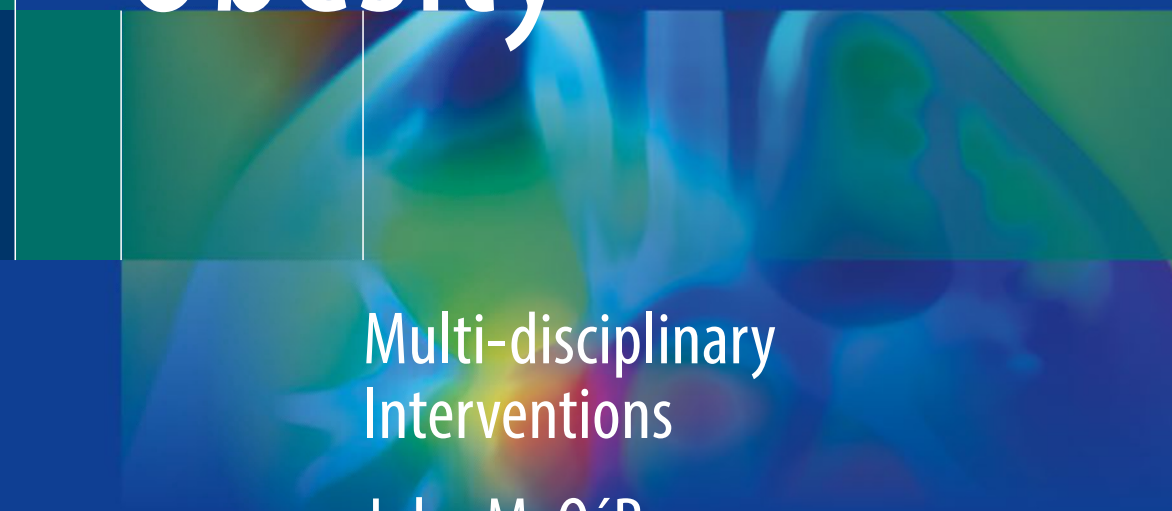
Musculoskeletal Disease and Obesity



Multi-disciplinary Interventions

John M. O´Byrne
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To my wife, Dr. Fiona Kearns, who has filled my life with joy.

—John M. O'Byrne

To my wife Claire and my children Oscar, Quinn, Molly, and Teddy: these are the days!

—Fiachra Rowan

To my wife Leann and my children Eve and Rebecca. Thank you for everything.

—Alan Molloy

Foreword

With obesity reaching epidemic proportions in many developed countries, there are significant implications on how we approach the surgical treatment of orthopaedic patients. Obesity is an independent risk factor for the development of osteoarthritis. As its prevalence rises so does the need for joint replacements and orthopaedic interventions in obese patients.

A significant issue is that the obese patient population provides unique challenges for successful surgical treatment. Physically, due to the patient's size and body habitus, the surgical approach and exposure, positioning of the patient, and execution of the procedure are significantly more challenging compared to non-obese patients. These physical challenges mandate the use of more resources in their care. Medically, obese patients have more comorbidities, putting them at risk for adverse surgical outcomes. These comorbidities, including sleep apnea diabetes, and cardiovascular issues require higher levels of care and preoperative evaluation. While there is no clear consensus, some studies have shown that these patients may be subject to higher rates of complications and poor outcomes.

A thorough understanding of the challenges in execution and management of surgery in the obese is required for successful outcomes. It is not adequate to simply repeat the usual steps performed in the average patient. The authors present a thoughtful consideration of the technical aspects necessary for successful management of obese patients and how to overcome the challenges inherent in their care. This book gives a complete review on orthopaedic subspecialty and a review of implications for anaesthesia and rehabilitation issues. For clinicians caring for the obese, this book provides an essential strategic outline for success.

Hospital for Special Surgery
New York, NY, USA

Michael L. Parks

Preface

As editors, we are delighted to present this textbook outlining the range of musculoskeletal challenges in patients living with obesity.

The epidemic of obesity is well described and increasingly realised to be a challenge for healthcare professionals and healthcare providers. The early chapters in this textbook look at the epidemiology and pathogenesis and also examine medical and surgical treatments. The musculoskeletal chapters focus on the different parts of the body where obesity contributes to the pathology and complicates the management. Specific chapters focus on paediatrics, oncology, and sports injuries. Imaging of patients living with obesity and also anaesthetic issues arising in this group of patients are highlighted. Rehabilitation of patients living with obesity is also described.

Obesity continues to challenge patients, clinicians, and providers, and we hope this textbook will prove a useful guide to anyone involved in the care of musculoskeletal conditions that arise in this patient population.

We thank all the chapter authors, and we particularly acknowledge the invaluable support of Ms. Ursula Gormally in the preparation of this textbook.

Dublin, Ireland — John M. O'Byrne
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Pathogenesis, Prevalence and Epidemiology of Obesity

1

Brian Quinn, Laura Browne, and Donal O'Shea

1.1 Introduction

Obesity is defined by the World Health Organization (WHO) as an excessive accumulation of body fat which presents a risk to health [1]. The WHO recommend measuring a person's body fat content using the body mass index (BMI) calculation [1, 2]. The different classifications of weight are shown in Table 1.1.

$$\text{BMI} = \frac{\text{Weight}\left(\text{in kg}\right)}{\left(\text{Height in Metres}\right)^2}$$

Table 1.1 WHO BMI ranges and classifications [1]

WHO classification	BMI range
Underweight	<18.4
Normal	18.5–24.9
Overweight	25–29.9
Obese	>30

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Although BMI is the gold standard used for assessing obesity, it has several flaws with its implementation. BMI measures body mass but does not distinguish between lean body mass and fat body mass. Therefore, using BMI to measure an athlete or bodybuilder would lead that individual to be classified as clinically obese. Additionally, the ranges specified by the WHO for BMI do not adequately account for height variability that occurs between phenotypes. This means that BMI can be properly employed as a measuring tool in America and Europe [3–6] while it fails to adequately assess obesity prevalence in Asia [1, 2]. A proposed remedy for this is to lower the cutoff ranges of overweight and obese BMI limits so that they reflect the country's average height ranges [7].

Alternative methods of measuring body fat are body fat percentage, which more accurately assesses fat content, and hip:waist ratio. However, these measurements are not broadly employed to assess a person's body fat content [2].

1.2 Incidence and Prevalence of Obesity in the Modern Day

Obesity has been recognised as a risk to health since the nineteenth century [1], and yet the prevalence of obesity has gradually increased over the past 100 years. As of 2016, almost 2 billion people were found to be overweight worldwide [14]. Current projections have the global population reaching 1 billion obese adults by 2025 [14]. The continents with the highest obese population are shown in Table 1.2.

As the prevalence of obesity rises globally, so does its disease burden. Obesity has been recognised as a pro-inflammatory condition which can in turn promote the development of musculoskeletal disease, type 2 diabetes and specific cancers [1–4]. Furthermore, it is a significant contributor to cardiovascular disease and respiratory disorders. Additionally, a frequently overlooked aspect of obesity is the effect it has on an individual's mental health. As of 2014 the economic burden of obesity was estimated at US$2 trillion globally [17]. This figure was generated based on the number of days missed due to sick leave, combined with the cost of medical interventions for individuals in which obesity was a contributing factor to their illness.

Table 1.2 Prevalence of obesity globally as of 2015 [7]

	Geographical location	Overweight prevalence as of 2015 (%)	Obese prevalence as of 2015 (%)
1	Americas	64.2	28.3
2	Europe	59.6	22.9
3	East Mediterranean	49.6	19.6
4	Africa	34	12.7
5	West Pacific	28.2	4.9
6	Southeast Asia	22	5

1.3 Pathogenesis

The metabolic pathway for the generation of fat is called lipogenesis. It begins in the brain, where hormones regulating starvation/satiety levels in the body will dictate whether the body is hungry or not. The individual will feel hungry and eat [7, 8]. Food is then metabolised. The process by which the body will turn ingested nutrients into energy is called the Krebs cycle [8]. This will lead to the generation of energy from the breakdown of glucose.

When there is an excess of glucose, the body will then attempt to store it in the form of fat. It is the body's attempt to create stores of energy that can be utilised in times of need [8]. Glucose is converted to fatty acids which are then turned into triglycerides. This occurs in the liver and fat cells (adipocytes).

Lipogenesis has a counterpart called lipolysis. It is the dissolution of lipids to generate energy. This is how the body will access its stores of energy.

The balance between lipogenesis and lipolysis is a dynamic one. In times of abundant food access and low-energy demand, lipogenesis will prevail over lipolysis. The opposite is also true.

1.4 Factors Contributing to Obesity

1.4.1 Diet

Typically, high-calorie diets will accelerate lipogenesis and thus increase the prevalence of obesity [7–12]. Foods such as unprocessed red meats, processed meats, sugar-sweetened foods and foods with high levels of saturated fats, trans fats, dietary cholesterol and sodium have been linked with higher rates of obesity [9–12]. Similarly, foods that will reduce rates of obesity are fruits, vegetables, beans, legumes, nuts, seeds, wholegrain, milk, polyunsaturated fats, fish and food containing omega-3 and high-content dietary fibre [9–12].

Diet is highly dependent on geographical factors [9–11]. Typically, a country will eat what it can make. Countries living by the sea will have selections of seafood while in landlocked countries, seafood is more difficult to come by and therefore more expensive. Diet is also dependent on a country's socio-economic status. A rich country that cannot grow exotic fruits can import them for a high price. Conversely, a developing country will have to rely on seasonal fruits and vegetables rather than importing fruits and vegetables all year round.

A factor common to all countries is that the availability of cheap but nutrient-poor food is becoming more and more abundant [9, 10]. Processed food with long shelf lives can be made, transported and stored with relatively low cost and difficulty. The same cannot be said for fresh ingredients such as fruit. This leads there to be an ease of access to nutrient-poor, lipogenic foodstuffs [9, 10].

So, to briefly summarise, a person's diet is limited to the food that is available in that country and is further limited to what that person can afford. This means that a wealthy individual in a wealthy country will have a wide variety of food to choose from and conversely, a poor individual in a poor country will be limited to the cheaper foodstuffs available in that country [9].

Surprisingly, countries with the highest degree of food availability also have the highest rates of obesity [5]. Continents such as the Americas and Europe have the highest prevalence of obesity and contain the wealthiest countries in the world [7]. In general, rich countries will have a higher degree of obesity than poorer countries [3, 7, 11]. However, the rate of change of obesity prevalence within those countries is different [7, 11]. Rich countries are generally seeing a decline in the rate of change of obesity prevalence [7, 11]. This means that, although people are still gaining weight, there are less of them with each passing year. Conversely, in developing countries the rate of change is increasing. Some of the factors contributing to this phenomenon are access to healthcare, food education and access to better foodstuffs [7, 9].

1.4.2 Genetics

A person's genetic makeup has a variety of effects on their growth and development. Weight gain is influenced by monogenic mutations and polygenic mutations [13–16]. Monogenic mutations are single genes that differ from the standard version of the gene. Their mutation leads to their expression patterns being different to what would be considered normal. The role of these genes is so impactful that they can drastically alter an individual's phenotype [15]. Examples of monogenic mutations are as follows: leptin (LEP), leptin receptor (LEPR), proopiomelanocortin (POMC) and melanocortin 4 receptor (MC4R) [13, 14]. Possessing any one of these mutations has been directly linked to excessive lipogenesis [13, 14]. These genetic mutations are rare and usually associated with developmental disabilities such as Prader–Willi syndrome [13]. Individuals with these genetic mutations will present as obese in early life, around late childhood or adolescence [13]. These genes display a high degree of heritability, meaning that an individual with these mutations has a 40–70% chance of passing the mutations on to their offspring [15].

Polygenic genes are multiple mutations that will collectively contribute to an increase in lipogenesis [15]. They are more commonly seen and can include hundreds of single-nucleotide polymorphisms (SNPs) [15]. Compared to monogenic mutations, these polygenic mutations will have a modest effect on lipogenesis [15]. Furthermore, they have a significantly lower heritability compared to their monogenic counterparts [15]. There is evidence showing that polygenic mutations are strongly linked to environmental factors [15]. A direct comparison between the two types of influential genes is presented in Fig. 1.1.

Monogenic	Polygenic
• Early Onset disease (paediatric)	• Late onset disease
• Single gene mutation has huge impact	• Hundreds of genetic mutations have small impact
• High genetic burden/penetrance	• Low genetic burden/penetrance
• Only a handful of genes	• Hundreds of variants
• Influenced by inheritance	• Influenced by environment
• Rare	• Common

Fig. 1.1 Comparison between monogenic vs. polygenic mutations [15]

1.4.3 Current Disease Modelling

Previous disease modelling on obesity was a simple input/output model [12]. An individual will ingest a specific number of calories a day. The individual will utilise a certain amount of those calories as energy throughout the day. The remainder of the calories are stored or excreted. This model assumed a linear correlation between energy input and output. However, more recent research has shown the correlation to be more dynamic and that it is influenced by factors such as basal metabolic rate, thermic effect of feeding and physical activity [12].

This could explain why obese individuals find it so difficult to continuously lose weight with diet and exercise. When an obese individual is put on a diet and exercise regimen, they will begin to lose weight quickly then stabilise and lose a lower but continuous amount of weight [12].

One of the models currently used for weight gain/weight loss is the constrained model of energy expenditure. This model states that the body will utilise a fixed constant amount of energy within a day. This energy will be split between active and passive processes. When more active energy is required, passive processes are altered to change the energy balance in favour of active processes [12].

1.5 Overview and Summary

To recap this chapter:

- Body mass is assessed using the body mass index equation.
- Obesity is the excessive accumulation of fat tissue that presents a risk to health.
- The generation of fat tissue is called lipogenesis.