD-related comorbidities—CC, sarcopenia, and dysphagia—also contribute to greater muscle degradation (Chen et al., 2023; Lim et al., 2025; Umay et al., 2019; Yilmaz et al., 2023). Importantly, the trunk-focused studies included in this review highlight a distinct trajectory of involvement in axial musculature; while most paraspinal and abdominal muscles appear structurally preserved in PD, reduced lumbar paraspinal thickness was observed specifically in individuals with chronic CC (Yilmaz et al., 2023), indicating that axial muscles may

become compromised when postural deformities are present. This pattern is consistent with biopsy evidence showing chronic myopathic changes, fibre disorganisation, and increased connective tissue in the paraspinal muscles of people with PD-related camptocormia (Spuler et al., 2010), supporting the possibility of direct muscle involvement in this phenotype. In PD without such deformities, trunk-muscle alterations were modest, although some studies reported associations between spinal alignment, muscle quality, and reduced activation of deep abdominal muscles. Taken together, these findings suggest that axial muscles, while generally preserved in typical PD, may be disproportionately vulnerable to chronic postural load, rigidity, and neuromuscular disruption when postural abnormalities develop. Accordingly, muscle-group-specific patterns should be considered when interpreting MA in PD.

Architectural impairments become apparent during muscular contractions in patients with PD compared to healthy controls. Monte et al. (2023) and Magris et al. (2024) suggested that the inability of the muscle to change shape in PD results from increased muscle stiffness (rigid hypertonia), although a direct mechanistic link has not yet been confirmed. Evidence from spastic cerebral palsy populations (Åhblom et al., 2024) suggest that increased stiffness (spasticity) is frequently accompanied by shorter fascicles, reduced PA, and restricted joint range of motion, supporting the hypothesis that hypertonia alters the mechanical behaviour of muscle fibres. However, whether this mechanism fully explains PD-specific architectural impairments requires further investigation. Furthermore, at rest, studies show consistent bilateral MA symmetry in patients with PD (Chen et al., 2023; Magris et al., 2024; Monte et al., 2023; Yin et al., 2021); this bilateral symmetry suggests that, in the rested state, muscle morphology alterations in PD may manifest more as a systemic characteristic rather than unilateral deficits. In contrast, the ability of the muscle to change shape is impaired on the more affected limb in PD (Magris et al., 2024; Monte et al., 2023); importantly, these alterations occurred even though maximal isometric torque was comparable bilaterally, indicating that PD primarily limits dynamic, rather than static, force-generating capacity. Supporting this interpretation, the more affected limb exhibited slower and diminished torque development, reduced power output, and lower EMG amplitudes, all of which point to deficient neuromuscular activation rather than intrinsic contractile impairment. Collectively, these findings suggest that PD disrupts the interaction between neural drive and functional architectural adjustments, particularly during tasks requiring rapid or high-intensity muscle activation.

4.3.1. Exercise and muscle architecture in PD

Evidence examining how exercise influences MA in PD is limited, with only two studies directly investigating this relationship (Calaway et al., 2025; Göz et al., 2023); as a result, it is insufficient to draw clinical implications. Nevertheless, their findings suggest that exercise and/or physical activity can promote hypertrophic adaptations, as reflected by increased MT; this suggests that despite neurodegenerative constraints, the skeletal muscle of individuals with PD retains the capacity for remodelling when stimulated. These findings align with evidence from broader exercise physiology and biomechanics research, where resistance strength and aerobic training induce architectural adaptations in both healthy individuals (Aagaard et al., 2001; Alegre et al., 2006; Gondin et al., 2005; Kawakami et al., 1995; Nunes et al., 2024; Timmins et al., 2016) and those with chronic conditions (Alcazar et al., 2019; Krase et al., 2022; Westerberg et al., 2018). In PD, these findings underscore the potential of exercise to counteract disuse-related atrophy and neuromuscular inefficiency, but the current evidence base remains sparse and short-term. Longitudinal trials integrating pre-post US assessments, EMG, and functional outcomes are essential to clarify whether architectural improvements translate into better motor control and performance. Future studies should also examine how training variables—such as contraction mode (e.g., concentric, eccentric), range of motion, and velocity—shape fascicle and pennation adaptations in PD

muscle, as these factors critically influence force production and movement efficiency in other populations (DE Oliveira et al., 2023; Lieber and Fridén, 2001; Timmins et al., 2016).

4.3.2. MA and clinical manifestations of PD

Utilising US to identify alterations in MA, and examining how these relate to clinical manifestations of PD, holds promise for improving early detection of muscular impairments that may contribute to functional decline. Research directly linking MA with PD manifestations is still in the early stages. Across the available studies, relationships between MA and clinical severity appear to be both muscle-specific and symptom-dependent, suggesting that PD does not affect all muscles uniformly. Findings such as the association between reduced distal lower-limb MT and higher UPDRS motor scores support the idea that progressive motor impairment may contribute to disuse-related atrophy in muscles critical for gait and postural control (Masaki et al., 2023a). Conversely, correlations between IO thickness and rigidity or bradykinesia imply that axial musculature may undergo compensatory or maladaptive adaptations in response to core PD symptoms (Aktar et al., 2023); this aligns with prior work suggesting that trunk muscle activation patterns are altered in PD, potentially as a strategy to maintain postural stability amid impaired motor control (Cole et al., 2017). In contrast, the absence of a relationship between knee extensor MT and clinical severity in the study by Magris et al. (2024) may reflect the moderating influence of physical activity levels. Given that participants in Magris et al. (2024) study were physically active, it is plausible that regular physical exercise attenuated the expected muscle loss or dysfunction, a hypothesis consistent with studies showing preserved lower limb function because of exercise and/or physical activity in PD populations (Falvo et al., 2008; Lauzé et al., 2016). Overall, these early findings point toward a complex interaction between disease severity, muscle-group function, and behavioural factors, underscoring the need for more targeted research to clarify how MA relates to clinical progression in PD.

4.3.3. MA and functional performance in PD

Using US-derived MA measures to understand functional capacity in PD is increasingly promising, although research remains limited. Available evidence indicates that relationships between MA and function are muscle- and task-specific, rather than uniform. Findings from Aktar et al. (2023) show that dynamic activation of deep abdominal muscles, rather than resting thickness, relates more strongly to core endurance and mobility, highlighting the importance of neuromuscular control. Trunk-focused studies similarly suggest that reduced lumbar extensor thickness and quality (echo intensity) is linked to poorer balance and postural alignment (Masaki et al., 2023a; Masaki et al., 2023b), consistent with broader ageing literature in which reduced paraspinal MT and quality (echo intensity) are associated to impaired balance and sagittal alignment (Kim et al., 2019a; Kim et al., 2019b). Lower-limb studies also support a functional role for MA, with greater MT or CSA associated with better gait speed, strength, and balance—particularly in those who are PD sarcopenic (Lim et al., 2025). However, several studies report no clear associations, reflecting heterogeneity in disease severity, assessment methods, and physical activity levels. Overall, the emerging evidence suggests that both muscle structure and activation capacity contribute to functional performance in PD, underscoring the need for integrated assessments in future research.

It is evident there's a strong interest in understanding how skeletal muscle characteristics relate to both functional abilities and clinical symptoms; however, to fully grasp muscle's influence on overall health and mobility in individuals with PD, a comprehensive view of muscle health, encompassing its morphological and underlying biological processes like metabolism and cellular functions, is essential (Caviness et al., 2000; Glendinning and Enoka, 1994; Murphy and Lynch, 2023; Nicoletti et al., 2021; Seo and Yeo, 2021). Focusing on morphological aspects, PD affects muscle function which is influenced by MA (e.g., FL, PA, MT) which is closely associated with muscle strength, power, and

rate of torque development (RTD), as it reflects the structural characteristics that underpin muscular force generation (Ando et al., 2015; Freilich et al., 1995; Strasser et al., 2013). This is important as people with PD are characterised by muscle weakness, demonstrating significant impairments in mechanical muscle function—including strength, power, and RTD (Gamborg et al., 2023). These impairments in muscle function in individuals with PD have been described in terms of low peak force or torque, as assessed during isokinetic (dynamic) and isometric maximal voluntary contractions (Koller and Kase, 1986; Manca and Deriu, 2025; Monte et al., 2023; Skinner et al., 2019; Smart et al., 2020; Stelmach et al., 1989; Stelmach and Worringham, 1988), slow RTD (Monte et al., 2023; Park and Stelmach, 2006; Stelmach et al., 1989; Stelmach and Worringham, 1988), and altered force variability (Skinner et al., 2019; Smart et al., 2020; Stelmach et al., 1989). Future research should focus on deeper exploration of the role of MA in force production and strength, as the current evidence is scarce.

4.4. Limitations of the review

This review is not without limitations. Despite an extensive literature review, some relevant studies may have been missed due to database selection, search terms, and applied filters. For instance, only articles published in English were included, which may limit the generalisability of the findings to studies conducted in other languages, regions, or populations. There is also a risk of bias as study selection was conducted by one reviewer, however to minimise the impact of this all articles where eligibility was uncertain were decided in combination with co-authors. Additionally, the exclusion of conference proceedings and short communications may have led to the omission of emerging research in this area. Since this is a scoping review, all original research studies were included regardless of study design or methodological quality, which presents a potential risk of bias from lower-quality studies. Assessing study quality, performing statistical analyses, or evaluating the robustness of statistical methods used in the included studies was beyond the scope of this review. The reporting of ICCs for reliability analyses and statistically significant correlations was intended to summarise research activity rather than to draw direct statistical comparisons across studies.

4.5. Future research considerations and recommendations

This review suggests that understanding PD muscle data is nearly as intricate as PD itself. Several factors complicate the synthesis of our current knowledge and a consistent interpretation of findings. These include wide variations in study characteristics, methodology, US techniques, the specific muscle architectural variables measured, how results are reported (whether static or dynamic), and the reporting conditions. Adding to this complexity are the inherent heterogeneity of PD among individuals and the naturally dynamic and adaptable nature of skeletal muscle.

We recommend:

- 1) Transparent and detailed reporting, including key patient information—such as age, gender, height, body mass, race, health history, disease severity and duration, participation in treatment or therapy, physical activity levels, and exercise prior to testing—to enhance comparability and interpretation across studies.
- 2) Tailoring the choice of MA measurement and selected muscles to the research question's goals. Muscles should be considered within their functional groups, rather than individually. During assessment, it is crucial to either eliminate or report on potential confounding variations in muscle measurement, such as joint positioning.
- 3) Future research should evaluate the necessity and methods for normalising MA measurements to ensure meaningful comparisons across individuals and studies. Studies must clearly report how MA values are derived, including fascicle angle measurements and any extrapolation techniques used.

4) Longitudinal studies measuring MA should be carried out to gain insights into how muscle changes progress as PD advances.

5) Future studies should recruit larger, more demographically diverse, and clinically representative cohorts, including individuals with advanced PD and greater motor impairment. Current research is skewed toward early-to-moderate disease stages. Additionally, greater emphasis should be placed on underexplored muscle groups—particularly upper limb and fine motor muscles—which are clinically significant yet often overlooked in existing literature.

6) More studies of dynamic muscle function are needed if we are to understand how limb muscles function during activities of daily living.

7) People with PD experience gait difficulty, we propose that future studies should explore the relationship between MA and walking gait and turning.

8) Future research should integrate US with wearable sensors, EMG, and inertial or force-based technologies to enable multimodal, longitudinal monitoring of muscle structure and function, supporting the development of personalised and adaptive rehabilitation strategies in PD.

9) Interventional studies involving exercise, pharmacological, neuromodulation, and neurosurgical approaches are needed to clarify causal links between treatments, muscle adaptation, and clinical outcomes, including the potential for neuromodulatory interventions such as AMPS to influence MA.

10) Further investigation is needed to determine how MA contributes to force generation, strength, power, peripheral fatigue and RTD, as the mechanical implications of architectural changes in PD remain unclear despite established biomechanical principles.

11) Finally, further research is needed to examine the relationship between MA and PD's clinical manifestations, identifying how motor symptoms such as tremor, bradykinesia, and muscle stiffness contribute to alterations in MA.

CRedit authorship contribution statement

Mahyar Behraznia: Writing – review & editing, Writing – original draft, Resources, Methodology, Investigation, Conceptualization, Data curation, Formal analysis. **Massimiliano Ditroilo:** Writing – review & editing, Supervision, Methodology, Investigation, Conceptualization, Validation, Formal analysis. **Tina Smith:** Writing – review & editing, Validation, Supervision, Methodology, Investigation, Conceptualization, Formal analysis.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.clinbiomech.2025.106733>.

References

- Aagaard, P., Andersen, J.L., Dyhre-Poulsen, P., Leffers, A.M., Wagner, A., Peter Magnusson, S., et al., 2001. A mechanism for increased contractile strength of human pennate muscle in response to strength training: changes in muscle architecture. *J. Physiol.* 534, 613–623. <https://doi.org/10.1111/j.1469-7793.2001.t01-1-00613.x>.
- Abbruzzese, G., Marchese, R., Avanzino, L., Pelosin, E., 2016. Rehabilitation for Parkinson's disease: current outlook and future challenges. *Parkinsonism Relat. Disord.* 22, S60–S64. <https://doi.org/10.1016/j.parkreldis.2015.09.005>.
- Abraham, O., Souza, D.M., da Costa Alves, W.M.G., da Silva, A.F., 2020. Muscle thickness and functional performance of patients with Parkinson's disease. *Revista Brasileira de Cineantropometria e Desempenho Humano* 22, e60774. <https://doi.org/10.1590/1980-0037.2020v22e60774>.

- Åhblom, A., Pontén, E., Destro, A., Petersson, S., von Walden, F., Wang, R., et al., 2024. Exploration of the triceps surae muscle in ambulatory children with cerebral palsy using instrumented measurements of stiffness and diffusion tensor magnetic resonance imaging for muscle architecture. *BMC Musculoskelet. Disord.* 25, 1–12. <https://doi.org/10.1186/s12891-024-07890-4>.
- Aktar, B., Ozyurek, S., Goz, E., Colakoglu, B.D., Balci, B., 2023. The relationship between transversus abdominis and internal oblique thickness and disease-related characteristics in Parkinson's disease: an ultrasound-based study. *Neurol. Sci. Neurophysiol.* 40, 9–14. <https://doi.org/10.4103/nsn.nsn.97.22>.
- Alcazar, J., Losa-Reyna, J., Rodriguez-Lopez, C., Navarro-Cruz, R., Alfaro-Acha, A., Ara, I., et al., 2019. Effects of concurrent exercise training on muscle dysfunction and systemic oxidative stress in older people with COPD. *Scand. J. Med. Sci. Sports* 29, 1591–1603. <https://doi.org/10.1111/sms.13494>.
- Alegre, L.M., Jiménez, F., Gonzalo-Orden, J.M., Martín-Acero, R., Aguado, X., 2006. Effects of dynamic resistance training on fascicle length and isometric strength. *J. Sports Sci.* 24, 501–508. <https://doi.org/10.1080/02640140500189322>.
- Allen, N.E., Canning, C.G., Sherrington, C., Fung, V.S.C., 2009. Bradykinesia, muscle weakness and reduced muscle power in Parkinson's disease. *Mov. Disord.* 24, 1344–1351. <https://doi.org/10.1002/mds.22609>.
- Allen, N.E., Sherrington, C., Canning, C.G., Fung, V.S.C., 2010. Reduced muscle power is associated with slower walking velocity and falls in people with Parkinson's disease. *Parkinsonism Relat. Disord.* 16, 261–264. <https://doi.org/10.1016/j.parkreldis.2009.12.011>.
- Ando, R., Saito, A., Umemura, Y., Akima, H., 2015. Local architecture of the vastus intermedius is a better predictor of knee extension force than that of the other quadriceps femoris muscle heads. *Clin. Physiol. Funct. Imaging* 35, 376–382. <https://doi.org/10.1111/CPF.12173>.
- Atkinson, R.A., Srinivas-Shankar, U., Roberts, S.A., Connolly, M.J., Adams, J.E., Oldham, J.A., et al., 2010. Effects of testosterone on skeletal muscle architecture in intermediate-frail and frail elderly men. *J. Gerontology: Ser. A* 65A, 1215–1219. <https://doi.org/10.1093/gerona/gdq118>.
- Beitz, J.M., 2014. Parkinson's disease: a review. *Front. Biosci.* 6 S, 65–74. <https://doi.org/10.2741/s415>.
- Bhidayasiri, R., Martinez-Martin, P., 2017. Clinical assessments in Parkinson's Disease: scales and monitoring. *Int. Rev. Neurobiol.* 132, 129–182. <https://doi.org/10.1016/bs.irn.2017.01.001>.
- Bland, D.C., Prosser, L.A., Bellini, L.A., Alter, K.E., Damiano, D.L., 2011. Tibialis anterior architecture, strength, and gait in individuals with cerebral palsy. *Muscle Nerve* 44, 509–517. <https://doi.org/10.1002/MUS.22098>.
- Blazevich, A.J., Sharp, N.C.C., 2006. Understanding muscle architectural adaptation: macro- and micro-level research. *Cells Tissues Organs* 181, 1–10. <https://doi.org/10.1159/000089964>.
- Blazevich, A.J., Gill, N.D., Zhou, S., 2006. Intra- and intermuscular variation in human quadriceps femoris architecture assessed in vivo. *J. Anat.* 209, 289–310. <https://doi.org/10.1111/J.1469-7580.2006.00619.X>.
- Bolsterlee, B., Gandevia, S.C., Herbert, R.D., 2016. Effect of transducer orientation on errors in ultrasound image-based measurements of human medial gastrocnemius muscle fascicle length and pennation. *PLoS One* 11, e0157273. <https://doi.org/10.1371/JOURNAL.PONE.0157273>.
- Calaway, C., Walls, K., Levitt, H., Caplan, J., Mann, B., Martinez, K., et al., 2025. Velocity-based-training frequency impacts changes in muscle morphology, neuromuscular performance, and functional capability in persons with Parkinson's disease. *J. Strength Cond. Res.* 39, 99–106. <https://doi.org/10.1519/JSC.0000000000004951>.
- Caviness, J.N., Smith, B.E., Stevens, J.C., Adler, C.H., Caselli, R.J., Reiners, C.A., et al., 2000. Motor unit changes in sporadic idiopathic Parkinson's disease. *Mov. Disord.* 15, 238–243. [https://doi.org/10.1002/1531-8257\(200003\)15:2<238::aid-mds1006>3.0.co;2-j](https://doi.org/10.1002/1531-8257(200003)15:2<238::aid-mds1006>3.0.co;2-j).
- Charles, J., Kissane, R., Hoehfurner, T., Bates, K.T., 2022. From fibre to function: are we accurately representing muscle architecture and performance? *Biol. Rev.* 97, 1640–1676. <https://doi.org/10.1111/BRV.12856>.
- Chen, N., Hu, H., Li, L., 2015. Ultrasound imaging of muscle-tendon architecture in neurological disease: theoretical basis and clinical applications. *Curr. Med. Imaging* 10, 246–251. <https://doi.org/10.2174/157340561004150121124300>.
- Chen, Y., He, L., Xu, K., Li, J., Guan, B., Tang, H., 2018. Comparison of calf muscle architecture between Asian children with spastic cerebral palsy and typically developing peers. *PLoS One* 13. <https://doi.org/10.1371/JOURNAL.PONE.0190642>.
- Chen, M., Liu, X., Liu, Q., Ding, C., Zhao, P., Zhang, Y., et al., 2023. The value of ultrasound measurement of muscle thickness at different sites and shear wave elastography in Parkinson's disease with sarcopenia: a pilot study. *Front. Neurosci.* 17, 1254859. <https://doi.org/10.3389/fnins.2023.1254859>.
- Coenning, C., Rieg, V., Siebert, T., Wank, V., 2024. Impact of contraction intensity and ankle joint angle on calf muscle fascicle length and pennation angle during isometric and dynamic contractions. *Sci. Rep.* 14, 24929. <https://doi.org/10.1038/s41598-024-75795-2>.
- Cole, M.H., Naughton, G.A., Silburn, P.A., 2017. Neuromuscular impairments are associated with impaired head and trunk stability during gait in Parkinson fallers. *Neurorehabil. Neural Repair* 31, 34–47. <https://doi.org/10.1177/1545968316656057>.
- Connolly, B.S., Lang, A.E., 2014. Pharmacological treatment of Parkinson disease. *JAMA* 311, 1670. <https://doi.org/10.1001/jama.2014.3654>.
- Cronin, N.J., Lichtwark, G., 2013. The use of ultrasound to study muscle-tendon function in human posture and locomotion. *Gait Posture* 37, 305–312. <https://doi.org/10.1016/j.gaitpost.2012.07.024>.

- Dalise, S., Azzollini, V., Chisari, C., 2020. Brain and muscle: how central nervous system disorders can modify the skeletal muscle. *Diagnostics* 10, 1047. <https://doi.org/10.3390/DIAGNOSTICS10121047>.
- David, F.J., Rafferty, M.R., Robichaud, J.A., Prodoehl, J., Kohrt, W.M., Vaillancourt, D. E., et al., 2011. Progressive resistance exercise and Parkinson's disease: a review of potential mechanisms. *Parkinsons Dis.* 2012, 124527. <https://doi.org/10.1155/2012/124527>.
- de Almeida, Sá R., de Sá, Ferreira A., Lemos, T., de Oliveira, L.A.S., 2022. Correlation analysis of lower-limb muscle function with clinical status, balance tests, and quality of life in people with Parkinson Disease. *Top Geriatr. Rehabil.* 38, 56–64. <https://doi.org/10.1097/TGR.0000000000000343>.
- de Boer, M.D., Seynnes, O.R., di Prampero, P.E., Pišot, R., Mekjavić, I.B., Biolo, G., et al., 2008. Effect of 5 weeks horizontal bed rest on human muscle thickness and architecture of weight bearing and non-weight bearing muscles. *Eur. J. Appl. Physiol.* 104, 401–407. <https://doi.org/10.1007/S00421-008-0703-0>.
- DE Oliveira, S.N., GMB, Zapello, Knihš, D.A., Fischer, G., ARP, Moro, 2023. Muscle architecture and maximal strength between male practitioners of functional fitness training and strength training. *Int. J. Exerc. Sci.* 16, 1142. <https://doi.org/10.70252/QKEB4935>.
- Dick, T.J.M., Hug, F., 2023. Advances in imaging for assessing the design and mechanics of skeletal muscle in vivo. *J. Biomech.* 155. <https://doi.org/10.1016/j.jbiomech.2023.111640>.
- Ding, C.W., Wang, C.S., Zhao, P., Chen, M.L., Zhang, Y.C., Liu, C.F., 2024. Shear wave elastography characteristics of the gastrocnemius muscle in postural instability gait disorder vs tremor dominant Parkinson's disease patients. *Acta Neurol. Belg.* 124, 1875–1884. <https://doi.org/10.1007/s13760-024-02547-4>.
- Dirkx, M.F., Bologna, M., 2022. The pathophysiology of Parkinson's disease tremor. *J. Neurol. Sci.* 435, 120196. <https://doi.org/10.1016/j.jns.2022.120196>.
- Donizetti Verri, E., da Silva, G.P., Marianetti Fioco, E., Soares da Silva, N., Valin Fabrin, S.C., Augusto Bueno Zanella, C., et al., 2019. Effects of Parkinson's disease on molar bite force, electromyographic activity and muscle thickness of the masseter, temporal and sternocleidomastoid muscles: a case-control study. *J. Oral Rehabil.* 46, 912–919. <https://doi.org/10.1111/JOOR.12824>.
- Dorsey, E.R., Sherer, T., Okun, M.S., Bloem, B.R., 2018. The emerging evidence of the Parkinson pandemic. *J. Parkinsons Dis.* 8, S3–S8. <https://doi.org/10.3233/JPD-181474>.
- Dunabestia, I., González-Devesa, D., Blanco-Martínez, N., Ayán-Pérez, C., 2025. The effects of stretching in Parkinson's disease: A systematic review of randomized controlled trials. *Parkinsonism Relat. Disord.* 134. <https://doi.org/10.1016/j.parkreldis.2025.107796>.
- Duranti, E., Villa, C., 2024. From brain to muscle: the role of muscle tissue in neurodegenerative disorders. *Biol.-Basel* 13, 719. <https://doi.org/10.3390/BIOLOGY13090719>.
- Ellis, T., Boudreau, J.K., DeAngelis, T.R., Brown, L.E., Cavanaugh, J.T., Earhart, G.M., et al., 2013. Barriers to exercise in people with parkinson disease. *Phys. Ther.* 93, 628–636. <https://doi.org/10.2522/PTJ.20120279>.
- Falvo, M.J., Schilling, B.K., Earhart, G.M., 2008. Parkinson's disease and resistive exercise: rationale, review, and recommendations. *Mov. Disord.* 23, 1–11. <https://doi.org/10.1002/MDS.21690>.
- Finni, T., Ikegaw, S., Lepola, V., Komi, P., 2001. In vivo behavior of vastus lateralis muscle during dynamic performances. *Eur. J. Sport Sci.* 1, 1–13. <https://doi.org/10.1080/17461390100071101>.
- Folland, J.P., Haas, B., Castle, P.C., 2011. Strength and activation of the knee musculature in Parkinson's disease: effect of medication. *NeuroRehabilitation* 29, 405–411. <https://doi.org/10.3233/NRE-2011-0719>.
- Franchi, M.V., Raiteri, B.J., Longo, S., Sinha, S., Narici, M.V., Csapo, R., 2018. Muscle architecture assessment: strengths, shortcomings and new frontiers of in vivo imaging techniques. *Ultrasound Med. Biol.* 44, 2492–2504. <https://doi.org/10.1016/j.ultrasmedbio.2018.07.010>.
- Franchi, M.V., Fitze, D.P., Raiteri, B.J., Hahn, D., Spörri, J., 2020. Ultrasound-derived biceps femoris long head fascicle length: extrapolation pitfalls. *Med. Sci. Sports Exerc.* 52, 233–243. <https://doi.org/10.1249/MSS.0000000000002123>.
- Freilich, R.J., Kirsner, R.L.G., Byrne, E., 1995. Isometric strength and thickness relationships in human quadriceps muscle. *Neuromuscul. Disord.* 5, 415–422. [https://doi.org/10.1016/0960-8966\(94\)00078-N](https://doi.org/10.1016/0960-8966(94)00078-N).
- Fukunaga, T., Kawakami, Y., Kuno, S., Funato, K., Fukashiro, S., 1997. Muscle architecture and function in humans. *J. Biomech.* 30, 457–463. [https://doi.org/10.1016/S0021-9290\(96\)00171-6](https://doi.org/10.1016/S0021-9290(96)00171-6).
- Gamborg, M., Hvid, L.G., Thru, C., Johansson, S., Franzén, E., Dalgas, U., et al., 2023. Muscle strength and power in people with Parkinson disease: a systematic review and meta-analysis. *J. Neurol. Phys. Ther.* 47, 3–15. <https://doi.org/10.1097/NPT.0000000000000421>.
- Gao, F., Zhao, H., Gaebler-Spira, D., Zhang, L.Q., 2011. In vivo evaluations of morphologic changes of gastrocnemius muscle fascicles and achilles tendon in children with cerebral palsy. *Am. J. Phys. Med. Rehabil.* 90, 364–371. <https://doi.org/10.1097/PHM.0B013E318214F699>.
- Gillett, J.G., Barrett, R.S., Lichtwark, G.A., 2013. Reliability and accuracy of an automated tracking algorithm to measure controlled passive and active muscle fascicle length changes from ultrasound. *Comput. Methods Biomech. Biomed. Engin.* 16, 678–687. <https://doi.org/10.1080/10255842.2011.633516>.
- Glendinning, D.S., Enoka, R.M., 1994. Motor unit behavior in Parkinson's disease. *Phys. Ther.* 74, 61–70. <https://doi.org/10.1093/PTJ/74.1.61>.
- Gondin, J., Guette, M., Ballay, Y., Martin, A., 2005. Electromyostimulation training effects on neural drive and muscle architecture. *Med. Sci. Sports Exerc.* 37, 1291–1299. <https://doi.org/10.1249/01.MSS.0000175090.49048.41>.
- Göz, E., Özyürek, S., Aktar, B., Dönmez Çolakoglu, B., Balci, B., 2023. The effects of Pilates training on abdominal muscle thickness and core endurance in patients with Parkinson's disease: a single-blind controlled clinical study. *Turk J. Med. Sci.* 53, 990–1000. <https://doi.org/10.55730/1300-0144.5663>.
- Hannaford, A., Pavey, N., Menon, P., van den Bos, M.A.J., Kiernan, M.C., Simon, N., et al., 2025. Muscle ultrasound aids diagnosis in amyotrophic lateral sclerosis. *Clin. Neurophysiol.* 170, 234–243. <https://doi.org/10.1016/J.CLINPH.2024.11.008>.
- Heckmatt, J.Z., Dubowitz, V., Leeman, S., 1980. Detection of pathological change in dystrophic muscle with B-scan ultrasound imaging. *Lancet* 315, 1389–1390. [https://doi.org/10.1016/S0140-6736\(80\)92656-2](https://doi.org/10.1016/S0140-6736(80)92656-2).
- Heckmatt, J.Z., Leeman, S., Dubowitz, V., 1982. Ultrasound imaging in the diagnosis of muscle disease. *J. Pediatr.* 101, 656–660. [https://doi.org/10.1016/S0022-3476\(82\)80286-2](https://doi.org/10.1016/S0022-3476(82)80286-2).
- Heckmatt, J.Z., Pier, N., Dubowitz, V., 1988. Real-time ultrasound imaging of muscles. *Muscle Nerve* 11, 56–65. <https://doi.org/10.1002/MUS.880110110>.
- Herbert, R.D., Gandevia, S.C., 1995. Changes in pennation with joint angle and muscle torque: in vivo measurements in human brachialis muscle. *J. Physiol.* 484 (Pt 2), 523–532. <https://doi.org/10.1113/JPHYSIOL.1995.SP020683>.
- Inkster, L.M., Eng, J.J., MacIntyre, D.L., Jon, Stoessl A., 2003. Leg muscle strength is reduced in Parkinson's disease and relates to the ability to rise from a chair. *Mov. Disord.* 18, 157–162. <https://doi.org/10.1002/MDS.10299>.
- Jankovic, J., 2008. Parkinson's disease: clinical features and diagnosis. *J. Neurol. Neurosurg. Psychiatry* 79, 368–376. <https://doi.org/10.1136/jnnp.2007.131045>.
- Kalia, L.V., Lang, A.E., 2015. Parkinson's disease. *Lancet* 386, 896–912. [https://doi.org/10.1016/S0140-6736\(14\)61393-3](https://doi.org/10.1016/S0140-6736(14)61393-3).
- Katzberg, H.D., Bril, V., Breiner, A., 2016. Ultrasound in neuromuscular disorders. *J. Clin. Neurophysiol.* 33, 80–85. <https://doi.org/10.1097/WNP.0000000000000234>.
- Kawakami, Y., Abe, T., Kuno, S.Y., Fukunaga, T., 1995. Training-induced changes in muscle architecture and specific tension. *Eur. J. Appl. Physiol. Occup. Physiol.* 72, 37–43. <https://doi.org/10.1007/BF00964112>.
- Kim, J.C., Lee, S.U., Jung, S.H., Lim, J.Y., Kim, D.H., Lee, S.Y., 2019a. Natural aging course of paraspinal muscle and back extensor strength in community-dwelling older adults (sarcopenia of spine, SarcoSpine): a prospective cohort study protocol. *BMJ Open* 9, e032443. <https://doi.org/10.1136/BMJOPEN-2019-032443>.
- Kim, D.H., Lee, S.Y., Park, S.J., Lee, Y.S., 2019b. Relationships between spinal sarcopenia and spinal sagittal balance in older women. *Ann. Geriatr. Med. Res.* 23, 141. <https://doi.org/10.4235/AGMR.19.0030>.
- Kimura, A., Yokozawa, T., Ozaki, H., 2021. Clarifying the biomechanical concept of coordination through comparison with coordination in motor control. *Front. Sports Act Living* 3, 753062. <https://doi.org/10.3389/fspor.2021.753062>.
- Kirmaci, Z.I.K., Firat, T., Özkur, H.A., Neyal, A.M., Neyal, A., Ergun, N., 2022. Muscle architecture and its relationship with lower extremity muscle strength in multiple sclerosis. *Acta Neurol. Belg.* 122, 1521–1528. <https://doi.org/10.1007/S13760-021-01768-1>.
- Klamroth, S., Steib, S., Devan, S., Pfeifer, K., 2016. Effects of exercise therapy on postural instability in Parkinson disease: a meta-analysis. *J. Neurol. Phys. Ther.* 40, 3–14. <https://doi.org/10.1097/NPT.0000000000000117>.
- Koller, W., Kase, S., 1986. Muscle strength testing in Parkinson's disease. *Eur. Neurol.* 25, 130–133. <https://doi.org/10.1159/000115998>.
- Krase, A.A., Terzis, G., Giannaki, C.D., Stasinaki, A.N., Wilkinson, T.J., Smith, A.C., et al., 2022. Seven months of aerobic intradialytic exercise training can prevent muscle loss in haemodialysis patients: an ultrasonography study. *Int. Urol. Nephrol.* 54, 447–456. <https://doi.org/10.1007/S11255-021-02931-6>.
- Kubo, K., Kanehisa, H., Azuma, K., Ishizu, M., Kuno, S.Y., Okada, M., et al., 2003. Muscle architectural characteristics in young and elderly men and women. *Int. J. Sports Med.* 24, 125–130. <https://doi.org/10.1055/S-2003-38204>.
- Kwah, L.K., Pinto, R.Z., Diong, J., Herbert, R.D., 2013. Reliability and validity of ultrasound measurements of muscle fascicle length and pennation in humans: a systematic review. *J. Appl. Physiol.* 114, 761–769. <https://doi.org/10.1152/JAPPLPHYSIOL.01430.2011>.
- Laroche, M., Delisle, M.B., Aziza, R., Lagarrigue, J., Mazieres, B., 1995. Is camptocormia a primary muscular disease? *Spine* 20, 1011–1016. <https://doi.org/10.1097/00007632-199505000-00007>.
- Larsson, L., Degens, H., Li, M., Salviati, L., Il, Lee Y., Thompson, W., et al., 2019. Sarcopenia: aging-related loss of muscle mass and function. *Physiol. Rev.* 99, 427–511. <https://doi.org/10.1152/PHYSREV.00061.2017>.
- Lauzé, M., Daneault, J.F., Duval, C., 2016. The effects of physical activity in Parkinson's disease: a review. *J. Parkinsons Dis.* 6, 685–698. <https://doi.org/10.3233/JPD-160790>.
- Levac, D., Colquhoun, H., O'Brien, K.K., 2010. Scoping studies: advancing the methodology. *Implement. Sci.* 5, 69. <https://doi.org/10.1186/1748-5908-5-69>.
- Lieber, R.L., Blevins, F.T., 1989. Skeletal muscle architecture of the rabbit hindlimb: functional implications of muscle design. *J. Morphol.* 199, 93–101. <https://doi.org/10.1002/JMOR.1051990108>.
- Lieber, R.L., Fridén, J., 2000. Functional and clinical significance of skeletal muscle architecture. *Muscle Nerve* 23, 1647–1666. [https://doi.org/10.1002/1097-4598\(200011\)23:11<1647::AID-MUS1>3.0.CO;2-M](https://doi.org/10.1002/1097-4598(200011)23:11<1647::AID-MUS1>3.0.CO;2-M).
- Lieber, R.L., Fridén, J., 2001. Clinical significance of skeletal muscle architecture. *Clin. Orthop. Relat. Res.* 383, 140–151. <https://doi.org/10.1097/00003086-200102000-00016>.
- Lim, L.S., Lu, C.-H., Lai, Y.-R., Kan, N.-N., Liao, C.-C., Lee, S.Y., 2025. Muscle ultrasonography as a diagnostic tool for assessing sarcopenia in Parkinson's disease. *J. Mov. Disord.* 18. <https://doi.org/10.14802/JMD.25072>.
- Lima, L.O., Scianni, A., Rodrigues-de-Paula, F., 2013. Progressive resistance exercise improves strength and physical performance in people with mild to moderate

- Parkinson's disease: a systematic review. *Aust. J. Phys.* 59, 7–13. [https://doi.org/10.1016/S1836-9553\(13\)70141-3](https://doi.org/10.1016/S1836-9553(13)70141-3).
- Magris, R., Nardello, F., Bombieri, F., Monte, A., Zamparo, P., 2024. Characterization of the vastus lateralis torque-length, and knee extensors torque-velocity and power-velocity relationships in people with Parkinson's disease. *Front. Sports Act Living* 6, 1380864. <https://doi.org/10.3389/fspor.2024.1380864>.
- Manca, A., Deriu, F., 2025. Isokinetics in select neurological disorders. In: Dvir, Z. (3rd Eds.), *Isokinetics: Muscle Testing, Interpretation and Clinical Applications*. Routledge, New York, pp. 345–375. <https://doi.org/10.4324/9781003380719-15>.
- Margraf, N.G., Wrede, A., Deuschl, G., Schulz-Schaeffer, W.J., 2016. Pathophysiological concepts and treatment of camptocormia. *J. Parkinsons Dis.* 6, 485–501. <https://doi.org/10.3233/JPD-160836>.
- Markowitz, J.E., 2011. Probe selection, machine controls, and equipment. In: Carmody, K.A., Moore, C.L., David, F.-K. (Eds.), *Handbook of Critical Care and Emergency Ultrasound*. McGraw-Hill, New York, pp. 25–38. <https://www.semanticscholar.org/paper/Probe-Selection-%2C-Machine-Controls-%2C-and-Equipment-Markowitz/fc5845de682b8f0e7a173c00abc533e0871f98a>.
- Martignon, C., Ruzzante, F., Giurini, G., Laginestra, F.G., Pedrinolla, A., Di Vico, I.A., et al., 2021. The key role of physical activity against the neuromuscular deterioration in patients with Parkinson's disease. *Acta Physiol.* 231, e13630. <https://doi.org/10.1111/apha.13630>.
- Masaki, M., Kasahara, M., Takeuchi, M., Minakawa, K., Inagaki, Y., Ogawa, Y., et al., 2022. Comparison of the mass and amount of intramuscular non-contractile tissue of the trunk and lower extremity muscles between patients with Parkinson's disease and community-dwelling older adults. *Neurol. Sci.* 43, 3629–3640. <https://doi.org/10.1007/S10072-021-05828-5>.
- Masaki, M., Takeuchi, M., Kasahara, M., Minakawa, K., Inagaki, Y., Ogawa, Y., et al., 2023a. Association of activities of daily living, mobility and balance ability, and symptoms of Parkinson's disease with the masses and amounts of intramuscular non-contractile tissue of the trunk and lower extremity muscles in patients with Parkinson's disease. *J. Med. Ultrason.* (50), 551–560. <https://doi.org/10.1007/S10396-023-01356-1>.
- Masaki, M., Kasahara, M., Inagaki, Y., Yokota, M., Takeuchi, M., 2023b. Association of sagittal spinal alignment in the standing position with the masses and amounts of intramuscular non-contractile tissue of the trunk and lower extremity muscles in patients with Parkinson's disease. *Clin. Biomech.* 101. <https://doi.org/10.1016/j.clinbiomech.2022.105868>.
- Mazzoni, P., Shabbott, B., Cortés, J.C., 2012. Motor control abnormalities in Parkinson's disease. *Cold Spring Harb. Perspect. Med.* 2, a009282. <https://doi.org/10.1101/cshperspect.a009282>.
- McCarty, E.B., Chao, T.N., 2021. Dysphagia and swallowing disorders. *Med. Clin. North Am.* 105, 939–954. <https://doi.org/10.1016/j.mcna.2021.05.013>.
- McGregor, M.M., Nelson, A.B., 2019. Circuit mechanisms of Parkinson's disease. *Neuron* 101, 1042–1056. <https://doi.org/10.1016/j.neuron.2019.03.004>.
- Monte, A., Magris, R., Nardello, F., Bombieri, F., Zamparo, P., 2023. Muscle shape changes in Parkinson's disease impair function during rapid contractions. *Acta Physiol.* 238, e13957. <https://doi.org/10.1111/apha.13957>.
- Murphy, K.T., Lynch, G.S., 2023. Impaired skeletal muscle health in Parkinsonian syndromes: clinical implications, mechanisms and potential treatments. *J. Cachexia. Sarcopenia Muscle* 14, 1987–2002. <https://doi.org/10.1002/JCSM.13312>.
- Nicoletti, V., Palermo, G., Del Prete, E., Mancuso, M., Ceravolo, R., 2021. Understanding the multiple role of mitochondria in Parkinson's disease and related disorders: lesson from genetics and protein–interaction network. *Front. Cell Dev. Biol.* 9, 636506. <https://doi.org/10.3389/fcell.2021.636506>.
- Nocera, J.R., Buckley, T., Waddell, D., Okun, M.S., Hass, C.J., 2010. Knee extensor strength, dynamic stability, and functional ambulation: are they related in Parkinson's disease? *Arch. Phys. Med. Rehabil.* 91, 589. <https://doi.org/10.1016/J.APMR.2009.11.026>.
- Nunes, J.P., Blazevich, A.J., Schoenfeld, B.J., Kassiano, W., Costa, B.D.V., Ribeiro, A.S., et al., 2024. Determining changes in muscle size and architecture after exercise training: one site does not fit all. *J. Strength Cond. Res.* 38, 787–790. <https://doi.org/10.1519/JSC.0000000000004722>.
- Oh, E.H., Seo, J.S., Kang, H.J., 2016. Assessment of oropharyngeal dysphagia in patients with Parkinson disease: use of ultrasonography. *Ann. Rehabil. Med.* 40, 190. <https://doi.org/10.5535/ARM.2016.40.2.190>.
- Paolucci, T., Sbardella, S., La Russa, C., Agostini, F., Mangone, M., Tramontana, L., et al., 2020. Evidence of rehabilitative impact of progressive resistance training (prt) programs in parkinson disease: an umbrella review. *Parkinsons Dis.* 2020. <https://doi.org/10.1155/2020/9748091>.
- Park, J.H., Stelmach, G.E., 2006. Force development during target-directed isometric force production in Parkinson's disease. *Neurosci. Lett.* 412, 173. <https://doi.org/10.1016/J.NEULET.2006.11.009>.
- Paul, S.S., Sherrington, C., Fung, V.S.C., Canning, C.G., 2013. Motor and cognitive impairments in Parkinson disease. *Neurorehabil. Neural Repair* 27, 63–71. <https://doi.org/10.1177/1545968312446754>.
- Pillen, S., van Alfen, N., 2011. Skeletal muscle ultrasound. *Neurol. Res.* 33, 1016–1024. <https://doi.org/10.1179/1743132811Y.0000000010>.
- Ponsoni, A., Sardeli, A.V., Costa, F.P., Mourão, L.F., 2023. Prevalence of sarcopenia in Parkinson's disease: a systematic review and meta-analysis. *Geriatr. Nurs. (Minneapolis)* 49, 44–49. <https://doi.org/10.1016/j.gerinurse.2022.11.006>.
- Potier, T.G., Alexander, C.M., Seynnes, O.R., 2009. Effects of eccentric strength training on biceps femoris muscle architecture and knee joint range of movement. *Eur. J. Appl. Physiol.* 105, 939–944. <https://doi.org/10.1007/S00421-008-0980-7>.
- Rawson, K.S., McNeely, M.E., Duncan, R.P., Pickett, K.A., Perlmutter, J.S., Earhart, G.M., 2019. Exercise and Parkinson disease: comparing tango, treadmill and stretching. *J. Neurol. Phys. Ther.* 43, 26. <https://doi.org/10.1097/NPT.0000000000000245>.
- Ritsche, P., Franchi, M.V., Faude, O., Finni, T., Seynnes, O., Cronin, N.J., 2024. Fully automated analysis of muscle architecture from b-mode ultrasound images with DL-Track US. *Ultrasound Med. Biol.* 50, 258–267. <https://doi.org/10.1016/j.ultrasmedbio.2023.10.011>.
- Roth, S.M., 2012. Genetic aspects of skeletal muscle strength and mass with relevance to sarcopenia. *Bonekey Rep.* 1, 58. <https://doi.org/10.1038/BONEKEY.2012.58>.
- Royall, N.A., Farrin, E., Bahner, D.P., Stawicki, S.P.A., 2011. Ultrasound-assisted musculoskeletal procedures: a practical overview of current literature. *World J. Orthop.* 2, 57. <https://doi.org/10.5312/wjo.v2.i7.57>.
- Sarto, F., Spörri, J., Fitze, D.P., Quinlan, J.I., Narici, M.V., Franchi, M.V., 2021. Implementing ultrasound imaging for the assessment of muscle and tendon properties in elite sports: practical aspects, methodological considerations and future directions. *Sports Med.* 51, 1151–1170. <https://doi.org/10.1007/s40279-021-01436-7>.
- Seo, M.H., Yeo, S., 2021. Triadin decrease impairs the expression of E-C coupling related proteins in muscles of MPTP-induced Parkinson's disease mice. *Front. Neurosci.* 15, 649688. <https://doi.org/10.3389/fnins.2021.649688>.
- Sharma, V.D., Patel, M., Miocinovic, S., 2020. Surgical treatment of Parkinson's disease: devices and lesion approaches. *Neurotherapeutics* 17, 1525–1538. <https://doi.org/10.1007/s13311-020-00939-x>.
- Shortland, A.P., Harris, C.A., Gough, M., Robinson, R.O., 2002. Architecture of the medial gastrocnemius in children with spastic diplegia. *Dev. Med. Child Neurol.* 44, 158. <https://doi.org/10.1111/j.1469-8749.2002.tb00779.x>.
- Şirin Ahışa, B., Kesiktaş, N., Paker, N., 2025. Ultrasonographic assessment of dysphagia in Parkinson's disease: a controlled study. *Neurologi. Sci.* 46, 4405–4414. <https://doi.org/10.1007/S10072-025-08280-X>.
- Skinner, J.W., Christou, E.A., Hass, C.J., 2019. Lower extremity muscle strength and force variability in persons with Parkinson disease. *J. Neurol. Phys. Ther.* 43, 56–62. <https://doi.org/10.1097/NPT.0000000000000244>.
- Smart, R.R., Richardson, C.M., Wile, D.J., Dalton, B.H., Jakobi, J.M., 2020. Importance of maximal strength and muscle-tendon mechanics for improving force steadiness in persons with Parkinson's disease. *Brain Sci.* 10, 471. <https://doi.org/10.3390/BRAINSCI10080471>.
- Son, J., Ward, S.R., Lieber, R.L., 2024. Scaling relationships between human leg muscle architectural properties and body size. *J. Exp. Biol.* 227. <https://doi.org/10.1242/JEB.246567>.
- Spuler, S., Krug, H., Klein, C., Medialdea, I.C., Jakob, W., Ebersbach, G., et al., 2010. Myopathy causing camptocormia in idiopathic Parkinson's disease: a multidisciplinary approach. *Mov. Disord.* 25, 552–559. <https://doi.org/10.1002/mds.22913>.
- Srivanthapoom, P., Hallett, M., 2016. Camptocormia in Parkinson's disease: definition, epidemiology, pathogenesis and treatment modalities. *J. Neurol. Neurosurg. Psychiatry* 87, 75–85. <https://doi.org/10.1136/jnnp-2014-310049>.
- Stelmach, G.E., Worringham, C.J., 1988. The preparation and production of isometric force in Parkinson's disease. *Neuropsychologia* 26, 93–103. [https://doi.org/10.1016/0028-3932\(88\)90033-4](https://doi.org/10.1016/0028-3932(88)90033-4).
- Stelmach, G.E., Teasdale, N., Phillips, J., Worringham, C.J., 1989. Force production characteristics in Parkinson's disease. *Exp. Brain Res.* 76, 165–172. <https://doi.org/10.1007/BF00253633>.
- Stevens-Lapsley, J., Kluger, B.M., Schenkman, M., 2012. Quadriceps muscle weakness, activation deficits, and fatigue with parkinson disease. *Neurorehabil. Neural Repair* 26, 533–541. <https://doi.org/10.1177/1545968311425925>.
- Strasser, E.M., Draskovits, T., Praschak, M., Quittan, M., Graf, A., 2013. Association between ultrasound measurements of muscle thickness, pennation angle, echogenicity and skeletal muscle strength in the elderly. *Age (Omaha)* 35, 2377. <https://doi.org/10.1007/S11357-013-9517-Z>.
- Suttrup, I., Warnecke, T., 2016. Dysphagia in Parkinson's disease. *Dysphagia* 31, 24–32. <https://doi.org/10.1007/S00455-015-9671-9>.
- Tedeschi, R., 2023. Automated mechanical peripheral stimulation for gait rehabilitation in Parkinson's disease: a comprehensive review. *Clin. Park Relat. Disord.* 9. <https://doi.org/10.1016/j.prdoa.2023.100219>.
- Tedeschi, R., Donati, D., Giorgi, F., 2024. The role of AMPS in Parkinson's disease management: scoping review and meta-analysis. *Bioengineering* 12, 21. <https://doi.org/10.3390/BIOENGINEERING12010021>.
- Timmins, R.G., Shield, A.J., Williams, M.D., Lorenzen, C., Opar, D.A., 2016. Architectural adaptations of muscle to training and injury: a narrative review outlining the contributions by fascicle length, pennation angle and muscle thickness. *Br. J. Sports Med.* 50, 1467–1472. <https://doi.org/10.1136/bjsports-2015-094881>.
- Torres, F., De Oliveira, M., Gomes De Oliveira, C., Farinatti, P., 2017. Pennation angle of vastus lateralis during isometric contractions performed at two knee angles. *Fisioterapia Em Movimento* 30, 75–83. <https://doi.org/10.1590/1980-5918.030.S01.A007>.
- Tricco, A.C., Lillie, E., Zarin, W., O'Brien, K.K., Colquhoun, H., Levac, D., et al., 2018. PRISMA extension for scoping reviews (PRISMA-ScR): checklist and explanation. *Ann. Intern. Med.* 169, 467–473. <https://doi.org/10.7326/M18-0850>.
- Umay, E., Ozturk, E., Gurcay, E., Delibas, O., Celikel, F., 2019. Swallowing in Parkinson's disease: how is it affected? *Clin. Neurol. Neurosurg.* 177, 37–41. <https://doi.org/10.1016/j.clineuro.2018.12.015>.
- Van, Nimwegen M., Speelman, A.D., Hofman-Van Rossum, E.J.M., Overeem, S., Deeg, D. J.H., Borm, G.F., et al., 2011. Physical inactivity in Parkinson's disease. *J. Neurol.* 258, 2214–2221. <https://doi.org/10.7326/M11-6097-7>.
- Vaswani, P.A., Tropea, T.F., Dahodwala, N., 2020. Overcoming barriers to Parkinson disease trial participation: increasing diversity and novel designs for recruitment and retention. *Neurotherapeutics* 17, 1724–1735. <https://doi.org/10.1007/s13311-020-00960-0>.

- Ward, S.R., Eng, C.M., Smallwood, L.H., Lieber, R.L., 2009. Are current measurements of lower extremity muscle architecture accurate? *Clin. Orthop. Relat. Res.* 467, 1074–1082. <https://doi.org/10.1007/S11999-008-0594-8>.
- Westerberg, E., Molin, C.J., Nees, S.S., Widenfalk, J., Punga, A.R., 2018. The impact of physical exercise on neuromuscular function in Myasthenia gravis patients: a single-subject design study. *Medicine* 97. <https://doi.org/10.1097/MD.00000000000011510>.
- Yilmaz, R., Wolke, R., Puls, N., Sorgun, M.H., Deuschl, G., Berg, D., et al., 2023. Characterizing camptocormia in Parkinson's disease using muscle ultrasonography. *J. Parkinsons Dis.* 13, 819. <https://doi.org/10.3233/JPD-230037>.
- Yin, L., Du, L., Li, Y., Xiao, Y., Zhang, S., Ma, H., et al., 2021. Quantitative evaluation of gastrocnemius medialis stiffness during passive stretching using shear wave elastography in patients with Parkinson's disease: a prospective preliminary study. *Korean J. Radiol.* 22, 1841–1849. <https://doi.org/10.3348/kjr.2020.1338>.