REVIEW ARTICLE

Intersection of Sarcopenia and cognitive decline: An umbrella review using HOMA-IR and NIRS

Mukesh Kumar G[1],Daniel Raj D[2], Rishi Panday[3], Abdul Samad Hashmi[4], Lucky Patidar[5]

[1]*M.Sc. Nursing, Department of Psychiatry, College of Nursing, AIIMS Bhopal*
[2]*M.Sc. MLT Microbiology, Department of Microbiology, AIIMS Bhopal*
[3]*Psychiatric Social Worker, Department of Psychiatry, AIIMS Bhopal*
[4]*Health Educator (Social Psychologist), Department of Psychiatry, AIIMS Bhopal*
[5]*MD Psychiatry, Department of Psychiatry, AIIMS Bhopal.*

ABSTRACT

Sarcopenia and cognitive decline frequently co-occur in ageing populations and share overlapping biological pathways, including physical inactivity, insulin resistance, mitochondrial dysfunction, inflammation, and altered muscle-brain signalling. Skeletal muscles, as a key insulin-sensitive and endocrine organ, may play a central role in linking metabolic dysfunction to neurocognitive impairment. However, the mechanistic integration of systemic metabolic markers and muscle-specific physiological measures remains insufficiently synthesized. **Objective:** To synthesize existing evidence on the interrelationships between sarcopenia, insulin resistance and cognitive decline with a specific focus on the roles of the homeostatic model assessment of insulin resistance (HOMA-IR) and near-infrared spectroscopy (NIRS) as complementary metabolic and physiological assessment tools. **Methods:** A comprehensive umbrella review was conducted, including narrative reviews, systematic reviews, scoping review, methodological reviews and observational studies published between January 2000 and January 2026. Studies examining associations between physical inactivity or sarcopenia, insulin resistance, muscle or cerebral oxygenation and cognitive outcomes were included. Findings were synthesised qualitatively due to heterogeneity in study design and outcome measures. **Results:** Population-based studies consistently demonstrated strong associations between sarcopenia and elevated HOMA-IR, independent of obesity. Mechanistic and methodological reviews highlighted impaired muscle oxidative capacity and mitochondrial dysfunction as early features of inactivity and metabolic decline, reliably assessed using NIRS. Preclinical and integrative human evidence supported a muscle-brain axis mediated by insulin resistance, myokine dysregulation and neurotropic signalling, contributing to cognitive impairment. **Conclusion:** Sarcopenia represents a metabolically active contributor to cognitive decline rather than a passive consequence of ageing. Integrating HOMA-IR and NIRS provides a multidimensional framework for identifying high-risk individuals, monitoring intervention responses and advancing understanding of the muscle-brain axis across the lifespan.

Keywords: Brain health, Dementia, Mitochondrial dysfunction, Cognitive Dysfunction, Insulin Resistance.

ISSN: **2582-6891**

INTRODUCTION

The global COVID-19 pandemic profoundly altered Sarcopenia, characterised by progressive loss of skeletal muscle mass, strength, and function, is a major contributor to frailty, metabolic disease, and reduced quality of life in ageing populations.(Cruz-Jentoft et al., 2019) It has been recognised as a significant public health concern by the World Health Organization due to its association with disability, falls, and increased mortality among older adults.(World Report on Ageing and Health, n.d.) In parallel, cognitive decline and dementia represent growing global public health challenges, with the global burden of dementia rising rapidly as populations age worldwide.(Dementia, n.d.) Increasing evidence indicates that sarcopenia and scognitive impairment frequently co-exist and share

Corresponding Author- Dr. Rishi Panday, Psychiatric Social Worker, Department of Psychiatry, AIIMS Bhopal

Email id - rishiraj.lu@gmail.com

How to cite: Kumar M., Raj D., Panday R., Hashmi AS., Patidar L. (2026). Intersection of Sarcopenia and cognitive decline: An umbrella review using HOMA-IR and NIRS Pandemic. Journal of Psychosocial Wellbeing 7(1):87-95

DOI: https://doi.org/10.55242/JPSW.2026.7111

Received: 14.03.2026 **Revised:** 08.04.2026
Accepted: 11.05.2026 **Published:** 14.06.2026

common biological pathways, particularly those related to physical inactivity, insulin resistance, chronic inflammation, and mitochondrial dysfunction.(Barstow, 2019; Grassi & Quaresima, 2016; Li et al., 2024; Srikanthan et al., 2010)

Skeletal muscle is the largest insulin-sensitive organ in the human body, accounting for the majority of postprandial glucose uptake. Impairment in muscle mass and function, therefore, plays a central role in the development of insulin resistance and metabolic dysregulation.(Kullmann et al., 2015) Beyond its metabolic role, skeletal muscle functions as an endocrine organ by secreting myokines that exert autocrine, paracrine, and endocrine effects on distant organs, including the brain. These myokines are involved in neurogenesis, synaptic plasticity, and neuroinflammation, suggesting a mechanistic link between muscle health and cognitive function. Consequently, loss of muscle mass and function due to inactivity or sarcopenia may have systemic consequences that extend to brain health.(Adami & Rossiter, 2018; Sendra-Pérez et al., 2025; Tuesta et al., 2022)

The interaction between skeletal muscle, insulin resistance, and cognition has been increasingly explored using systemic metabolic indices, such as the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR), which provides an estimate of peripheral insulin resistance, and muscle-focused physiological tools, including near-infrared spectroscopy (NIRS), which enables non-invasive assessment of muscle oxygenation and mitochondrial function. (Homeostasis Model Assessment of Insulin Resistance in a General Adult Population in Korea: Additive Association of Sarcopenia and Obesity with Insulin Resistance - Kwon - 2017 - Clinical Endocrinology - Wiley Online Library, n.d.) Integrating these approaches provides a multidimensional perspective on the pathophysiology linking inactive skeletal muscle, metabolic dysfunction, and cognitive decline.(Liu et al., 2023; Nagase & Tohda, 2021; Park et al., 2022; Sui et al., 2020)

METHODS

Study Design

This study was conducted as an umbrella review to synthesise and interpret existing evidence on the interrelationships between muscle inactivity, sarcopenia, insulin resistance, and cognitive decline, with a particular focus on metabolic and neurophysiological markers such as the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) and near-infrared (NIR) spectroscopy. An umbrella review methodology was chosen as the available literature is heterogeneous with respect to study design, population characteristics, assessment tools, and outcome measures, making formal quantitative synthesis inappropriate. This approach enables a comprehensive, contextually grounded, and theory-driven integration of findings across disciplines, including geriatrics, endocrinology, neuroscience, and rehabilitation science.

Conceptual Framework and Research Questions

The review was guided by a structured framework integrating physical inactivity, muscle mass and function, metabolic dysregulation, and cognitive outcomes. A modified PICO framework was applied during the planning phase to refine the research questions:[I] Population (P): Adults and older adults, including healthy individuals and those with metabolic, neuromuscular, or cognitive impairments.[II] Exposure (I): Physical inactivity, muscle disuse, or sarcopenia.[III] Comparison (C): Physically active individuals or those without sarcopenia or insulin resistance (where applicable)[IV] Outcomes (O): Insulin resistance (measured using HOMA-IR), cognitive decline, brain function, and cerebral or muscular oxygenation assessed by NIR spectroscopy.

The primary research questions were:[I] How do physical inactivity and sarcopenia contribute to insulin resistance and cognitive decline?[II] What evidence supports the use of HOMA-IR as a metabolic marker linking muscle dysfunction and brain health?[III] How has near-infrared spectroscopy been used to assess muscle and cerebral oxygenation in relation to metabolic and cognitive outcomes?

Search Strategy

A comprehensive literature search was conducted across multiple electronic databases, including PubMed, ONOS, Google Scholar, and freely available online articles. The search covered studies published from January 2000 to January 2026, without restrictions on geographical location. Only studies published in English were considered. Search terms were developed from the research questions and included combinations of keywords and controlled vocabulary terms such as

physical inactivity, muscle disuse, sarcopenia, insulin resistance, HOMA-IR, cognitive decline, dementia, brain metabolism, and near-infrared spectroscopy. Search strategies were adapted to each database's indexing system. In addition, reference lists of relevant articles were manually screened to identify further eligible studies.

Eligibility Criteria

Studies were selected using predefined inclusion and exclusion criteria to ensure relevance and consistency.

Inclusion criteria

comprised scoping review, systematic review, narrative review, methodological review, critical narrative review, mechanistic narrative review, cross-sectional studies, and longitudinal observational studies that examined at least one of the following relationships: (1) physical inactivity or sarcopenia and insulin resistance, (2) insulin resistance and cognitive decline, or (3) muscle or cerebral oxygenation assessed using near-infrared spectroscopy in relation to metabolic or cognitive outcomes.

Exclusion criteria

included editorials, letters to the editor, case reports and studies lacking relevant metabolic or cognitive outcome measures.

Study Selection and Data Extraction

Study selection was conducted in a multistep process involving title screening, abstract review, and full-text assessment. Relevant data were extracted and organised using reference management software to remove duplicates and ensure systematic documentation. Extracted information included publication details, study design, sample characteristics, assessment methods for sarcopenia, insulin resistance and cognition, use of HOMA-IR and NIR spectroscopy, and key findings.

Data Synthesis

Given the heterogeneity of the included studies, findings were synthesised qualitatively. Studies were grouped thematically according to (1) physical inactivity and sarcopenia, (2) insulin resistance and metabolic dysfunction, (3) cognitive decline and brain health, and (4) applications of HOMA-IR and near-infrared spectroscopy. Patterns, consistencies, and gaps in the literature were identified and discussed narratively. Formal risk-of-bias assessment tools were not applied, consistent with umbrella review methodology.

RESULTS

This umbrella review synthesised evidence from twelve eligible studies published between 2010 and 2025, encompassing a broad range of study designs, populations and methodological approaches (Table 1). The included literature comprised three large population-based cross-sectional studies with medication analysis, one systematic review, five narrative reviews, one methodological review, one animal experiment, and one scoping review. Collectively, these studies integrated evidence from nationally representative human cohorts, clinical adult populations, and animal models, providing a comprehensive perspective on the interrelationships among sarcopenia, insulin resistance, muscle physiology, and cognitive outcomes. Across population-based studies, sample sizes ranged from 5,574 to 15,779 adults, primarily derived from the National Health and Nutrition Examination Survey (NHANES), enabling robust examination of associations among sarcopenia, obesity, and insulin resistance using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR).(Kuo et al., 2007) Mechanistic and methodological reviews predominantly focused on muscle oxidative capacity and oxygen utilization, assessed using near-infrared spectroscopy (NIRS), while preclinical studies explored muscle inactivity, insulin resistance and myokine-mediated muscle-brain signalling in relation to cognitive decline.

Given the heterogeneity in study designs, outcome measures and assessment tools, a qualitative synthesis of results was undertaken. Evidence was organised thematically into major themes such as Sarcopenia and insulin resistance, Muscle Oxidative Capacity, Physical Inactivity, and Metabolic Decline, Near-Infrared Spectroscopy as a Tool to Assess Inactive and Sarcopenic Muscle, and The Muscle–Brain Axis: Mechanistic Evidence Linking Sarcopenia to Cognition. This approach enabled integration of epidemiological, physiological and mechanistic findings to elucidate shared pathways underpinning the muscle-metabolism-brain axis.

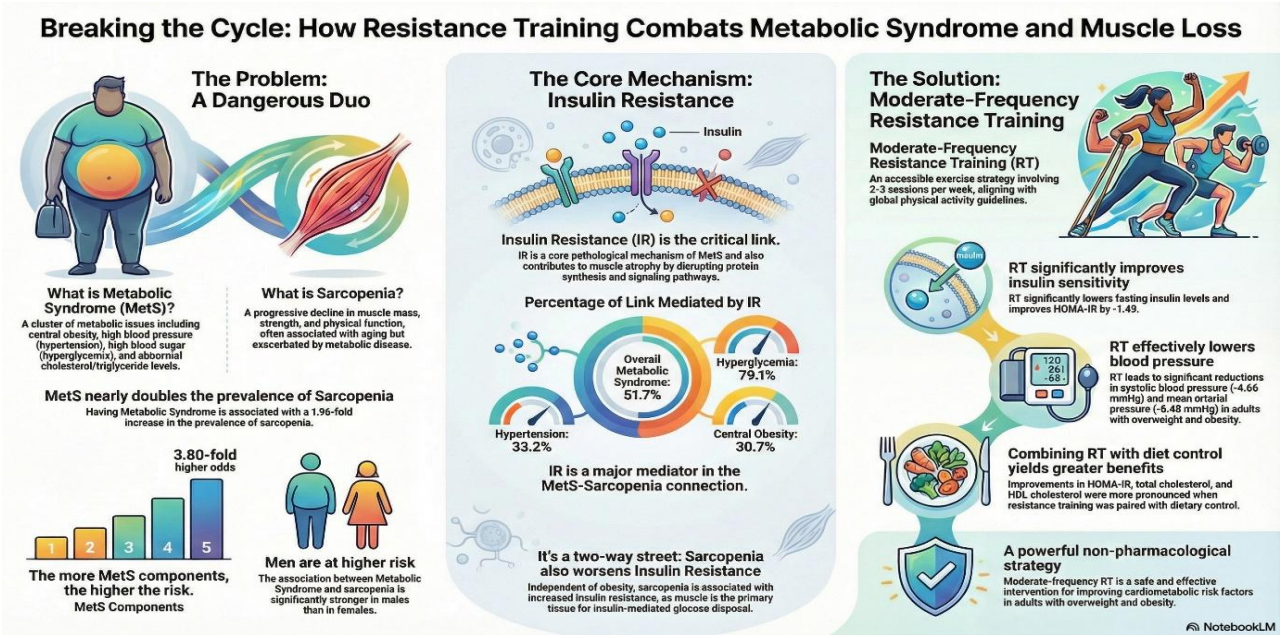
Table 1: Characteristics of included studies						
Author (Year)	**Study Type**	**Population / Model**	**Muscle/ Inactivity Variable**	**Insulin Resistance (HOMA-IR / Metabolic Marker)**	**NIRS / Muscle oxygenation**	**Cognitive / Brain Outcome**
Zhang et al., 2024	Cross-sectional (NHANES)	5,574 adults	Sarcopenic obesity	HOMA-IR ↑; strong association with SO	Not used	Not assessed
Srikanthan et al., 2010	Cross-sectional (NHANES III)	14,528 adults	Sarcopenia ± obesity	HOMA-IR ↑ in sarcopenic individuals	Not used	Not assessed
Li et al., 2024	Cross-sectional + mediation analysis	15,779 adults	Sarcopenia	HOMA-IR mediates MetS-sarcopenia link (30–79%)	Not used	Not assessed
Grassi & Quaresima, 2016	Narrative review	Humans & patients	Muscle oxidative metabolism	Indirect (mitochondrial dysfunction)	Yes – NIRS (O_2 extraction, oxidative capacity)	Exercise tolerance (indirect brain relevance)
Adami & Rossiter, 2017	Methodological review	Humans	Muscle oxidative capacity	Metabolic dysfunction context	Yes – NIRS ($m\dot{V}O_2$ recovery kinetics)	Not assessed
Barstow, 2019	Review (CORP)	Humans	Muscle oxygen delivery/utilisation	Insulin resistance is discussed mechanistically	Yes – NIRS	Cerebral implications discussed
Sendra-Pérez et al., 2025	Critical narrative review	Athletes & clinical	Muscle oxygenation	Not directly measured	Yes – NIRS (SmO_2, O_2Hb, HHb)	Not assessed
Tuesta et al., 2022	Systematic review	Clinical adults	Exercise-induced muscle adaptation	Metabolic disease populations	Yes – NIRS	Functional outcomes
Park et al., 2022	Animal experiment	Mice (physical inactivity model)	Muscle inactivity	Indirect (irisin ↓)	Not used	Cognitive decline, ↓BDNF
Liu et al., 2023	Scoping review (pre-clinical)	Rodents	Muscle atrophy, insulin resistance	Muscle insulin resistance model	Not used	Memory decline, ↓neurogenesis
Sui et al., 2021	Narrative review	Human studies	Sarcopenia components	IR discussed mechanistically	Not used	Cognitive impairment, dementia
Oudbier et al., 2022	Mechanistic narrative review	Humans & animals	Low muscle mass	Insulin metabolism, IR	Mitochondrial oxidative capacity	Cognitive impairment, dementia

FOOTNOTE

NHANES – National Health and Nutrition Examination Survey; HOMA-IR – Homeostatic Model Assessment of Insulin Resistance; IR – Insulin Resistance; MetS – Metabolic Syndrome; SO – Sarcopenic Obesity; NIRS – Near-Infrared Spectroscopy; SmO_2 – Muscle Oxygen Saturation; O_2Hb – Oxygenated Haemoglobin; HHb – Deoxygenated Haemoglobin; $m\dot{V}O_2$ – Muscle Oxygen Consumption; BDNF – Brain-Derived Neurotrophic Factor; CORP – Control of Oxygenation and Respiration in Peripheral tissues.

Sarcopenia and Insulin Resistance: Evidence from Population Studies

Robust epidemiological evidence demonstrates that sarcopenia is strongly associated with insulin resistance, independent of obesity. Using data from the National Health and Nutrition Examination Survey (NHANES III), Srikanthan, Hevener, and Karlamang la (2010) showed that sarcopenia was associated with significantly higher HOMA-IR values in both obese and non-obese adults.(Srikanthan et al., 2010) Import antly, the association was strongest in individuals younger than 60 years, suggesting that low muscle mass may be an early predictor of metabolic dysfuncti on rather than a consequence of ageing alone. (NotebookLM, n.d.)



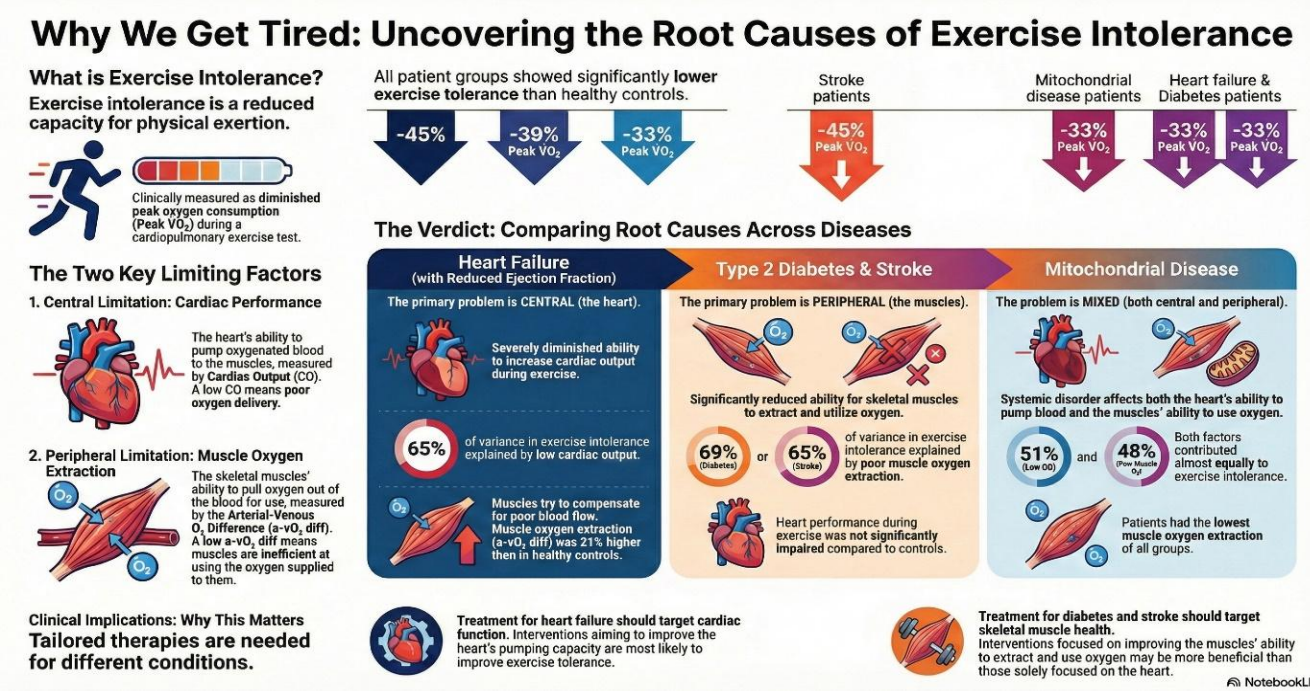
Extending these findings, Li, Ji, Liu, and Wu (2024) analysed NHANES data from 1999–2006 and 2011–2018 and reported that metabolic syndrome nearly doubled the prevalence of sarcopenia.(Li et al., 2024) Their mediation analysis revealed that HOMA-IR accounted for 30–80% of the association between central obesity, hypertension, hyperglycemia, and sarcopenia, indicating that insulin resistance is a central mechanistic pathway linking metabolic syndrome to muscle loss. Collectively, these studies establish insulin resistance, quantified by HOMA-IR, as a key systemic marker of sarcopenic risk and highlight the bidirectional relationship between metabolic dysfunction and skeletal muscle deterioration.

Muscle Oxidative Capacity, Physical Inactivity, and Metabolic Decline

Beyond muscle mass, skeletal muscle oxidative capacity is a critical determinant of metabolic health and physical function.(Pedersen, 2019) Loss of oxidative capacity is consistently observed in association with ageing, physical inactivity, and chronic disease.(NotebookLM, n.d.)

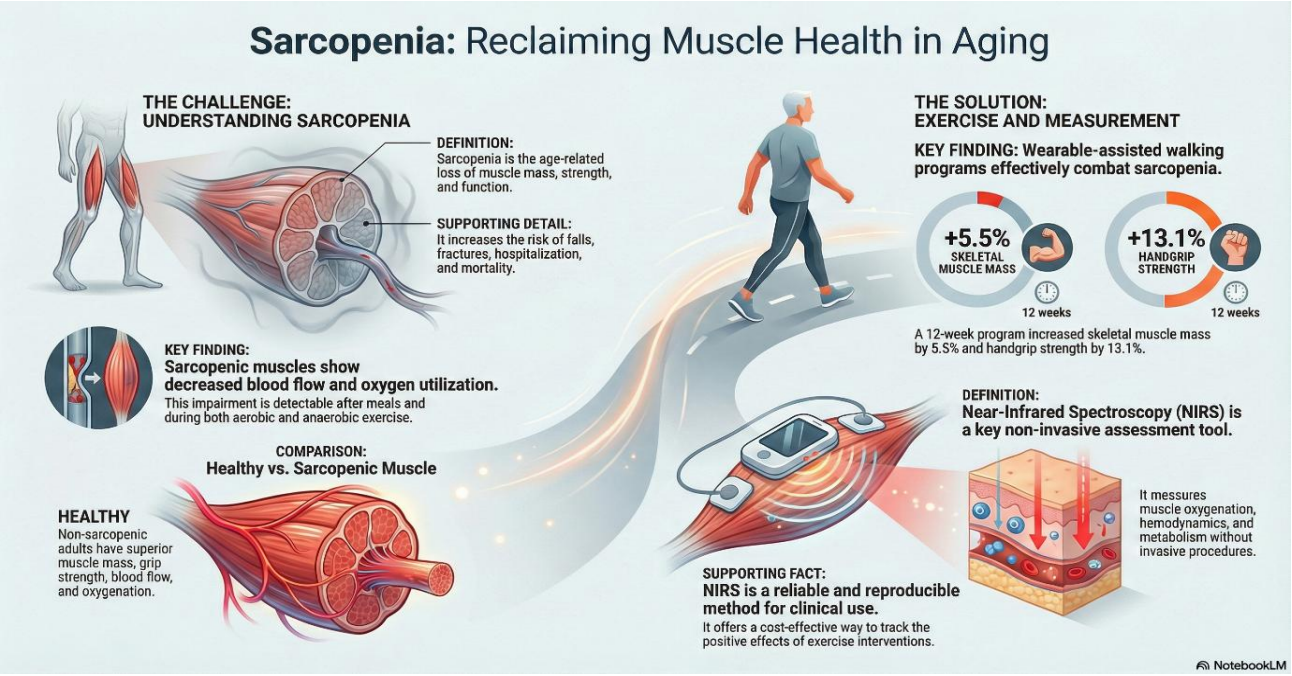
Grassi and Quaresima (2016) described skeletal muscle oxidative metabolism as highly plastic and sensitive to physical activity status, noting that declines in oxidative capacity often precede overt functional impairment.(Grassi & Quaresima, 2016) Reduced oxidative capacity limits exercise tolerance, reinforcing sedentary behaviour and exacerbating insulin resistance and metabolic inflexibility

In a comprehensive methodological review, Barstow (2019) outlined the physiological basis for how near-infrared spectroscopy captures the balance between oxygen delivery and utilisation in skeletal muscle.(Barstow, 2019) He emphasised that impaired muscle oxygen extraction and utilisation reflect mitochondrial dysfunction and reduced metabolic flexibility—hallmarks of inactive and sarcopenic muscle.



Near-Infrared Spectroscopy as a Tool to Assess Inactive and Sarcopenic Muscle

Near-infrared spectroscopy (NIRS) enables non-invasive, real-time assessment of muscle oxygenation, perfusion, and oxidative metabolism by tracking changes in oxyhaemoglobin, deoxyhaemoglobin, and tissue oxygen saturation. Unlike systemic metabolic markers, NIRS provides localised functional information about skeletal muscle health.(NotebookLM, n.d.)



Adami and Rossiter (2018) demonstrated that NIRS-derived post-exercise recovery kinetics of muscle oxygen consumption closely reflect intrinsic mitochondrial oxidative capacity and correlate with gold-standard techniques such as ^{31}P magnetic resonance spectroscopy. (Adami & Rossiter, 2018). They highlighted the feasibility and tolerability of NIRS in frail and clinically compromised populations, making it particularly suitable for sarcopenic individuals

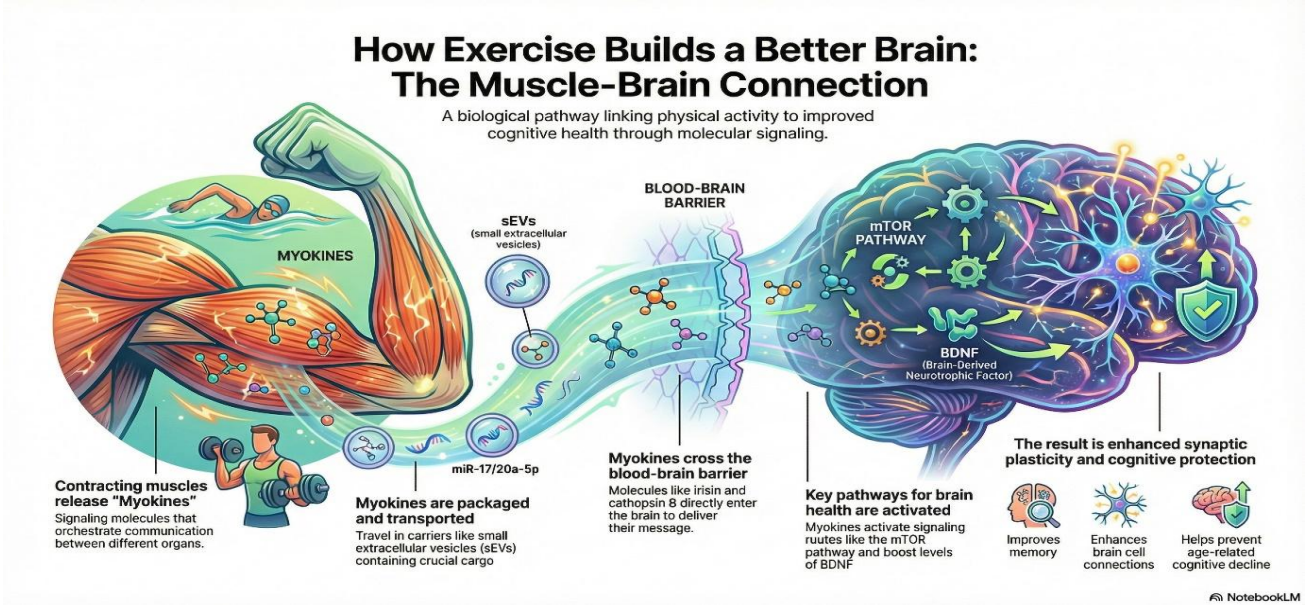
A systematic review by Tuesta and colleagues (2022) confirmed that NIRS reliably detects exercise-induced adaptations in muscle oxygenation and metabolism across diverse clinical populations.(Tuesta et al., 2022) Their

findings support NIRS as a sensitive tool for monitoring rehabilitation and therapeutic exercise interventions in inactive and metabolically impaired individuals

The Muscle–Brain Axis: Mechanistic Evidence Linking Sarcopenia to Cognition

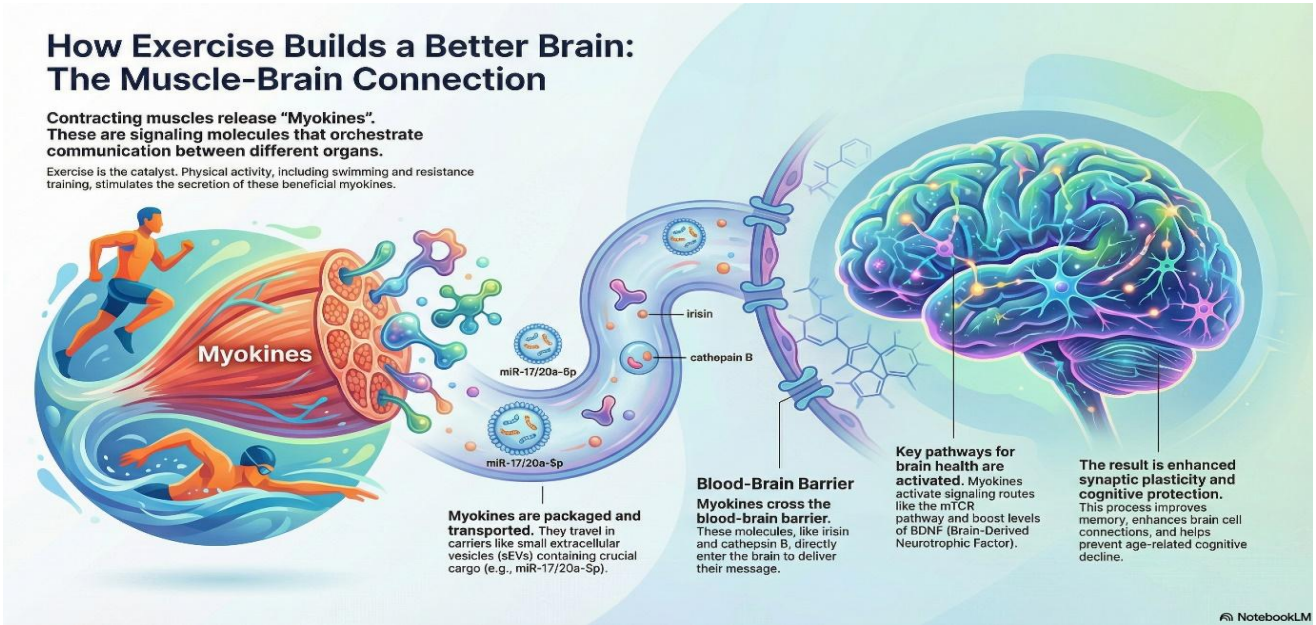
Muscle Atrophy and Cognitive Impairment

Direct mechanistic evidence linking skeletal muscle loss to cognitive decline has emerged from preclinical studies. Nagase and Tohda (2021) demonstrated that experimentally induced muscle atrophy accelerated cognitive impairment in Alzheimer's disease model mice.(Nagase & Tohda, 2021) They identified hemopexin, a muscle-derived protein, as a key mediator linking muscle atrophy to hippocampal neuroinflammation and memory deficits, providing causal evidence for muscle-to-brain signalling.(NotebookLM, n.d.)



Protective Role of Exercise-Induced Myokines

Conversely, exercise-induced myokines exert neuroprotective effects. Park, Kim, and Mikami (2022) showed that physical inactivity reduced circulating irisin levels, leading to anxiety-like behaviour, depressive states, and working memory impairment in mice.(Park et al., 2022) Restoration of exercise increased skeletal muscle FNDC5/irisin expression, enhanced hippocampal brain-derived neurotrophic factor (BDNF), and reversed cognitive decline. Neutralisation of irisin abolished these benefits, confirming its causal role in maintaining cognitive function.(NotebookLM, n.d.)



Integrative Reviews of Sarcopenia and Cognitive Decline

A scoping review by Liu, Wong, Chow, Cheung, and Wong (2023) synthesised preclinical evidence demonstrating that muscle atrophy, muscle insulin resistance, and altered myokine secretion impair hippocampal neurogenesis and synaptic plasticity.(Liu et al., 2023) Their findings support the view that skeletal muscle is an active mediator of cognitive function rather than a passive target of neurodegeneration.

Similarly, Sui, Williams, Holloway-Kew, Hyde, and Pasco (2021) highlighted shared biological pathways linking sarcopenia and cognitive decline, including inflammation, oxidative stress, mitochondrial dysfunction, and impaired myokine signalling.(Sui et al., 2020) Physical inactivity emerged as a key modifiable risk factor common to both conditions.
Building on these findings, Oudbier and colleagues (2022) proposed a unifying pathophysiological model in which dysfunctional myokine secretion, insulin resistance, impaired protein metabolism, and mitochondrial dysfunction jointly explain the association between low skeletal muscle mass and cognitive impairment.(Oudbier et al., 2022) They emphasised restoration of muscle activity as a potential therapeutic strategy to preserve cognitive health in ageing populations

Integrating HOMA-IR and NIRS: Clinical and Research Implications

The evidence synthesised in this review supports a complementary biomarker framework:[I] HOMA-IR captures systemic insulin resistance, a central driver of sarcopenia and metabolic dysfunction (Srikanthan et al.; Li et al.).(Srikanthan et al., 2010).[II] NIRS provides localised, functional assessment of muscle oxidative health and inactivity-related decline (Barstow, Adami & Rossiter, Tuesta et al.).(Barstow, 2019)[III]Muscle-derived signalling molecules directly influence brain inflammation, neurotrophin expression, and cognition (Nagase & Tohda; Park et al.).(Nagase & Tohda, 2021).

Integrating HOMA-IR and NIRS may enable the early identification of high-risk individuals, improve monitoring of exercise and rehabilitation interventions, and provide a deeper mechanistic understanding of the muscle–brain axis.

CONCLUSION

Inactive and sarcopenic muscle is strongly linked to insulin resistance and cognitive decline through interconnected pathways involving impaired insulin signalling, mitochondrial dysfunction, inflammation, and altered myokine secretion. HOMA-IR provides a robust systemic marker of metabolic dysfunction, while near-infrared spectroscopy offers a non-invasive window into local muscle metabolic health. Together, these tools provide a powerful framework for advancing research and clinical strategies to preserve metabolic and cognitive health throughout the lifespan.

Authors contributions

Conceptualisation, design and literature search: MKG and DRD, RP and ASH

Data acquisition, manuscript preparation, drafting and revision: LP, DRD

Manuscript editing and manuscript review: MKG and LP

Financial support and sponsorship

Nil

Conflict of interest

There is no conflict of interest

REFERENCE

Adami, A., & Rossiter, H. B. (2018). Principles, insights, and potential pitfalls of the noninvasive determination of muscle oxidative capacity by near-infrared spectroscopy. Journal of Applied Physiology, 124(1), 245–248.

Barstow, T. J. (2019). Understanding near infrared spectroscopy and its application to skeletal muscle research. Journal of Applied Physiology, 126(5), 1360–1376.

Cruz-Jentoft, A. J., Bahat, G., Bauer, J., Boirie, Y., Bruyère, O., Cederholm, T., Cooper, C., Landi, F., Rolland, Y., Sayer, A. A., Schneider, S. M., Sieber, C. C., Topinkova, E., Vandewoude, M., Visser, M., Zamboni, M., Writing Group for the European Working Group on Sarcopenia in Older People 2 (EWGSOP2), and the Extended Group for EWGSOP2, Bautmans, I., Baeyens, J.-P., … Schols, J. (2019). Sarcopenia: Revised European consensus on definition and diagnosis. Age and Ageing, 48(1), 16–31.

Dementia. (n.d.). Retrieved January 15, 2026, from https://www.who.int/news-room/fact-sheets/detail/dementia

Grassi, B., & Quaresima, V. (2016). Near-infrared spectroscopy and skeletal muscle oxidative function in vivo in health and disease: A review from an exercise physiology perspective. Journal of Biomedical Optics, 21(9), 091313.

Homeostasis model assessment of insulin resistance in a general adult population in Korea: Additive association of sarcopenia and obesity with insulin resistance—Kwon—2017—Clinical Endocrinology—Wiley Online Library. (n.d.). Retrieved January 12, 2026, from https://onlinelibrary.wiley.com/doi/epdf/10.1111/cen.13233

Kullmann, S., Heni, M., Fritsche, A., & Preissl, H. (2015). Insulin action in the human brain: Evidence from neuroimaging studies. Journal of Neuroendocrinology, 27(6), 419–423.

Kuo, H.-K., Leveille, S. G., Yu, Y.-H., & Milberg, W. P. (2007). Cognitive Function, Habitual Gait Speed, and Late-Life Disability in the National Health and Nutrition Examination Survey (NHANES) 1999–2002. Gerontology, 53(2), 102–110.

Li, M., Ji, R., Liu, X., & Wu, Y. (2024). Associations of metabolic syndrome and its components with sarcopenia, and the mediating role of insulin resistance: Findings from NHANES database. BMC Endocrine Disorders, 24(1), 203.

Liu, C., Wong, P. Y., Chow, S. K. H., Cheung, W. H., & Wong, R. M. Y. (2023). Does the regulation of skeletal muscle influence cognitive function? A scoping review of pre-clinical evidence. Journal of Orthopaedic Translation, 38, 76–83.

Nagase, T., & Tohda, C. (2021). Skeletal muscle atrophy-induced hemopexin accelerates onset of cognitive impairment in Alzheimer's disease. Journal of Cachexia, Sarcopenia and Muscle, 12(6), 2199–2210.

NotebookLM. (n.d.). Retrieved January 23, 2026, from https://notebooklm.google.com/?icid=home_maincta

Oudbier, S. J., Goh, J., Looijaard, S. M. L. M., Reijnierse, E. M., Meskers, C. G. M., & Maier, A. B. (2022). Pathophysiological Mechanisms Explaining the Association Between Low Skeletal Muscle Mass and Cognitive Function. The Journals of Gerontology: Series A, 77(10), 1959–1968.

Park, J., Kim, J., & Mikami, T. (2022). Exercise hormone irisin prevents physical inactivity-induced cognitive decline in mice. Behavioural Brain Research, 433, 114008.

Pedersen, B. K. (2019). Physical activity and muscle-brain crosstalk. Nature Reviews. Endocrinology, 15(7), 383–392.

Sendra-Pérez, C., Encarnación-Martínez, A., & Priego-Quesada, J. I. (2025). Critical Narrative Review of the Applications of Near-Infrared Spectroscopy Technology in Sports Science. Sensors, 25(21), 6798.

Srikanthan, P., Hevener, A. L., & Karlamangla, A. S. (2010). Sarcopenia Exacerbates Obesity-Associated Insulin Resistance and Dysglycemia: Findings from the National Health and Nutrition Examination Survey III. PLoS ONE, 5(5), e10805.

Sui, S. X., Williams, L. J., Holloway-Kew, K. L., Hyde, N. K., & Pasco, J. A. (2020). Skeletal Muscle Health and Cognitive Function: A Narrative Review. International Journal of Molecular Sciences, 22(1), 255.

Tuesta, M., Yáñez-Sepúlveda, R., Verdugo-Marchese, H., Mateluna, C., & Alvear-Ordenes, I. (2022). Near-Infrared Spectroscopy Used to Assess Physiological Muscle Adaptations in Exercise Clinical Trials: A Systematic Review. Biology, 11(7), 1073.