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Judul	:	ARTIKEL TWO FACES OF SARCOPENIA: DISTINCT METABOLIC AND CARDIOMETABOLIC SIGNATURES ACROSS OBESE AND NON-OBESE PHENOTYPES IN A MULTI-DISTRICT URBAN POPULATION IN JAKARTA-A CROSS SECTIONAL STUDY
Nama Media	:	OBESITIES
Penerbit	:	MDPI
Volume/Tahun	:	6/5/2026
URL Repository	:	https://www.mdpi.com/2673-4168/6/5/60

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31 Agustus 2026
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Message from the Editor-in-Chief

Obesities covers a wide range of research fields related to obesity, including cell experiments, animal experiments, and clinical research. As a researcher in the field of food science, I believe that diet is important for preventing and reducing the incidence of obesity; therefore, I would like to expand the scope of this journal to include not only dietary therapy but also the physiological functions of food components that lead to a reduction in the incidence of obesity and its related diseases. We encourage you to submit papers on any topic related to obesity and adipocytes. As *Obesities* is an open access journal, your article will be published online immediately upon acceptance. Readers will have unlimited free access to the content once published. We would be pleased to welcome you as one of our authors.

Editor-in-Chief
Prof. Dr. Nobuyuki Takahashi

Aims

Obesities (ISSN 2673-4168) is an open access journal that provides an advanced forum for studies of obesity in the areas of nutrition, diabetes, bariatric surgery, public health, pediatrics, basic science, exercise, eating disorders, psychology, genetics, biochemistry, physiology, molecular, metabolic, epidemiological aspects of obesity, and related disorders. We encourage scientists to publish their experimental and theoretical results in as much detail as possible. The full experimental details must be provided so that the results can be reproduced. There is no restriction on the length of the papers. We also encourage the publication of timely reviews and commentaries on topics of interest to obesity research.

Publications can be in the form of reviews, regular research papers, communications, conference reports. Large electronic files, software, movies, etc., that cannot be published in the main paper can be posted as supplementary material.

Scope

- Obesity Biology and Integrated Physiology
- Clinical Trials and Investigations
- Pediatric Obesity
- Epidemiology/Genetics
- Nutritional medicine
- Clinical nutrition medicine
- Genetics and nutrition
- Nutritional epidemiology
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Website (https://dbs.nodai.ac.jp/html/100000859_en.html)
Editor-in-Chief
Laboratory of Physiology and Metabolism, Department of Food Safety and Nutritional Science, Faculty of Applied Bioscience, Tokyo University of Agriculture, Tokyo, Japan
Interests: nutrition; phytochemicals; lipids; nuclear receptors; lipid metabolisms; inflammation; insulin resistance; hyperlipidemia; adipocytes; hepatocytes; enterocytes
Special Issues, Collections and Topics in MDPI journals

Dr. Deodato Assanelli
Website (https://www.unibs.it/en/ugov/person/661)
Editorial Board Member
Sport-Internal Medicine, Department of Clinical and Experimental Sciences, University of Brescia, 25121 Brescia, Italy
Interests: exercise in diabetes; digital ECG, exercise in elderly; oxidative stress; cardiopulmonary test

ara Baldassano
Website (https://www.unipa.it/persone/docenti/b/sara.baldassano)
Editorial Board Member
Dipartimento di Scienze e Tecnologie Biologiche Chimiche e Farmaceutiche, Università degli Studi di Palermo, 90133 Palermo, PA, Italy
Interests: human nutrition; micronutrients; biofortified food; physical activity; gut peptides; bone remodeling and metabolism; glucose homeostasis; lipid homeostasis;

systemic homeostasis; human physiology

Special Issues, Collections and Topics in MDPI journals

tefano Ballestri

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Website (https://www.researchgate.net/profile/Stefano_Ballestri)

Editorial Board Member

Division of Internal Medicine, Hospital of Pavullo, 41126 Modena, Italy

Interests: epidemiology; pathogenesis; diagnosis and treatment of fatty liver (metabolic and viral); genetic determinants of fatty liver; metabolic syndrome; insulin resistance; association between fatty liver, metabolic syndrome, diabetes and cardiovascular diseases; liver ultrasonography

Dr. Nicholas Bello

Editorial Board Member

Rutgers, The State University of New Jersey, Piscataway, NJ, USA

Special Issues, Collections and Topics in MDPI journals

iccardo Dalle Grave

Website (http://www.dallegrave.it/en/)

Editorial Board Member

Department of Eating and Weight Disorders, Villa Garda Hospital, Garda, Italy

Interests: eating disorders; obesity; nutrition; cognitive behavior therapy; lifestyle modification; digital treatment; weight loss drugs

Special Issues, Collections and Topics in MDPI journals

Dr. Carmine Finelli

Editorial Board Member

Department of Internal Medicine, Ospedale Cav. R. Apicella–ASL Napoli 3 Sud, Via di Massa, 1, Pollena, 80040 Napoli, Italy

Interests: obesity biology and integrated physiology; clinical trials and investigations; epidemiology/genetics; nutritional medicine; clinical nutrition medicine; genetics and nutrition; nutritional epidemiology; pharmacology; adipogenesis; behavioral

epidemiology; biophysics and lipid metabolism; exercise and human physiology; phenotyping; fat cell physiology; aging; metabolic syndrome; nutrition behavior; eating disorder; over weight; adipocyte cell biology; etiology; psychological and epidemiological aspects of obesity and related disorders; diabetes; obesity; food science and technology; global health; appetite; dietary surveys; macronutrients; micronutrients; malnutrition; weight control

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Dr. Michel Fontés

Website (http://www.cadureso.com/actualite/actualite-sante/927-professeur-michel-fontes-therapie-genique-et-maladies-rares)

Editorial Board Member

Obesity and Risk of Thrombosis Laboratory, Aix Marseille Universite, Marseille, France

Interests: peripheral neuropathies; animal models; drug development; pathophysiology of inherited disorders

Special Issues, Collections and Topics in MDPI journals

Mathieu Gayda

Website (https://esc365.escardio.org/person/203643)

Editorial Board Member

Cardiovascular Prevention and Rehabilitation Center, Montreal Heart Institute and Université de Montréal, 5055 St Zotique Street East, Montreal, QC H1T 1N6, Canada

Interests: the links between heart rate recovery, CV mortality and glycaemic status in patients with CHD; the effects of Mediterranean diet counselling and exercise training programs on cardiometabolic parameters, aerobic and muscular function, fat mass loss or QT dispersion; the validity and reproducibility of exercise parameters during deep water running and aquabiking; Optimisation and prescription of high intensity interval exercise in cardiac patients; muscle microvascular function and cardiovascular risk; the effects of cardiovascular risks or diseases on cognitive function, brain oxygenation/perfusion, cardiac function, exercise capacity and the effects of exercise training intervention on those pa; Metabolic and cardiovascular responses to acute and chronic high intensity interval exercise and moderate intensity continuous exercise in healthy, obese and patients with CHD

Alexandra Gouveia

Website (https://www.cienciavitae.pt/portal/en/2C15-98D1-4FA9)

Editorial Board Member

Department of Biomedicine, Faculty of Medicine and IBMC/I3S- Instituto de Investigação e Inovação em Saúde, University of Porto, Porto, Portugal

Interests: obesity; melanocortins; white and brown adipose tissue; browning mechanism; inflammation; autophagy; stress response; ageing

Andrew Gray

Website (https://www.otago.ac.nz/healthsciences/expertise/profile?id=769)

Editorial Board Member

Dunedin School of Medicine, University of Otago, Dunedin 9054, New Zealand

Interests: biostatistics; statistics; epidemiology; public health; obesity; nutrition; sleep; physical activity; respiratory health

Dr. Nadim Haboubi

Website (https://easo.org/expert/nadim-haboubi/)

Editorial Board Member

Clinical Nutrition and Obesity, University of South Wales, Pontypridd CF37 1DL, UK

Interests: IBS; obesity; nutrition; coeliac; bariatric

Prof. Dr. Andrew John Hill

Website (https://medicinehealth.leeds.ac.uk/medicine/staff/425/professor-andrew-hill)

Editorial Board Member

Division of Psychological & Social Medicine, Institute of Health Sciences, University of Leeds School of Medicine, Level 10, Worsley Building, Clarendon Way, Leeds LS2 9NL, UK

Interests: obesity; eating disorders; psychology; body image; food craving; weight bias in children

Dr. Phillip B. Hylemon

Editorial Board Member

Department of Microbiology/Immunology, Medical College of Virginia, Virginia Commonwealth University, Richmond, VA, USA

Interests: physiology, metabolism and genetics of intestinal anaerobic bacteria; isolation and characterization of antibiotics compounds secreted by human gut bacteria; the role of the human gut microbiome in cirrhosis; regulation of bile acid activated cell signaling pathways

Terry D. Hinds, Jr.

Website (https://medicine.uky.edu/users/tdhi230)

Editorial Board Member

Department of Pharmacology and Nutritional Sciences, University of Kentucky College of Medicine, Lexington, KY 40508, USA

Interests: heme oxygenase; bilirubin; oxidative stress; nuclear receptors; fatty liver; glucocorticoid receptor; PPARs

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Dr. Krissy Kendall

Website (https://www.ecu.edu.au/schools/medical-and-health-sciences/our-staff/profiles/lecturers/dr-kristina-kendall)

Editorial Board Member

School of Medical and Health Sciences, Edith Cowan University, Perth, Australia

Interests: ergogenic aids; body composition; resistance training; high-intensity interval training

Prof. Gerard A. Kennedy

Website (https://federation.edu.au/institutes-and-schools/ihw/staff-profiles/staff-profiles2/kennedy)

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Psychology, School of Health and Life Sciences, Federation University, Mt Helen, Australia

Interests: sleep; sleep disorders; circadian rhythms; insomnia; sleep apnoea; diabetes; obesity; wellbeing; clinical psychology; physical activity

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Dr. Marion Korach-André

Website (https://www.univ-fcomte.fr/valorisation-et-transfert-de-technologie)

Editorial Board Member

Department of Research and Innovation, University Marie and Louis Pasteur, UFR ST - Bât DF - 16 route de Gray - 25030 Besançon CEDEX - France

Interests: lipid metabolism; lipidomic; sex differences; sex hormones; nutrition; metabolic syndrome

Special Issues, Collections and Topics in MDPI journals

Dr. Evangelos Liberopoulos

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Editorial Board Member

Department of Internal Medicine, Faculty of Medicine, School of Health Sciences, University of Ioannina, 45110 Ioannina, Greece

Interests: lipids; diabetes; obesity; cholesterol; cardiovascular disease; atherosclerosis

Dr. Paolo Marzullo

Editorial Board Member

Department of Translational Medicine, Università del Piemonte Orientale "A. Avogadro", Novara, Italy

Dr. Clarika Massaro

Website (https://onlinelibrary.wiley.com/doi/abs/10.1002/9780470988473.ch1)

Editorial Board Member

Institute of Clinical Physiology (IFC), National Research Council (CNR), 73100 Lecce, Italy

Interests: pathogenesis of chronic disese; nutri- and pharmacogenomics applied to obesity and cardiovascular disease prevention; analysis of the anti-aterogenic and anti-inflammatory properties of nutritional fatty acids and plant food bioactives in the field of vascular biology and physical exercise; analysis of dietary patterns and nutritional status in health and chronic disease

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Dr. Laurent Metzinger

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Editorial Board Member

UR-UPJV 4666, HEMATIM, CURS, Université de Picardie Jules Verne, 80025 Amiens, France

Interests: gene regulation; non-coding RNA; genetics; diabetes; chronic kidney disease; cardiovascular disease; multi-omics

Special Issues, Collections and Topics in MDPI journals

Prof. Dr. Vincenzo Mollace

Website (https://www.researchgate.net/profile/Vincenzo_Mollace)

Editorial Board Member

Institute of Research for Food Safety & Health (IRC-FSH), University of Catanzaro “Magna Graecia”, Catanzaro, Italy

Interests: neurological diseases; neurodegeneration; natural compounds; poliphenols; reactive oxygen species; apoptosis; autophagy; mitochondria; endoplasmic reticulum; blood brain barrier; endothelium involvement

Special Issues, Collections and Topics in MDPI journals

Justin B. Moore

Website (https://school.wakehealth.edu/Faculty/M/Justin-B-Moore)

Editorial Board Member

Wake Forest School of Medicine, Winston-Salem, NC27101, USA

Interests: obesity; sleep; physical activity; healthy eating; lifestyle intervention; mHealth; implementation

Special Issues, Collections and Topics in MDPI journals

Dr. Ju-Ock Nam

Website (https://scholar.google.com/citations?user=jPi5rsoAAAAJ&hl=en)

Editorial Board Member

Department of Food Science and Biotechnology, Kyungpook National University, Daegu 41566, Republic of Korea

Interests: tumor microenvironment; obesity; cell signaling

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Dr. Kerry O'Brien

Website (http://orcid.org/0000-0003-3145-6038)

Editorial Board Member

Behavioural Sciences Research Laboratory, Monash University, Clayton, VIC 3800, Australia

Interests: stigma; discrimination and health; obesity stigma; alcohol and gambling marketing and harms; body image; psychological and social aspects of endometriosis; public health; addiction

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Prof. Dr. Kye Won Park

Editorial Board Member

Department of Food Science and Biotechnology, Sungkyunkwan University, Suwon, Republic of Korea

Interests: adipocytes; phytochemicals; PPAR

Dr. Zoltan Pataky

Website1 (https://www.unige.ch/medecine/diabetescentre/en/members/scientific-advisory-board/zoltan-pataky) Website2 (https://www.hug.ch/education-therapeutique) Website3 (https://zpataky.com)

Editorial Board Member

Unit of Therapeutic Patient Education (Diabetes and Obesity), WHO Collaborating Centre, University Hospitals of Geneva, Gabrielle-Perret-Gentil 4, 14, CH–1211 Geneva, Switzerland

Interests: obesity; food addiction; weight loss treatment and management; patient education

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Dr. Gu Seob Roh

Editorial Board Member

Department of Anatomy and Convergence Medical Science, College of Medicine, Gyeongsang National University, Jinju, Gyeongnam, Republic of Korea

Interests: obesity; diabetes; NAFLD; diabetic

Dr. Manuel Sánchez Santos

Website (https://www.ugr.es/en/staff/manuel-sanchez-santos)

Editorial Board Member

1. Department of Pharmacology, School of Pharmacy, University of Granada, 18071 Granada, Spain

2. Research Collaborator, Health Research Institute of Granada (ibs.GRANADA), Center of Biomedical Research (CIBM), Granada, Spain

Interests: polyphenols; flavonoids; prebiotics and probiotics; cardiovascular disease; endothelial function; oxidative stress; dysbiosis; microbiota; obesity; hypertension; systemic lupus erythematosus; type 2 diabetes

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E-ISSN: 2673-4168

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☆	#1	MMWR Recommendations and Reports	131.2	99th percentile
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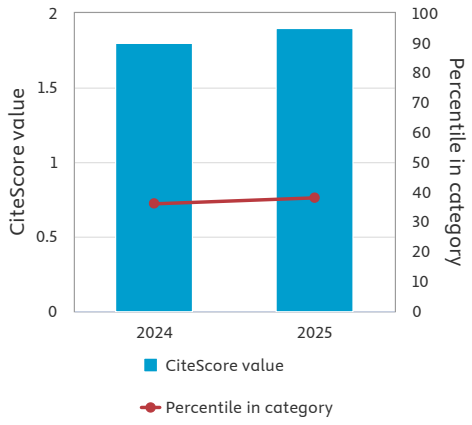
☆	#4	Clinical Microbiology Reviews	28.9	97th percentile
☆	#5	The Lancet HIV	18.9	97th percentile
☆	#6	European Journal of Preventive Cardiology	16.1	96th percentile
☆	#7	Emerging Microbes and Infections	14.1	96th percentile
☆	#8	Alzheimer's and Dementia	13.9	95th percentile
☆	#9	Eurosurveillance	13.6	95th percentile
☆	#10	Journal of the American Society of Nephrology	12.9	94th percentile

CiteScore trend



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☆	#11	Journal of Exposure Science and Environmental Epidemiology	11.7	93rd percentile
☆	#12	International Journal of Epidemiology	11.5	93rd percentile
☆	#13	European Journal of Epidemiology	11.2	92nd percentile
☆	#14	Clinical Journal of the American Society of Nephrology	10.0	92nd percentile
☆	#15	Emerging Infectious Diseases	9.5	91st percentile
☆	#16	Journal of Clinical Epidemiology	9.4	91st percentile
☆	#17	Epidemiology and Psychiatric Sciences	9.3	90th percentile
☆	#18	Global Health Research and Policy	8.4	89th percentile
☆	#19	Journal of Health Monitoring	8.4	89th percentile
☆	#20	American Journal of Preventive Medicine	8.0	88th percentile
☆	#21	Social Psychiatry and Psychiatric Epidemiology	7.7	88th percentile
☆	#22	Communications Medicine	7.7	87th percentile
☆	#23	Influenza and other Respiratory Viruses	7.6	86th percentile
☆	#24	Journal of Epidemiology and Community Health	7.5	86th percentile
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☆	#30	Epidemiologic Reviews	7.0	82nd percentile
☆	#31	Epidemiology	6.8	82nd percentile
☆	#32	Clinical Epidemiology	6.7	81st percentile
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☆	#34	Paediatric and Perinatal Epidemiology	6.7	80th percentile

Rank	Source title	CiteScore 2025	Percentile
#35	Neuroepidemiology	6.6	80th percentile
#36	BMC Medical Research Methodology	6.6	79th percentile
#37	Health Care Science	6.6	78th percentile
#38	Global Heart	6.3	78th percentile
#39	Microbial Risk Analysis	6.1	77th percentile
#40	Infection Control and Hospital Epidemiology	5.8	77th percentile
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#42	Health Promotion and Chronic Disease Prevention in Canada : Research, Policy and Practice	5.6	76th percentile
#43	Health Services Research and Managerial Epidemiology	5.5	75th percentile
#44	Genetic Epidemiology	5.5	74th percentile
#45	Infectious Agents and Cancer	5.4	74th percentile
#46	Cancer Epidemiology Biomarkers and Prevention	5.4	73rd percentile
#47	Environmental Epidemiology	5.4	73rd percentile
#48	Zoonoses and Public Health	5.3	72nd percentile
#49	Epidemics	5.2	71st percentile
#50	American Journal of Infection Control	5.2	71st percentile
#51	Journal of Immigrant and Minority Health	5.2	70th percentile
#52	Food and Environmental Virology	5.2	70th percentile
#53	Journal of Epidemiology and Global Health	5.0	69th percentile
#54	European Journal of Cancer Prevention	4.7	69th percentile
#55	Cancer Epidemiology	4.6	68th percentile
#56	Environmental and Molecular Mutagenesis	4.6	67th percentile
#57	Clinical Practice and Epidemiology in Mental Health	4.6	67th percentile

Rank	Source title	CiteScore 2025	Percentile
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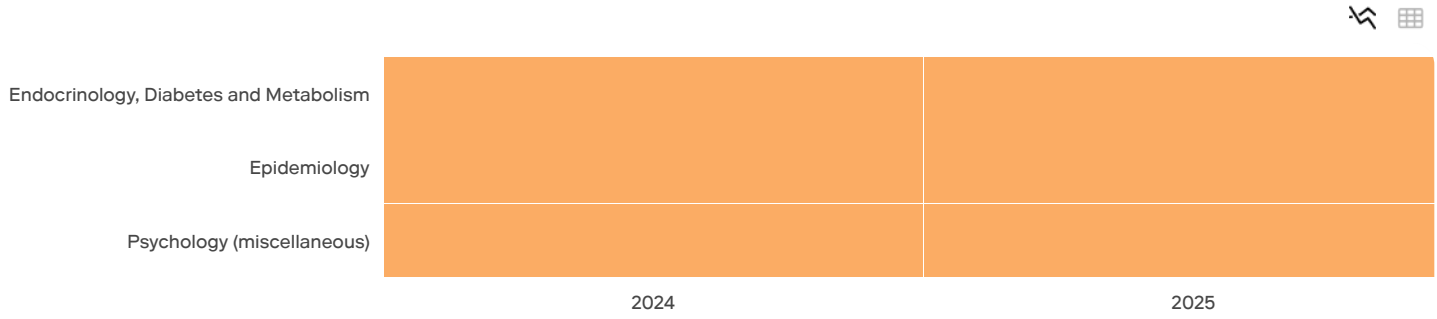

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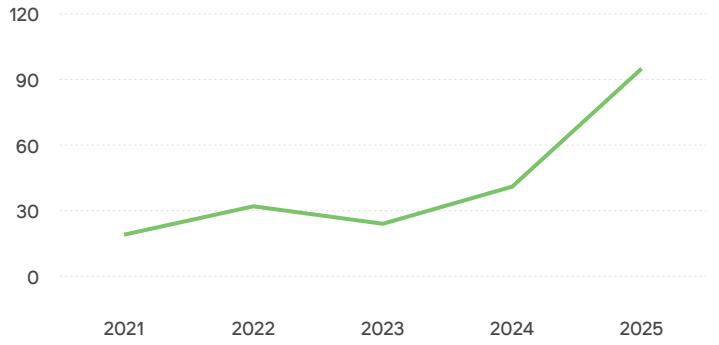

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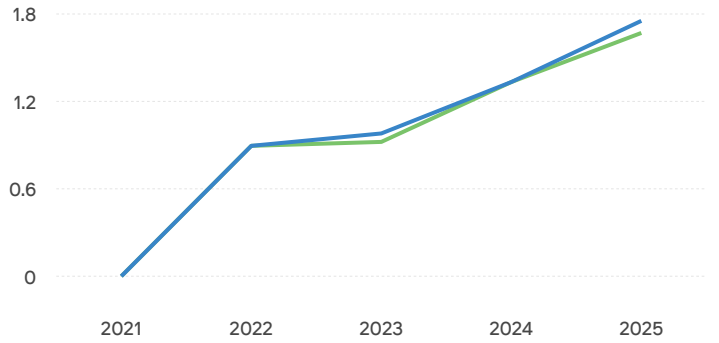

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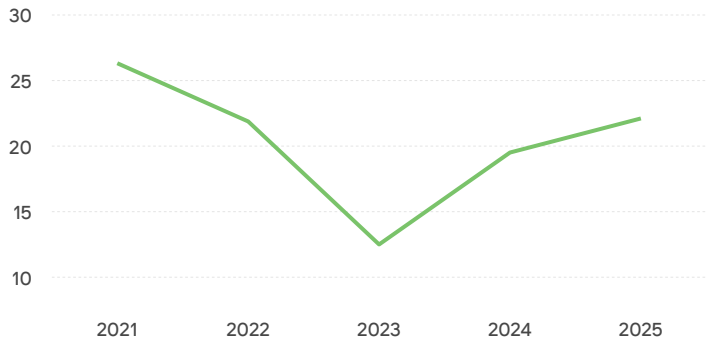

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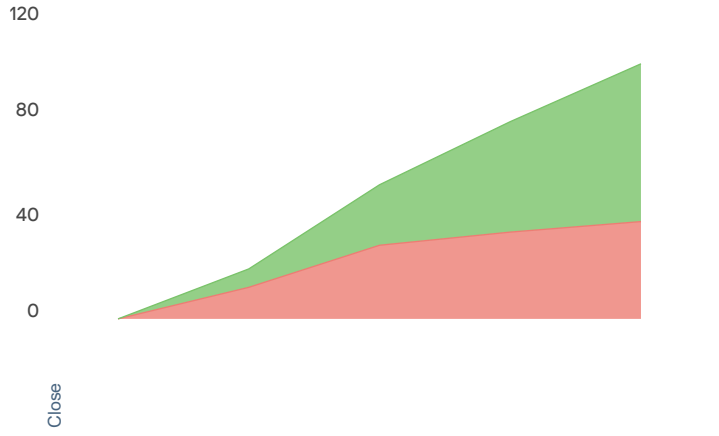

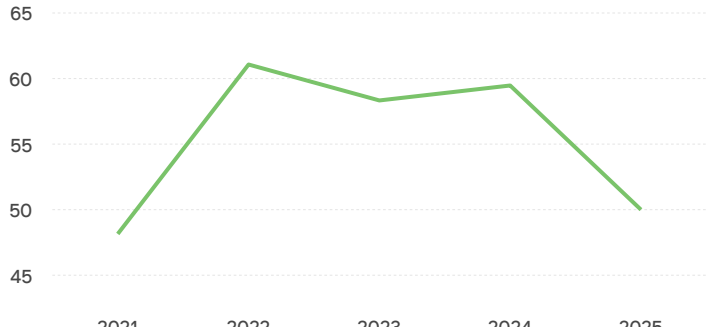

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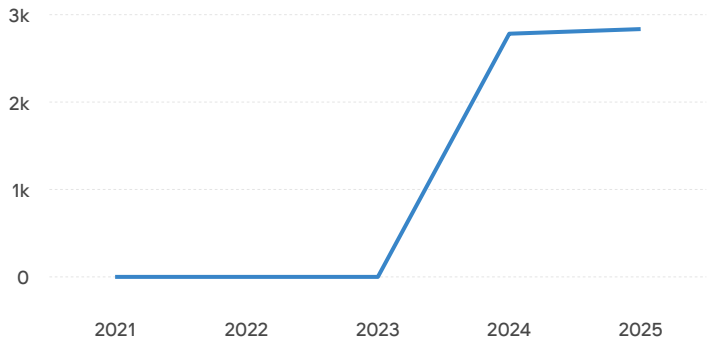

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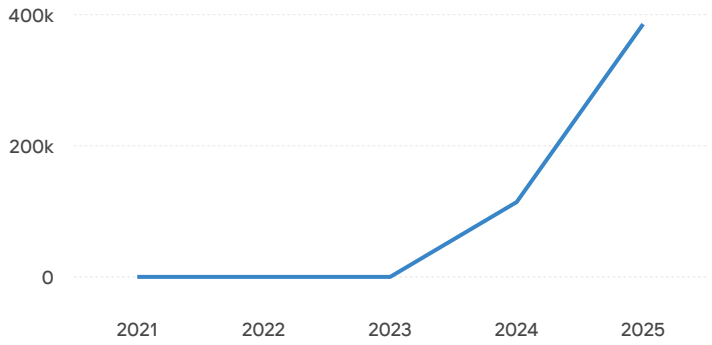

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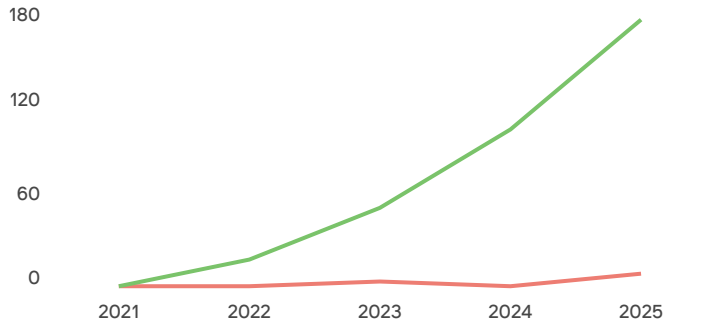

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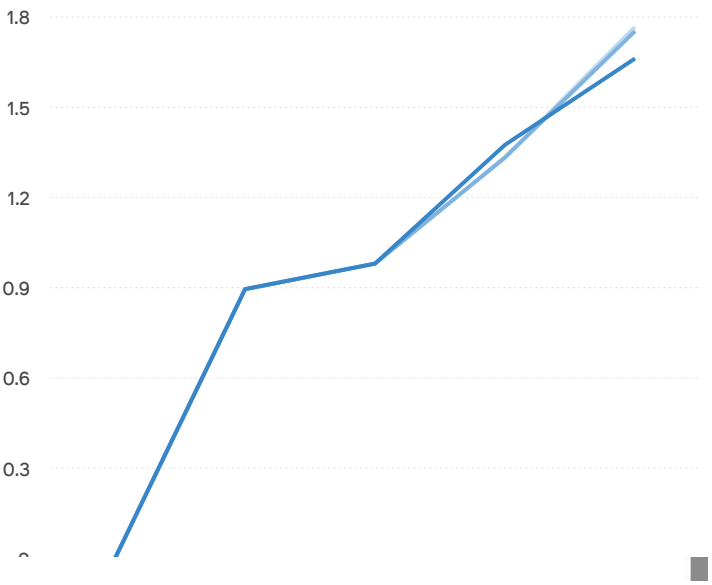

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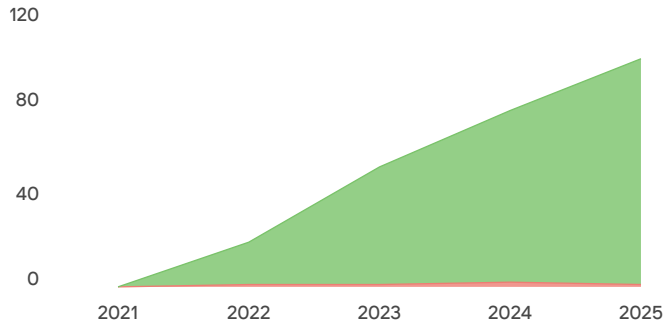

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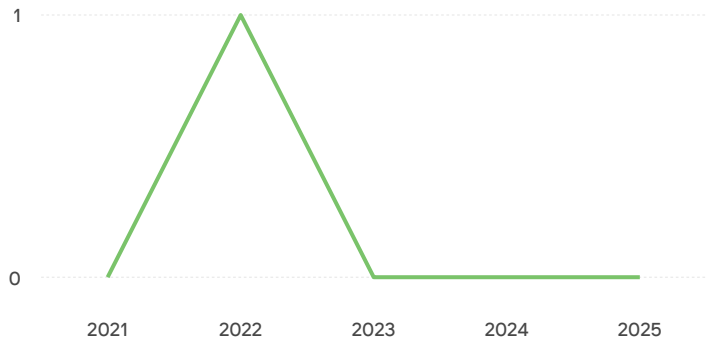

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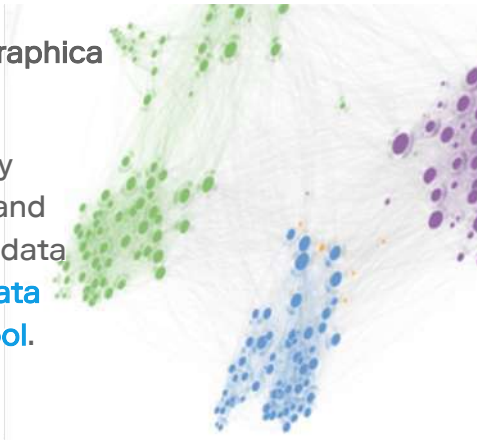

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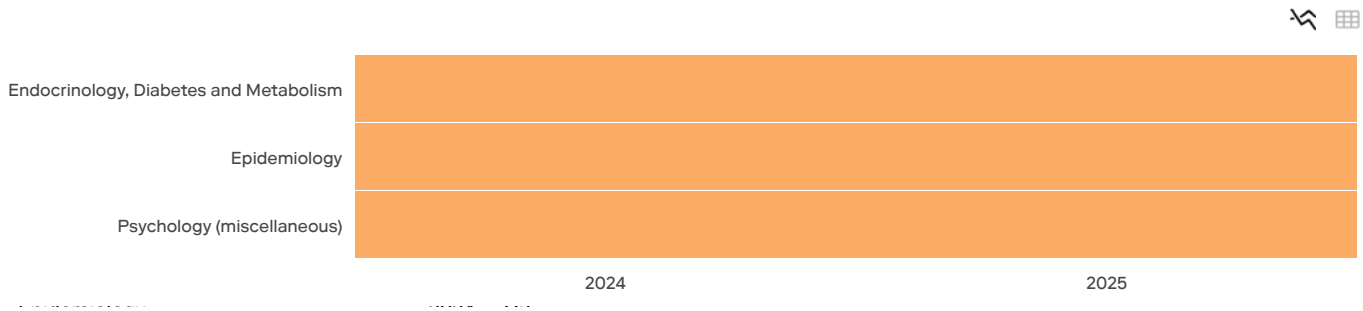

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Two Faces of Sarcopenia: Distinct Metabolic and Cardiometabolic Signatures across Obese and Non-Obese Phenotypes in a Multi-District Urban Population in Jakarta – A Cross Sectional Study

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Abstract

Introduction: Sarcopenia and obesity are increasingly recognized as overlapping conditions that contribute to adverse health outcomes, particularly in rapidly urbanizing populations. The coexistence of both conditions, termed sarcopenic obesity, represents a complex phenotype with potentially greater metabolic burden, yet its differentiation from non-obese sarcopenia remains insufficiently explored. **Aim:** This study aimed to compare metabolic and cardiometabolic profiles between sarcopenic obesity and non-obese sarcopenia in an urban population. **Methods:** A cross-sectional study was conducted using secondary data from the PRIMBON 2025 database in Jakarta, Indonesia. Participants were adults aged ≥18 years diagnosed with sarcopenia based on the 2025 Asian Working Group for Sarcopenia (AWGS) criteria. Sarcopenic obesity was defined according to the 2025 AOASO–IAGG consensus using BMI ≥25 kg/m² and visceral fat level >10. A total of approximately 604 participants were included from five administrative regions of Jakarta. Data were analyzed using Mann–Whitney U test and Chi-square test, with results presented as median (min–max), mean rank, and odds risk (OR). A dot-and-whisker plot was used to visualize effect sizes. **Results:** Sarcopenic obesity was associated with older age and a higher proportion of males ($p < 0.01$). Significant differences were observed in systolic and diastolic blood pressure, fasting blood glucose, triglycerides, and HDL (all $p < 0.01$), indicating a more adverse cardiometabolic profile. No significant differences were found for hemoglobin, total cholesterol, LDL, uric acid, and handgrip strength. **Conclusion:** Sarcopenic obesity represents a more severe metabolic phenotype compared to non-obese sarcopenia.

Keywords: sarcopenia; sarcopenic obesity; metabolic profile; cardiometabolic risk; urban population; AWGS 2025; AOASO-IAGG 2025

https://doi.org/10.3390/xxxxx

1. Introduction

Sarcopenia is a progressive and generalized loss of skeletal muscle mass and strength associated with adverse outcomes such as falls, fractures, frailty, and mortality. Although commonly associated with aging, muscle decline begins as early as the fourth decade of life, with an annual loss of 1–2% after the age of 50 years.(1) Globally, the prevalence of sarcopenia varies widely due to heterogeneity in diagnostic criteria, ranging from 10% to 27% based on a meta-analysis of 151 studies involving more than 692,000 individuals.(2) This variability highlights both the magnitude of the problem and the ongoing challenge in establishing standardized definitions and measurements of sarcopenia.(3)

In parallel, obesity has reached pandemic proportions, with its prevalence more than doubling worldwide since 1980 and currently affecting approximately 30% of the global population.(4) Obesity significantly increases the risk of chronic diseases such as type 2 diabetes, cardiovascular disease, cancer, dementia, and depression, and contributes to increased healthcare costs by up to 30% compared to individuals with normal body mass index.(5) However, the "obesity paradox" suggests a more complex relationship between body mass index and mortality, particularly in older populations, where both low and high BMI are associated with increased mortality risk, forming a U-shaped curve.(6–8) This paradox indicates that BMI alone may inadequately capture body composition and cardiometabolic risk, as individuals with sarcopenia may present with markedly different adiposity despite having similar degrees of muscle loss. Consequently, sarcopenia may manifest as two distinct phenotypes, non-obese sarcopenia and sarcopenic obesity, which are hypothesized to exhibit different metabolic and cardiometabolic profiles. (9,10)

The coexistence of sarcopenia and obesity, termed sarcopenic obesity, represents a more severe and clinically relevant phenotype due to its compounded adverse effects. The prevalence of sarcopenic obesity varies widely depending on diagnostic criteria, ranging from 0.8% to 22.3% in women and 1.3% to 15.4% in men, with increasing prevalence at older ages.(11,12) A global meta-analysis estimated its prevalence at approximately 11% among older adults.(13) Importantly, sarcopenic obesity has been associated with significantly higher risks of cardiovascular disease, reduced bone mineral density, and all-cause mortality compared to either condition alone.(14–16) Large cohort studies, including NHANES III and UK Biobank, consistently demonstrate that individuals with sarcopenic obesity have a markedly increased mortality risk, emphasizing its clinical and public health importance.(17,18) In addition to these clinical outcomes, sarcopenic obesity is characterized by metabolic and hematologic alterations that may reflect its underlying pathophysiology. Cardiometabolic biomarkers, including blood pressure, fasting blood glucose, lipid profile, serum uric acid, and hemoglobin, provide important insights into metabolic homeostasis, systemic inflammation, oxidative stress, and nutritional status. (19,20)

The development of sarcopenia and sarcopenic obesity is multifactorial, involving complex interactions between metabolic, hormonal, inflammatory, and lifestyle-related factors. Both conditions are strongly associated with metabolic and cardiovascular disorders, including diabetes, dyslipidemia,(21) metabolic syndrome (22), restrictive lung disease,(23) osteoporosis (24). Mechanistically, chronic low-grade inflammation, insulin resistance, mitochondrial dysfunction, and alterations in lipid and glucose metabolism contribute to muscle degradation and fat accumulation, creating a vicious cycle that exacerbates disease progression.(4,25,26)

Recent advances in diagnostic criteria, particularly the 2025 Asian Working Group for Sarcopenia (AWGS) consensus, have strengthened the conceptual framework of

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sarcopenia through a life-course approach, simplified diagnostic algorithms, and recognition of skeletal muscle as a central organ interacting with multiple physiological systems.(27,28) In parallel, the Asia–Oceania Association for the Study of Obesity (AOASO) and the International Association of Gerontology and Geriatrics Asia/Oceania Region (IAGG-AOR) consensus has established sarcopenic obesity as a distinct clinical entity requiring standardized diagnostic criteria and targeted management strategies.(29) Within this updated framework, the concept of the "Two Faces of Sarcopenia" emerges, distinguishing between non-obese sarcopenia and sarcopenic obesity, which may exhibit fundamentally different metabolic and cardiometabolic profiles.

In addition to conventional cardiometabolic biomarkers such as glycemic status, lipid profile, and blood pressure, hemoglobin and serum uric acid may provide complementary insights into the biological mechanisms underlying these phenotypes. Hemoglobin reflects oxygen-carrying capacity and tissue oxygenation, which are essential for skeletal muscle metabolism and function, whereas serum uric acid is closely linked to oxidative stress, chronic inflammation, insulin resistance, and metabolic syndrome. (30,31) Therefore, this study aims to explore how various metabolic factors (including glycemic status, lipid profile, hemoglobin, uric acid, and blood pressure) are differentially associated with these two phenotypes, providing deeper insight into the heterogeneity of sarcopenia and its clinical implications in a large urban population in Jakarta, Indonesia, where rapid urbanization and lifestyle transitions may further shape these metabolic patterns.

2. Material and Methods

2.1. Study Design and Setting

This study employed a cross-sectional design using secondary data obtained from the PRIMBON (Partnership for Research, Innovation, and Multisectoral Benefit for Outreach and Networking) database, developed by Universitas Tarumanagara since June 2024. The dataset utilized in this study corresponds to the 2025 data collection period. Data were derived from five administrative regions of Jakarta (West, North, South, Central, and East Jakarta), with three representative districts selected from each region to ensure adequate geographic representation of the urban population.

2.2. Population, Sample, and Sampling Technique

The study population consisted of adult individuals aged ≥18 years residing in Jakarta who were identified as having sarcopenia based on the 2025 AWGS criteria and recorded in the PRIMBON database.(27,28) The minimum sample size was calculated using the formula:

$$n = \frac{Z^2 p(1-p)}{d^2}$$

Assuming a confidence level of 95% ($Z = 1.96$), estimated prevalence ($p = 0.5$), and precision ($d = 0.05$), the minimum required sample size was 384 participants. To enhance statistical power and account for potential data exclusion, a total sample of approximately 500 participants was targeted. A clustered sampling approach was applied across the selected districts to ensure proportional representation.

2.3. Inclusion and Exclusion Criteria

Participants were included if they were adults aged ≥18 years residing in Jakarta, identified as having sarcopenia based on the AWGS 2025 criteria (27,28), registered in the PRIMBON 2025 database, and had complete data on anthropometric, muscle, and metabolic parameters. Because the primary objective of this study was to compare the metabolic and cardiometabolic characteristics of the two clinically recognized sarcopenia phenotypes (non-obese sarcopenia and sarcopenic obesity), only participants meeting the

diagnostic criteria for sarcopenia were included. Accordingly, healthy individuals and obese participants without sarcopenia were not included, as comparisons with these groups were beyond the scope of the present study. Information regarding medical history used for the exclusion criteria was obtained from a standardized self-reported health questionnaire completed by participants during the PRIMBON 2025 health screening. Participants were excluded if they had incomplete or missing data, resided outside Jakarta, or self-reported conditions that could compromise the validity of muscle strength, body composition, or metabolic measurements, including disorders affecting handgrip strength (e.g., upper limb injury or neuromuscular diseases such as myasthenia gravis), conditions interfering with body composition assessment (e.g., metallic implants or prosthetic devices), secondary hypertension or other conditions influencing blood pressure measurements, hematological disorders affecting hemoglobin levels (e.g., polycythemia vera or aplastic anemia), hereditary or severe lipid metabolism disorders, type 1 diabetes mellitus, or other clinical conditions known to significantly alter metabolic biomarkers such as fasting blood glucose, lipid profile, and uric acid.

2.4. Data Collection Procedures

Data were extracted from the PRIMBON 2025 database following a standardized protocol. The dataset included demographic characteristics, anthropometric measurements, muscle function (handgrip strength), body composition assessed by bioelectrical impedance analysis (BIA) (skeletal muscle index and visceral fat level), and cardiometabolic parameters (blood pressure, hemoglobin, lipid profile, fasting blood glucose, and uric acid). Data cleaning and validation were performed prior to analysis, including screening for completeness, plausibility, and consistency across variables. Only eligible participants meeting the predefined criteria were included in the final analysis.

2.5. Operational Definitions and Classification of Sarcopenia and Sarcopenic Obesity

Sarcopenia was defined based on the AWGS 2025 criteria using appendicular skeletal muscle mass index (ASM/height²) and handgrip strength. Low skeletal muscle mass was defined as <7.6 kg/m² for men and <5.7 kg/m² for women aged <65 years, and <7.0 kg/m² for men and <5.7 kg/m² for women aged ≥65 years. Low handgrip strength was defined as <34 kg for men and <20 kg for women aged <65 years, and <28 kg for men and <18 kg for women aged ≥65 years.(27,28) Sarcopenic obesity was defined according to the 2025 consensus of the AOA-AO and the IAGG-AOR, whereby participants fulfilled the AWGS criteria for sarcopenia in addition to obesity defined as body mass index (BMI) ≥25 kg/m² and visceral fat level >10 as an alternative to waist circumference.(29) Based on these criteria, participants in this study were categorized into two groups: non-obese sarcopenia and sarcopenic obesity. These definitions were adapted to align with the measurement specifications of the devices used in the standardized PRIMBON 2025 data collection.

2.6. Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 27.0 (IBM Corp., Released 2020. IBM SPSS Statistics for Windows, Version 27.0. Armonk, NY, USA: IBM Corp.) and Microsoft Excel 365 (Microsoft Corporation, Redmond, WA, USA). Descriptive (univariate) analysis was conducted to summarize the characteristics of the study population. Numerical variables were presented as median (minimum–maximum) due to non-normal distribution, while categorical variables were expressed as frequencies and percentages.

Bivariate analysis was performed to compare differences between sarcopenic obesity and non-obese sarcopenia groups. The Mann–Whitney U test was used for continuous

variables, with results interpreted based on mean rank differences, while categorical variables were analyzed using the Chi-square test (with Yates' correction when appropriate). Effect estimates for categorical variables were reported as Odds Ratio (OR).

To visually represent the magnitude and direction of differences between groups, a dot-and-whisker plot was constructed, illustrating effect estimates and their corresponding confidence intervals. Statistical significance was defined as a two-tailed p-value <0.05, with an alpha level of 5% and a statistical power of 80%.

2.7. Ethical Approval

This study was approved by the Universitas Tarumanagara Human Research Ethics Committee, Institute of Research and Community Engagement, under ethical approval number 015-UTHREC/UNTAR/IV/2026. The study utilized secondary data from the PRIMBON database, and all data were anonymized prior to analysis to ensure participant confidentiality. As no direct interaction with participants was conducted, informed consent was waived by the ethics committee. All procedures were carried out in accordance with the ethical principles outlined in the Declaration of Helsinki.

3. Results

A total of 604 participants met the inclusion criteria and were included in the analysis (Figure 1). The median age was 41.5 years, with a predominance of females (61.9%). Most participants were employed (66.3%) and married (70.9%). Sarcopenic obesity was identified in 13.1% of participants. Median and Interquartiles cardiometabolic parameters, including blood pressure, lipid profile, glucose, hemoglobin, and uric acid, are presented in Table 1.

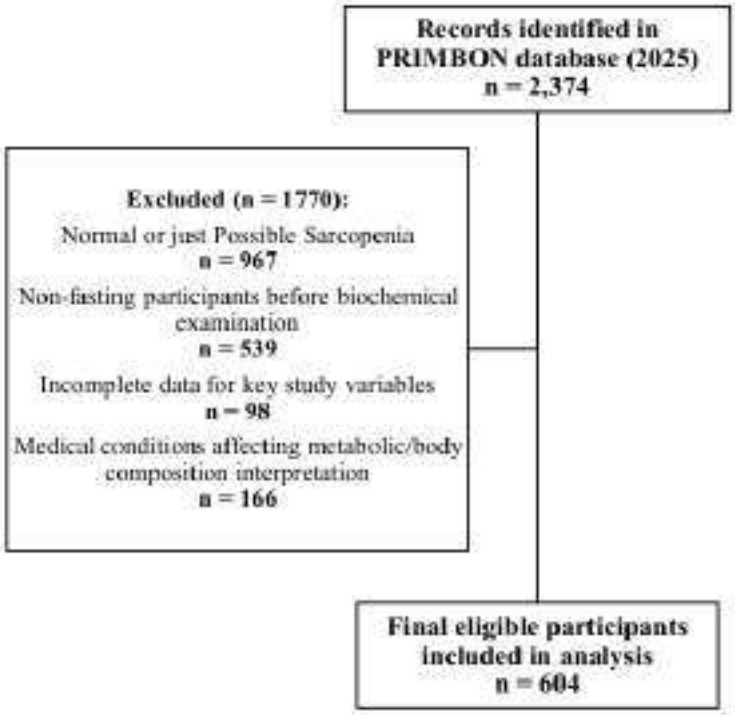

Figure 1. Flow diagram of participant selection.

Table 1. Baseline Demographic, Anthropometric, Muscle, and Cardiometabolic Profiles of the Study Population.

Parameter	Sarcopenic Obesity	Sarcopenia

	N (%)	Med (IQR)	N (%)	Med (IQR)
Age, years	79 (100)	59 (45 – 70)	525 (100)	38 (26 – 54.5)
Gender				
• Male	42 (53.2)		188 (35.8)	
• Female	37 (46.8)		337 (64.2)	
Occupation				
• Employee	27 (34.2)		313 (59.6)	
• Self-employed	4 (5.1)		22 (4.2)	
• Unemployed	48 (60.8)		190 (36.2)	
Marital status				
• Married	76 (96.2)		352 (67)	
• Unmarried	3 (3.8)		173 (33)	
Body weight, kg	79 (100)	65 (60 – 70)	525 (100)	52.7 (48 – 59)
Body height, cm	79 (100)	154.7 (148.7 – 160.3)	525 (100)	157.8 (152.1 – 164)
Body mass index, kg/m²	79 (100)	26.4 (25.5 – 28.1)	525 (100)	21.5 (19.41 – 23.25)
Handgrip strength, kg	79 (100)	20 (16 – 26.6)	525 (100)	21.7 (17.6 – 26.7)
Skeletal muscle index, kg/m²	79 (100)	5.6 (5.4 – 7.3)	525 (100)	5.4 (4.9 – 6.2)
Visceral Fat Level	79 (100)	12 (11 – 15)	525 (100)	5 (3 - 7)
Systolic Blood Pressure, mmHg	79 (100)	130 (120 – 144)	525 (100)	120 (110 – 129)
Diastolic Blood Pressure, mmHg	79 (100)	80 (72 – 86)	525 (100)	77 (70 – 84)
Triglycerides, mg/dL	79 (100)	159 (124 – 203)	525 (100)	131 (99 – 168.5)
Low Density Lipoprotein, mg/dL	79 (100)	117 (95 – 137)	525 (100)	111.4 (93.7 – 130)
High Density Lipoprotein, mg/dL	79 (100)	48 (40 – 60)	525 (100)	55 (45.1 – 64.5)
Total Cholesterol, mg/dL	79 (100)	197 (176 – 232)	525 (100)	195 (177 – 219)
Fasting blood Glucose, mg/dL	79 (100)	101 (87 – 122)	525 (100)	89 (82 – 100)
Hemoglobin, g/dL	79 (100)	12.5 (11.2 – 14.5)	525 (100)	12.9 (11.2 – 14.2)
Uric Acid, mg/dL	79 (100)	4.5 (3.9 – 5.5)	525 (100)	4.5 (3.8 – 5.2)

Table 2 and Figure 2 demonstrate significant metabolic and cardiometabolic differences between sarcopenic obesity and non-obese sarcopenia based on Mann–Whitney U test analysis. Individuals with sarcopenic obesity showed significantly higher mean ranks for age, systolic and diastolic blood pressure, triglycerides, and fasting blood glucose (all $p < 0.01$), indicating a more adverse cardiometabolic profile. In contrast, HDL levels were significantly lower in the obese group ($p = 0.002$), while no significant differences were observed for hemoglobin, total cholesterol, LDL, uric acid, and handgrip strength. Sex distribution also differed significantly, with a higher proportion of males in the sarcopenic obesity group (OR 2.035, $p = 0.005$). These findings are further visualized in Figure 2, where dot-and-whisker plot estimates confirm the direction and magnitude of differences, highlighting triglycerides, blood pressure, and glucose as the most prominent discriminating factors between the two phenotypes (see details in Table 2 and Figure 2).

Table 2. Metabolic and Cardiometabolic Differences Between Sarcopenic Obesity and Non-Obese Sarcopenia.

Parameter	Type of Sarcopenia		Mean Rank	*p*-value
	Obesity	Non-Obesity		
Age	59 (22 – 84)	38 (18 – 96)	430.71 vs 282.21	<0.001
Systolic Blood pressure	130 (100 - 210)	120 (81 – 192)	398.34 vs 288.08	<0.001

Diastolic Blood pressure	80 (60 - 105)	77 (60 – 115)	351.63 vs 295.11	0.007
Triglycerides	159 (62 – 524)	131 (45 – 496)	375.44 vs 292.52	<0.001
Low Density Lipoprotein	117 (43 – 180)	111.4 (40 – 233)	323.13 vs 299.40	0.260
High Density Lipoprotein	48 (21 – 88.4)	55 (19 – 85.8)	244.72 vs 311.19	0.002
Total Cholesterol	197 (111 – 333)	195 (96 – 320)	309.69 vs 301.42	0.694
Fasting Blood Glucose	101 (62 – 319)	89 (61 – 313)	397.72 vs 288.17	<0.001
Uric Acid	4.5 (2.9 – 10.3)	4.5 (2.7 – 11.3)	326.09 vs 298.95	0.197
Hemoglobin	12.5 (7.4 – 16.3)	12.9 (5.4 – 18.8)	292.49 vs 304.01	0.584
Handgrip	20 (7.9 – 46)	21.7 (7.1 – 59.3)	270.52 vs 307.31	0.081
Gender			2.035	0.005
• Male	42 (18,3%)	188 (81.7%)	(1.225 – 2.782)	
• Female	37 (9.9%)	337 (90.1%)		

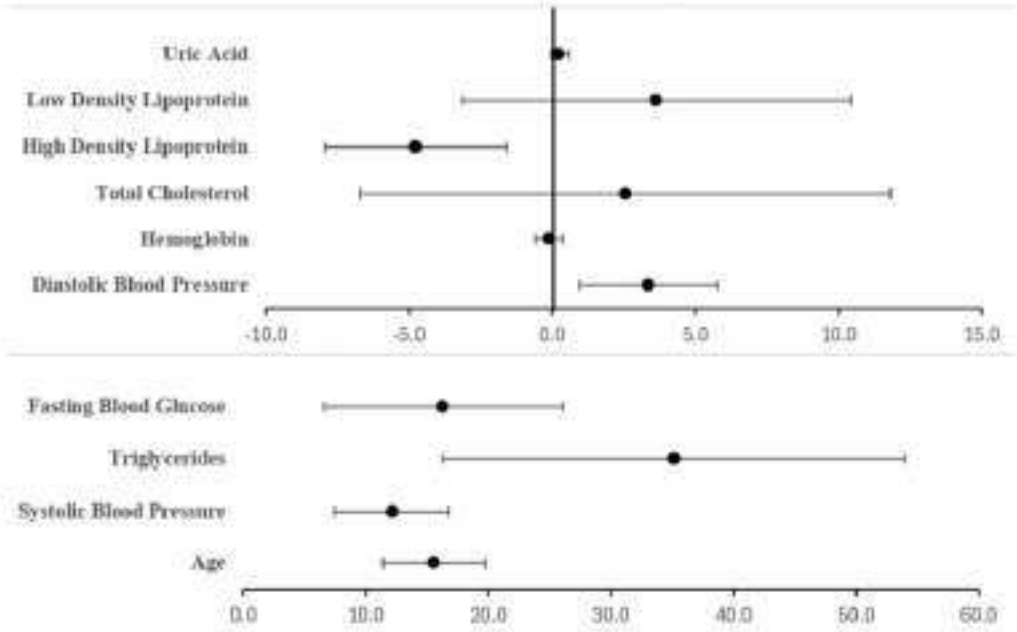

Figure 2. Dot-and-Whisker Plot of Metabolic and Cardiometabolic Differences Between Sarcopenic Obesity and Non-Obese Sarcopenia.

4. Discussion

In this study, the sarcopenic obesity group exhibited higher blood pressure compared to the non-obese sarcopenia group, both in systolic values (130 vs 120 mmHg; mean rank 398.34 vs 288.08; $p < 0.001$) and diastolic values (80 vs 77 mmHg; mean rank 351.63 vs 295.11; $p = 0.007$). These findings are consistent with the study by Han et al., which reported the highest prevalence of hypertension in sarcopenic obesity at 74.7%, compared to 60.9% in non-obese sarcopenia, with an increased risk up to OR 3.0 (95% CI 2.48–3.63).(32) A meta-analysis by T. Bai et al. also reported that sarcopenia is associated with an increased risk of hypertension, with a pooled OR of 1.29 (95% CI 1.00–1.67), and an even stronger association in Asian populations (OR 1.50; 95% CI 1.35–1.67).(33) Furthermore, an NHANES-based study demonstrated that improved muscle quality was associated with a reduction in hypertension prevalence by up to 7% and a decrease in systolic blood pressure by 1.88 mmHg (95% CI −3.56 to −0.20),(34) while Luo et al. identified an inverse relationship between relative muscle strength and hypertension (OR 0.68 in males

and 0.81 in females).(35) Therefore, part of the observed differences may reflect demographic differences between groups rather than obesity status alone. Furthermore, because the analyses were based on unadjusted bivariate comparisons, the independent contribution of obesity could not be determined.

A prospective study by Park et al., involving 25,103 hypertension-free participants, also showed that increased muscle strength was associated with lower blood pressure and reduced risk of hypertension, particularly in women.(36) However, some studies have demonstrated more complex relationships, such as Ji et al., who reported a positive association between handgrip strength and blood pressure in overweight/obese men,(37) and Zhang et al., who observed a protective effect in older women.(38) From a pathophysiological perspective, this condition reflects the interaction between reduced muscle mass and increased adiposity, which promotes insulin resistance, chronic inflammation, adipokine–myokine dysregulation, as well as increased sympathetic activity and vascular stiffness.(39) A recent study by Dlamini et al. further demonstrated that the interaction between muscle and adipose tissue is mediated by various biological proteins, where LEP, FABP4, IL6, and GGH are positively associated with both visceral adipose tissue and muscle mass, while ACAN, CELA3A, PLA2G1B, and NCAM1 show negative associations, and specific proteins such as NOTCH3, COL1A1, and DKK3 are more closely related to adipose tissue, whereas IGFBP1 is associated with muscle mass, illustrating the complex molecular regulation underlying the relationship between body composition and blood pressure.(40)

The sarcopenic obesity group in this study exhibited higher triglyceride levels and lower HDL compared to the non-obese sarcopenia group (TG: 159 vs 131 mg/dL; mean rank 375.44 vs 292.52; $p < 0.001$; HDL: 48 vs 55 mg/dL; mean rank 244.72 vs 311.19; $p = 0.002$), while total cholesterol and LDL did not differ significantly. These findings indicate a more pronounced lipid metabolism dysfunction in sarcopenic obesity, particularly characterized by hypertriglyceridemia and reduced HDL, reflecting an atherogenic profile.(41–43) This is consistent with the study by Huang et al., which demonstrated that elevated triglyceride levels are significantly associated with an increased risk of sarcopenia (p for trend = 0.001) in a non-linear dose–response pattern, and are also linked to reduced muscle mass.(44) Furthermore, Yin et al. reported that lipid ratios such as TG/HDL and LDL/HDL are strongly associated with sarcopenia, with ORs of 1.02 (95% CI 1.02–1.04) and 1.27 (95% CI 1.11–1.45), respectively, emphasizing the importance of lipid interactions in determining risk.(45) Li et al. also found that an increased LDL/HDL ratio is associated with up to a threefold higher risk of sarcopenia (OR 3.01; 95% CI 2.66–3.41).(46) On the other hand, the relationship between HDL and sarcopenia appears to be complex; a longitudinal study by Wang et al. showed that higher HDL levels were associated with a 42% increased risk of sarcopenia (OR 1.42; 95% CI 1.28–1.58),(47) while Huh et al. similarly reported increased risk in the highest HDL quartile (OR 1.36; 95% CI 1.05–1.75).(48) From a pathophysiological perspective, these findings reflect disrupted lipid metabolism driven by insulin resistance, chronic inflammation, and alterations in adipokine–myokine signaling, which affect lipolysis, fatty acid oxidation, and lipid distribution. Elevated triglycerides contribute to lipotoxicity in skeletal muscle, while HDL dysfunction may impair mitochondrial function and muscle energy metabolism.(49) In addition, chronic inflammation accelerates muscle protein degradation and worsens metabolic dysfunction, creating a vicious cycle that promotes the progression of sarcopenic obesity.(4,50)

Fasting blood glucose levels were higher in the sarcopenic obesity group compared to the non-obese sarcopenia group (101 vs 89 mg/dL; mean rank 397.72 vs 288.17; $p < 0.001$). These findings indicate that glycemic dysfunction is more pronounced in sarcopenic obesity, reflecting a greater degree of insulin resistance compared to non-obese

sarcopenia.(51–53) This is consistent with a meta-analysis showing that individuals with diabetes have a higher risk of sarcopenia (OR 1.518; 95% CI 1.110–2.[illegible]),(54) as well as longitudinal evidence demonstrating that elevated fasting glucose is associated with an increased risk of sarcopenia (OR [illegible].52; 95% CI 1.17–1.96).(55) Additionally, studies in diabetic populations have shown that poor glycemic control is significantly associated with sarcopenia (62.3% vs 47.9%; $p = 0.007$), with an increased risk up to OR 1.79 (95% CI 1.17–2.75).(51) Other studies have also demonstrated that glucose fluctuations and elevated mean glucose are independent risk factors for sarcopenia and are associated with reductions in muscle mass and strength.(56,57) Furthermore, the triglyceride–glucose (TyG) index, as a marker of insulin resistance, has been shown to be strongly associated with sarcopenic obesity, with individuals in the highest tertile having a 1.82-fold higher risk compared to those in the lowest tertile(53), and up to 3.369-fold in males and 3.157-fold in females in other analyses(52), emphasizing the central role of insulin resistance in this phenotype(58,59) From a pathophysiological perspective, this condition reflects a bidirectional relationship between hyperglycemia and sarcopenia, in which insulin resistance, chronic inflammation, oxidative stress, and the accumulation of advanced glycation end-products (AGEs) impair muscle protein synthesis, mitochondrial function, and tissue perfusion. Conversely, reduced muscle mass decreases peripheral glucose uptake, thereby exacerbating hyperglycemia and reinforcing the metabolic cycle underlying sarcopenic obesity.(58,60–62)

Sarcopenic obesity represents a phenotype with a more complex cardiometabolic burden, characterized by elevated blood pressure, atherogenic dyslipidemia (high triglycerides and low HDL), and more severe glucose disturbances, which interact within the spectrum of metabolic syndrome.(16,63–65)] This is consistent with multiple studies showing that sarcopenic obesity, or related phenotypes such as dynapenic obesity, has a higher prevalence of metabolic syndrome compared to non-sarcopenic or non-obese groups, as reported by Sénéchal et al. (183/566 vs 469/1397)(66), Chung et al. (449/666 vs 192/337)(67), and Kim et al. (53/110 vs 73/169)(68), all of which consistently demonstrate a greater proportion of metabolic disturbances in individuals with combined muscle loss and obesity. Other studies, including those by Baek et al., Park et al., and Kang et al., further show that the majority of individuals with sarcopenic obesity meet the criteria for metabolic syndrome (up to >60–70%), reflecting the clustering of risk factors such as hypertension, hyperglycemia, and dyslipidemia within a single clinical entity.(65,69,70)

In this study, no significant differences were observed between sarcopenic obesity and non-obese sarcopenia in hemoglobin levels ($p = 0.584$) or uric acid levels ($p = 0.197$), which may be attributed to the homogeneity of the study population, as all participants already met the AWGS 2025 criteria for sarcopenia, resulting in a more restricted biological variation. Most existing literature has demonstrated an association between hemoglobin and sarcopenia,(71–84) where lower hemoglobin levels are linked to an increased risk of sarcopenia (OR 0.95; 95% CI 0.90–0.99)(71) as well as reduced muscle mass and function, while anemia has been associated with a higher risk (OR 2.4; 95% CI 1.2–4.9).(72) In contrast, the relationship between uric acid and sarcopenia appears inconsistent, with some studies reporting a protective effect of higher uric acid levels (HR 0.57–0.72 in higher quartiles)(85), whereas others have shown positive or non-significant associations after adjusting for confounders.(85–87) The lack of significant findings in this study may suggest that hemoglobin and uric acid function more as general systemic markers or modulators of muscle status, but are not sufficiently sensitive to distinguish obesity-based sarcopenia phenotypes, particularly when compared to metabolic parameters such as blood pressure, lipid profile, and glucose, which more directly reflect metabolic dysfunction.(85,87,88)

This study found that the sarcopenic obesity group was older than the non-obese sarcopenia group (59 vs 38 years; $p < 0.001$) and had a significantly different sex

distribution, with a higher proportion of males (OR 2.035; $p = 0.005$). These findings are consistent with the majority of the literature identifying age as a primary determinant of sarcopenia, with prevalence increasing markedly after the age of 50 years and reaching up to 16.7% in individuals aged 80–89 years(12), as well as demonstrating sex-related differences, with prevalence ranging from 0.8–22.3% in females and 1.3–15.4% in males.(11) This pattern is closely related to age-associated changes in body composition, characterized by a decline in muscle mass and an increase in fat mass, which are not fully captured by indices such as BMI, thereby contributing to the "obesity paradox" and the U-shaped relationship between BMI and mortality.(4,8,89) The combination of older age and sex differences is clinically important, as sarcopenic obesity has been associated with a higher risk of cardiovascular disease, reduced bone mineral density, and increased mortality compared to either condition alone, with large cohort studies such as NHANES III and the UK Biobank consistently demonstrating a significantly elevated mortality risk in this population.(14,17,18)

Furthermore, routine assessment of muscle strength, body composition, blood pressure, and metabolic parameters should be considered, particularly in older adults and individuals with obesity or other cardiometabolic risk factors. Early identification of sarcopenic obesity provides an opportunity to implement targeted interventions, including resistance exercise, adequate protein intake, weight management, and optimization of blood pressure, lipid profile, and glycemic control. Integrating these measures into primary care and community-based health programs may help slow disease progression, preserve physical function, and reduce the long-term burden of metabolic syndrome, cardiovascular disease, disability, and premature mortality.

This study has several important strengths. It utilizes a large, multi-district urban dataset from Jakarta, allowing for a more comprehensive and representative assessment of sarcopenia phenotypes in a real-world metropolitan population. The use of updated diagnostic frameworks, including AWGS 2025 and the AOASO–IAGG 2025 consensus for sarcopenic obesity, enhances the clinical relevance and contemporary validity of the findings.(27–29) Additionally, the study adopts an integrated metabolic approach by simultaneously evaluating multiple cardiometabolic parameters, enabling a more holistic understanding of the "two faces of sarcopenia".

However, several limitations should also be acknowledged. First, the cross-sectional design precludes causal inference and does not permit evaluation of temporal relationships. Second, the analyses were based primarily on unadjusted bivariate comparisons without multivariable adjustment for important confounding variables, particularly age and sex, which differed significantly between groups and may have influenced the observed metabolic differences. Participants with sarcopenic obesity were older and more frequently male than those with non-obese sarcopenia. Rather than representing primary findings, these differences should be considered important potential confounding factors because both age and sex are independently associated with blood pressure, glucose metabolism, lipid profile, and body composition. Consequently, part of the observed metabolic differences between groups may reflect demographic differences rather than obesity status alone.

Furthermore, the secondary database did not include several potentially important confounders, including age, sex, dietary intake, physical activity, smoking status, alcohol consumption, medication use, and socioeconomic factors. Consequently, the observed differences should be interpreted as associations rather than independent effects of obesity. Finally, because the study population consisted exclusively of individuals with sarcopenia from an urban population in Jakarta, the findings may not be generalizable to rural populations or other ethnic groups with different demographic and lifestyle characteristics. Future studies incorporating multivariable analyses are needed to

determine whether these associations remain after adjustment for age, sex, and other potential confounders.

5. Conclusions

This study demonstrates that the two phenotypes of sarcopenia (non-obese sarcopenia and sarcopenic obesity) exhibit distinct metabolic and cardiometabolic profiles in an urban Jakarta population. Compared with individuals with non-obese sarcopenia, those with sarcopenic obesity were older, more frequently male, and exhibited higher blood pressure, fasting blood glucose, and triglyceride levels, along with lower HDL concentrations. In contrast, hemoglobin, total cholesterol, LDL, uric acid, and handgrip strength did not differ significantly between groups. These findings suggest that sarcopenic obesity is associated with a less favorable cardiometabolic profile; however, the observed differences may also be influenced by potential confounding factors, including age, medication use, physical activity, dietary habits, smoking status, and other lifestyle-related factors that were not available in the present dataset. Therefore, these results should be interpreted as associative rather than causal. Future studies incorporating multivariable analyses and longitudinal designs are warranted to determine the independent contribution of obesity to metabolic dysfunction among individuals with sarcopenia.

Author Contributions: **AHS** and **YF** conceptualized and designed the study. **YF** supervised the overall research process and provided critical revisions to the manuscript. **TS**, **AM**, **AP**, **BAW**, **DD**, and **MFB** contributed to data acquisition, data cleaning, and field coordination. **EE**, **SMDN**, **ATE** and **HH** contributed to methodological development, data interpretation, and validation of findings. **AHS** and **BAW** performed the statistical analysis and drafted the initial manuscript. All authors contributed to manuscript writing, critically reviewed the content, and approved the final version for publication.

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Informed Consent Statement: All data were fully anonymized prior to analysis, ensuring that no individual participant could be identified. The authors confirm that the dissemination of aggregated findings poses no risk to participants and is intended solely for scientific and academic purposes.

Data Availability Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Declaration of Use of AI in Academic Writing: The authors declare that artificial intelligence tools were used solely for minor grammatical and language refinement (similar to grammar-checking tools such as Grammarly). All aspects of the manuscript, including study design, data analysis, interpretation, and writing, were conducted and critically reviewed manually by the authors.

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Abstract

Introduction: Sarcopenia and obesity are increasingly recognized as overlapping conditions that contribute to adverse health outcomes, particularly in rapidly urbanizing populations. The coexistence of both conditions, termed sarcopenic obesity, represents a complex phenotype with potentially greater metabolic burden, yet its differentiation from non-obese sarcopenia remains insufficiently explored. Aim: This study aimed to compare metabolic and cardiometabolic profiles between sarcopenic obesity and non-obese sarcopenia in an urban population. Methods: A cross-sectional study was conducted using secondary data from the PRIMBON 2025 database in Jakarta, Indonesia. Participants were adults aged ≥18 years diagnosed with sarcopenia based on the 2025 Asian Working Group for Sarcopenia (AWGS) criteria. Sarcopenic obesity was defined according to the 2025 AOASO–IAGG consensus using BMI ≥ 25 kg/m^2 and visceral fat level > 10. A total of approximately 604 participants were included from five administrative regions of Jakarta. Data were analyzed using Mann–Whitney U test and Chi-square test, with results presented as median (min–max), mean rank, and odds risk (OR). A dot-and-whisker plot was used to visualize effect sizes. Results: Sarcopenic obesity was associated with older age and a higher proportion of males ($p < 0.01$). Significant differences were observed in systolic and diastolic blood pressure, fasting blood glucose, triglycerides, and HDL (all $p < 0.01$), indicating a more adverse cardiometabolic profile. No significant differences were found for hemoglobin, total cholesterol, LDL, uric acid, and handgrip strength. Conclusions: Sarcopenic obesity represents a more severe metabolic phenotype compared to non-obese sarcopenia.

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1. Introduction

Sarcopenia is a progressive and generalized loss of skeletal muscle mass and strength associated with adverse outcomes such as falls, fractures, frailty, and mortality. Although commonly associated with aging, muscle decline begins as early as the fourth decade of life, with an annual loss of 1–2% after the age of 50 years [1]. Globally, the prevalence of sarcopenia varies widely due to heterogeneity in diagnostic criteria, ranging from 10% to 27% based on a meta-analysis of 151 studies involving more than 692,000 individuals [2]. This variability highlights both the magnitude of the problem and the ongoing challenge in establishing standardized definitions and measurements of sarcopenia [3].

In parallel, obesity has reached pandemic proportions, with its prevalence more than doubling worldwide since 1980 and currently affecting approximately 30% of the global population [4]. Obesity significantly increases the risk of chronic diseases such as type 2 diabetes, cardiovascular disease, cancer, dementia, and depression, and contributes to increased healthcare costs by up to 30% compared to individuals with normal body mass index [5]. However, the "obesity paradox" suggests a more complex relationship between body mass index and mortality, particularly in older populations, where both low and high BMI are associated with increased mortality risk, forming a U-shaped curve [6–8]. This paradox indicates that BMI alone may inadequately capture body composition and cardiometabolic risk, as individuals with sarcopenia may present with markedly different adiposity despite having similar degrees of muscle loss. Consequently, sarcopenia may manifest as two distinct phenotypes, non-obese sarcopenia and sarcopenic obesity, which are hypothesized to exhibit different metabolic and cardiometabolic profiles [9,10].

The coexistence of sarcopenia and obesity, termed sarcopenic obesity, represents a more severe and clinically relevant phenotype due to its compounded adverse effects. The prevalence of sarcopenic obesity varies widely depending on diagnostic criteria, ranging from 0.8% to 22.3% in women and 1.3% to 15.4% in men, with increasing prevalence at older ages [11,12]. A global meta-analysis estimated its prevalence at approximately 11% among older adults [13]. Importantly, sarcopenic obesity has been associated with significantly higher risks of cardiovascular disease, reduced bone mineral density, and all-cause mortality compared to either condition alone [14–16]. Large cohort studies, including NHANES III and UK Biobank, consistently demonstrate that individuals with sarcopenic obesity have a markedly increased mortality risk, emphasizing its clinical and public health importance [17,18]. In addition to these clinical outcomes, sarcopenic obesity is characterized by metabolic and hematologic alterations that may reflect its underlying pathophysiology. Cardiometabolic biomarkers, including blood pressure, fasting blood glucose, lipid profile, serum uric acid, and hemoglobin, provide important insights into metabolic homeostasis, systemic inflammation, oxidative stress, and nutritional status [19,20].

The development of sarcopenia and sarcopenic obesity is multifactorial, involving complex interactions between metabolic, hormonal, inflammatory, and lifestyle-related factors. Both conditions are strongly associated with metabolic and cardiovascular disorders, including diabetes, dyslipidemia [21], metabolic syndrome [22], restrictive lung disease [23], osteoporosis [24]. Mechanistically, chronic low-grade inflammation, insulin resistance, mitochondrial dysfunction, and alterations in lipid and glucose metabolism contribute to muscle degradation and fat accumulation, creating a vicious cycle that exacerbates disease progression [4,25,26].

Recent advances in diagnostic criteria, particularly the 2025 Asian Working Group for Sarcopenia (AWGS) consensus, have strengthened the conceptual framework of sarcopenia through a life-course approach, simplified diagnostic algorithms, and recognition of skeletal muscle as a central organ interacting with multiple physiological systems [27,28]. In parallel, the Asia–Oceania Association for the Study of Obesity (AOASO) and the International Association of Gerontology and Geriatrics Asia/Oceania Region (IAGG-AOR) consensus has established sarcopenic obesity as a distinct clinical entity requiring standardized diagnostic criteria and targeted management strategies [29]. Within this updated framework, the concept of the "Two Faces of Sarcopenia" emerges, distinguishing between non-obese sarcopenia and sarcopenic obesity, which may exhibit fundamentally different metabolic and cardiometabolic profiles.

In addition to conventional cardiometabolic biomarkers such as glycemic status, lipid profile, and blood pressure, hemoglobin and serum uric acid may provide complementary insights into the biological mechanisms underlying these phenotypes. Hemoglobin reflects oxygen-carrying capacity and tissue oxygenation, which are essential for skeletal muscle metabolism and function, whereas serum uric acid is closely linked to oxidative stress, chronic inflammation, insulin resistance, and metabolic syndrome [30,31]. Therefore, this study aims to explore how various metabolic factors (including glycemic status, lipid profile, hemoglobin, uric acid, and blood pressure) are differentially associated with these two phenotypes, providing deeper insight into the heterogeneity of sarcopenia and its clinical implications in a large urban population in Jakarta, Indonesia, where rapid urbanization and lifestyle transitions may further shape these metabolic patterns.

2. Material and Methods

2.1. Study Design and Setting

This study employed a cross-sectional design using secondary data obtained from the PRIMBON (Partnership for Research, Innovation, and Multisectoral Benefit for Outreach and Networking) database, developed by Universitas Tarumanagara since June 2024. The dataset utilized in this study corresponds to the 2025 data collection period. Data were derived from five administrative regions of Jakarta (West, North, South, Central, and East Jakarta), with three representative districts selected from each region to ensure adequate geographic representation of the urban population.

2.2. Population, Sample, and Sampling Technique

The study population consisted of adult individuals aged ≥18 years residing in Jakarta who were identified as having sarcopenia based on the 2025 AWGS criteria and recorded in the PRIMBON database [27,28]. The minimum sample size was calculated using the formula:

$$n = \frac{Z^2\, p(1-p)}{d^2}$$

Assuming a confidence level of 95% (Z = 1.96), estimated prevalence (p = 0.5), and precision (d = 0.05), the minimum required sample size was 384 participants. To enhance statistical power and account for potential data exclusion, a total sample of approximately 500 participants was targeted. A clustered sampling approach was applied across the selected districts to ensure proportional representation.

2.3. Inclusion and Exclusion Criteria

Participants were included if they were adults aged ≥18 years residing in Jakarta, identified as having sarcopenia based on the AWGS 2025 criteria [27,28], registered in the PRIMBON 2025 database, and had complete data on anthropometric, muscle, and

metabolic parameters. Because the primary objective of this study was to compare the metabolic and cardiometabolic characteristics of the two clinically recognized sarcopenia phenotypes (non-obese sarcopenia and sarcopenic obesity), only participants meeting the diagnostic criteria for sarcopenia were included. Accordingly, healthy individuals and obese participants without sarcopenia were not included, as comparisons with these groups were beyond the scope of the present study. Information regarding medical history used for the exclusion criteria was obtained from a standardized self-reported health questionnaire completed by participants during the PRIMBON 2025 health screening. Participants were excluded if they had incomplete or missing data, resided outside Jakarta, or self-reported conditions that could compromise the validity of muscle strength, body composition, or metabolic measurements, including disorders affecting handgrip strength (e.g., upper limb injury or neuromuscular diseases such as myasthenia gravis), conditions interfering with body composition assessment (e.g., metallic implants or prosthetic devices), secondary hypertension or other conditions influencing blood pressure measurements, hematological disorders affecting hemoglobin levels (e.g., polycythemia vera or aplastic anemia), hereditary or severe lipid metabolism disorders, type 1 diabetes mellitus, or other clinical conditions known to significantly alter metabolic biomarkers such as fasting blood glucose, lipid profile, and uric acid.

2.4. Data Collection Procedures

Data were extracted from the PRIMBON 2025 database following a standardized protocol. The dataset included demographic characteristics, anthropometric measurements, muscle function (handgrip strength), body composition assessed by bioelectrical impedance analysis (BIA) (skeletal muscle index and visceral fat level), and cardiometabolic parameters (blood pressure, hemoglobin, lipid profile, fasting blood glucose, and uric acid). Data cleaning and validation were performed prior to analysis, including screening for completeness, plausibility, and consistency across variables. Only eligible participants meeting the predefined criteria were included in the final analysis.

2.5. Operational Definitions and Classification of Sarcopenia and Sarcopenic Obesity

Sarcopenia was defined based on the AWGS 2025 criteria using appendicular skeletal muscle mass index (ASM/height^2) and handgrip strength. Low skeletal muscle mass was defined as <7.6 kg/m^2 for men and <5.7 kg/m^2 for women aged <65 years, and <7.0 kg/m^2 for men and <5.7 kg/m^2 for women aged ≥65 years. Low handgrip strength was defined as <34 kg for men and <20 kg for women aged <65 years, and <28 kg for men and <18 kg for women aged ≥65 years [27,28]. Sarcopenic obesity was defined according to the 2025 consensus of the AOASO and the IAGG-AOR, whereby participants fulfilled the AWGS criteria for sarcopenia in addition to obesity defined as body mass index (BMI) ≥25 kg/m^2 and visceral fat level >10 as an alternative to waist circumference [29]. Based on these criteria, participants in this study were categorized into two groups: non-obese sarcopenia and sarcopenic obesity. These definitions were adapted to align with the measurement specifications of the devices used in the standardized PRIMBON 2025 data collection.

2.6. Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics version 27.0 (IBM Corp., Released 2020. IBM SPSS Statistics for Windows, Version 27.0, Armonk, NY, USA: IBM Corp.) and Microsoft Excel 365 (Microsoft Corporation, Redmond, WA, USA). Descriptive (univariate) analysis was conducted to summarize the characteristics of the study population. Numerical variables were presented as median (minimum–maximum) due

to non-normal distribution, while categorical variables were expressed as frequencies and percentages.

Bivariate analysis was performed to compare differences between sarcopenic obesity and non-obese sarcopenia groups. The Mann–Whitney U test was used for continuous variables, with results interpreted based on mean rank differences, while categorical variables were analyzed using the Chi-square test (with Yates' correction when appropriate). Effect estimates for categorical variables were reported as Odds Ratio (OR).

To visually represent the magnitude and direction of differences between groups, a dot-and-whisker plot was constructed, illustrating effect estimates and their corresponding confidence intervals. Statistical significance was defined as a two-tailed p-value < 0.05, with an alpha level of 5% and a statistical power of 80%.

2.7. Ethical Approval

This study was approved by the Universitas Tarumanagara Human Research Ethics Committee, Institute of Research and Community Engagement, under ethical approval number 015-UTHREC/UNTAR/IV/2026. The study utilized secondary data from the PRIMBON database, and all data were anonymized prior to analysis to ensure participant confidentiality. As no direct interaction with participants was conducted, informed consent was waived by the ethics committee. All procedures were carried out in accordance with the ethical principles outlined in the Declaration of Helsinki.

3. Results

A total of 604 participants met the inclusion criteria and were included in the analysis (Figure 1). The median age was 41.5 years, with a predominance of females (61.9%). Most participants were employed (66.3%) and married (70.9%). Sarcopenic obesity was identified in 13.1% of participants. Median and Interquartiles cardiometabolic parameters, including blood pressure, lipid profile, glucose, hemoglobin, and uric acid, are presented in Table 1.

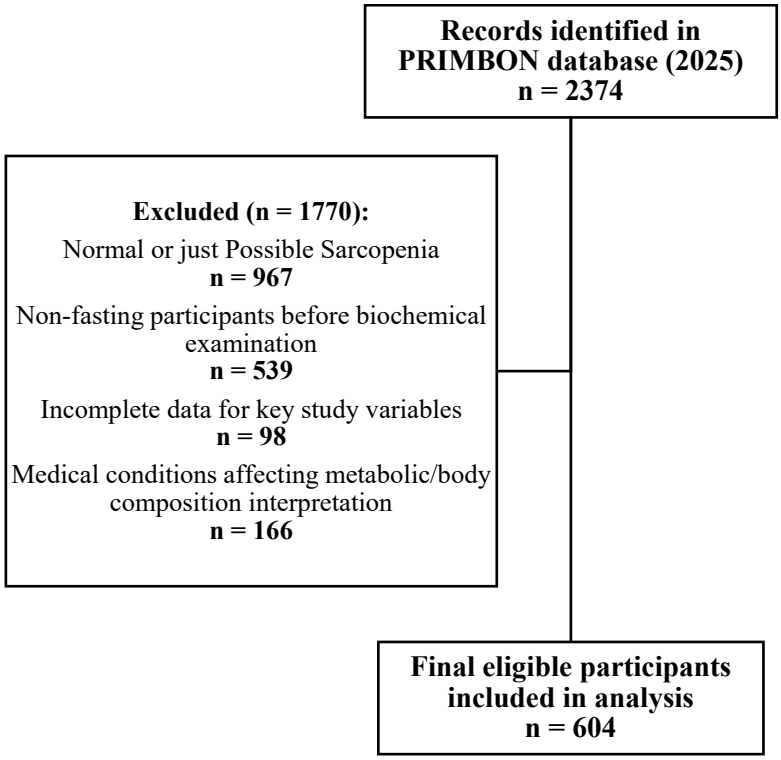

Figure 1. Flow diagram of participant selection.

Table 1. Baseline Demographic, Anthropometric, Muscle, and Cardiometabolic Profiles of the Study Population.

Parameter	Sarcopenic Obesity		Sarcopenia	
	N (%)	Med (IQR)	N (%)	Med (IQR)
Age, years	79 (100)	59 (45–70)	525 (100)	38 (26–54.5)
Gender				
• Male	42 (53.2)		188 (35.8)	
• Female	37 (46.8)		337 (64.2)	
Occupation				
• Employee	27 (34.2)		313 (59.6)	
• Self-employed	4 (5.1)		22 (4.2)	
• Unemployed	48 (60.8)		190 (36.2)	
Marital status				
• Married	76 (96.2)		352 (67)	
• Unmarried	3 (3.8)		173 (33)	
Body weight, kg	79 (100)	65 (60–70)	525 (100)	52.7 (48–59)
Body height, cm	79 (100)	154.7 (148.7–160.3)	525 (100)	157.8 (152.1–164)
Body mass index, kg/m^2	79 (100)	26.4 (25.5–28.1)	525 (100)	21.5 (19.41–23.25)
Handgrip strength, kg	79 (100)	20 (16–26.6)	525 (100)	21.7 (17.6–26.7)
Skeletal muscle index, kg/m^2	79 (100)	5.6 (5.4–7.3)	525 (100)	5.4 (4.9–6.2)
Visceral Fat Level	79 (100)	12 (11–15)	525 (100)	5 (3–7)
Systolic Blood Pressure, mmHg	79 (100)	130 (120–144)	525 (100)	120 (110–129)
Diastolic Blood Pressure, mmHg	79 (100)	80 (72–86)	525 (100)	77 (70–84)
Triglycerides, mg/dL	79 (100)	159 (124–203)	525 (100)	131 (99–168.5)
Low-Density Lipoprotein, mg/dL	79 (100)	117 (95–137)	525 (100)	111.4 (93.7–130)
High-Density Lipoprotein, mg/dL	79 (100)	48 (40–60)	525 (100)	55 (45.1–64.5)
Total Cholesterol, mg/dL	79 (100)	197 (176–232)	525 (100)	195 (177–219)
Fasting blood Glucose, mg/dL	79 (100)	101 (87–122)	525 (100)	89 (82–100)
Hemoglobin, g/dL	79 (100)	12.5 (11.2–14.5)	525 (100)	12.9 (11.2–14.2)
Uric Acid, mg/dL	79 (100)	4.5 (3.9–5.5)	525 (100)	4.5 (3.8–5.2)

Table 2 and Figure 2 demonstrate significant metabolic and cardiometabolic differences between sarcopenic obesity and non-obese sarcopenia based on Mann–Whitney U test analysis. Individuals with sarcopenic obesity showed significantly higher mean ranks for age, systolic and diastolic blood pressure, triglycerides, and fasting blood glucose (all $p < 0.01$), indicating a more adverse cardiometabolic profile. In contrast, HDL levels were significantly lower in the obese group ($p = 0.002$), while no significant differences were observed for hemoglobin, total cholesterol, LDL, uric acid, and handgrip strength. Sex distribution also differed significantly, with a higher proportion of males in the sarcopenic obesity group (OR 2.035, $p = 0.005$). These findings are further visualized in Figure 2, where dot-and-whisker plot estimates confirm the direction and magnitude of differences, highlighting triglycerides, blood pressure, and glucose as the most prominent discriminating factors between the two phenotypes (see details in Table 2 and Figure 2).

Table 2. Metabolic and Cardiometabolic Differences Between Sarcopenic Obesity and Non-Obese Sarcopenia.

Parameter	Type of Sarcopenia		Mean Rank	*p*-Value
	Obesity	Non-Obesity		
Age	59 (22–84)	38 (18–96)	430.71 vs. 282.21	<0.001
Systolic Blood pressure	130 (100–210)	120 (81–192)	398.34 vs. 288.08	<0.001
Diastolic Blood pressure	80 (60–105)	77 (60–115)	351.63 vs. 295.11	0.007
Triglycerides	159 (62–524)	131 (45–496)	375.44 vs. 292.52	<0.001
Low-Density Lipoprotein	117 (43–180)	111.4 (40–233)	323.13 vs. 299.40	0.260
High-Density Lipoprotein	48 (21–88.4)	55 (19–85.8)	244.72 vs. 311.19	0.002
Total Cholesterol	197 (111–333)	195 (96–320)	309.69 vs. 301.42	0.694

Table 2. *Cont.*

Parameter	Type of Sarcopenia		Mean Rank	*p*-Value
	Obesity	Non-Obesity		
Fasting Blood Glucose	101 (62–319)	89 (61–313)	397.72 vs. 288.17	<0.001
Uric Acid	4.5 (2.9–10.3)	4.5 (2.7–11.3)	326.09 vs. 298.95	0.197
Hemoglobin	12.5 (7.4–16.3)	12.9 (5.4–18.8)	292.49 vs. 304.01	0.584
Handgrip	20 (7.9–46)	21.7 (7.1–59.3)	270.52 vs. 307.31	0.081
Gender				
• Male	42 (18.3%)	188 (81.7%)	2.035 (1.225–2.782)	0.005
• Female	37 (9.9%)	337 (90.1%)		

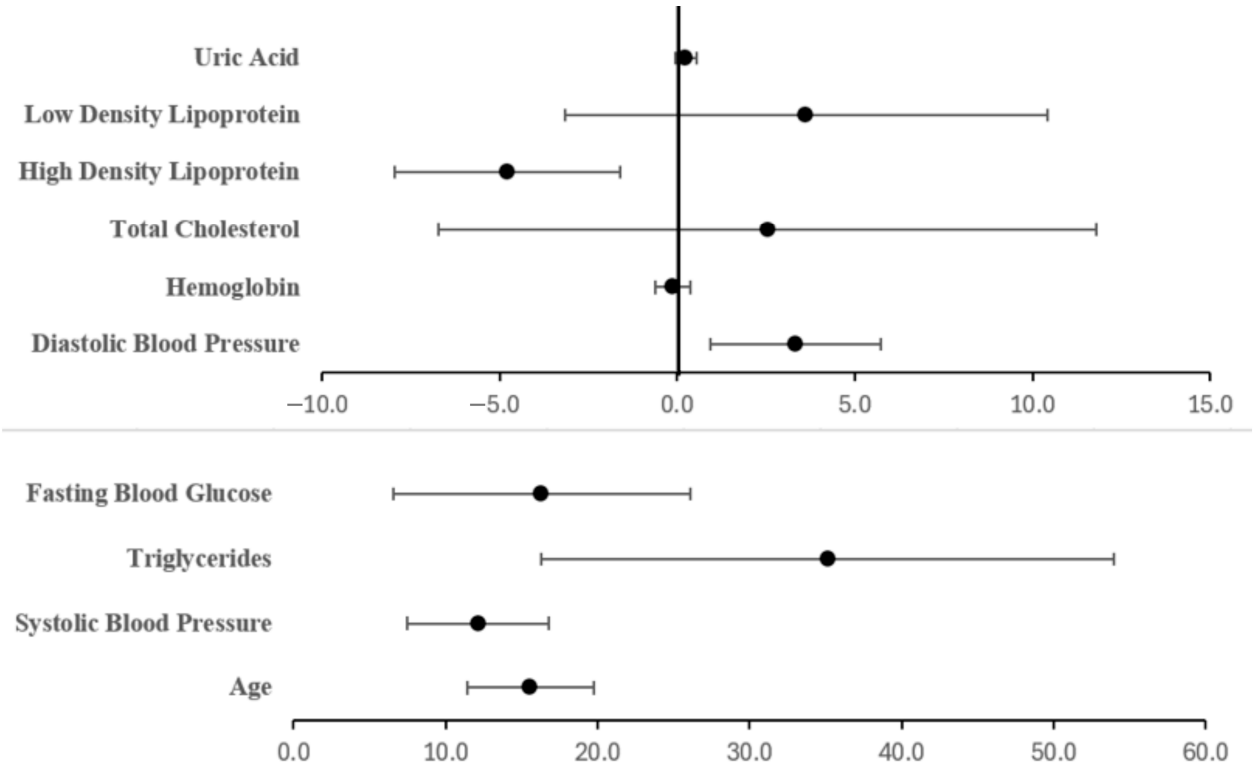

Figure 2. Dot-and-Whisker Plot of Metabolic and Cardiometabolic Differences Between Sarcopenic Obesity and Non-Obese Sarcopenia.

4. Discussion

In this study, the sarcopenic obesity group exhibited higher blood pressure compared to the non-obese sarcopenia group, both in systolic values (130 vs. 120 mmHg; mean rank 398.34 vs. 288.08; $p < 0.001$) and diastolic values (80 vs. 77 mmHg; mean rank 351.63 vs. 295.11; $p = 0.007$). These findings are consistent with the study by Han et al., which reported the highest prevalence of hypertension in sarcopenic obesity at 74.7%, compared to 60.9% in non-obese sarcopenia, with an increased risk up to OR 3.0 (95% CI 2.48–3.63) [32]. A meta-analysis by T. Bai et al. also reported that sarcopenia is associated with an increased risk of hypertension, with a pooled OR of 1.29 (95% CI 1.00–1.67), and an even stronger association in Asian populations (OR 1.50; 95% CI 1.35–1.67) [33]. Furthermore, an NHANES-based study demonstrated that improved muscle quality was associated with a reduction in hypertension prevalence by up to 7% and a decrease in systolic blood pressure by 1.88 mmHg (95% CI −3.56 to −0.20) [34], while Luo et al. identified an inverse relationship between relative muscle strength and hypertension (OR 0.68 in males and 0.81 in females) [35]. Therefore, part of the observed differences

may reflect demographic differences between groups rather than obesity status alone. Furthermore, because the analyses were based on unadjusted bivariate comparisons, the independent contribution of obesity could not be determined.

A prospective study by Park et al., involving 25,103 hypertension-free participants, also showed that increased muscle strength was associated with lower blood pressure and reduced risk of hypertension, particularly in women [36]. However, some studies have demonstrated more complex relationships, such as Ji et al., who reported a positive association between handgrip strength and blood pressure in overweight/obese men [37], and Zhang et al., who observed a protective effect in older women [38]. From a pathophysiological perspective, this condition reflects the interaction between reduced muscle mass and increased adiposity, which promotes insulin resistance, chronic inflammation, adipokine–myokine dysregulation, as well as increased sympathetic activity and vascular stiffness [39]. A recent study by Dlamini et al. further demonstrated that the interaction between muscle and adipose tissue is mediated by various biological proteins, where LEP, FABP4, IL6, and GGH are positively associated with both visceral adipose tissue and muscle mass, while ACAN, CELA3A, PLA2G1B, and NCAM1 show negative associations, and specific proteins such as NOTCH3, COL1A1, and DKK3 are more closely related to adipose tissue, whereas IGFBP1 is associated with muscle mass, illustrating the complex molecular regulation underlying the relationship between body composition and blood pressure [40].

The sarcopenic obesity group in this study exhibited higher triglyceride levels and lower HDL compared to the non-obese sarcopenia group (TG: 159 vs. 131 mg/dL; mean rank 375.44 vs. 292.52; $p < 0.001$; HDL: 48 vs. 55 mg/dL; mean rank 244.72 vs. 311.19; $p = 0.002$), while total cholesterol and LDL did not differ significantly. These findings indicate a more pronounced lipid metabolism dysfunction in sarcopenic obesity, particularly characterized by hypertriglyceridemia and reduced HDL, reflecting an atherogenic profile [41–43]. This is consistent with the study by Huang et al., which demonstrated that elevated triglyceride levels are significantly associated with an increased risk of sarcopenia (p for trend = 0.001) in a non-linear dose–response pattern, and are also linked to reduced muscle mass [44]. Furthermore, Yin et al. reported that lipid ratios such as TG/HDL and LDL/HDL are strongly associated with sarcopenia, with ORs of 1.02 (95% CI 1.02–1.04) and 1.27 (95% CI 1.11–1.45), respectively, emphasizing the importance of lipid interactions in determining risk [45]. Lu et al. also found that an increased LDL/HDL ratio is associated with up to a threefold higher risk of sarcopenia (OR 3.01; 95% CI 2.66–3.41) [46]. On the other hand, the relationship between HDL and sarcopenia appears to be complex; a longitudinal study by Wang et al. showed that higher HDL levels were associated with a 42% increased risk of sarcopenia (OR 1.42; 95% CI 1.28–1.58) [47], while Huh et al. similarly reported increased risk in the highest HDL quartile (OR 1.36; 95% CI 1.05–1.75) [48]. From a pathophysiological perspective, these findings reflect disrupted lipid metabolism driven by insulin resistance, chronic inflammation, and alterations in adipokine–myokine signaling, which affect lipolysis, fatty acid oxidation, and lipid distribution. Elevated triglycerides contribute to lipotoxicity in skeletal muscle, while HDL dysfunction may impair mitochondrial function and muscle energy metabolism [49]. In addition, chronic inflammation accelerates muscle protein degradation and worsens metabolic dysfunction, creating a vicious cycle that promotes the progression of sarcopenic obesity [4,50].

Fasting blood glucose levels were higher in the sarcopenic obesity group compared to the non-obese sarcopenia group (101 vs. 89 mg/dL; mean rank 397.72 vs. 288.17; $p < 0.001$). These findings indicate that glycemic dysfunction is more pronounced in sarcopenic obesity, reflecting a greater degree of insulin resistance compared to non-obese sarcopenia [51–53]. This is consistent with a meta-analysis showing that individuals with diabetes have a

higher risk of sarcopenia (OR 1.518; 95% CI 1.110–2.076) [54], as well as longitudinal evidence demonstrating that elevated fasting glucose is associated with an increased risk of sarcopenia (OR 1.52; 95% CI 1.17–1.96) [55]. Additionally, studies in diabetic populations have shown that poor glycemic control is significantly associated with sarcopenia (62.3% vs. 47.9%; $p = 0.007$), with an increased risk up to OR 1.79 (95% CI 1.17–2.75) [51]. Other studies have also demonstrated that glucose fluctuations and elevated mean glucose are independent risk factors for sarcopenia and are associated with reductions in muscle mass and strength [56,57]. Furthermore, the triglyceride–glucose (TyG) index, as a marker of insulin resistance, has been shown to be strongly associated with sarcopenic obesity, with individuals in the highest tertile having a 1.82-fold higher risk compared to those in the lowest tertile [53], and up to 3.369-fold in males and 3.157-fold in females in other analyses [52], emphasizing the central role of insulin resistance in this phenotype [58,59]. From a pathophysiological perspective, this condition reflects a bidirectional relationship between hyperglycemia and sarcopenia, in which insulin resistance, chronic inflammation, oxidative stress, and the accumulation of advanced glycation end-products (AGEs) impair muscle protein synthesis, mitochondrial function, and tissue perfusion. Conversely, reduced muscle mass decreases peripheral glucose uptake, thereby exacerbating hyperglycemia and reinforcing the metabolic cycle underlying sarcopenic obesity [58,60–62].

Sarcopenic obesity represents a phenotype with a more complex cardiometabolic burden, characterized by elevated blood pressure, atherogenic dyslipidemia (high triglycerides and low HDL), and more severe glucose disturbances, which interact within the spectrum of metabolic syndrome [16,63–65]. This is consistent with multiple studies showing that sarcopenic obesity, or related phenotypes such as dynapenic obesity, has a higher prevalence of metabolic syndrome compared to non-sarcopenic or non-obese groups, as reported by Sénéchal et al. (183/566 vs. 469/1397) [66], Chung et al. (449/666 vs. 192/337) [67], and Kim et al. (53/110 vs. 73/169) [68], all of which consistently demonstrate a greater proportion of metabolic disturbances in individuals with combined muscle loss and obesity. Other studies, including those by Baek et al., Park et al., and Kang et al., further show that the majority of individuals with sarcopenic obesity meet the criteria for metabolic syndrome (up to >60–70%), reflecting the clustering of risk factors such as hypertension, hyperglycemia, and dyslipidemia within a single clinical entity [65,69,70].

In this study, no significant differences were observed between sarcopenic obesity and non-obese sarcopenia in hemoglobin levels ($p = 0.584$) or uric acid levels ($p = 0.197$), which may be attributed to the homogeneity of the study population, as all participants already met the AWGS 2025 criteria for sarcopenia, resulting in a more restricted biological variation. Most existing literature has demonstrated an association between hemoglobin and sarcopenia [71–84], where lower hemoglobin levels are linked to an increased risk of sarcopenia (OR 0.95; 95% CI 0.90–0.99) [71] as well as reduced muscle mass and function, while anemia has been associated with a higher risk (OR 2.4; 95% CI 1.2–4.9) [72]. In contrast, the relationship between uric acid and sarcopenia appears inconsistent, with some studies reporting a protective effect of higher uric acid levels (HR 0.57–0.72 in higher quartiles) [85], whereas others have shown positive or non-significant associations after adjusting for confounders [85–87]. The lack of significant findings in this study may suggest that hemoglobin and uric acid function more as general systemic markers or modulators of muscle status, but are not sufficiently sensitive to distinguish obesity-based sarcopenia phenotypes, particularly when compared to metabolic parameters such as blood pressure, lipid profile, and glucose, which more directly reflect metabolic dysfunction [85,87,88].

This study found that the sarcopenic obesity group was older than the non-obese sarcopenia group (59 vs. 38 years; $p < 0.001$) and had a significantly different sex distribution, with a higher proportion of males (OR 2.035; $p = 0.005$). These findings are consistent with

the majority of the literature identifying age as a primary determinant of sarcopenia, with prevalence increasing markedly after the age of 50 years and reaching up to 16.7% in individuals aged 80–89 years [12], as well as demonstrating sex-related differences, with prevalence ranging from 0.8–22.3% in females and 1.3–15.4% in males [11]. This pattern is closely related to age-associated changes in body composition, characterized by a decline in muscle mass and an increase in fat mass, which are not fully captured by indices such as BMI, thereby contributing to the "obesity paradox" and the U-shaped relationship between BMI and mortality [4,8,89]. The combination of older age and sex differences is clinically important, as sarcopenic obesity has been associated with a higher risk of cardiovascular disease, reduced bone mineral density, and increased mortality compared to either condition alone, with large cohort studies such as NHANES III and the UK Biobank consistently demonstrating a significantly elevated mortality risk in this population [14,17,18].

Furthermore, routine assessment of muscle strength, body composition, blood pressure, and metabolic parameters should be considered, particularly in older adults and individuals with obesity or other cardiometabolic risk factors. Early identification of sarcopenic obesity provides an opportunity to implement targeted interventions, including resistance exercise, adequate protein intake, weight management, and optimization of blood pressure, lipid profile, and glycemic control. Integrating these measures into primary care and community-based health programs may help slow disease progression, preserve physical function, and reduce the long-term burden of metabolic syndrome, cardiovascular disease, disability, and premature mortality.

This study has several important strengths. It utilizes a large, multi-district urban dataset from Jakarta, allowing for a more comprehensive and representative assessment of sarcopenia phenotypes in a real-world metropolitan population. The use of updated diagnostic frameworks, including AWGS 2025 and the AOASO–IAGG 2025 consensus for sarcopenic obesity, enhances the clinical relevance and contemporary validity of the findings [27–29]. Additionally, the study adopts an integrated metabolic approach by simultaneously evaluating multiple cardiometabolic parameters, enabling a more holistic understanding of the "two faces of sarcopenia".

However, several limitations should also be acknowledged. First, the cross-sectional design precludes causal inference and does not permit evaluation of temporal relationships. Second, the analyses were based primarily on unadjusted bivariate comparisons without multivariable adjustment for important confounding variables, particularly age and sex, which differed significantly between groups and may have influenced the observed metabolic differences. Participants with sarcopenic obesity were older and more frequently male than those with non-obese sarcopenia. Rather than representing primary findings, these differences should be considered important potential confounding factors because both age and sex are independently associated with blood pressure, glucose metabolism, lipid profile, and body composition. Consequently, part of the observed metabolic differences between groups may reflect demographic differences rather than obesity status alone.

Furthermore, the secondary database did not include several potentially important confounders, including age, sex, dietary intake, physical activity, smoking status, alcohol consumption, medication use, and socioeconomic factors. Consequently, the observed differences should be interpreted as associations rather than independent effects of obesity. Finally, because the study population consisted exclusively of individuals with sarcopenia from an urban population in Jakarta, the findings may not be generalizable to rural populations or other ethnic groups with different demographic and lifestyle characteristics. Future studies incorporating multivariable analyses are needed to determine whether these associations remain after adjustment for age, sex, and other potential confounders.

5. Conclusions

This study demonstrates that the two phenotypes of sarcopenia (non-obese sarcopenia and sarcopenic obesity) exhibit distinct metabolic and cardiometabolic profiles in an urban Jakarta population. Compared with individuals with non-obese sarcopenia, those with sarcopenic obesity were older, more frequently male, and exhibited higher blood pressure, fasting blood glucose, and triglyceride levels, along with lower HDL concentrations. In contrast, hemoglobin, total cholesterol, LDL, uric acid, and handgrip strength did not differ significantly between groups. These findings suggest that sarcopenic obesity is associated with a less favorable cardiometabolic profile; however, the observed differences may also be influenced by potential confounding factors, including age, medication use, physical activity, dietary habits, smoking status, and other lifestyle-related factors that were not available in the present dataset. Therefore, these results should be interpreted as associative rather than causal. Future studies incorporating multivariable analyses and longitudinal designs are warranted to determine the independent contribution of obesity to metabolic dysfunction among individuals with sarcopenia.

Author Contributions: A.H.S. and Y.F. conceptualized and designed the study. Y.F. supervised the overall research process and provided critical revisions to the manuscript. T.S., A.M., A.P., B.A.W., D.D. and M.F.D. contributed to data acquisition, data cleaning, and field coordination. E., S.M.D.N., A.T.E. and H.H. contributed to methodological development, data interpretation, and validation of findings. A.H.S. and B.A.W. performed the statistical analysis and drafted the initial manuscript. All authors have read and agreed to the published version of the manuscript.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki, and approved by the Institutional Review Board (or Ethics Committee) of Universitas Tarumanagara Human Research Ethics Committee Institute of Reserch and Community Engagement (UTHRECT) (016-UTHRECT/UNTAR/IV/2026, 1 April 2026).

Informed Consent Statement: All data were fully anonymized prior to analysis, ensuring that no individual participant could be identified. The authors confirm that the dissemination of aggregated findings poses no risk to participants and is intended solely for scientific and academic purposes.

Data Availability Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

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Conflicts of Interest: The authors declare no conflicts of interest.

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