STARZYK, Agnieszka and CHARZEWSKI, Piotr. Understanding Obesity and the Role of Pharmacological Treatments: Insights for Health and Fitness Enthusiasts. Quality in Sport. 2025;38:58211. eISSN 2450-3118.
https://doi.org/10.12775/QS.2025.38.58211
https://apcz.umk.pl/QS/article/view/58211

The journal has been 20 points in the Ministry of Higher Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Higher Education and Science of 05.01.2024. No. 32553.
Has a Journal's Unique Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 - aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 r. Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398.
Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 19.01.2025. Revised: 02.02.2025. Accepted: 07.02.2025 Published: 07.02.2025.

Understanding Obesity and the Role of Pharmacological Treatments: Insights for Health and Fitness Enthusiasts

Agnieszka Starzyk
Medical University of Warsaw
Żwirki i Wigury 61, 02-091 Warsaw, Poland
astarz.st@gmail.com
https://orcid.org/0009-0002-8696-4187

Piotr Charzewski
Kozminski University
Jagiellońska 57, 03-301 Warsaw, Poland
charzewskip@gmail.com
https://orcid.org/0009-0007-5170-3899

ABSTRACT

Introduction: Obesity results from disrupted central mechanisms regulating body weight, influenced by the interplay between genetic predisposition and environmental factors. Genetic forms of obesity, including monogenic and syndromic types, represent rare and complex neuroendocrine disorders where genetic contributions play a dominant role. Characterized by severe, early-onset obesity often accompanied by eating disorders and multiple comorbidities, these conditions pose significant diagnostic and therapeutic challenges. Historically, the management of genetic obesity has relied predominantly on lifestyle interventions, focusing on nutrition and physical activity. However, recent advancements in therapeutic options, including targeted pharmacological treatments, offer new hope for improving outcomes and quality of life in affected individuals. These developments underscore the importance of integrating genetic diagnostics into clinical practice to enable personalized care approaches. This review explores the current strategies for managing genetic obesity, examines the evidence underpinning these approaches, and provides an overview of emerging therapies under evaluation.

Materials and Methods: This synthesis is based on a comprehensive review of three academic articles addressing obesity subtypes, genetic obesity, and their associated treatment strategies. A systematic approach was employed to identify relevant studies from PubMed and other databases, focusing on publications within the last two decades. The search terms included "obesity subtypes," "genetic obesity," "cardiometabolic risk," "biomarkers," and "treatment strategies." Articles were selected based on their relevance to obesity classification, molecular biomarkers, and therapeutic approaches.

Conclusions: The reviewed articles underscore the importance of understanding obesity as a heterogeneous condition requiring tailored medical approaches. Obesity subtypes, including metabolically healthy, metabolically abnormal, and syndromic obesity, exhibit distinct pathophysiological mechanisms and risk profiles, necessitating personalized treatment strategies. Advances in medical treatments, such as GLP-1 receptor agonists, MC4R agonists, and other targeted pharmacotherapies, have shown promise in addressing specific obesity subtypes, particularly genetic and syndromic forms. These therapies, combined with lifestyle and surgical interventions, represent a significant step forward in managing obesity more effectively.

The integration of genetic diagnostics into routine clinical practice is critical to identifying suitable candidates for these therapies and optimizing outcomes. Continued research into the molecular mechanisms underlying obesity and its subtypes is essential to refine medical treatments and improve their accessibility and efficacy. Such efforts will contribute to reducing the burden of obesity and improving the quality of life for affected individuals.

Keywords: obesity, body fat, biomarkers, cardiovascular diseases, heart failure, diabetes mellitus, genetic obesity

INTRODUCTION

Obesity has emerged as one of the most significant global public health challenges, with its prevalence increasing dramatically over the past several decades. Characterized by excessive or abnormal fat accumulation, obesity is a multifaceted condition associated with an elevated risk of chronic diseases, including type 2 diabetes mellitus (T2DM), cardiovascular disease (CVD), dyslipidemia, hypertension, and certain cancers [1, 2]. Although body mass index (BMI) is widely used to define and classify obesity, it has well-documented limitations, as it does not account for variations in fat distribution, muscle mass, or the underlying metabolic profile of individuals [5, 6]. Nevertheless, BMI remains a convenient metric, with a range of 18.5–24.9 kg/m² considered normal, 25–29.9 kg/m² categorized as overweight, and ≥30 kg/m² classified as obese. While BMI provides a general framework for assessing weight status, it often fails to differentiate between individuals with varying metabolic and clinical profiles within the same category [6, 14].

Growing evidence highlights the importance of recognizing obesity as a heterogeneous condition encompassing distinct subtypes, each with unique metabolic, genetic, and clinical characteristics [1, 2, 3]. These subtypes include metabolically healthy obese (MHO), metabolically abnormal obese (MAO), metabolically obese normal weight (MONW), and sarcopenic obese (SO). For instance, individuals with MHO maintain a normal metabolic profile despite increased adiposity, while those with MONW exhibit metabolic abnormalities such as insulin resistance and dyslipidemia despite having a normal BMI [8, 9]. Sarcopenic obesity, characterized by low muscle mass combined with excess fat, is particularly associated with aging populations and carries additional risks of functional impairment and frailty [10, 13].

Understanding the molecular mechanisms underlying these subtypes has been a focus of research, with particular attention given to biomarkers such as adipokines (e.g., adiponectin, leptin), inflammatory markers (e.g., C-reactive protein, interleukin-6), and genetic factors identified through genome-wide association studies (GWAS) [1, 7, 11]. These markers offer insights into the diverse metabolic pathways contributing to obesity phenotypes, though standardization in their diagnostic use remains limited [2, 12]. Environmental and lifestyle factors, including diet, physical activity, and psychosocial stressors, further modulate the expression and progression of obesity subtypes [3,18].

Despite advances in pharmacological treatments for obesity, including the use of GLP-1 receptor agonists and MC4R agonists, medical management alone is often insufficient to address the multifactorial nature of the disease [3, 23]. Combining medical treatments with structured exercise programs offers a promising avenue for improving outcomes in individuals with different obesity phenotypes. Regular physical activity not only enhances the efficacy of pharmacotherapy by improving metabolic flexibility, lipid profiles, and insulin sensitivity but also mitigates the risk of comorbidities associated with obesity, such as CVD and musculoskeletal disorders [1, 20].

This review explores the diverse subtypes of obesity, their associated biomarkers, and the underlying genetic and molecular pathways. Emphasis is placed on integrating medical treatments and exercise as a synergistic approach to managing obesity and its related health risks. A combined strategy that incorporates pharmacological interventions with personalized exercise regimens offers the potential to achieve more effective and sustainable outcomes for individuals across various obesity subtypes [3, 9, 23].

MATERIALS AND METHODS

This review synthesizes findings from three comprehensive research articles on obesity subtypes, associated biomarkers, and therapeutic strategies [1, 3, 9]. A systematic approach was employed to identify relevant literature through database searches in PubMed, Embase, and Web of Science. Keywords used in the search included “obesity subtypes,” “metabolically healthy obese,” “biomarkers in obesity,” “genetic obesity,” “cardiometabolic risk,” and “obesity treatment strategies.” Studies published in English over the past two decades, focusing on adult populations, were considered [6, 18].

Inclusion criteria prioritized peer-reviewed articles that explored distinct obesity phenotypes, their underlying molecular mechanisms, and clinical management approaches, including pharmacological and lifestyle interventions [3, 7, 18]. Studies employing genome-wide association studies (GWAS), epigenetic analyses, and biomarker identification to characterize obesity subtypes were particularly emphasized [1, 7]. Additionally, research addressing the integration of exercise with medical treatments for obesity was included [10, 23].

Data from the selected articles were analyzed to highlight key findings regarding the role of biomarkers such as adipokines (e.g., leptin, and adiponectin) and inflammatory markers (e.g., interleukin-6, and C-reactive protein) in distinguishing obesity subtypes [1, 11, 19]. Information on pharmacological interventions, including GLP-1 receptor agonists and MC4R agonists, and their effectiveness in treating specific subtypes of obesity was synthesized [3, 23]. The role of structured exercise regimens as an adjunct to medical therapy was also reviewed to provide a comprehensive understanding of combined treatment strategies [10, 20].

The methodologies of the included studies were critically evaluated, focusing on sample size, study design, and analytical techniques. This synthesis aims to integrate findings on obesity subtypes, biomarkers, and therapeutic approaches to offer a holistic perspective on managing obesity and its related comorbidities [3, 23].

RESULTS

Obesity is increasingly recognized as a heterogeneous condition, encompassing distinct phenotypes that differ in their metabolic, genetic, and clinical characteristics [1, 2, 3]. Key obesity subtypes include metabolically healthy obese (MHO), metabolically abnormal obese (MAO), metabolically obese normal weight (MONW), and sarcopenic obese (SO) [3, 9, 10]. These phenotypes reflect the diverse pathophysiology and risk profiles within obesity, underscoring the limitations of BMI as a sole diagnostic criterion [5, 6].

Cardiometabolic imaging studies reviewed in this article emphasize the need for a more nuanced understanding of obesity in the context of precision medicine [1, 2, 15]. While BMI remains a useful initial metric for classifying individuals into weight categories, it is insufficient to fully capture the complexities of obesity-related health risks. Adding waist circumference as an additional measure significantly enhances the assessment of cardiovascular risk by identifying individuals with central or abdominal fat accumulation, which is strongly associated

with visceral obesity and ectopic fat deposition [1, 9]. The combination of an elevated waistline and features of metabolic syndrome serves as a reliable indicator of higher cardiometabolic risk [1, 15].

Given the limited resources available for managing obesity, prioritizing the clinical management of high-risk groups, including overweight and moderately obese individuals with central adiposity, is critical [9, 16]. At the same time, the growing prevalence of severe obesity warrants particular attention due to its substantial health burden and associated comorbidities. These findings underscore the importance of moving away from a one-size-fits-all approach to obesity and adopting the principles of precision medicine. By recognizing obesity as a collection of distinct subtypes—or "obesities"—rather than a single condition, healthcare providers can better tailor interventions to meet the specific needs of diverse patient populations [1, 9 , 17].

Phenotypes and Characteristics

1. **Metabolically Healthy Obese (MHO):**

MHO individuals exhibit excess adiposity but maintain a normal metabolic profile, including insulin sensitivity, normal blood pressure, and favorable lipid levels. Despite their apparent health, longitudinal studies suggest that many MHO individuals transition to metabolically abnormal states over time, particularly if obesity persists [2, 9, 15].

2. **Metabolically Abnormal Obese (MAO):**

MAO is characterized by obesity accompanied by metabolic dysfunction, including insulin resistance, dyslipidemia, systemic inflammation, and hypertension. These individuals face an elevated risk of developing cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) [2, 3].

3. **Metabolically Obese Normal Weight (MONW):**

MONW individuals have a normal BMI but exhibit metabolic abnormalities associated with visceral adiposity and ectopic fat deposition. This phenotype challenges traditional notions of

obesity, as individuals with normal weight can experience significant metabolic and cardiovascular risks [8, 9].

4. **Sarcopenic Obese (SO):**

SO combines low muscle mass with excess adiposity, primarily affecting older adults. This phenotype is associated with impaired physical function, frailty, and increased cardiometabolic risk, further complicating its management [10, 13].

Individuals with severe obesity (BMI ≥40 kg/m²), who may be 100 to 200 pounds (45–90 kg) or more overweight, face significant cardiovascular challenges that complicate their management within healthcare systems. Approximately one-third of individuals with severe obesity exhibit clinical evidence of heart failure, with the risk of developing heart failure increasing markedly with the duration of obesity. Prevalence rates exceed 70% after 20 years and rise to 90% after 30 years of severe obesity [15]. This condition profoundly affects cardiac structure and function, contributing to left ventricular remodeling, reduced systolic function, and diastolic dysfunction, which can ultimately progress to heart failure. Additionally, severe obesity is associated with systemic metabolic, inflammatory, and neurohormonal alterations that further impair cardiovascular health. Histological analyses reveal cardiomyocyte hypertrophy, myocardial fat infiltration, and fibrosis in the hearts of individuals with severe obesity. Furthermore, increased pulmonary vascular resistance and elevated pulmonary artery pressure result in higher right ventricular afterload, leading to right ventricular dysfunction [1-18].

Weight loss, achieved through lifestyle changes, pharmacotherapy, or surgical interventions, has been shown to improve cardiac structure and function, potentially reversing remodeling and reducing cardiovascular risk. Bariatric surgery, in particular, has demonstrated significant benefits in decreasing cardiac morbidity and mortality among patients with severe obesity and pre-existing cardiac disease. Although current heart failure guidelines place limited emphasis on weight reduction, they acknowledge the elevated risk posed by severe obesity [8-17]. The likelihood of developing atrial fibrillation also rises significantly with increasing BMI, with a BMI ≥30 kg/m² associated with a 49% higher lifetime risk of atrial fibrillation. Among individuals with severe obesity, those undergoing weight-loss surgery exhibit lower rates of new-onset atrial fibrillation compared to those receiving standard care. Severe obesity is also linked to higher mortality rates following in-hospital cardiac arrest, particularly in cases

involving nonventricular or ventricular fibrillation arrhythmias. These findings highlight the critical importance of effective weight management strategies in mitigating the cardiovascular complications associated with severe obesity [22-31].

Treatment Strategies and Considerations

Despite advancements in medical management and lifestyle interventions, clinical outcomes for patients with severe obesity and associated cardiac disease or heart failure remain suboptimal [23, 25]. Treatment options for severe obesity include lifestyle modification therapy, pharmacotherapy, and bariatric surgery. While lifestyle modifications can effectively improve cardiovascular risk factors and comorbid conditions, they often fail to produce sustainable weight loss or long-lasting metabolic improvements. For instance, the Look AHEAD trial demonstrated that individuals with severe obesity who achieved a 4-year weight loss of ≥10% of their initial body weight experienced significant cardiovascular benefits. However, only 25% of participants in the trial maintained this level of weight loss, while the majority (75%) regained weight within the same period [3, 23].

Pharmacological treatments, although beneficial in certain cases, similarly struggle to produce durable results, with weight loss frequently regained within 12 months of treatment cessation. Lifestyle interventions do, however, play an essential role in supporting long-term weight maintenance, particularly when combined with bariatric surgery. Prospective studies have shown that lifestyle interventions implemented within 12 months of bariatric surgery, whether pre- or postoperatively, can enhance both weight loss and its maintenance over time. For example, a structured lifestyle intervention program initiated after bariatric surgery has been shown to significantly improve weight loss outcomes [1 - 6].

Although lifestyle modification alone may not be sufficient for addressing severe obesity, its integration with bariatric surgery highlights its critical role in comprehensive obesity management. By supporting sustained behavioral changes, lifestyle interventions can amplify the benefits of surgical treatments, contributing to improved long-term health outcomes for individuals with severe obesity.

1. **Lifestyle Interventions:**

Exercise and dietary modifications are foundational to managing all obesity subtypes. For MHO, these interventions aim to maintain metabolic health, while for MAO and MONW, they address underlying inflammation and insulin resistance. Resistance training is particularly beneficial for SO, improving muscle mass and physical function. However, lifestyle interventions alone often fall short for severe obesity or in individuals with significant metabolic dysfunction, necessitating adjunct therapies [18, 20].

2. **Pharmacological Treatments:**

Emerging pharmacotherapies, including GLP-1 receptor agonists (e.g., liraglutide, semaglutide) and MC4R agonists (e.g., setmelanotide), have shown promise in managing obesity. These treatments are particularly effective in MAO and genetic obesity, as they target appetite regulation and energy expenditure. While these drugs offer significant weight reduction and metabolic improvements, their high costs and potential side effects, such as gastrointestinal discomfort, limit widespread accessibility and patient adherence [23, 25].

3. **Bariatric Surgery:**

For severe obesity or refractory cases, bariatric surgery remains a highly effective treatment, particularly for MAO and SO phenotypes. Procedures such as gastric bypass and sleeve gastrectomy not only reduce weight but also improve insulin sensitivity and lipid profiles. However, surgery carries inherent risks, including complications and long-term nutritional deficiencies, necessitating careful patient selection and follow-up [23-31].

Summary of Benefits and Limitations

Each treatment strategy offers unique advantages and limitations depending on the obesity phenotype. Lifestyle interventions are safe and broadly applicable but may have limited efficacy in advanced or severe cases. Pharmacological treatments provide targeted benefits for metabolic dysfunction but can be costly and associated with side effects. Bariatric surgery is highly effective but invasive and requires substantial post-operative commitment. A phenotype-specific approach that combines these strategies, tailored to individual needs, holds the greatest potential for improving outcomes and reducing the burden of obesity-related comorbidities.

Results Summary

Until recently, the management of syndromic and monogenic obesity primarily relied on multicomponent lifestyle interventions, which remain the cornerstone of clinical care [3, 23]. These interventions, focusing on dietary modifications, physical activity, and behavioral therapies, aim to address the complex needs of these patients. However, the emergence of innovative, targeted treatments in recent years has significantly altered this paradigm, paving the way for a more personalized approach to managing these rare and complex conditions [3, 7, 18]. Advances in pharmacotherapy, including the development of drugs targeting specific genetic pathways, represent a promising alternative to traditional methods, offering hope for improved outcomes in this challenging patient population.

Bariatric surgery, once considered the only effective option for severe cases, now faces competition from pharmaceutical interventions. While surgery remains a viable treatment for some, the potential for irreversible anatomical changes and uncertain long-term outcomes necessitates caution in its application, especially in younger patients [26]. Targeted pharmacological treatments, such as those modulating the leptin-melanocortin pathway, may offer safer and more reversible alternatives. However, the precise role of each treatment in the management of syndromic and monogenic obesity remains unclear, requiring further research to establish evidence-based guidelines tailored to these conditions [23].

A major priority for improving outcomes in this population is early and accurate genetic diagnosis. Identifying the genetic basis of obesity in children who exhibit rapid weight gain and other suggestive clinical features is crucial, as it allows timely access to specialized multidisciplinary care, novel therapeutic agents, and clinical trials [3, 7]. Early intervention can prevent the progression of obesity-related complications, reduce the likelihood of failure with conservative treatments, and alleviate the stigma faced by patients and their families. Genetic testing should be routinely offered to children with severe obesity and associated syndromic features to ensure they receive appropriate and timely care [18].

CONCLUSIONS

Intensive lifestyle interventions remain an integral component of management, particularly when delivered in outpatient settings close to the patient’s home. These programs can help mitigate the progression of obesity-related complications and improve quality of life. Specific

healthcare pathways, such as those implemented in France, provide a structured framework for integrating lifestyle interventions with specialized care, offering a model for improving long-term outcomes in patients with syndromic or monogenic obesity [1-3].

Ongoing research continues to produce new therapeutic options, fostering hope for the future. Advances in gene-editing technologies, such as CRISPR-mediated gene editing and induced pluripotent stem cell therapies, hold significant potential for addressing the root causes of monogenic obesity. These approaches could enable direct correction of defective genes, offering a curative option for patients with severe genetic forms of obesity. While these technologies remain experimental, they underscore the importance of collaboration between clinicians and scientists in developing innovative solutions [23].

The clinical severity and lifelong impact of syndromic and monogenic obesity necessitate a comprehensive and multidisciplinary approach. Greater involvement from healthcare providers, researchers, and policymakers is essential to optimize treatment strategies, improve access to care, and ensure better prognoses for affected individuals. Through continued innovation and cooperation, it is possible to address the unique challenges faced by this vulnerable population and enhance their overall health and well-being [31].

Author`s contribution:

Conceptualization: Agnieszka Starzyk
Methodology: Agnieszka Starzyk, Piotr Charzewski
Software: Piotr Charzewski
Check: Agnieszka Starzyk
Formal analysis: Agnieszka Starzyk, Piotr Charzewski
Investigation: Piotr Charzewski
Resources: Agnieszka Starzyk, Piotr Charzewski
Data curation: Agnieszka Starzyk
Writing-rough preparation: Agnieszka Starzyk
Writing-review and editing: Piotr Charzewski
Supervision: Agnieszka Starzyk
Project administration: Agnieszka Starzyk

All authors have read and agreed with the published version of the manuscript.

Founding Statement: The study did not receive funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Conflict of Interest Statement: The authors declare no conflicts of interest.

Acknowledgments: Not applicable.

Bibliography:

1. Mayoral LP, Andrade GM, Mayoral EP, Huerta TH, Canseco SP, Rodal Canales FJ, Cabrera-Fuentes HA, Cruz MM, Pérez Santiago AD, Alpuche JJ, Zenteno E, Ruíz HM, Cruz RM, Jeronimo JH, Perez-Campos E. Obesity subtypes, related biomarkers & heterogeneity. Available from: https://pubmed.ncbi.nlm.nih.gov/32134010/
2. Piché ME, Tchernof A, Després JP. Obesity Phenotypes, Diabetes, and Cardiovascular Diseases. Available from: https://pubmed.ncbi.nlm.nih.gov/32437302/
3. Faccioli N, Poitou C, Clément K, Dubern B. Current Treatments for Patients with Genetic Obesity. Available from: https://pubmed.ncbi.nlm.nih.gov/37191347/
4. Shukla A, Kumar K, Singh A. Association between obesity and selected morbidities: a study of BRICS countries. Available from: https://pubmed.ncbi.nlm.nih.gov/24718033/
5. Romero-Corral A, Somers VK, Sierra-Johnson J, Thomas RJ, Collazo-Clavell ML, Korinek J, Allison TG, Batsis JA, Sert-Kuniyoshi FH, Lopez-Jimenez F. Accuracy of body mass index in diagnosing obesity in the adult general population. Available from: https://pubmed.ncbi.nlm.nih.gov/18283284/
6. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: executive summary. Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults. Available from: https://pubmed.ncbi.nlm.nih.gov/9771869/
7. Zhang YP, Zhang YY, Duan DD. From Genome-Wide Association Study to Phenome-Wide Association Study: New Paradigms in Obesity Research. Available from: https://pubmed.ncbi.nlm.nih.gov/27288830/
8. Du T, Yu X, Zhang J, Sun X. Lipid accumulation product and visceral adiposity index are effective markers for identifying the metabolically obese normal-weight phenotype. Available from: https://pubmed.ncbi.nlm.nih.gov/25690647/
9. Conus F, Rabasa-Lhoret R, Péronnet F. Characteristics of metabolically obese normal-weight (MONW) subjects. Available from: https://pubmed.ncbi.nlm.nih.gov/17332780/

10. Lee DC, Shook RP, Drenowatz C, Blair SN. Physical activity and sarcopenic obesity: definition, assessment, prevalence and mechanism. Available from: https://pubmed.ncbi.nlm.nih.gov/28031974/
11. Phillips CM, Perry IJ. Does inflammation determine metabolic health status in obese and nonobese adults? Available from: https://pubmed.ncbi.nlm.nih.gov/23979951/
12. Kjaer IG, Kolle E, Hansen BH, Anderssen SA, Torstveit MK. Obesity prevalence in Norwegian adults assessed by body mass index, waist circumference and fat mass percentage. Available from: https://pubmed.ncbi.nlm.nih.gov/26153357/
13. Sakuma K, Yamaguchi A. Sarcopenic obesity and endocrinal adaptation with age. Available from: https://pubmed.ncbi.nlm.nih.gov/23690769/
14. Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. Available from: https://pubmed.ncbi.nlm.nih.gov/22106927/
15. Bradshaw PT, Monda KL, Stevens J. Metabolic syndrome in healthy obese, overweight, and normal weight individuals: the Atherosclerosis Risk in Communities Study. Available from: https://pubmed.ncbi.nlm.nih.gov/23505187/
16. Smith U. Abdominal obesity: a marker of ectopic fat accumulation. Available from: https://pubmed.ncbi.nlm.nih.gov/25932676/
17. Keys A, Fidanza F, Karvonen MJ, Kimura N, Taylor HL. Indices of relative weight and obesity. Available from: https://pubmed.ncbi.nlm.nih.gov/24691951/
18. Dombrowski SU, Knittle K, Avenell A, Araújo-Soares V, Sniehotta FF. Long term maintenance of weight loss with non-surgical interventions in obese adults: systematic review and meta-analyses of randomised controlled trials. Available from: https://pubmed.ncbi.nlm.nih.gov/25134100/
19. Yoshikane H, Yamamoto T, Ozaki M, Matsuzaki M. [Clinical significance of high-sensitivity C-reactive protein in lifestyle-related disease and metabolic syndrome]. Available from: https://pubmed.ncbi.nlm.nih.gov/17941193/
20. Ricciardi R, Talbot LA. Use of bioelectrical impedance analysis in the evaluation, treatment, and prevention of overweight and obesity. Available from: https://pubmed.ncbi.nlm.nih.gov/17489956/
21. Lin D, Chun TH, Kang L. Adipose extracellular matrix remodelling in obesity and insulin resistance. Available from: https://pubmed.ncbi.nlm.nih.gov/27179976/
22. Anhê FF, Jensen BAH, Varin TV, Servant F, Van Blerk S, Richard D, Marceau S, Surette M, Biertho L, Lelouvier B, Schertzer JD, Tchernof A, Marette A. Type 2 diabetes influences

bacterial tissue compartmentalisation in human obesity. Available from: https://pubmed.ncbi.nlm.nih.gov/32694777/

23. Sjöström L, Peltonen M, Jacobson P, Sjöström CD, Karason K, Wedel H, Ahlin S, Anveden Å, Bengtsson C, Bergmark G, Bouchard C, Carlsson B, Dahlgren S, Karlsson J, Lindroos AK, Lönroth H, Narbro K, Näslund I, Olbers T, Svensson PA, Carlsson LM. Bariatric surgery and long-term cardiovascular events. Available from: https://pubmed.ncbi.nlm.nih.gov/22215166/
24. Warnes CA, Roberts WC. The heart in massive (more than 300 pounds or 136 kilograms) obesity: analysis of 12 patients studied at necropsy. Available from: https://pubmed.ncbi.nlm.nih.gov/6496330/
25. Ashrafian H, le Roux CW, Darzi A, Athanasiou T. Effects of bariatric surgery on cardiovascular function. Available from: https://pubmed.ncbi.nlm.nih.gov/19001033/
26. Aggarwal R, Harling L, Efthimiou E, Darzi A, Athanasiou T, Ashrafian H. The Effects of Bariatric Surgery on Cardiac Structure and Function: a Systematic Review of Cardiac Imaging Outcomes. Available from: https://pubmed.ncbi.nlm.nih.gov/26328532/
27. Rider OJ, Francis JM, Tyler D, Byrne J, Clarke K, Neubauer S. Effects of weight loss on myocardial energetics and diastolic function in obesity. Available from: https://pubmed.ncbi.nlm.nih.gov/23269470/
28. Morricone L, Malavazos AE, Coman C, Donati C, Hassan T, Caviezel F. Echocardiographic abnormalities in normotensive obese patients: relationship with visceral fat. Available from: https://pubmed.ncbi.nlm.nih.gov/12055325/
29. Wong CY, O'Moore-Sullivan T, Leano R, Hukins C, Jenkins C, Marwick TH. Association of subclinical right ventricular dysfunction with obesity. Available from: https://pubmed.ncbi.nlm.nih.gov/16458145/
30. Michaud A, Grenier-Larouche T, Caron-Dorval D, Marceau S, Biertho L, Simard S, Richard D, Tchernof A, Carpentier AC. Biliopancreatic diversion with duodenal switch leads to better postprandial glucose level and beta cell function than sleeve gastrectomy in individuals with type 2 diabetes very early after surgery. Available from: https://pubmed.ncbi.nlm.nih.gov/28764844/
31. Angrisani L, Santonicola A, Iovino P, Vitiello A, Zundel N, Buchwald H, Scopinaro N. Bariatric Surgery and Endoluminal Procedures: IFSO Worldwide Survey 2014. Available from: https://pubmed.ncbi.nlm.nih.gov/28405878/