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OBESITY AND STROKE BURDEN IN HUNGARY: EPIDEMIOLOGICAL, CARDIOMETABOLIC AND HEALTH-ECONOMIC PERSPECTIVES

PhD thesis

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Table of contents

1. Introduction 6

1.1 Definition and classification of stroke 6

1.2 Epidemiology of stroke 7

1.3 Cardiometabolic risk factors of stroke 8

1.4 Obesity as an independent and mediating risk factor 8

1.5 Limitations of BMI in risk assessment 10

1.6 Bioimpedance analysis, body composition and metabolic risk 11

1.7 Predictive performance and screening implications 13

1.8 Economic burden of stroke 15

2. Objectives 17

3. Methods 18

3.1 Study design and population 18

3.2 Data collection and measurements 19

3.3 Definition of variables 20

3.3.1 Exposure variables 20

3.3.2 Outcome variables 21

3.3.3 Conceptual linkage of stroke risk 21

3.4 Statistical analysis 21

3.4.1 Descriptive analysis 21

3.4.2 Logistic regression modelling 22

3.4.3 ROC analysis and diagnostic performance 22

3.4.4 Inflection point analysis (LOESS regression) 22

3.4.5 Integration of analytical approaches 23

3.4.6 Bootstrap procedures 23

3.5 Economic modelling 23

3.5.1 Conceptual framework 23
3.5.2 Population-attributable fraction (PAF) 23
3.5.3 Economic burden estimation 24
3.5.4 Monte Carlo simulation 24
3.5.5 Sensitivity analyses and robustness 24
3.6 Model parameters and implementation details 24
3.7 Bias control and methodological considerations 25
3.8 Ethical considerations 26
4. Results 27
4.1 National screening cohort used for prevalence and economic modelling 27
4.2 Weighted BMI distribution and estimated prevalence of excess body mass 28
4.3 Population-attributable fraction of stroke and projected economic burden 29
4.4 Body-composition cohort with complete cardiometabolic phenotyping 30
4.5 Demographic composition of the analytical cohorts 32
4.6 Overall predictive performance of BMI and InBody-derived parameters 32
4.7 BMI inflection points and risk-escalation zones identified by LOESS analysis .. 35
4.8 Stratified ROC analyses across BMI categories 37
4.9 Age-restricted sensitivity analysis in participants aged ≥45 years 38
4.10 Non-linear relationships between BMI and body-composition parameters across the adiposity spectrum 40
4.11 Integrated interpretation of complementary analytical approaches 42
4.12 Modelled implementation scenario of selective body-composition assessment. 43
5. Discussion 46
5.1 Main findings 46
5.2 The Hungarian obesity burden 47
5.3 Hungary-specific economic interpretation 48

5.4 Cardiometabolic risk as the mechanistic link 50
5.5 Risk identification in a high-burden population 51
5.5.1 BMI as a first-line screening tool: historical context and current utility 51
5.5.2 Biological limitations of BMI and the role of body-composition assessment 51
5.5.3 Sex-specific patterns of early cardiometabolic risk emergence 52
5.5.4 Implications for future risk-identification strategies 53
5.6 Policy and clinical implications 53
5.6.1 Implications for clinical thresholds and intervention strategies 53
5.6.2 Implications for population-level policy and resource allocation 54
5.7 Limitations 55
6. Conclusions 58
7. Summary 60
8. References 61
9. Bibliography of the candidate's publications 78
9.1 Original publications related to the dissertation 78
9.2 Other publications 78
10. Acknowledgements 85

List of Abbreviations

ABI	Ankle–brachial index
ADA	American Diabetes Association
AF	Atrial fibrillation
AUC	Area under the curve
BIA	Bioelectrical impedance analysis
BMI	Body mass index
CHARLS	China Health and Retirement Longitudinal Study
CI	Confidence interval
CT	Computed tomography
DXA	Dual-energy X-ray absorptiometry
EAS	European Atherosclerosis Society
EHIS	European Health Interview Survey
ESC	European Society of Cardiology
ESC/ESH	European Society of Cardiology / European Society of Hypertension
EUR	Euro
EUROSTAT	Statistical Office of the European Union
HUF	Hungarian forint
INTERSTROKE	International Study of Risk Factors for Stroke
IQR	Interquartile range
KSH	Hungarian Central Statistical Office
LDL	Low-density lipoprotein
LOESS	Locally estimated scatterplot smoothing
MRI	Magnetic resonance imaging
PAF	Population-attributable fraction
ROC	Receiver operating characteristic
RR	Relative risk
VLDL	Very-low-density lipoprotein
WHO	World Health Organization
yrs	Years

1. Introduction

1.1 Definition and classification of stroke

Stroke represents one of the most significant challenges in modern medicine and public health, due to its high incidence, substantial mortality, and long-term consequences for affected individuals (1-3). It was historically defined by the World Health Organization as a rapidly developing clinical syndrome characterized by focal or global disturbance of cerebral function, lasting for more than 24 hours or leading to death, with no apparent cause other than vascular origin (4, 5). However, this classic clinical definition is now considered outdated, because it relies solely on the 24-hour symptom duration and does not account for modern neuroimaging findings. Current definitions instead use a tissue-based approach, where stroke is defined as brain infarction demonstrated by neuroimaging (CT or MRI), even if symptoms resolve earlier or if clinical signs are transient (4, 6).

From a pathophysiological perspective, stroke can be divided into two major categories: ischemic and hemorrhagic stroke (2, 7, 8). Ischemic stroke accounts for approximately 85–90% of all cases and is caused by an interruption of cerebral blood flow, most commonly due to thrombotic or embolic occlusion of cerebral arteries (2, 9, 10). Thrombotic strokes typically occur in the context of atherosclerotic disease, where plaque formation leads to vessel narrowing and eventual occlusion (11-15). Embolic strokes, on the other hand, are often associated with cardiac sources, such as atrial fibrillation, where emboli travel through the bloodstream and obstruct cerebral vessels (4, 12, 16).

Hemorrhagic stroke, although less frequent, is associated with higher mortality rates (1, 2). It results from the rupture of intracranial blood vessels, leading to bleeding within the brain tissue (intracerebral hemorrhage) or into the subarachnoid space (subarachnoid hemorrhage) (2, 4, 17). The underlying causes of hemorrhagic stroke often include chronic hypertension, vascular malformations, and aneurysm rupture (9, 12, 18).

The distinction between ischemic and hemorrhagic stroke is clinically crucial, as it determines both acute management and long-term preventive strategies (2, 4, 19). However, despite differences in etiology, both subtypes share common underlying vascular risk factors, emphasizing the importance of addressing these factors in primary prevention (9, 11, 20, 21).

1.2 Epidemiology of stroke

Stroke is a leading cause of death and disability worldwide, imposing a substantial burden on individuals, healthcare systems, and societies (1-3). According to global estimates, approximately 12 million new stroke cases occur annually, and more than 6 million deaths are attributed to stroke each year (1, 2, 22). In addition to mortality, stroke is a major contributor to long-term disability, significantly impacting quality of life and healthcare resource utilization (2, 3, 23).

Over recent decades, the epidemiology of stroke has undergone important changes (1, 24). While age-standardized incidence and mortality rates have declined in many high-income countries due to improved prevention and treatment strategies, the absolute number of stroke cases continues to rise (1, 2, 25). This increase is largely driven by demographic changes, particularly population aging, as well as the growing prevalence of modifiable risk factors such as obesity, diabetes, and sedentary lifestyle (1, 9, 26).

In Europe, stroke remains a major public health issue, with considerable heterogeneity across regions (27, 28). Western European countries have generally experienced improvements in stroke outcomes, reflecting advances in acute care and risk factor management (27, 29). In contrast, Central and Eastern European countries continue to exhibit higher incidence and mortality rates, highlighting persistent health inequalities (1, 27).

Hungary is among the countries with a particularly high burden of stroke (1, 10, 30, 31). Epidemiological data indicate elevated rates of both incidence and mortality compared to the European average (27, 30, 32). This can be attributed to a combination of factors, including a high prevalence of cardiometabolic risk factors, unfavorable lifestyle patterns, and socioeconomic determinants (33-35). Furthermore, disparities in access to preventive and healthcare services may contribute to the observed differences (33, 36, 37).

These findings underscore the urgent need for effective primary prevention strategies tailored to the Hungarian population (31, 38-40).

1.3 Cardiometabolic risk factors of stroke

The development of stroke is strongly influenced by a range of modifiable cardiometabolic risk factors (2, 9, 11). Among these, hypertension is widely recognized as the most significant contributor, with a well-established causal relationship with both ischemic and hemorrhagic stroke (2, 9, 41). Elevated blood pressure leads to vascular damage, endothelial dysfunction, and increased risk of both atherosclerosis and vessel rupture (11, 42-44).

Diabetes mellitus represents another major risk factor, contributing to stroke through mechanisms such as accelerated atherosclerosis, microvascular damage, and increased thrombogenicity (9, 42, 45, 46). Similarly, dyslipidaemia, particularly elevated low-density lipoprotein (LDL) cholesterol levels, plays a key role in the development of atherosclerotic plaques (9, 42, 47).

Atrial fibrillation is an important cardiac risk factor for stroke, significantly increasing the risk of embolic events (4, 12, 48, 49). The irregular heart rhythm associated with atrial fibrillation promotes thrombus formation in the atria, which can subsequently embolize to cerebral vessels (4, 12, 16).

Importantly, these risk factors frequently coexist within individuals, forming a cluster of metabolic abnormalities often referred to as metabolic syndrome (50, 51). The presence of multiple risk factors has a synergistic effect, leading to a substantially increased risk of stroke compared to individual factors alone (9, 52, 53). This clustering highlights the importance of comprehensive risk assessment and integrated preventive approaches (54, 55).

1.4 Obesity as an independent and mediating risk factor

Obesity has emerged as one of the most important public health challenges of the 21st century and plays a central role in the development of cardiometabolic diseases (50, 54, 56-58). It is increasingly recognized not only as a risk factor in itself but also as a mediator that contributes to the development of other risk factors, including hypertension, diabetes, and dyslipidaemia (11, 56, 59, 60).

The relationship between obesity and stroke is complex and involves multiple pathophysiological mechanisms (2, 54, 61). A key element of this relationship is the

qualitative nature of adipose tissue, particularly the distinction between visceral and subcutaneous fat compartments (51, 62).

Visceral adipose tissue, located within the abdominal cavity and surrounding internal organs, is metabolically highly active and exerts a disproportionate effect on cardiometabolic risk (51, 52, 63). Unlike subcutaneous fat, visceral fat is characterized by increased lipolytic activity and releases free fatty acids directly into the portal circulation (51, 62, 64, 65). This promotes hepatic insulin resistance, increased very-low-density lipoprotein (VLDL) production, and dysregulation of glucose metabolism (42, 51, 64). In parallel, visceral fat acts as an endocrine organ, secreting pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha, as well as adipokines that disrupt metabolic homeostasis (52, 56, 66).

This chronic low-grade inflammatory state contributes to endothelial dysfunction, characterized by reduced nitric oxide bioavailability, increased oxidative stress, and impaired vascular reactivity (52, 54, 67). In addition, obesity is associated with activation of prothrombotic pathways, including increased fibrinogen levels, platelet activation, and reduced fibrinolysis, which together create a thrombogenic environment conducive to ischemic events (52, 68, 69).

Subcutaneous adipose tissue, particularly in peripheral regions, is generally considered less harmful and may even exert partially protective effects by acting as a metabolic buffer for excess energy storage (51, 70, 71). However, when the storage capacity of subcutaneous fat is exceeded, the resulting lipid overflow into visceral depots and ectopic sites, such as the liver and muscle, further accelerates metabolic dysfunction (51, 62, 72).

Obesity is also closely linked to insulin resistance, which represents a central mechanism in the development of cardiometabolic disease (42, 56, 73). Insulin resistance contributes to hyperglycaemia, dyslipidaemia, and vascular inflammation, thereby increasing the risk of both macrovascular and microvascular complications (42, 54, 74). These processes collectively contribute to the development of both ischemic and, to a lesser extent, hemorrhagic stroke (9, 54, 75).

Importantly, obesity does not act in isolation but amplifies the effect of other risk factors (54, 56, 76). It increases the likelihood of developing hypertension through mechanisms such as sympathetic nervous system activation, renin–angiotensin–aldosterone system dysregulation, and sodium retention (11, 56). It also predisposes to type 2 diabetes and

atherogenic lipid profiles, thereby acting as a central node in the network of stroke risk factors (42, 56).

The global prevalence of obesity has increased dramatically in recent decades, reaching epidemic levels (77-79). Hungary is among the most affected countries in Europe, with prevalence rates exceeding 70% when overweight and obesity are combined (31, 35, 80). This high prevalence represents not only a clinical challenge but also a population-level shift in baseline vascular risk (54, 56, 81). In such a context, obesity functions less as an individual anomaly and more as a structural determinant of disease patterns, underscoring the need for effective and early screening strategies (37, 39, 79, 82).

1.5 Limitations of BMI in risk assessment

Body mass index (BMI) is the most widely used measure for assessing overweight and obesity in both clinical and epidemiological settings (83-85). It is calculated as weight in kilograms divided by the square of height in meters and provides a simple and standardized method for classifying individuals into weight categories (78, 86).

Importantly, BMI was not originally developed as a medical or diagnostic tool. The concept was introduced in the early 19th century by Adolphe Quetelet, who aimed to describe the "average man" (l'homme moyen) from a statistical perspective (86-88). Its primary purpose was population-level characterization rather than individual health assessment (86, 89, 90). The clinical application of BMI emerged much later, particularly in the mid-20th century, when it was adopted in epidemiological studies and subsequently validated as a predictor of mortality (87, 91). However, these early validations were largely based on cohorts of middle-aged, predominantly white Western men, and focused primarily on all-cause mortality rather than morbidity or cardiometabolic risk profiles (89, 92). This historical context is important, as it highlights that BMI thresholds were not originally designed to detect early metabolic disease across diverse populations (84, 86).

Despite its widespread use, BMI has several important limitations (83, 86, 93). One of the primary limitations is its inability to distinguish between fat mass and lean body mass (83, 84, 94). As a result, individuals with high muscle mass may be misclassified as overweight or obese, while individuals with normal BMI but elevated body fat percentage, often referred to as "normal-weight obesity", may be incorrectly considered low risk (70, 83, 95).

Another key limitation of BMI is its inability to capture fat distribution (83, 84, 96). Central adiposity, particularly visceral fat accumulation, is known to be more strongly associated with cardiometabolic risk than overall body weight alone (51, 52). However, BMI provides no information regarding fat distribution, limiting its ability to reflect important differences in metabolic risk between individuals with similar BMI values (83, 84).

Furthermore, BMI does not adequately account for individual variability in body composition related to age, sex, and ethnicity (83, 97). For example, women generally have a higher body fat percentage than men at the same BMI, while aging is associated with sarcopenia and increased fat mass despite stable body weight (70, 97). Ethnic differences are also well documented (98, 99). In several Asian populations, cardiometabolic risk has been shown to occur at lower BMI values compared to European populations, leading the World Health Organization to propose lower BMI cut-off points (e.g., 23 kg/m² for overweight and 27.5 kg/m² for obesity in certain Asian groups) (86, 99). These findings suggest that the conventional BMI thresholds of 25 and 30 kg/m² may not be universally applicable.

Taken together, these limitations indicate that while BMI remains a practical and valuable tool for large-scale screening, it provides an incomplete representation of individual metabolic risk, particularly in populations with high prevalence of overweight and obesity.

1.6 Bioimpedance analysis, body composition and metabolic risk

Given the growing recognition of the importance of fat distribution and body composition in cardiometabolic disease, methods capable of assessing these parameters have gained increasing attention in both clinical and epidemiological research (83, 100, 101). Among these, bioelectrical impedance analysis (BIA) represents one of the most widely used and practically applicable techniques in both clinical and population-based settings (100, 102, 103).

BIA is based on the principle that different biological tissues exhibit distinct electrical conductive properties (100, 102, 103). Lean tissue, which contains a high proportion of water and electrolytes, conducts electrical current efficiently, whereas adipose tissue, with lower water content, presents higher resistance (100, 104). During measurement, a low-intensity alternating electrical current is passed through the body, and the resulting

impedance is recorded. This impedance consists of resistance, reflecting total body water, and reactance, which is related to cell membrane integrity and capacitance. These parameters are then used in validated prediction models to estimate body composition components (100, 105).

Modern BIA systems, including multi-frequency and segmental devices, have significantly improved the resolution of these measurements (100, 106). By applying multiple frequencies, it becomes possible to differentiate between intracellular and extracellular water compartments, while segmental analysis allows independent assessment of the trunk and limbs (100, 104). This is particularly relevant because central fat accumulation is more strongly associated with metabolic risk than peripheral fat distribution (51, 62, 107).

Devices such as the InBody system implement segmental multi-frequency BIA technology and provide a detailed body composition profile within a short measurement time (106, 108). These systems estimate multiple parameters that are directly relevant to cardiometabolic risk assessment (83, 102). Body fat percentage reflects total adiposity and allows identification of individuals with excess fat accumulation even in the absence of elevated BMI (70, 83, 109). Visceral fat estimates, although indirect, aim to capture central adiposity, which is closely linked to insulin resistance, systemic inflammation, and vascular risk (51, 52, 110). Parameters such as trunk fat ratio further characterize fat distribution and provide insight into central versus peripheral fat dominance (51, 62).

In addition to adiposity-related measures, BIA devices also quantify skeletal muscle mass, which is an important determinant of metabolic health (70, 83, 111). Muscle tissue plays a central role in glucose uptake and insulin sensitivity, and reduced muscle mass, particularly when combined with increased fat mass, may indicate sarcopenic obesity, a phenotype associated with elevated cardiometabolic risk (70, 112-114).

From a practical perspective, BIA offers several advantages. It is non-invasive, rapid, relatively inexpensive, and easy to implement in large-scale screening programmes (83, 100, 115). These features make it particularly suitable for real-world applications where more precise but resource-intensive methods, such as dual-energy X-ray absorptiometry, are not feasible (100, 116, 117).

However, BIA measurements are subject to several limitations (100, 102, 108). Results are influenced by hydration status, recent food intake, and physical activity, all of which

can affect impedance and thereby alter estimated body composition (83, 100, 118, 119). Standardization of measurement conditions is therefore essential. The presence of implanted electronic devices may contraindicate measurement, and metallic implants can introduce measurement variability (100, 119). Furthermore, different devices use proprietary algorithms, which may lead to systematic differences between systems, limiting comparability across devices (83, 100). Finally, as an indirect method, BIA provides estimates rather than direct measurements, and its accuracy may be reduced in individuals with extreme body compositions (83, 100, 108).

Despite these limitations, BIA represents a practical and physiologically informative method for body composition assessment in both clinical and population-based settings (83, 100, 120). Its ability to estimate parameters related to fat distribution and skeletal muscle mass has contributed to increasing interest in its potential role in cardiometabolic risk assessment (83, 84, 121-123).

1.7 Predictive performance and screening implications

The question of whether body composition parameters provide superior predictive performance compared to BMI remains an area of active debate in the literature (83, 84, 124). While BMI has long been established as a simple and scalable screening tool, its limitations have prompted investigations into whether more physiologically relevant measures, such as body fat percentage, visceral adiposity, and muscle mass, can improve risk prediction (84, 97, 116, 117, 125).

Several studies have demonstrated that body composition parameters may offer improved prediction of cardiometabolic outcomes in specific populations (51, 122, 126). Measures reflecting central adiposity have been reported to show stronger associations with several cardiometabolic outcomes than BMI alone in selected populations (51, 62, 127). Similarly, body fat percentage has been suggested to identify individuals at increased risk despite having a normal BMI, thereby capturing phenotypes that would otherwise remain undetected (70, 122).

However, the evidence is not uniform. In many studies, the overall improvement in predictive performance offered by body composition parameters over BMI has been modest at the population level (83, 97, 128). While statistically significant differences are sometimes observed, these do not always translate into clinically meaningful gains in discrimination (83, 84, 129). This has led to ongoing debate regarding whether the added

complexity and cost of body composition assessment are justified in routine screening (84, 100, 130).

Importantly, predictive performance appears to vary across populations (83, 97). Ethnic differences play a significant role in this context (98, 99). For example, in Asian populations, increased cardiometabolic risk has been observed at lower BMI values, and body composition measures may provide earlier detection of metabolic abnormalities (99, 131, 132). In contrast, in European populations, the incremental value of such parameters over BMI has often been less pronounced in whole-cohort analyses (27, 133, 134). These findings suggest that the utility of body composition is context-dependent and may be influenced by underlying population characteristics, including baseline adiposity, fat distribution patterns, and genetic factors (99, 135, 136).

Another important aspect is the heterogeneity within BMI categories (133, 135, 137). Individuals with similar BMI values may exhibit markedly different metabolic profiles depending on fat distribution and muscle mass (70, 83, 138). In such cases, body composition analysis may provide additional discriminatory value, particularly in intermediate BMI ranges where risk classification based on BMI alone is less reliable (83, 84).

From a screening perspective, these observations raise a key question: should body composition assessment replace BMI, or should it be used selectively? Current evidence suggests that a universal replacement is unlikely to be efficient or necessary (83, 100). Instead, a more targeted approach may be warranted, in which BMI serves as a first-line screening tool, and body composition parameters are applied in selected subgroups where additional information is most likely to influence clinical decision-making (37, 83, 135). This approach aligns with the broader goal of improving risk stratification without compromising feasibility (83, 100, 139). In large-scale or resource-limited settings, maintaining simplicity is essential, while in higher-risk or ambiguous cases, more detailed phenotyping may be justified (37, 83, 140).

Taken together, the existing literature suggests that body composition analysis does not universally outperform BMI, but it has the potential to enhance risk prediction in specific contexts (57, 83, 84). Identifying these contexts represents a key challenge and provides the rationale for the present investigation.

1.8 Economic burden of stroke

Stroke represents one of the leading causes of mortality and long-term disability worldwide and imposes a substantial economic burden on healthcare systems (1, 2). This burden encompasses both direct and indirect costs (3, 68). Direct costs include acute hospitalization, diagnostic procedures, medical treatment, rehabilitation, and long-term institutional or home-based care (3, 141). Indirect costs arise from productivity loss, long-term disability, informal caregiving, and premature mortality (3, 29).

In Hungary, the economic impact of stroke is particularly pronounced (3, 30). The annual cost has been estimated at approximately €1.016 billion, reflecting not only the high incidence of stroke but also the long-term consequences of disability and recurrent events (30, 31). This places stroke among the most economically burdensome diseases in the Hungarian healthcare system and highlights its relevance as a public health priority (30, 37).

A defining feature of stroke epidemiology is that a large proportion of cases are attributable to modifiable risk factors (2, 9). Conditions such as hypertension, diabetes mellitus, dyslipidaemia, and obesity play a central role in the development of cerebrovascular disease (9, 54, 142). Among these, excess body weight is of particular importance, not only as an independent risk factor but also as a driver of multiple downstream metabolic and vascular abnormalities (31, 56, 143).

From a public health perspective, this creates a critical opportunity for prevention (144-146). However, preventive strategies must be evaluated not only in terms of clinical effectiveness but also in terms of economic efficiency (56, 147). In resource-constrained healthcare systems, including Hungary, interventions must compete for limited funding, making it essential to prioritize those that offer the greatest potential health benefit relative to their cost (147-151).

In this context, understanding the proportion of disease burden attributable to specific risk factors becomes highly relevant (1, 31). Estimating the contribution of overweight and obesity to stroke burden allows for a more precise assessment of the potential impact of preventive strategies targeting body weight and metabolic health (31, 56). Such analyses provide a bridge between epidemiological evidence and health policy decision-making, supporting the allocation of resources toward interventions with the highest expected return in terms of disease reduction and cost savings (144).

Taken together, these observations highlight important challenges in current obesity-related risk assessment and stroke prevention strategies. In populations characterized by high prevalence of excess body weight, effective risk stratification requires approaches that are both physiologically meaningful and feasible at the population level (56, 83, 84). At the same time, the substantial clinical and economic burden of stroke further emphasizes the importance of improving early identification of cardiometabolic risk (2, 31). These considerations provide the rationale for the present study, which aims to integrate epidemiological, clinical, and economic perspectives in order to evaluate obesity-related stroke risk and screening strategies in the Hungarian population.

2. Objectives

The overarching aim of the present dissertation was to evaluate the role of excess body weight in the development and prevention of stroke-related cardiometabolic risk in the Hungarian adult population. Particular emphasis was placed on epidemiological burden, economic consequences, and the predictive performance of anthropometric and body composition-based screening approaches.

To address this aim, the study pursued the following specific objectives:

First, to quantify the population-level burden of overweight and obesity in Hungary and to place these findings into an international context, comparing national prevalence estimates with European data.

Second, to estimate the contribution of excess body weight to stroke occurrence and its associated economic burden, using population-attributable modelling approaches to translate this burden into healthcare cost implications.

Third, to investigate whether conventional BMI thresholds adequately capture the emergence of stroke-related cardiometabolic risk factors—such as hypertension, diabetes mellitus, and atherosclerosis—in the Hungarian population by identifying data-driven inflection points.

Fourth, to examine sex-specific differences in risk emergence and to assess whether uniform BMI thresholds are appropriate for early risk detection in both men and women.

Fifth, to evaluate the predictive performance of BMI in comparison with body composition parameters derived from bioelectrical impedance analysis, assessing whether measures such as body fat percentage and visceral fat provide improved discrimination of cardiometabolic risk.

Sixth, to explore non-linear relationships between BMI, body composition, and cardiometabolic outcomes by identifying risk inflection zones and escalation ranges using non-parametric modelling techniques.

3. Methods

3.1 Study design and population

The present study was a cross-sectional, population-based screening investigation conducted within a nationwide mobile health programme in Hungary between June 1 and August 31, 2022. The programme provided free, on-site preventive services across 108 geographically distributed screening locations, covering both urban and rural regions. The geographical distribution of screening sites is presented in Figure 1, illustrating the nationwide coverage of the programme.

The screening programme included comprehensive health assessments, including anthropometric measurements, cardiometabolic risk evaluation, and body composition analysis using bioelectrical impedance. Data were collected under routine screening conditions, reflecting a real-world preventive setting.

The study population consisted of adult individuals aged 18 years and above who voluntarily participated in the screening programme. Following data cleaning and validation procedures, a total of 2,442 participants were included in the primary analytical dataset. This population was used for analyses related to BMI distribution and the estimation of overweight and obesity prevalence (31).

For analyses requiring detailed body composition and cardiometabolic data, a subpopulation was defined. This subgroup included 868 participants aged 30 years or older who underwent bioelectrical impedance analysis and had complete data available for body composition parameters and outcome variables. This analytical sample was used for the evaluation of predictive performance and risk associations (152).

Data collection was conducted according to standardized operational protocols. Personnel involved in the measurements received structured training in anthropometric assessment, body composition analysis, and data recording procedures to ensure consistency across all screening sites.

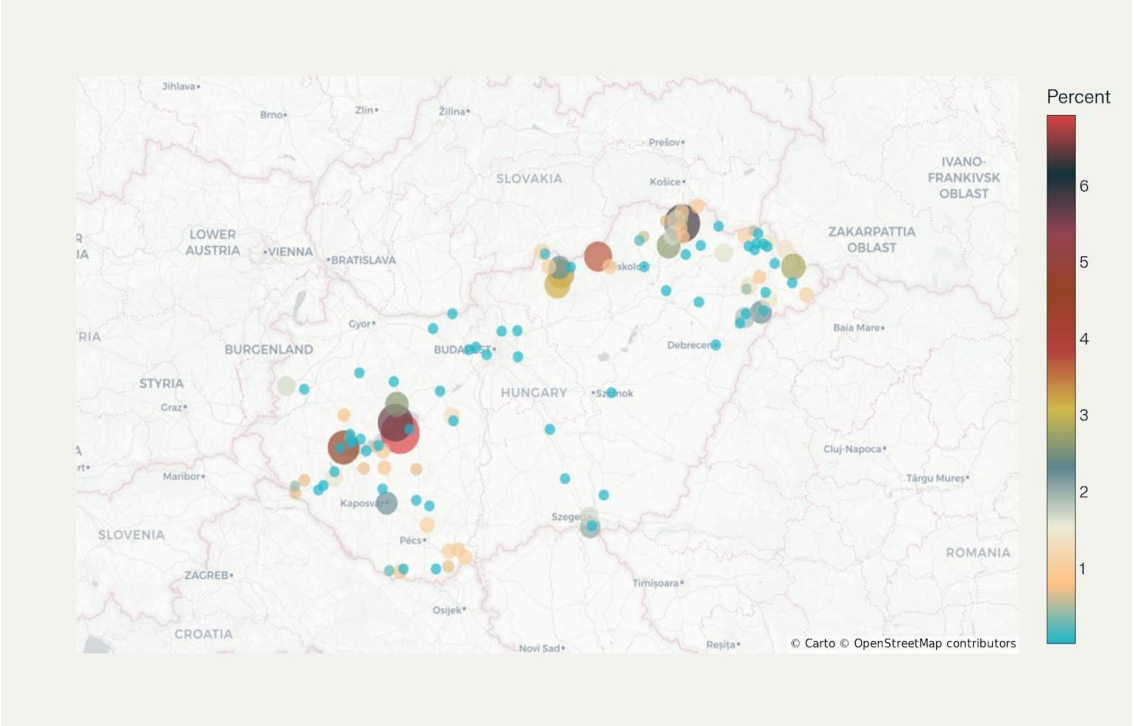


Figure 1. Geographic distribution of screening sites across Hungary.

The map presents all 108 screening site locations that participated in the "Bringing Screening to You" programme from June 1 to August 31, 2022. The size of each indicator corresponds to its proportional contribution to the study population (N=2,442).

3.2 Data collection and measurements

Data collection was performed using standardized procedures across all screening sites. Structured questionnaires administered by trained personnel were used to collect demographic and clinical information, including age, biological sex, medication use, and the presence of known chronic conditions.

Anthropometric measurements were obtained using calibrated instruments. Body weight was measured to the nearest 0.1 kg, and height was recorded to the nearest 0.5 cm using a portable stadiometer. Participants were measured without shoes and wearing light clothing. Body mass index (BMI) was calculated using the standard formula:

$$BMI = weight(kg)/height(m)^2$$

Blood pressure was measured using an automated cuff-based system (MESI mTABLET ABI system, MESI Development of Medical Devices Ltd., Ljubljana, Slovenia). Fasting blood glucose, total cholesterol, and triglyceride levels were assessed using a point-of-care analyzer (Multicare IN, Biomedical Systems International, Arezzo, Italy).

Carotid duplex ultrasound examinations were performed using a GE Versana Active 4D ultrasound system (GE HealthCare, Chicago, IL, USA) to assess the presence of atherosclerosis.

Body composition was evaluated using a segmental multi-frequency bioelectrical impedance analyzer (InBody 270, InBody Inc., Seoul, South Korea). Measurements were performed in a standing position, with participants barefoot and maintaining direct contact with both hand and foot electrodes. Arms were held slightly abducted from the torso to ensure standardized measurement conditions.

The following body composition parameters were extracted for analysis:

Body mass index (BMI): calculated as weight (kg) divided by height squared (m^2), and categorized according to World Health Organization (WHO) thresholds.

Body fat percentage (%): proportion of total body mass consisting of adipose tissue.

Skeletal muscle mass (kg): estimated total mass of skeletal muscle tissue.

Visceral fat level (device-specific scale): indirect estimate of intra-abdominal adiposity.

Trunk fat ratio: proportion of fat mass located in the trunk relative to total body fat.

Total fat mass (kg): absolute mass of adipose tissue.

All measurements were performed according to predefined protocols to ensure consistency across sites.

3.3 Definition of variables

3.3.1 Exposure variables

The primary exposure variables included body mass index (BMI) and body composition parameters derived from bioelectrical impedance analysis.

BMI was analysed both as a continuous variable and as a categorical variable according to World Health Organization (WHO) classification thresholds. Continuous modelling was used in the primary analyses to preserve variability and to allow the identification of non-linear relationships.

Body composition parameters were included to capture aspects of adiposity not reflected by BMI. These included body fat percentage, visceral fat level, skeletal muscle mass, total fat mass, and trunk fat ratio.

Where appropriate, exposure variables were analysed both continuously and within predefined BMI strata to explore heterogeneity in predictive performance.

3.3.2 Outcome variables

Cardiometabolic risk factors were defined as binary outcomes according to international clinical guidelines.

Hypertension was defined according to the 2018 European Society of Cardiology/European Society of Hypertension (ESC/ESH) guidelines as systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg, or current use of antihypertensive medication.

Diabetes mellitus was defined according to the 2021 American Diabetes Association (ADA) criteria as fasting plasma glucose ≥ 7.0 mmol/L, random glucose ≥ 11.1 mmol/L, or use of antidiabetic medication.

Dyslipidemia was defined based on the 2019 ESC/EAS guidelines as total cholesterol > 5.2 mmol/L or triglycerides > 1.7 mmol/L.

Atherosclerosis was defined as the presence of carotid arterial plaque confirmed by ultrasound, including intima–media thickness > 1.5 mm, focal atherosclerotic lesions, or documented evidence in medical history.

3.3.3 Conceptual linkage of stroke risk

Stroke events were not directly available in the body-composition cohort. Therefore, established cardiometabolic determinants of stroke, including hypertension, diabetes mellitus, dyslipidaemia and carotid atherosclerosis, were used as proxy endpoints for early cerebrovascular risk.

3.4 Statistical analysis

All statistical analyses were performed using R statistical software (version 4.1.2; R Foundation for Statistical Computing, Vienna, Austria). A two-sided p-value < 0.05 was considered statistically significant.

3.4.1 Descriptive analysis

Descriptive statistics were used to summarize the characteristics of the study population. Continuous variables were assessed for normality and reported as means with standard deviations or medians with interquartile ranges, as appropriate. Categorical variables were presented as absolute counts and percentages.

All primary descriptive analyses were conducted using post-stratification weights to reflect the demographic structure of the Hungarian adult population.

3.4.2 Logistic regression modelling

Associations between exposure variables (BMI and body composition parameters) and cardiometabolic outcomes were evaluated using logistic regression models. Both univariate and multivariate models were constructed.

Multivariate models were adjusted for age and sex as primary confounders. Continuous modelling of exposure variables was applied where appropriate to assess dose–response relationships.

Model assumptions, including linearity in the logit, were assessed using graphical methods. Where necessary, non-linear modelling approaches were applied.

3.4.3 ROC analysis and diagnostic performance

Receiver operating characteristic (ROC) analysis was used to evaluate the discriminatory performance of BMI and body composition parameters for cardiometabolic outcomes.

The area under the ROC curve (AUC) was calculated as a measure of discrimination. Comparisons between correlated ROC curves were performed using DeLong's test.

Stratified ROC analyses were conducted across predefined BMI categories to assess whether the performance of body composition parameters varied across levels of adiposity.

To assess the robustness of the primary findings with respect to age, the ROC and paired DeLong analyses were repeated in participants aged 45 years or older (n = 717; 266 men and 451 women). The same predictor variables, outcomes, sex-specific analyses, predefined BMI strata, and criteria for clinically relevant incremental discrimination were applied as in the primary analysis. This age-restricted analysis was considered a post hoc sensitivity analysis.

3.4.4 Inflection point analysis (LOESS regression)

Non-linear relationships between BMI and cardiometabolic risk were explored using locally estimated scatterplot smoothing (LOESS) regression. LOESS curves were generated separately for each outcome and stratified by sex. The smoothing parameter (span) was optimized through leave-one-out cross-validation by comparing span values between 0.25 and 0.40, with span = 0.30 providing the best balance between overfitting and oversmoothing.

Inflection points were identified using a predefined derivative-based algorithm. First derivatives of the LOESS curves were calculated across the BMI range, and baseline

slope characteristics were determined within the normal BMI range (18.5–22.0 kg/m²). The inflection point was defined as the lowest BMI value at which the slope exceeded the baseline mean by more than two standard deviations and remained continuously increasing over the subsequent BMI interval, thereby representing a sustained acceleration of cardiometabolic risk rather than a transient fluctuation.

To assess robustness, sensitivity analyses were performed using alternative smoothing parameters, and bootstrap resampling (n = 1000) was used to evaluate the stability of the identified thresholds.

3.4.5 Integration of analytical approaches

The analytical framework combined regression modelling, ROC analysis, and non-linear modelling techniques to evaluate associations, predictive performance, and threshold effects within the same dataset.

3.4.6 Bootstrap procedures

Sampling variability in prevalence estimates was assessed using stratified non-parametric bootstrap resampling.

Bootstrap samples were generated within age and sex strata to preserve the demographic structure of the study population. Prevalence estimates were recalculated for each replicate using the weighted dataset.

The resulting distributions were used to derive confidence intervals for key prevalence estimates.

3.5 Economic modelling

3.5.1 Conceptual framework

An economic modelling framework was developed to estimate the burden of stroke attributable to excess body weight in the Hungarian population. The model integrates population-level exposure data with externally derived relative risk estimates to quantify the impact of overweight and obesity on stroke incidence.

3.5.2 Population-attributable fraction (PAF)

The population-attributable fraction (PAF) was used to estimate the proportion of stroke cases attributable to excess body weight. PAF was calculated using standard epidemiological formulations incorporating exposure prevalence and relative risk estimates:

$$PAF = \sum_{i=1}^{n} p_i \cdot \frac{(RR_i - 1)}{RR_i}$$

where p_i represents the prevalence of exposure in category i and RR_i denotes the corresponding relative risk.

Relative risk estimates were obtained from large-scale international studies and applied to the weighted BMI distribution derived from the study population.

3.5.3 Economic burden estimation

PAF estimates were applied to national stroke incidence and cost data to quantify the annual economic burden attributable to overweight and obesity. The primary outcome of the model was the estimated annual stroke-related economic burden attributable to excess body weight.

3.5.4 Monte Carlo simulation

Monte Carlo simulation was performed to account for uncertainty in model parameters. A total of 10,000 simulation iterations were conducted.

Input parameters included BMI category prevalence, relative risk estimates, and demographic weighting factors. Relative risks were modelled using log-normal distributions, while BMI category proportions were modelled using Dirichlet distributions. Where applicable, correlated parameters were incorporated using multivariate distributions.

3.5.5 Sensitivity analyses and robustness

Sensitivity analyses were performed to evaluate the stability of the model under varying assumptions. These included the use of alternative relative risk estimates and scenario-based analyses.

In addition, model robustness was assessed using repeated simulations and comparison of results across different input configurations.

3.6 Model parameters and implementation details

Relative risk estimates linking BMI to stroke were derived from five published international models, including the HUNT Study (153), CHARLS cohort (154), Physicians' Health Study (155), INTERSTROKE study (9), and a large-scale meta-analysis (156).

Category-specific relative risks were derived from model-specific estimates and harmonized across studies to align with WHO BMI categories. The normal weight category (18.5–24.9 kg/m²) was used as the reference group.

The model-specific relative risk values used in the analysis are presented in Table 1

Table 1. International BMI–stroke relative risk models used in PAF estimation.

Relative risk (RR) estimates represent the association between body mass index (BMI) and stroke risk as reported in the original studies. All values are expressed per 5 kg/m² increase in BMI. Abbreviations: RR, relative risk; CI, confidence interval.

Model	Population	Stroke Outcome	RR (per 5 kg/m²)	95% CI
HUNT Study	Norway, 14 139 adults (65% F)	Ischemic	1.16	1.04–1.30
CHARLS-Male Cohort	China, 3 724 men	Total stroke	1.29	1.13–1.48
Physicians' Health Study	US male physicians (n = 21 867)	Total stroke	1.42	1.18–1.70
INTERSTROKE	32 countries, 13 462 cases	Total stroke	1.34	1.24–1.45
Wang et al. Meta-analysis	97 cohorts, 1.8 million	Total stroke	1.27	1.20–1.33

These relative risk estimates were used to derive category-specific risk values applied in the PAF calculations.

To account for uncertainty in model parameters, Monte Carlo simulation was applied.

A total of 10,000 simulation iterations were performed. Input parameters included BMI category prevalence derived from the weighted study sample, relative risk estimates obtained from the selected models, and demographic weighting factors.

Relative risks were modelled using log-normal distributions, reflecting their multiplicative nature, while BMI category proportions were modelled using Dirichlet distributions to preserve the compositional structure of categorical data.

Simulation outputs were expressed as distributions of population-attributable fraction (PAF) estimates, which were subsequently translated into economic outcomes.

3.7 Bias control and methodological considerations

Potential sources of bias were considered during study design and analysis.

Selection bias may be present due to voluntary participation in the screening programme. To mitigate this, post-stratification weighting was applied based on age and sex distributions of the national population.

Measurement variability was minimized through standardized protocols and training across all screening sites. Residual measurement error, particularly in bioelectrical impedance analysis, is expected to be non-differential.

Potential confounding was addressed through multivariate modelling with adjustment for age and sex.

3.8 Ethical considerations

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and its subsequent amendments.

Ethical approval was obtained from the institutional ethics committee (ETT TUKEB; approval No. IV/2395-3/2022/EKU).

Participation in the screening programme was voluntary, and all participants provided informed consent prior to data collection.

All data were collected and processed in anonymized form. Personal identifiers were not included in the analytical dataset, and data handling complied with applicable data protection regulations.

4. Results

4.1 National screening cohort used for prevalence and economic modelling

The economic modelling cohort included 2,442 adults examined between June 1 and August 31, 2022 within the nationwide mobile screening programme. The mean age of the analytical sample was 52.9 ± 14.5 years, with a median age of 54 years (IQR 42–64) and an age range of 18–87 years. Women represented 1,737 participants (71.1%), while men accounted for 705 participants (28.9%). Mean BMI was 28.6 ± 6.0 kg/m², with a median BMI of 28.0 kg/m² (IQR 24.4–32.1). A total of 2,617 screened individuals were excluded because of incomplete or invalid data. Excluded participants were slightly younger than the analytical cohort (51.2 ± 18.7 vs. 52.9 ± 14.5 years; $t = 2.70$, $p = 0.007$), whereas sex distribution did not differ significantly between groups ($\chi^2(1) = 3.77$, $p = 0.053$). Detailed cohort characteristics are presented in Table 2.

Table 2. Key demographic characteristics of included and excluded participants.
Abbreviations: n, number of participants; BMI, body mass index; IQR, interquartile range; χ^2, chi-square test; t, Student's t-test; p, p-value.

Characteristic	Included sample (n = 2,442)	Excluded sample (n = 2,617)	Between-group comparison
Mean age (years)	52.9 ± 14.5	51.2 ± 18.7	t=2.70; p=0.007
Median age (years)	54 (IQR 42 – 64)	52 (IQR 38 – 64)	—
Age range (years)	18 – 87	1 – 87	—
Sex distribution – Women	1,737 (71.1%)	1751 (66.9%)	$\chi^2(1)$=3.77; p=0.053
Sex distribution – Men	705 (28.9%)	866 (33.1%)	—
Mean BMI (kg/m²)	28.6 ± 6.0	27.7 ± 6.1	—
Median BMI (kg/m²)	28.0 (IQR 24.4 – 32.1)	27.1 (IQR 23.5 – 31.4)	—
BMI range (kg/m²)	14.7 – 73.9	15.4 – 52.9	—

Post-stratification weighting was subsequently applied using 2022 Hungarian census data to correct for demographic imbalance within the screening population. Women were down-weighted (0.764), whereas men were up-weighted (1.505). Age-specific weighting factors were 1.162 for participants aged 18–44 years, 0.707 for those aged 45–64 years, and 0.835 for those aged ≥65 years (Table 3).

Table 3. Post-stratification weighting factors applied in the economic modelling.
Abbreviation: yrs, years.

Dimension	Census (%)	Sample (%)	Weight
Women	52.0	68.1	0.764
Men	48.0	31.9	1.505
Age 18–44 yrs	33.0	28.4	1.162
Age 45–64 yrs	33.0	46.7	0.707
Age ≥ 65 yrs	20.8	24.9	0.835

4.2 Weighted BMI distribution and estimated prevalence of excess body mass

Post-stratification weighting aligned the analytical sample with the 2022 Hungarian adult population with respect to age and sex; however, the volunteer screening cohort was not nationally representative with regard to other demographic, regional, or socioeconomic characteristics. The weighted mean BMI was 29.7 ± 6.4 kg/m², with a median BMI of 28.9 kg/m² (IQR 25.1–33.6). BMI values ranged from 15.4 to 59.8 kg/m². The weighted prevalence of underweight was 2.3% (95% CI 1.7–3.0), while 27.0% (95% CI 25.2–28.9) of participants were classified as having normal weight. Overweight accounted for 30.5% (95% CI 28.6–32.4), and obesity for 40.3% (95% CI 38.3–42.3). The combined prevalence of overweight and obesity was 72.5% (95% CI 70.6–74.4) (Table 4).

Table 4. Age- and sex-weighted BMI distribution in the screening cohort.
Abbreviation: confidence interval, CI.

BMI category	Weighted prevalence (%)	95% CI
Underweight	2.3	1.7-3.0
Normal weight	27.0	25.2-28.9
Overweight	30.5	28.6-32.4
Obesity	40.3	38.3-42.3
Overweight + obesity	72.5	70.6-74.4

Regional comparison with EUROSTAT data demonstrated that the weighted prevalence observed in the present study was among the highest reported values in Europe (Table 5).

Table 5. Regional comparison of overweight and obesity prevalence.

Data derived from EUROSTAT European Health Interview Survey (EHIS) 2019 datasets (28) and the present study.

Country/Region	Overweight + obesity (%)	Year	Source
Hungary (present study)	72.5	2022	Present study
Malta	72.1	2019	EUROSTAT
Hungary (EHIS)	64.1	2019	EUROSTAT
Slovakia	59.2	2019	EUROSTAT
Czech Republic	56.8	2019	EUROSTAT
Romania	54.7	2019	EUROSTAT
EU average	51.6	2019	EUROSTAT
Austria	46.3	2019	EUROSTAT

4.3 Population-attributable fraction of stroke and projected economic burden

Application of the five international BMI–stroke risk models yielded population-attributable fraction (PAF) estimates ranging from 9.7% to 20.4%. The lowest estimate was obtained using the HUNT Study model, with a PAF of 9.7% (95% CI 7.4–12.5), while the highest estimate was generated by the Physicians' Health Study model, with a PAF of 20.4% (95% CI 14.3–26.1). Intermediate estimates were 16.3% (95% CI 8.2–23.8) for the CHARLS model, 18.7% (95% CI 15.9–21.5) for INTERSTROKE, and 20.1% (95% CI 3.4–35.6) for the meta-analysis model. When applied to the estimated 2022 Hungarian national stroke expenditure of 1.016 billion euros, these PAF estimates corresponded to an attributable annual stroke-related economic burden ranging from 99 million euros (95% CI 75–127 million) to 207 million euros (95% CI 145–265 million), depending on the selected risk model (Table 6). The mean estimate across all five models corresponded to a PAF of 17.0% and an attributable annual stroke-related economic burden of 173 million euros.

Table 6. Population-attributable fraction of stroke associated with excess BMI and estimated annual stroke-related economic burden.

Abbreviations: PAF, population-attributable fraction; CI, confidence interval; €, euro; €M, million euros; HUNT Study, the Nord-Trøndelag Health Study; CHARLS, China Health and Retirement Longitudinal Study; INTERSTROKE, International Study of Risk Factors for Stroke; Physicians' Health, Physicians' Health Study.

Model	PAF (%)	95% CI	Annual stroke-related economic burden (€M)	95% CI
HUNT Study	9.7	7.4–12.5	99	75–127
CHARLS (male)	16.3	8.2–23.8	166	83–242
INTERSTROKE	18.7	15.9–21.5	190	162–218
Meta-analysis	20.1	3.4–35.6	204	35–362
Physicians' Health	20.4	14.3–26.1	207	145–265

Leave-one-out sensitivity analysis demonstrated limited variability across model configurations, with estimated burden values ranging from −16% to +8% relative to the all-model mean (Table 7).

Table 7. Leave-one-out sensitivity analysis of the five BMI-stroke risk models.

Abbreviations: PAF, population-attributable fraction; Δ, percentage difference relative to the all-model mean estimate; €, euro; €M, million euros; HUNT Study, the Nord-Trøndelag Health Study; CHARLS, China Health and Retirement Longitudinal Study; INTERSTROKE, International Study of Risk Factors for Stroke; Physicians' Health, Physicians' Health Study.

Leave-one-out scenario	Mean PAF (%)	Annual stroke-related economic burden (€M)	Δ vs all-model mean
None (base)	17.0	173	-
Omit HUNT	18.1	184	+6%
Omit CHARLS	18.4	187	+8%
Omit INTERSTROKE	16.5	168	-3%
Omit Meta-analysis	14.4	148	-14%

4.4 Body-composition cohort with complete cardiometabolic phenotyping

The body-composition cohort included 868 adults with complete anthropometric, InBody, laboratory, and carotid ultrasound data. Women represented 547 participants (63.0%), while 321 participants (37.0%) were men. Median age in the overall cohort was 54.8 years (IQR 47.2–63.2). Median BMI was 27.6 kg/m² (IQR 24.5–31.4). No significant sex differences were observed for either age or BMI. Body-composition parameters differed substantially between sexes. Women had higher body-fat percentage, visceral fat level, and total fat mass, whereas men demonstrated higher skeletal muscle

mass and trunk fat ratio (all $p < 0.001$). Hypertension was present in 55.8% of participants, hypercholesterolaemia in 41.5%, hypertriglyceridaemia in 44.5%, atherosclerosis in 38.4%, and diabetes mellitus in 13.7%. Hypertension and atherosclerosis were more frequent among men, while hypercholesterolaemia was more common among women. Detailed cohort characteristics and outcome prevalence are presented in Table 8.

Table 8. Baseline characteristics and outcome prevalence by sex in the body-composition cohort

Abbreviations: BMI, body mass index; IQR, interquartile range; n, sample size; %, percentage; kg, kilograms. Data are median (IQR) or n (%). p-values from Mann-Whitney U test (continuous) or chi-square test (categorical).

Characteristic	Men (n=321)	Women (n=547)	All (n=868)	p-value
Age, median (IQR), years	55.2 (48.1-63.1)	54.2 (46.8-63.5)	54.8 (47.2-63.2)	0.420
BMI, median (IQR), kg/m²	28.2 (25.4-31.6)	27.3 (24.1-31.4)	27.6 (24.5-31.4)	0.080
Body fat %, median (IQR)	27.1 (22.6-31.5)	37.2 (32.2-42.7)	33.5 (27.0-40.2)	<0.001
Visceral fat level (1-20)	10.0 (8.0-14.0)	13.0 (9.0-17.0)	12.0 (9.0-16.0)	<0.001
Muscle mass, kg	36.7 (32.4-40.9)	25.0 (22.6-27.4)	27.6 (24.1-34.5)	<0.001
Fat mass, kg	23.6 (18.0-31.1)	28.2 (21.9-36.9)	26.6 (20.5-34.0)	<0.001
Trunk fat ratio	301.9 (230.7-387.8)	258.7 (192.2-332.5)	272.9 (202.1-352.0)	<0.001
Hypertension, n (%)	202 (62.9)	282 (51.6)	484 (55.8)	<0.001
Diabetes mellitus, n (%)	42 (13.1)	77 (14.1)	119 (13.7)	0.69
Hypercholesterolaemia, n (%)	117 (36.4)	243 (44.4)	360 (41.5)	0.03
Hypertriglyceridaemia, n (%)	150 (46.7)	236 (43.1)	386 (44.5)	0.34
Atherosclerosis, n (%)	156 (48.6)	177 (32.4)	333 (38.4)	<0.001

4.5 Demographic composition of the analytical cohorts

Both analytical cohorts showed female predominance. Compared with the 2022 Hungarian census population, middle-aged adults were markedly overrepresented in both cohorts, as shown in Figure 2.

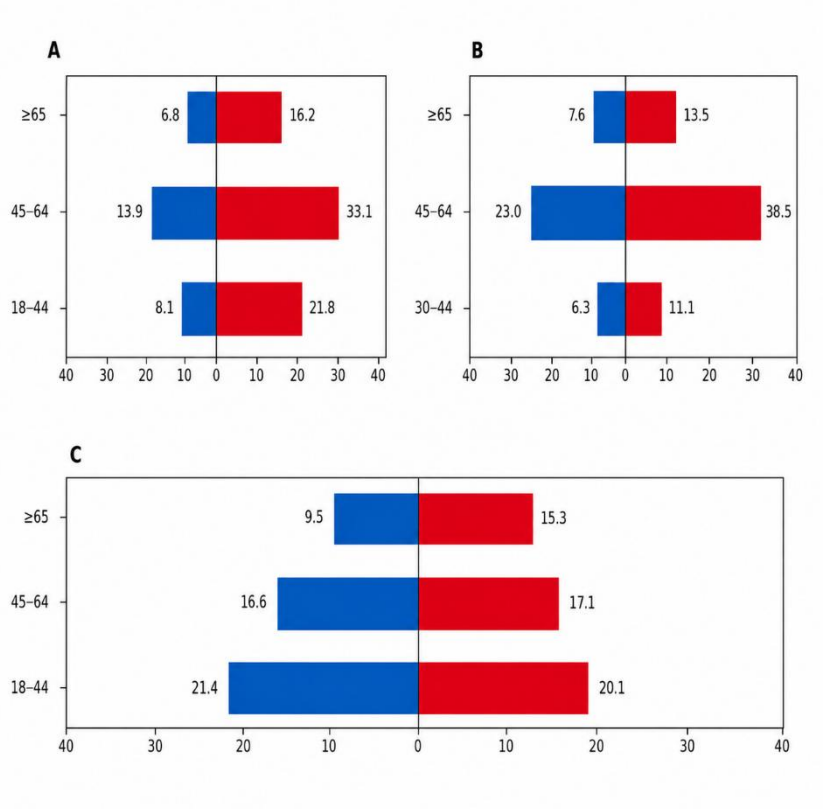


Figure 2. Age and sex distribution of the analytical cohorts compared with the 2022 census population.

Panel A presents the national screening cohort used for prevalence and economic modelling (n = 2442), Panel B the body-composition cohort (n = 868), and Panel C the 2022 Hungarian adult census population (N = 7,921,212). Blue bars represent men and red bars represent women. Values indicate the percentage of the total population shown in each panel. The body-composition cohort included participants aged 30 years or older; therefore, its youngest age category was 30–44 years.

4.6 Overall predictive performance of BMI and InBody-derived parameters

Across the full body-composition cohort, most anthropometric and body-composition parameters demonstrated statistically significant associations with cardiometabolic outcomes. However, overall discriminatory performance remained limited to moderate, with most AUC values ranging between approximately 0.55 and 0.70.

In contrast, discriminatory performance for hypercholesterolaemia was limited across all anthropometric and body-composition parameters. Most AUC values approximated 0.50, and several parameters did not differ significantly from chance-level discrimination.

Comparisons between BMI and the best-performing InBody-derived parameters demonstrated largely comparable predictive performance across hypertension, diabetes mellitus, and hypertriglyceridaemia in both sexes, with no statistically significant differences observed in DeLong comparisons.

The largest divergence between BMI and body-composition parameters was observed for male atherosclerosis. In this subgroup, body-fat percentage yielded an AUC of 0.656 (95% CI 0.597–0.716), compared with 0.576 (95% CI 0.514–0.639) for BMI, corresponding to a ΔAUC of +0.080 ($p = 0.001$).

Complete ROC results and DeLong comparisons are presented in Tables 9 and 10.

Table 9. Full ROC matrix for BMI and InBody-derived body-composition parameters by sex.

Abbreviations: AUC, area under the curve; CI, confidence interval; BMI, body mass index. *AUC values with 95% CI. $p<0.05$ vs. chance (AUC=0.5).

Risk factor	Parameter	Men AUC (95% CI)	Women AUC (95% CI)
Hypertension	BMI	0.658 (0.598-0.717)	0.690 (0.646-0.733)
	Body fat %	0.642 (0.581-0.703)	0.661 (0.616-0.707)
	Visceral fat level	0.654 (0.594-0.714)	0.657 (0.612-0.702)
	Trunk fat ratio	0.659 (0.599-0.719)	0.679 (0.635-0.724)
	Muscle mass	0.544 (0.479-0.608)	0.541 (0.492-0.589)
	Fat mass	0.648 (0.588-0.709)	0.656 (0.611-0.702)
Hypercholesterolemia	BMI	0.526 (0.460-0.592)	0.500 (0.451-0.548)
	Body fat %	0.540 (0.474-0.606)	0.505 (0.457-0.554)
	Visceral fat level	0.533 (0.467-0.599)	0.498 (0.449-0.547)
	Trunk fat ratio	0.512 (0.446-0.578)	0.478 (0.429-0.526)
	Muscle mass	0.533 (0.467-0.599)	0.501 (0.452-0.549)
	Fat mass	0.535 (0.469-0.601)	0.508 (0.459-0.557)
Diabetes mellitus	BMI	0.656 (0.561-0.751)	0.638 (0.568-0.709)

	Body fat %	0.648 (0.553-0.743)	0.603 (0.532-0.674)
	Visceral fat level	0.644 (0.549-0.740)	0.594 (0.523-0.665)
	Trunk fat ratio	0.643 (0.548-0.738)	0.617 (0.546-0.688)
	Muscle mass	0.517 (0.423-0.612)	0.562 (0.491-0.633)
	Fat mass	0.648 (0.553-0.743)	0.609 (0.538-0.680)
Hypertriglyceridemia	BMI	0.676 (0.617-0.735)	0.573 (0.524-0.621)
	Body fat %	0.630 (0.569-0.691)	0.563 (0.515-0.612)
	Visceral fat level	0.655 (0.594-0.715)	0.561 (0.512-0.610)
	Trunk fat ratio	0.664 (0.604-0.723)	0.575 (0.527-0.624)
	Muscle mass	0.586 (0.523-0.648)	0.515 (0.466-0.564)
	Fat mass	0.661 (0.601-0.721)	0.564 (0.515-0.612)
Atherosclerosis	BMI	0.576 (0.514-0.639)	0.547 (0.495-0.599)
	Body fat %	0.656 (0.597-0.716)	0.553 (0.501-0.605)
	Visceral fat level	0.599 (0.538-0.661)	0.540 (0.488-0.592)
	Trunk fat ratio	0.616 (0.555-0.677)	0.559 (0.507-0.611)
	Muscle mass	0.385 (0.324-0.446)	0.400 (0.351-0.449)
	Fat mass	0.609 (0.547-0.670)	0.515 (0.463-0.567)

Table 10. DeLong comparison of BMI with the best-performing InBody parameter for each outcome and sex.

Abbreviations: AUC, area under the curve; CI, confidence interval; BMI, body mass index; ΔAUC, difference in AUC (InBody − BMI). Statistically significant difference indicated in bold.

Outcome	Sex	BMI AUC (95% CI)	Best parameter	InBody AUC (95% CI)	ΔAUC	p-value
Hypertension	Men	0.658 (0.598-0.717)	Trunk fat ratio	0.659 (0.599-0.719)	+0.001	0.901
Hypertension	Women	0.690 (0.646-0.733)	Trunk fat ratio	0.679 (0.635-0.724)	-0.011	0.229
Atherosclerosis	Men	0.576 (0.514-0.639)	Body fat %	0.656 (0.597-0.716)	+0.080	**0.001**
Atherosclerosis	Women	0.547 (0.495-0.599)	Body fat %	0.553 (0.501-0.605)	+0.006	0.683
Diabetes mellitus	Men	0.656 (0.561-0.751)	Body fat %	0.648 (0.553-0.743)	-0.008	0.805
Diabetes mellitus	Women	0.638 (0.568-0.709)	Trunk fat ratio	0.617 (0.546-0.688)	-0.021	0.111
Hypertriglyceridemia	Men	0.676 (0.617-0.735)	Trunk fat ratio	0.664 (0.604-0.723)	-0.012	0.254
Hypertriglyceridemia	Women	0.573 (0.524-0.621)	Trunk fat ratio	0.575 (0.527-0.624)	+0.002	0.779
Hypercholesterolemia	Men	0.526 (0.460-0.592)	Body fat %	0.540 (0.474-0.606)	+0.014	0.529
Hypercholesterolemia	Women	0.500 (0.451-0.548)	Visceral fat level	0.508 (0.459-0.557)	+0.008	0.487

4.7 BMI inflection points and risk-escalation zones identified by LOESS analysis

LOESS analysis demonstrated non-linear and sex-specific relationships between BMI and cardiometabolic outcomes. In men, inflection points were identified within a relatively narrow BMI range between 23.8 and 24.3 kg/m² across the analysed outcomes. In women, inflection points consistently occurred at lower BMI values, ranging from 21.8 to 22.3 kg/m². Following the inflection points, progressive increases in cardiometabolic risk were observed until the corresponding Youden-optimised cut-off values. The width of these risk-escalation zones varied across outcomes, with the broadest ranges observed for hypertension and narrower transitions observed for diabetes mellitus. Across all analysed outcomes, women demonstrated earlier onset of risk acceleration compared with

men. Sex-specific LOESS curves are presented in Figure 3, while detailed inflection points and risk-escalation zones are summarized in Table 11.

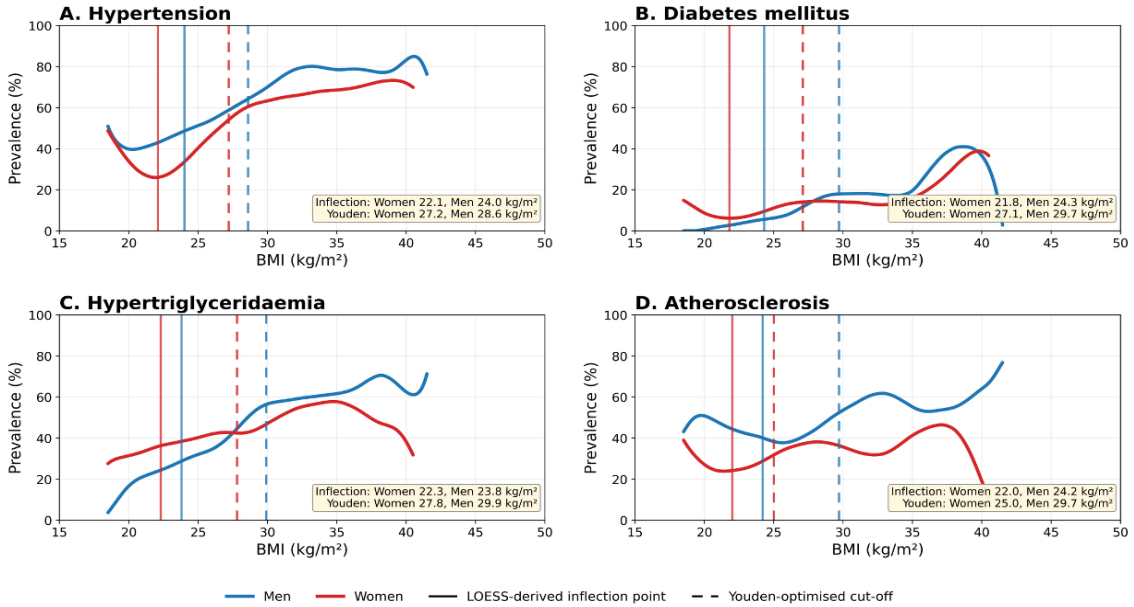


Figure 3. Sex-specific LOESS curves for BMI and cardiometabolic outcome prevalence. Panel A: hypertension; Panel B: diabetes mellitus; Panel C: hypertriglyceridemia; Panel D: atherosclerosis. Solid vertical lines indicate LOESS-derived inflection points. Dashed vertical lines indicate Youden-optimised cut-off values. Blue curves represent men and red curves represent women.

Table 11. LOESS-derived inflection points and risk-escalation zones for major cardiometabolic outcomes.

Inflection points represent population-level risk emergence; Youden cutoffs represent optimal clinical thresholds balancing sensitivity/specificity. Zone width indicates BMI range of progressive risk escalation.

Outcome	Sex	Inflection point (kg/m²)	Youden cutoff (kg/m²)	Zone width (kg/m²)
Hypertension	Men	24.0	29.9	5.9
Hypertension	Women	22.1	27.8	5.7
Diabetes mellitus	Men	24.3	28.6	4.3
Diabetes mellitus	Women	21.8	25.0	3.2
Hypertriglyceridemia	Men	23.8	29.2	5.4
Hypertriglyceridemia	Women	22.3	26.4	4.1
Atherosclerosis	Men	24.2	28.8	4.6
Atherosclerosis	Women	22.0	26.8	4.8

4.8 Stratified ROC analyses across BMI categories

Stratified ROC analyses were performed across non-overlapping 3 kg/m² BMI intervals to evaluate whether the discriminatory performance of body-composition parameters varied across different levels of adiposity. Although overall ROC analyses demonstrated broadly comparable performance between BMI and InBody-derived parameters, several subgroup-specific differences emerged after stratification. A limited number of BMI strata demonstrated higher discriminatory performance for selected body-composition parameters compared with BMI, particularly for male atherosclerosis and selected low- and high-BMI subgroups. The observed subgroup effects were heterogeneous across outcomes and sexes and were not uniformly distributed across the BMI spectrum. Detailed stratified ROC comparisons are presented in Table 12. Given the relatively small sample sizes within several BMI strata, these findings should be interpreted as exploratory and hypothesis-generating.

Table 12. Stratified differences in favor of InBody.

Abbreviations: BMI, body mass index; N = sample size, n = outcome positive cases; AUC, area under the curve; CI, confidence interval; ΔAUC, difference in AUC (InBody − BMI). BMI ranges defined as left-closed, right-open intervals (e.g., 24–27 = BMI ≥ 24 and <27 kg/m²). Only cases meeting both significance ($p < 0.05$) and clinical relevance (ΔAUC > 0.10) thresholds shown.

Sex	BMI Range (kg/m²)	Outcome	Parameter	N/n	BMI AUC (95% CI)	InBody AUC (95% CI)	ΔAUC	*p*-Value
Male	18–21	Hypertension	Visceral fat	24/11	0.438 (0.109–0.766)	0.838 (0.588–1.000)	0.400	0.048
Male	18–21	Hypercholesterolemia	Muscle mass	24/7	0.333 (0.026–0.641)	0.714 (0.427–1.000)	0.381	0.043
Male	27–30	Atherosclerosis	Trunk fat ratio	82/46	0.499 (0.374–0.624)	0.666 (0.547–0.784)	0.167	0.018
Male	27–30	Atherosclerosis	Body fat %	82/46	0.499 (0.374–0.624)	0.662 (0.543–0.782)	0.163	0.044
Male	33–36	Atherosclerosis	Body fat %	36/23	0.506 (0.274–0.739)	0.857 (0.708–1.000)	0.351	0.029
Male	33–36	Atherosclerosis	Trunk fat ratio	36/23	0.506 (0.274–0.739)	0.818 (0.651–0.985)	0.312	0.037
Female	36–39	Atherosclerosis	Trunk fat ratio	29/19	0.382 (0.167–0.597)	0.648 (0.439–0.858)	0.266	0.046

4.9 Age-restricted sensitivity analysis in participants aged ≥45 years

The age-restricted sensitivity analysis included 717 participants aged 45–87 years, comprising 266 men (37.1%) and 451 women (62.9%). Median age was 57.0 years (IQR 51.0–65.0), and median BMI was 27.7 kg/m² (IQR 24.6–31.4). Men had a higher prevalence of hypertension (66.5% vs. 58.5%, p = 0.041) and atherosclerosis (57.9% vs. 37.9%, p < 0.001), whereas the prevalence of diabetes mellitus and hypertriglyceridaemia was similar between sexes.

Discriminatory performance in the age-restricted cohort remained limited to moderate and was broadly consistent with the primary analysis. Among men, body-fat percentage demonstrated higher discrimination for atherosclerosis than BMI, with an AUC of 0.670 (95% CI 0.605–0.734), compared with 0.597 (95% CI 0.530–0.665) for BMI (ΔAUC = +0.073, p = 0.004). Although statistically significant, the difference remained below the predefined ΔAUC threshold of 0.10 for clinically relevant incremental discrimination.

Among women, BMI demonstrated slightly higher discrimination for diabetes mellitus than the best-performing InBody-derived parameter, total fat mass (AUC 0.631 [95% CI 0.559–0.702] vs. 0.605 [95% CI 0.529–0.680]; ΔAUC = −0.026, p = 0.013). No other comparison between BMI and the best-performing InBody-derived parameter reached nominal statistical significance. Detailed results are shown in Table 13.

Table 13. DeLong comparison of BMI with the best-performing InBody parameter for each outcome and sex in participants aged ≥45 years.

Abbreviations: AUC, area under the curve; CI, confidence interval; BMI, body mass index; ΔAUC, difference in AUC (InBody − BMI). Statistically significant difference indicated in bold.

Outcome	Sex	BMI AUC (95% CI)	Best parameter	InBody AUC (95% CI)	ΔAUC	p-value
Hypertension	Men	0.649 (0.579-0.718)	Trunk fat ratio	0.649 (0.578-0.720)	+0.000	0.998
Hypertension	Women	0.698 (0.649-0.748)	Trunk fat ratio	0.686 (0.637-0.736)	-0.012	0.174
Atherosclerosis	Men	0.597 (0.530-0.665)	Body fat %	0.670 (0.605-0.734)	+0.073	**0.004**
Atherosclerosis	Women	0.541 (0.487-0.595)	Trunk fat ratio	0.551 (0.497-0.605)	+0.010	0.264
Diabetes mellitus	Men	0.666 (0.583-0.750)	Fat mass	0.653 (0.566-0.740)	-0.013	0.543
Diabetes mellitus	Women	0.631 (0.559-0.702)	Fat mass	0.605 (0.529-0.680)	-0.026	**0.013**
Hypertriglyceridemia	Men	0.679 (0.616-0.743)	Visceral fat level	0.664 (0.599-0.728)	-0.016	0.355
Hypertriglyceridemia	Women	0.566 (0.513-0.619)	Trunk fat ratio	0.569 (0.516-0.622)	+0.003	0.714
Hypercholesterolemia	Men	0.539 (0.466-0.612)	Visceral fat level	0.547 (0.475-0.618)	+0.008	0.654
Hypercholesterolemia	Women	0.491 (0.438-0.545)	Body fat %	0.502 (0.448-0.555)	+0.010	0.434

The repeated BMI-stratified analysis identified seven comparisons meeting both the nominal significance criterion of $p < 0.05$ and the predefined effect-size criterion of $\Delta AUC > 0.10$. These included male atherosclerosis within the BMI range of 21–24 kg/m², male hypertension at BMI 24–27 kg/m², female hypertension at BMI 18–21 kg/m², and female diabetes mellitus within the BMI ranges of 33–36 and 39–42 kg/m². These localized findings were based on relatively small stratum-specific event counts and differed from the pattern observed in the complete body-composition cohort. Detailed data is shown in Table 14.

Table 14. Stratified differences in favor of InBody in participants aged ≥45 years.

Abbreviations: BMI, body mass index; N = sample size, n = outcome positive cases; AUC, area under the curve; CI, confidence interval; ΔAUC, difference in AUC (InBody − BMI). BMI ranges defined as left-closed, right-open intervals (e.g., 24–27 = BMI ≥ 24 and <27 kg/m^2). Only cases meeting both significance ($p < 0.05$) and clinical relevance (ΔAUC > 0.10) thresholds shown.

Sex	BMI range (kg/m²)	Outcome	Parameter	N/n	BMI AUC (95% CI)	InBody AUC (95% CI)	ΔAUC	p-value
Male	21-24	Atherosclerosis	Body fat %	32/17	0.522 (0.312-0.731)	0.722 (0.535-0.908)	+0.200	0.040
Male	24-27	Hypertension	Trunk fat ratio	57/33	0.456 (0.292-0.619)	0.620 (0.470-0.770)	+0.164	0.043
Female	18-21	Hypertension	Body fat %	29/11	0.338 (0.121-0.556)	0.644 (0.408-0.879)	+0.306	0.006
Female	18-21	Hypertension	Trunk fat ratio	29/11	0.338 (0.121-0.556)	0.576 (0.337-0.814)	+0.237	0.011
Female	33-36	Diabetes mellitus	Body fat %	41/6	0.445 (0.200-0.691)	0.690 (0.451-0.930)	+0.245	0.007
Female	33-36	Diabetes mellitus	Trunk fat ratio	41/6	0.445 (0.200-0.691)	0.667 (0.433-0.900)	+0.221	0.023
Female	39-42	Diabetes mellitus	Muscle mass	16/6	0.325 (0.029-0.621)	0.683 (0.375-0.992)	+0.358	0.039

Overall, restricting the analysis to participants aged 45 years or older did not materially alter the principal discriminatory findings and did not reveal a general superiority of InBody-derived parameters over BMI.

4.10 Non-linear relationships between BMI and body-composition parameters across the adiposity spectrum

Sex-specific LOESS analyses were performed to evaluate the relationships between BMI and major InBody-derived body-composition parameters, including body fat percentage, visceral fat level, skeletal muscle mass, and trunk fat ratio.

Positive associations were observed between BMI and all analysed body-composition parameters; however, several relationships demonstrated non-linear patterns across the BMI spectrum. Among women, body fat percentage and visceral fat level demonstrated visible changes in slope beginning approximately between BMI 23–26 kg/m². In men, similar transition patterns were observed at slightly higher BMI ranges, particularly for

visceral fat level between BMI 26–32 kg/m². By contrast, trunk fat ratio demonstrated a more stable and approximately linear relationship with BMI across the analysed range in both sexes. Skeletal muscle mass demonstrated weaker and less consistent relationships with BMI, particularly among men, where plateau-like patterns were observed at higher BMI values. Overall, several body-composition parameters demonstrated distinct transition zones and changes in slope across intermediate BMI ranges rather than stable linear relationships throughout the full BMI spectrum.

The approximate linear and non-linear transition ranges are summarized in Table 15, while sex-specific LOESS relationships between BMI and body-composition parameters are presented in Figure 4.

Table 15. Sex-specific linear and non-linear BMI regions for body-composition parameters identified by LOESS modelling.

Abbreviations: BMI, body mass index; R^2, coefficient of determination. Linear regions represent BMI intervals in which the relationship between BMI and the respective body-composition parameter remained approximately linear according to LOESS modelling.

Sex	Parameter	BMI Linear Region (kg/m²)	Linear R^2	BMI Non-linear Region (kg/m²)
Women	Muscle mass	19.5–26.0	0.56	26.0–40.5
Women	Body fat percentage	19.5–23.5	0.67	23.5–40.5
Women	Visceral fat level	19.5–25.5	0.61	25.5–40.5
Women	Trunk segmental fat	19.5–40.5	0.85	None observed
Women	Fat mass	19.5–40.5	0.90	None observed
Men	Muscle mass	19.5–25.0	0.20	25.0–40.5
Men	Body fat percentage	19.5–24.0	0.42	24.0–40.5
Men	Visceral fat level	19.5–26.0	0.64	26.0–40.5
Men	Trunk segmental fat	19.5–40.5	0.88	None observed
Men	Fat mass	19.5–40.5	0.88	None observed

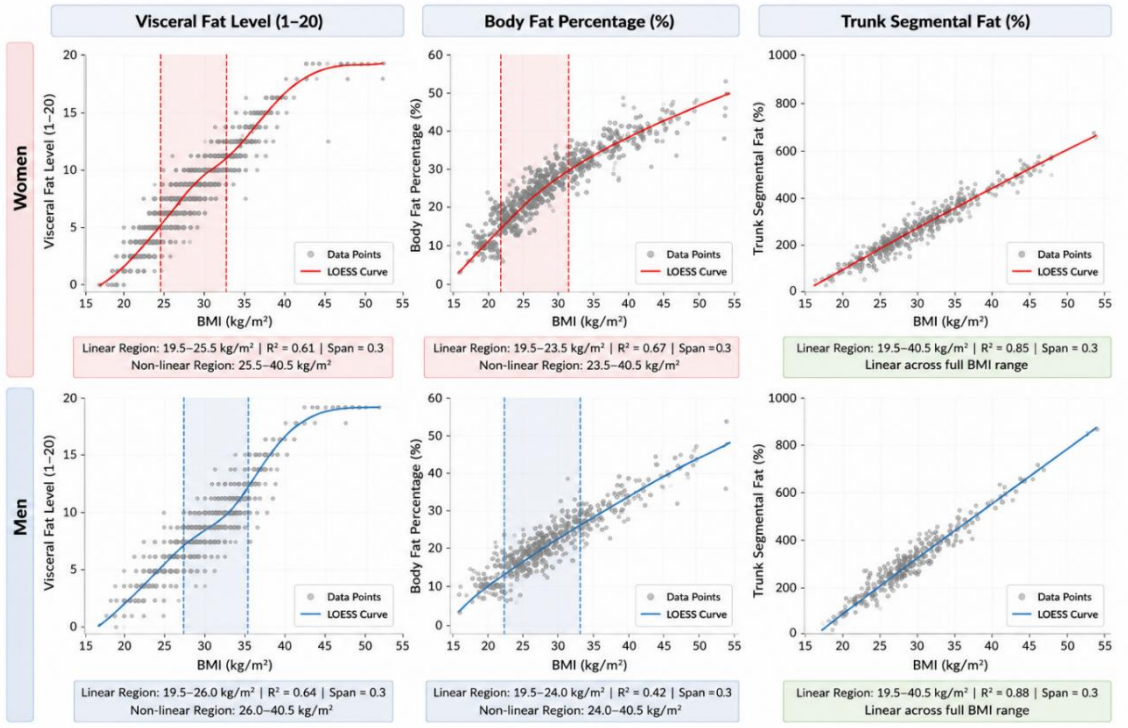


Figure 4. Sex-specific LOESS relationships between BMI and body composition parameters.

Top row shows women; bottom row shows men. Left panels show visceral fat level, middle panels show body fat percentage, and right panels show trunk segmental fat according to BMI. Grey points represent individual observations, and solid lines represent LOESS-smoothed curves. Red curves and shaded areas indicate women, while blue curves and shaded areas indicate men. Dashed vertical lines mark the estimated transition boundaries between the approximately linear and non-linear BMI ranges. Green annotation boxes indicate parameters with an approximately linear relationship across the analysed BMI range.

Abbreviations: BMI, body mass index; LOESS, locally estimated scatterplot smoothing; R^2, coefficient of determination.

4.11 Integrated interpretation of complementary analytical approaches

The combined interpretation of the overall ROC analyses, LOESS-derived cardiometabolic risk curves, BMI–body composition LOESS analyses, and stratified ROC analyses revealed a consistent pattern across multiple analytical approaches.

Overall ROC analyses demonstrated that BMI showed broadly comparable discriminatory performance to InBody-derived body-composition parameters for most cardiometabolic outcomes. Apart from male atherosclerosis, no consistent overall

superiority of body-composition parameters over BMI was observed across the analysed outcomes, supporting the continued utility of BMI as a first-line population-level screening parameter.

At the same time, LOESS analyses demonstrated that the prevalence of major cardiometabolic outcomes began to increase at BMI ranges lower than the conventional WHO overweight threshold, particularly among women. In parallel, sex-specific LOESS analyses between BMI and body-composition parameters demonstrated visible non-linear transition zones and changes in slope across intermediate BMI ranges, particularly for body fat percentage and visceral fat level.

These observations suggest that the relationship between BMI and underlying body-composition phenotype may become less stable across selected intermediate BMI ranges. Stratified ROC analyses further demonstrated that localized discriminatory differences between BMI and body-composition parameters were concentrated within similar BMI intervals, particularly among men between BMI 27–30 kg/m².

Taken together, the convergence of earlier cardiometabolic risk emergence, BMI–body composition non-linearity, and localized subgroup-specific discriminatory differences identified intermediate BMI ranges that may warrant further investigation in region-specific preventive screening models.

These findings should be interpreted as exploratory and hypothesis-generating rather than as evidence supporting modification of current BMI-based intervention thresholds or screening recommendations.

4.12 Modelled implementation scenario of selective body-composition assessment

A hypothetical implementation scenario was constructed to estimate the potential operational implications of selective body-composition assessment within the intermediate BMI ranges identified by the integrated analytical framework. In this theoretical scenario, individuals outside the predefined intermediate BMI ranges would undergo standard BMI-based assessment only, whereas participants within the identified BMI intervals would additionally receive InBody-based body-composition analysis. Based on the integrated findings of the preceding analyses, the selected BMI ranges were 22–30 kg/m² for women and 24–30 kg/m² for men. Using post-stratification weighting derived from the Hungarian adult population, these BMI ranges corresponded to

approximately 4.11 million adults, representing 51.9% of the national adult population (Table 16).

Table 16. Estimated population distribution within the intermediate BMI ranges identified by the integrated analytical framework.

Abbreviations: BMI, body mass index; n, sample size; N, weighted population size.

Group	BMI Criterion	Sample n (%)	Weighted N (Hungary)	% Adult Population
Males	BMI 24-30 kg/m²	159 (49.5%)	1,883,000	23.8%
Females	BMI 22-30 kg/m²	302 (55.2%)	2,230,000	28.1%
Total indicated	-	461 (53.1%)	4,113,000	51.9%

Operational estimates were based on the average duration of measurements performed during the screening programme. BMI-only assessment was estimated to require approximately 2 minutes per participant, whereas InBody-based body-composition assessment was estimated to require approximately 5 minutes per participant. Labour-cost estimates were calculated using the average Hungarian gross wage reported by the Hungarian Central Statistical Office (KSH) for 2022. Based on these assumptions, the estimated labour cost of BMI-only assessment was 148 HUF (0.37 EUR) per participant, compared with 371 HUF (0.94 EUR) for universal InBody assessment. Under the selective assessment scenario, the weighted mean examination time was estimated at 3.56 minutes per participant, corresponding to an estimated throughput of 22.7 participants per hour. The estimated labour cost was 264 HUF (0.67 EUR) per participant. Compared with BMI-only screening, the mixed BMI plus selective InBody approach corresponded to approximately 178.4% of the unit cost of BMI-only screening, while estimated screening throughput decreased to 75.7% of BMI-only capacity, representing an estimated 24.3% reduction in screening throughput. Detailed time, labour-cost, and throughput estimates for the alternative assessment scenarios are presented in Table 17.

Table 17. Estimated operational characteristics of modelled BMI-based and selective body-composition assessment scenarios.

Abbreviations: BMI, body mass index; min, minutes; HUF, Hungarian forint; EUR, euro.

Method	Time (min)	Cost (HUF)	Cost (EUR)	Patients/hour
BMI-only	2	148	0.37	30
InBody-only	5	371	0.94	12
Mixed BMI + targeted InBody	3.56	264	0.67	22.7

Despite these operational trade-offs, the selective strategy remained substantially less resource-intensive than universal body-composition assessment of the entire screened population. The identified BMI ranges should not be interpreted as novel intervention thresholds, but rather as exploratory high-phenotypic-heterogeneity zones identified through the convergence of multiple analytical approaches. Further studies are required to determine whether these observations translate into clinically meaningful improvement in risk stratification and whether region-specific implementation strategies would be feasible in preventive practice.

5. Discussion

5.1 Main findings

The present dissertation provides a multi-level evaluation of excess body weight in the Hungarian population, integrating epidemiological burden, body-composition phenotyping, predictive modelling, and health-economic analyses. While the body-composition component focused on established cardiometabolic determinants of stroke, the health-economic component directly quantified the stroke burden attributable to excess body weight.

The findings demonstrate a high burden of overweight and obesity in the study population, with an age- and sex-weighted estimate approaching three quarters of the screening cohort. This exceptionally high prevalence indicates that excess adiposity is not confined to specific subgroups, but instead constitutes a major structural determinant of cardiometabolic disease burden within the Hungarian population.

Despite its known limitations, BMI demonstrated broadly consistent discriminatory performance across multiple cardiometabolic outcomes, supporting its continued role as a practical and scalable first-line screening tool in both clinical and public health settings. Overall, body-composition parameters did not demonstrate clear universal superiority over BMI, although selected subgroup-specific advantages were observed for certain outcomes. These findings were further supported by the age-restricted sensitivity analysis among participants aged ≥45 years, which yielded a broadly similar pattern and did not demonstrate general superiority of InBody-derived parameters over BMI.

At the same time, several findings indicated that the relationship between BMI, body composition, and cardiometabolic risk may not be uniformly linear across the BMI spectrum. LOESS-based analyses demonstrated earlier emergence of cardiometabolic risk than predicted by conventional WHO thresholds, particularly among women. In parallel, sex-specific analyses of BMI and body-composition relationships identified visible non-linear transition zones and partial decoupling between BMI and selected body-composition parameters across intermediate BMI ranges. Stratified ROC analyses further suggested that localized discriminatory differences between BMI and body-composition measures were concentrated within similar BMI intervals.

Taken together, these observations support the interpretation that intermediate BMI ranges may represent zones of increasing phenotypic heterogeneity, in which similar BMI

values may reflect differing underlying body-composition and metabolic profiles. While these findings do not challenge the central utility of BMI in population-level screening, they suggest that risk emergence and phenotype distribution may be more complex than implied by uniform threshold-based classification alone.

Finally, the population-attributable fraction analyses demonstrated that excess body weight contributes substantially to the national stroke burden in Hungary. Depending on the applied risk model, approximately 10–20% of stroke cases were attributable to overweight and obesity, corresponding to a considerable annual economic burden. These findings reinforce the public health importance of improving early risk identification and prevention strategies in high-burden populations.

5.2 The Hungarian obesity burden

The most appropriate starting point for interpreting the present findings is the extraordinary population burden of excess body weight in Hungary. In the age- and sex-weighted estimate derived from the voluntary screening sample, 72.5% of participants were classified as overweight or obese, with 30.5% falling into the overweight category and a further 40.3% into the obese category. Only 27.0% were within the normal BMI range, while 2.3% were underweight. Because the sample was not probability-based and post-stratification accounted only for age and sex, these figures should be interpreted as weighted estimates from the screening cohort rather than definitive nationally representative prevalence estimates. Nevertheless, the magnitude of the observed burden is consistent with previous evidence indicating that Hungary is among the European countries most affected by excess body weight.

The magnitude of this burden becomes particularly apparent in international comparison. The Hungarian estimate of 72.5% exceeds the EU average of 51.6% by 20.9 percentage points and is also higher than neighboring Central and Eastern European countries included in the regional comparison, including Slovakia (59.2%), the Czech Republic (56.8%), and Romania (54.7%) (28). The weighted estimate was 8.4 percentage points higher than the 2019 Hungarian EHIS estimate of 64.1%; however, this difference should not be interpreted as evidence of a temporal increase because of differences in sampling strategy and anthropometric assessment methods.

These observations are important not merely because they describe a high-risk population, but because they define the epidemiological context in which all downstream

interpretations of cardiometabolic risk must be placed. In a country where nearly three out of four adults carry excess body mass, obesity cannot be conceptualized solely as an individual lifestyle issue. Rather, it becomes a structural determinant of chronic disease epidemiology, shaping the overall distribution of vascular and metabolic risk within the population.

Taken together, these findings define Hungary as a high-burden obesity environment in which efficient, scalable, and economically feasible approaches to cardiometabolic risk identification become particularly important from both clinical and public health perspectives.

5.3 Hungary-specific economic interpretation

The second major interpretive dimension of the present work is economic. In the PAF-based modelling, overweight and obesity accounted for 9.7% to 20.4% of stroke cases depending on the selected international risk model, corresponding to an estimated annual stroke-related economic burden of €99 million to €207 million in Hungary under a theoretical complete-normalization scenario. The total annual stroke-related expenditure used in the model was €1.016 billion (157). Conservative estimates based on the HUNT model (153) yielded €99 million (95% CI: €75–127 million), whereas the Physicians' Health Study-based model (155) yielded €207 million (95% CI: €145–265 million). Intermediate estimates were generated by the CHARLS (154), INTERSTROKE (9), and meta-analytic models (156).

These estimates are important not merely because they quantify a large economic burden, but because they illustrate the scale at which excess adiposity may influence healthcare resource allocation within a high-burden population. In resource-constrained healthcare systems, preventable stroke expenditure represents not only an efficiency issue, but also a strategic allocation issue. Resources consumed by obesity-related stroke burden are consequently unavailable for other components of vascular prevention and care, including acute stroke pathways, rehabilitation services, primary care strengthening, or broader prevention programmes.

There is also a conceptual advantage to linking obesity prevalence directly to stroke-related expenditure rather than discussing obesity in generic economic terms. Stroke represents one of the most consequential vascular endpoints because it combines acute treatment costs with long-term disability, recurrent-event risk, rehabilitation demand,

caregiver burden, and productivity loss. Demonstrating that excess BMI contributes measurably to the national stroke-related expenditure therefore provides a more concrete public-health interpretation than simply stating that obesity is economically costly.

At the same time, the economic interpretation of these findings must remain methodologically disciplined. The primary modelling scenario assumed proportional cost reduction with decreasing stroke incidence, which represents a reasonable but simplified assumption. In practice, healthcare systems contain fixed and semi-fixed cost components, meaning that reductions in disease incidence do not necessarily translate directly into proportional financial savings. Sensitivity analyses partly addressed this uncertainty by demonstrating that alternative assumptions reduced mean projected savings to approximately €92 million under less extreme counterfactual scenarios, while leave-one-out validation showed that exclusion of any single risk model altered aggregate estimates by no more than ±16%. Similarly, exclusion of extreme BMI values changed cost estimates by only 0.8%, supporting the overall robustness of the broader economic signal (31).

Importantly, the key interpretation is not that Hungary could literally "recover" €207 million annually through obesity reduction alone. Rather, the findings indicate that the obesity burden is sufficiently large to generate stroke-related expenditure at a scale that is clearly relevant from a healthcare-policy perspective. Even partial reductions in obesity-related vascular risk could therefore produce meaningful economic benefit at the population level.

Beyond incidence reduction alone, the relationship between obesity and stroke-related expenditure may also be influenced by differences in stroke severity and long-term disability. Obesity is associated not only with increased stroke incidence, but also with more severe vascular phenotypes through the coexistence of hypertension, insulin resistance, endothelial dysfunction, chronic inflammation, and prothrombotic states (158, 159). These mechanisms may contribute to larger infarct burden, poorer collateral circulation, prolonged rehabilitation needs, and impaired recovery potential (160-163).

Consequently, even moderate reductions in obesity-related vascular risk may theoretically produce disproportionately large economic effects through shifts in severity distribution rather than simple reductions in event count alone. This interpretation further

reinforces the importance of improving early identification of high-risk individuals within high-burden populations such as Hungary.

5.4 Cardiometabolic risk as the mechanistic link

The relationship between excess body weight and stroke is complex and multifactorial. Obesity contributes to cerebrovascular risk both indirectly, through its role in the development of established cardiometabolic conditions such as hypertension, diabetes mellitus, dyslipidaemia, and atherosclerosis, and directly, through mechanisms including chronic low-grade inflammation, endothelial dysfunction, arterial stiffness, and prothrombotic states (9, 159, 164).

As an upstream determinant within this network, excess adiposity promotes the clustering of multiple vascular risk factors (42, 165, 166). In the present population, these conditions were highly prevalent and frequently co-occurred, consistent with previous observations in Central and Eastern European cohorts (35, 167). The coexistence of multiple cardiometabolic abnormalities is particularly important because combined vascular risk-factor burden is known to increase cerebrovascular risk more substantially than isolated abnormalities alone (9, 12, 42).

At the same time, the metabolic consequences of excess body weight are unlikely to be uniformly distributed across individuals with similar BMI values. Individuals with comparable BMI may still demonstrate differing body-composition profiles, visceral adiposity, inflammatory states, insulin resistance, and vascular phenotypes. Consequently, similar BMI values may be associated with differing cardiometabolic and vascular risk profiles.

This biological heterogeneity may partly contribute to several observations identified in the present analyses, including the earlier emergence of cardiometabolic risk, the non-linear relationships observed between BMI and selected body-composition parameters, and the localized subgroup-specific differences in discriminatory performance identified within intermediate BMI ranges.

In addition to its indirect cardiometabolic effects, obesity itself has also been identified as an independent risk factor for stroke in multiple large epidemiological studies (9, 42, 156). Adiposity-related alterations in vascular biology, including endothelial dysfunction, oxidative stress, chronic inflammation, and impaired fibrinolytic balance, may contribute

directly to cerebrovascular injury even in the absence of overt clinical comorbidity (165, 168, 169).

Taken together, these observations are consistent with the possibility that BMI alone may incompletely reflect adiposity-related biological heterogeneity in selected populations, particularly across intermediate BMI ranges characterized by increasing phenotypic variability.

5.5 Risk identification in a high-burden population

5.5.1 BMI as a first-line screening tool: historical context and current utility

Body mass index was originally developed by Adolphe Quetelet in the 19th century as a population-level descriptor of body habitus rather than as a direct clinical measure of adiposity (86). Its later adoption into modern medicine was driven primarily by large epidemiological studies demonstrating associations between BMI and long-term morbidity and mortality (89, 156). These observations ultimately formed the basis of the current World Health Organization (WHO) classifications for overweight and obesity (79, 99).

Despite its well-recognized limitations (including the inability to distinguish between fat and lean mass or to characterize adipose tissue distribution) BMI remains one of the most practical and scalable tools for population-level cardiometabolic risk assessment (65, 83, 84, 86). In the present analyses, BMI demonstrated broadly consistent discriminatory performance across multiple cardiometabolic outcomes, including hypertension, diabetes mellitus, hypertriglyceridaemia, and atherosclerosis. These findings support its continued utility as a pragmatic first-line screening approach, particularly in high-burden settings where simplicity, accessibility, and scalability are critical considerations (50, 112, 170, 171).

5.5.2 Biological limitations of BMI and the role of body-composition assessment

Given the known biological limitations of BMI, considerable attention has been directed toward body-composition analysis methods such as bioelectrical impedance analysis (BIA), which aim to characterize metabolically relevant adiposity patterns more directly through estimation of fat mass, visceral adiposity, and fat distribution.

Importantly, the present findings do not indicate clear universal superiority of body-composition assessment over BMI in cross-sectional cardiometabolic outcome

discrimination. Across the full cohort, InBody-derived parameters demonstrated broadly comparable AUC performance to BMI, with only limited subgroup-specific advantages observed for selected outcomes. These results therefore support the continued central role of BMI in large-scale screening frameworks.

At the same time, several observations suggest that BMI may incompletely reflect biologically relevant adiposity patterns across selected BMI ranges. In particular, LOESS-based analyses demonstrated non-linear relationships between BMI and several body-composition parameters, especially visceral fat level, indicating that similar BMI values may not necessarily correspond to equivalent metabolically active adiposity profiles.

Importantly, the absence of substantial overall AUC superiority indicates that the observed biological heterogeneity does not automatically translate into clinically meaningful improvements in cross-sectional outcome discrimination. Nevertheless, these findings remain biologically relevant because visceral adiposity represents a metabolically active compartment strongly linked to insulin resistance, systemic inflammation, endothelial dysfunction, and vascular risk.

Taken together, the present analyses therefore suggest not that body-composition assessment universally outperforms BMI, but rather that BMI may incompletely reflect metabolically relevant adiposity distribution across selected BMI regions despite similar overall discriminatory performance.

5.5.3 Sex-specific patterns of early cardiometabolic risk emergence

Beyond body-composition relationships alone, the present findings also demonstrated that cardiometabolic risk emergence occurred at lower and sex-specific BMI ranges than suggested by conventional WHO classifications. LOESS-based analyses identified earlier inflection regions for several stroke-related cardiometabolic outcomes, with these transition patterns occurring consistently at lower BMI values in women than in men.

These findings suggest that the relationship between BMI and cardiometabolic risk may follow a continuous and biologically heterogeneous trajectory rather than aligning strictly with fixed categorical thresholds. Importantly, the present analyses do not establish alternative clinical cut-offs or challenge existing guideline frameworks directly. Rather, they indicate that the onset and acceleration of cardiometabolic risk may occur earlier and in a sex-specific manner within the Hungarian population.

The coexistence of earlier risk emergence and non-linear BMI–body-composition relationships may be particularly relevant across intermediate BMI regions, where similar BMI values may correspond to differing metabolic and adiposity profiles.

5.5.4 Implications for future risk-identification strategies

Taken together, the convergence of sex-specific earlier cardiometabolic risk emergence, non-linear BMI–visceral fat relationships, and localized subgroup-specific discriminatory differences suggests that intermediate BMI ranges may represent biologically heterogeneous regions warranting further investigation.

Importantly, these observations should not be interpreted as evidence that body-composition assessment should replace BMI in routine large-scale screening. Rather, the findings suggest that certain metabolically relevant adiposity patterns may not be fully captured by BMI alone despite similar overall discriminatory performance in the present cross-sectional analyses. Restricting the analysis to participants aged ≥45 years, thereby reducing the potential influence of the latency between excess adiposity and clinically manifest cardiometabolic disease, did not materially change this interpretation; the persistent advantage of body-fat percentage for male atherosclerosis remained below the predefined threshold for clinically relevant incremental discrimination.

From a clinical and research perspective, these intermediate BMI regions may therefore represent candidate zones for future prospective investigation, particularly in relation to targeted body-composition assessment strategies, longitudinal vascular-risk trajectories, and individualized prevention approaches. Further prospective and interventional studies will be required to determine whether incorporation of body-composition assessment within selected BMI ranges can provide clinically meaningful improvements in risk prediction, prevention, or healthcare efficiency.

5.6 Policy and clinical implications

5.6.1 Implications for clinical thresholds and intervention strategies

Current clinical practice generally recommends intensified intervention at BMI ≥30 kg/m², or at BMI ≥27 kg/m² in the presence of established cardiometabolic risk factors. These thresholds have been widely adopted due to their simplicity and international comparability.

However, the findings of the present work suggest that these thresholds may not fully reflect the heterogeneity of cardiometabolic risk in all populations. In particular, the observed earlier emergence of risk, as well as the consistent sex-specific differences in inflection points, indicate that a uniform, one-size-fits-all approach may not be optimal for early risk identification.

Rather than challenging current guideline thresholds directly, these results suggest that additional attention may be warranted within specific BMI ranges where risk appears to begin to accelerate. In this context, the identification of intermediate BMI "windows" associated with early cardiometabolic changes may provide a useful framework for future investigation. Such windows could represent clinically relevant zones for earlier lifestyle intervention or targeted monitoring, even in the absence of overt disease.

Importantly, these observations should be considered hypothesis-generating. Prospective validation is required to determine whether earlier or sex-specific intervention thresholds would translate into improved clinical outcomes or more efficient prevention strategies in practice.

5.6.2 Implications for population-level policy and resource allocation

At the population level, the implications of the present findings extend beyond individual risk assessment to broader questions of healthcare policy and resource allocation. In a setting characterized by both a high prevalence of excess body weight and a substantial obesity-related stroke burden, prevention strategies must be evaluated not only in terms of clinical effectiveness, but also in relation to feasibility, scalability, and economic sustainability.

Public-health interventions targeting obesity (including fiscal measures, workplace-based prevention programmes, and structural initiatives improving access to healthy nutrition and physical activity) require robust epidemiological and economic data in order to support rational policy development.

In this context, quantifying the contribution of excess body weight to stroke-related healthcare expenditure provides an important foundation for future cost-effectiveness and prevention modelling. Without such data, preventive decision-making remains largely assumption-driven, limiting both strategic prioritization and long-term sustainability.

The present findings suggest that, given the scale of the obesity burden in Hungary, even moderate improvements in population-level adiposity patterns could potentially translate

into meaningful reductions in healthcare burden and vascular disease risk. At the same time, the findings also indicate that effective prevention may depend not only on reducing obesity prevalence itself, but also on improving identification of emerging cardiometabolic risk within high-burden populations.

Taken together, these findings indicate that in populations such as Hungary, the challenge extends beyond obesity reduction alone toward developing scalable and economically sustainable strategies capable of identifying cardiometabolic risk at earlier stages while preserving feasibility at the population level.

5.7 Limitations

Several limitations of the present work should be acknowledged.

First, the cross-sectional design of the study precludes causal inference. While the observed associations between BMI, body composition parameters, and cardiometabolic risk factors are biologically plausible and consistent with existing literature, the temporal relationship between exposure and outcome cannot be established. In particular, the duration and historical trajectory of excess body weight were not captured, which may influence the development of cardiometabolic conditions and attenuate observed associations.

Second, the study population was derived from a voluntary, mobile screening programme, which may introduce selection bias. Participants may differ from the general population in terms of health awareness, socioeconomic status, or access to healthcare. Although post-stratification weighting was applied to correct for age and sex distribution, residual confounding related to unmeasured variables—such as regional, educational, or socioeconomic factors—cannot be excluded. As a result, the representativeness of the findings at the national level may be limited. In addition, a substantial proportion of initially screened individuals were excluded from the primary analytical cohort due to missing anthropometric data or age below 18 years. Importantly, these exclusions primarily reflected incomplete participation in specific screening modules rather than systematic exclusion based on demographic or clinical characteristics. Comparison of included and excluded participants revealed no significant difference in sex distribution and only a modest difference in age (52.9 vs. 51.2 years). Nevertheless, some degree of selection bias cannot be fully excluded, and the observed prevalence estimates and risk profiles should be interpreted in light of this limitation.

Third, the use of bioelectrical impedance analysis (BIA) for body composition assessment introduces methodological constraints. While InBody measurements provide practical and accessible estimates of body composition, they may not be directly comparable to gold-standard methods such as dual-energy X-ray absorptiometry (DXA). Furthermore, because data collection was conducted within a nationwide mobile screening programme, measurement conditions could not be standardized as strictly as in a controlled laboratory setting. Participants may have differed with regard to recent food and fluid intake, hydration status, time since physical activity, bladder filling, and clothing, all of which may influence impedance-based estimates. Environmental conditions, including ambient temperature and humidity, may also have affected skin–electrode contact and peripheral fluid distribution. Although standardized operational instructions were used and participants were measured barefoot and in light clothing, residual measurement variability cannot be excluded.

Fourth, the discrimination analyses were based on cross-sectional associations, and statistical significance does not necessarily imply clinical utility. In particular, subgroup analyses (especially in smaller strata) may be subject to reduced statistical power and increased uncertainty. The observed differences between BMI and body composition parameters should therefore be interpreted cautiously and require prospective validation. Additionally, the age-restricted sensitivity analysis was performed post hoc and further reduced sample and event counts within several sex–BMI–outcome strata; therefore, its stratified findings should also be considered exploratory.

Fifth, the economic modelling approach relied on several simplifying assumptions. The application of population-attributable fraction (PAF) estimates to aggregate national stroke costs assumes proportional cost reduction with decreasing stroke incidence. In reality, healthcare systems include fixed and semi-fixed cost components, and marginal cost savings may not follow a strictly linear pattern. Furthermore, the PAF estimates were derived from externally validated BMI–stroke risk models originating from populations with different demographic, ethnic, and healthcare characteristics than Hungary. Although multiple independent cohorts and sensitivity analyses were used to improve robustness, some uncertainty related to the transferability of these relative risk estimates remains unavoidable.

Finally, the identification of lower and sex-specific BMI inflection points should be interpreted as hypothesis-generating. The present analysis does not establish optimal clinical thresholds, and prospective cohort studies and interventional trials are required to determine whether modified or population-specific cut-offs would improve risk prediction and clinical outcomes.

6. Conclusions

The present dissertation provides a multi-level evaluation of excess body weight as a determinant of cardiometabolic risk and stroke burden in Hungary, integrating epidemiological burden, economic consequences, and the predictive performance of anthropometric and body composition measures. While the body-composition analyses focused on upstream cardiometabolic determinants of stroke, the economic modelling component directly estimated stroke burden attributable to excess body weight.

Excess body weight emerges as a dominant population-level characteristic in Hungary. With more than 70% of adults classified as overweight or obese, adiposity represents a structural determinant of cardiometabolic risk rather than a marginal condition. In such a high-prevalence environment, a large proportion of the population is already exposed to elevated risk, emphasizing the importance of effective and scalable screening strategies.

Overweight and obesity contribute substantially to the national stroke burden and its associated economic consequences. Depending on the applied risk model, approximately 10% to 20% of stroke cases can be attributed to excess body weight, corresponding to an estimated annual economic burden of €99 million to €207 million. Even under conservative assumptions, this represents a policy-relevant and potentially modifiable component of healthcare expenditure.

The analyses further indicate that cardiometabolic risk may emerge at lower BMI levels than those defined by conventional WHO thresholds, with consistently earlier inflection points observed in women. These findings suggest that uniform threshold-based classification may not fully capture the underlying distribution of risk in the Hungarian population, and highlight the importance of considering sex-specific and population-adapted approaches in future research.

Despite its limitations, BMI remains a robust and practical first-line screening tool. Its consistent performance across multiple cardiometabolic outcomes, combined with its simplicity and accessibility, supports its continued central role in both clinical and public health settings.

The analyses further suggested that the relationship between BMI and selected metabolically relevant body-composition parameters, particularly visceral adiposity, may become non-linear across intermediate BMI ranges. These observations indicate that

similar BMI values may not necessarily correspond to equivalent metabolic phenotypes, despite broadly similar overall discriminatory performance in cross-sectional analyses.

However, body composition analysis did not demonstrate clear overall superiority over BMI, although it provided incremental value in selected subgroups. These findings suggest that the role of body composition assessment is best understood as complementary rather than substitutive, and may be most relevant in targeted clinical contexts rather than as a universal screening approach.

Taken together, the results of this dissertation support a refinement rather than a replacement of current risk assessment strategies. In high-burden populations such as Hungary, more nuanced interpretation of BMI, combined with selective use of additional assessment tools, may improve early risk identification while preserving feasibility at the population level.

Further prospective and interventional studies are required to determine whether population-specific and sex-sensitive adaptations of current screening approaches can lead to measurable improvements in clinical outcomes and cost-effectiveness in stroke prevention.

7. Summary

Stroke remains one of the leading causes of mortality and long-term disability worldwide, largely driven by modifiable cardiometabolic risk factors. Among these, excess body weight plays a central role both directly and through its association with hypertension, diabetes, and atherosclerosis. Although body mass index (BMI) remains the primary tool for population-level assessment of nutritional status, its inability to capture body composition and fat distribution has led to increasing interest in complementary methods such as bioelectrical impedance analysis. Hungary represents a particularly relevant setting for examining these questions due to its exceptionally high prevalence of overweight and obesity and the associated cardiometabolic and stroke burden. The present dissertation integrated epidemiological, economic, and analytical approaches to evaluate obesity-related cardiometabolic and stroke risk in the Hungarian population. The findings demonstrated that excess body weight is highly prevalent and contributes substantially to stroke-related healthcare expenditure. Despite its limitations, BMI remained a robust and practical first-line screening tool, showing broadly consistent performance across multiple cardiometabolic outcomes. At the same time, the analyses identified non-linear relationships between BMI and metabolically relevant body-composition parameters, suggesting that similar BMI values may not necessarily correspond to equivalent metabolic risk profiles within intermediate BMI ranges. Body-composition assessment did not demonstrate clear overall superiority over BMI in cross-sectional discrimination analyses, although selected subgroup-specific differences were observed. Furthermore, several cardiometabolic risk factors appeared at lower BMI values than those defined by conventional classification systems, particularly among women, indicating sex-specific variability in risk emergence. Taken together, these findings suggest that cardiometabolic risk may accelerate within specific BMI intervals preceding conventional thresholds. These “risk emergence windows” identify BMI ranges that warrant further investigation in relation to early risk detection and targeted prevention. Ultimately, the results of this dissertation suggest that optimizing the interpretation, rather than replacing the use, of BMI—combined with selective application of complementary methods—may contribute to more effective and sustainable stroke prevention strategies.

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9. Bibliography of the candidate's publications

9.1 Original publications related to the dissertation

Jarecsny Tamás, Egri Csilla Arvane, Kosik Roland, Schwab Richárd, Mechtler László, Szőllősi Gergő József, Schandl László, Tomasics Gyula, Gyuricskó István, Pázmándi Eszter Melinda, Fazekas Ferenc, Fekete Mónika

Economic burden of stroke attributable to excess body mass in Hungary: a population-attributable fraction analysis

BMC PUBLIC HEALTH 26: 1 Paper: 772, 10 p. (2026)

Közlemény: 36919416 | Szakcikk (Folyóiratcikk) | Tudományos

Scopus - Public Health, Environmental and Occupational Health SJR indikátor: Q1

IF: 4,4

Jarecsny Tamás, Al-Muhanna Nadim, Fábián Dóra Rebeka, Kosik Roland, Schwab Richard, Szőllősi Gergő József, Schandl László, Tomasics Gyula, Pázmándi Eszter Melinda, Folyovich András, Fazekas Ferenc, Fekete Mónika

Optimizing Public Health Screening: Population-Specific BMI Thresholds for Targeted Body Composition Assessment in Hungary

NUTRIENTS 18: 9 Paper: 1410, 15 p. (2026)

Közlemény: 37102317 | Szakcikk (Folyóiratcikk) | Tudományos

Scopus - Food Science SJR indikátor: D1

Scopus - Nutrition and Dietetics SJR indikátor: Q1

IF: 5,8

9.2 Other publications

Fazekas-Pongor Vince, Major Dávid, Varga János Tamás, Lehoczki Andrea, Varga Péter, **Jarecsny Tamás**, Lipécz Ágnes, Csípő Tamás, Szappanos Ágnes, Matiscsák Attila, Fekete Mónika

Physiological and Recovery Responses to Dietary Polyphenols in the Context of Exercise: Relevance for Muscle Aging and Sarcopenia

NUTRIENTS 18: 5 Paper: 788, 32 p. (2026)

Közlemény: 36982518 | Összefoglaló cikk (Folyóiratcikk) | Tudományos

Scopus - Food Science SJR indikátor: D1

Scopus - Nutrition and Dietetics SJR indikátor: Q1

IF: 5,8

Pázmándi Eszter, **Jarecsny Tamás**, Fekete Mónika

Polifenolokban gazdag élelmiszerek és rezisztenciatréning szinergikus hatásai a szarkopénia megelőzésében és kezelésében

ÚJ DIÉTA: A MAGYAR DIETETIKUSOK LAPJA (2001-) 35: 1 pp. 7-14. (2026)

Közlemény: 36946747 | Összefoglaló cikk (Folyóiratcikk) | Tudományos

Fekete Mónika1, **Jarecsny Tamás**1, Lehoczki Andrea, Major Dávid, Fazekas-Pongor Vince, Csípő Tamás, Lipécz Ágnes, Szappanos Ágnes, Pázmándi Eszter Melinda, Varga Péter, Varga János Tamás

Mediterranean Diet, Polyphenols, and Neuroprotection: Mechanistic Insights into Resveratrol and Oleuropein

NUTRIENTS 17: 24 Paper: 3929, 40 p. (2025)

Közlemény: 36501242 | Összefoglaló cikk (Folyóiratcikk) | Tudományos

Scopus - Food Science SJR indikátor: D1

Scopus - Nutrition and Dietetics SJR indikátor: Q1

1Megosztott első szerzők

IF: 5,8

Lehoczki Andrea, Fekete Mónika, **Jarecsny Tamás**, Zábó Virág, Szappanos Ágnes, Csípő Tamás, Lipécz Ágnes, Major Dávid, Fazekas-Pongor Vince, Varga Péter, Varga János Tamás

The Neuroprotective Role of Curcumin: From Molecular Pathways to Clinical Translation—A Narrative Review

NUTRIENTS 17: 17 Paper: 2884, 42 p. (2025)

Közlemény: 36324408 | Összefoglaló cikk (Folyóiratcikk) | Tudományos

Scopus - Food Science SJR indikátor: D1

Scopus - Nutrition and Dietetics SJR indikátor: Q1

IF: 5,8

Varga Péter, Lehoczki Andrea, Fekete Mónika, **Jarecsny Tamás**, Kryczyk-Poprawa Agata, Zábó Virág, Major Dávid, Fazekas-Pongor Vince, Csípő Tamás, Varga János T

The Role of Magnesium in Depression, Migraine, Alzheimer's Disease, and Cognitive Health: A Comprehensive Review

NUTRIENTS 17: 13 Paper: 2216, 27 p. (2025)

Közlemény: 36227770 | Összefoglaló cikk (Folyóiratcikk) | Tudományos

Scopus - Food Science SJR indikátor: D1

Scopus - Nutrition and Dietetics SJR indikátor: Q1

IF: 5,8

Folyovich A, Mátis R, Biczó D, Pálosi M, Béres-Molnár AK, Al-Muhanna N, **Jarecsny T**, Dudás E, Jánoska D, Toldi G, Páldy Anna

High average daily temperature in summer and the incidence of thrombolytic treatment for acute ischemic stroke

ENCEPHALE-REVUE DE PSYCHIATRIE CLINIQUE BIOLOGIQUE ET THERAPEUTIQUE 50: 5 pp. 510-515. (2024)

Közlemény: 34478430 | Szakcikk (Folyóiratcikk) | Tudományos

Scopus - Arts and Humanities (miscellaneous) SJR indikátor: Q1

Scopus - Psychiatry and Mental Health SJR indikátor: Q3

IF: 1,0

Folyovich András, Szabó Pál Tamás, Műhelyi Viktória, Pápai György, Csató Gábor, Győrfi Pál, Fábián Dóra, Róka Gabriella, Korda Lívia, **Jarecsny Tamás**, Van Der Merwe Jan, Pete Kinga Nedda, Bátyai Éva, Béres-Molnár Katalain Anna

„Stroke-ovi". A lakossági felvilágosítás új célközönsége: az óvodások [The new target population of stroke awareness campaign: Kindergarten students]

IDEGGYOGYASZATI SZEMLE / CLINICAL NEUROSCIENCE 76: 5-6 pp. 197-204. (2023)

Közlemény: 34059981 | Szakcikk (Folyóiratcikk) | Tudományos

Scopus - Neurology (clinical) SJR indikátor: Q4

Scopus - Neurology SJR indikátor: Q4

IF: 0,9

Szatmári Szabolcs, Hadnagy Viktória, Tóth Adrián, **Jarecsny Tamás**, Peták István, Mechtler László, Schwab Richárd

A bélmikrobiom szerepe a primer stroke-prevencióban, avagy az esszenciális magas vérnyomás oki kezelésének gyakorlati szempontjai

HÁZIORVOS TOVÁBBKÉPZŐ SZEMLE 28: 6 pp. 287-291. (2023)

Közlemény: 34453479 | Összefoglaló cikk (Folyóiratcikk) | Tudományos

Béres-Molnár AK, Simonné-Mátis RK, Al-Muhanna N, **Jarecsny T**, Dudás E, Jánoska D, Pálosi M, Toldi G, Folyovich A

Date of birth and the incidence of acute ischemic stroke in Hungary

REVISTA DE NEUROLOGIA 75: 6 pp. 143-147. (2022)

Közlemény: 33111199 | Szakcikk (Folyóiratcikk) | Tudományos

Scopus - Medicine (miscellaneous) SJR indikátor: Q3

Scopus - Neurology (clinical) SJR indikátor: Q3

IF: 1,2

Jarecsny Tamás, Trencséni Dalma, Peták István, Mechtler László, Frecska Ede, Schwab Richard

A mikrobiom-asszociált gyulladás és a migrén: a bél-agy tengely újabb állomása?

CENTRAL EUROPEAN JOURNAL OF GASTROENTEROLOGY AND HEPATOLOGY / GASZTROENTEROLÓGIAI ÉS HEPATOLÓGIAI SZEMLE 8: 1 pp. 17-22. (2022)

Közlemény: 33583650 | Összefoglaló cikk (Folyóiratcikk) | Tudományos

Schwab Richárd, Bán Orsolya, Stella Péter, **Jarecsny Tamás**, Turóczi Fanny, Peták István, Kempler Péter

A humán mikrobiom kóroki szerepe a 2-es típusú diabetes és annak szövődményspektruma kialakulásában

MAGYAR BELORVOSI ARCHIVUM 75: 5-6 pp. 210-217. (2022)

Közlemény: 33583653 | Összefoglaló cikk (Folyóiratcikk) | Tudományos

Al-Muhanna Nadim, Béres-Molnár Katalin Anna, **Jarecsny Tamás**, Folyovich András

Covid-19-asszociált Guillain–Barré-szindróma első hazai esete [The first Hungarian patient with Guillain-Barre syndrome after COVID-19]

IDEGGYOGYASZATI SZEMLE / CLINICAL NEUROSCIENCE 74: 1-2 pp. 64-66. (2021)

Közlemény: 31876152 | Rövid közlemény (Folyóiratcikk) | Tudományos

Scopus - Neurology (clinical) SJR indikátor: Q4

Scopus - Neurology SJR indikátor: Q4

IF: 0,708

Folyovich András, Matis Réka, Al-Muhanna Nadim, **Jarecsny Tamás**, Dudás Eszter, Jánoska Dorottya, Palosi Mihály, Béres-Molnár Anna K, Toldi Gergely

Christmas, acute ischemic stroke and stroke-related mortality in Hungary

BRAIN AND BEHAVIOR 11: 5 Paper: e02104, 6 p. (2021)

Közlemény: 31951854 | Szakcikk (Folyóiratcikk) | Tudományos

Scopus - Behavioral Neuroscience SJR indikátor: Q2

IF: 3,405

Folyovich A, Biczó D, **Jarecsny T**, Al-Muhanna N, Jánoska D, Béres-Molnár KA, Dudás E, Toldi G

Daylight saving time and the incidence of thrombolysis to treat acute ischemic stroke

REVUE NEUROLOGIQUE 176: 5 pp. 361-365. (2020)

Közlemény: 31396243 | Szakcikk (Folyóiratcikk) | Tudományos

Scopus - Neurology (clinical) SJR indikátor: Q2

Scopus - Neurology SJR indikátor: Q3

IF: 2,607

Folyovich András, Majoros Angéla, **Jarecsny Tamás**, Pánczél Gitta, Pápai Zsuzsanna, Rudas Gábor, Kozák Lajos, Barna Gábor, Béres-Molnár Katalin A, Vadasdi Károly, Liszkay Gabriella, Horváth Eszter, Toldi Gergely

Epileptic Seizure Provoked by Bone Metastasis of Chronic Lymphoid Leukemia and Merkel Cell Carcinoma

CASE REPORTS IN MEDICINE 2020 Paper: 4318638, 4 p. (2020)

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Scopus - Medicine (miscellaneous) SJR indikátor: Q4

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