1 Obesity Reduces Life Expectancy

The effect of obesity on survival has been recognized for over 2500 years since Hippocrates first noted that “sudden death is more common in those who are naturally fat than lean.” [1] Two centuries later, the physiologist Malcolm Flemyng described obesity as a disease “because it obstructs the free exercise of the animal functions and hath a tendency to shorten life” [1]. Indeed, obesity is associated with a striking reduction in life expectancy in both adult men and women and across racial and ethnic groups [2–4]. This observation has been confirmed in several large pooled analyses of prospective studies, including a meta-analysis of over 239 studies spanning 4 continents which found that every 5 kg/m² increase in body mass index (BMI) over 25 kg/m² is associated with a 29–39% increase in all-cause mortality [5–7]. The association of obesity and mortality even extends to individuals with so-called “metabolically healthy” obesity, who do not exhibit cardiometabolic abnormalities (e.g. high waist circumference, hypertension, hypertriglyceridemia, low high-density lipoprotein, or abnormal glycemic parameters) [8]. The effect of obesity on survival is mediated by a broad range of conditions with the predominant mediators being cardiovascular diseases, respiratory diseases and cancer [7].

The direct relationship between BMI over 25 kg/m² and mortality has been challenged by some studies reporting a protective effect of overweight and/or Class I obesity in cardiovascular disease, cancer, respiratory disease, renal disease and the elderly. These observations have been termed “the obesity paradox” [9].
However, the obesity paradox is largely debunked when accounting for the methodological issues in these studies (Table 1).

### 2 Obesity and Cardiovascular Disease

Most cardiovascular disease is increased in the setting of obesity, including coronary heart disease, heart failure with reduced ejection fraction (HFrEF), heart failure with preserved ejection fraction (HFP EF), atrial fibrillation and stroke [10–11]. Obesity contributes to these diseases via both indirect and direct effects on the cardiovascular system. The indirect effects are well known and include hyperlipidemia, dyslipidemia, arterial hypertension, insulin resistance, hyperglycemia, and systemic inflammation [10]. These cardiometabolic risk factors correlate with fat mass in obesity, and particularly with visceral and ectopic fat depots that are known to have systemic metabolic effects [12].

The direct effects of obesity on cardiovascular health have received less attention in clinical care but are increasingly recognized in the literature. The epicardial fat depot, in particular, has been found to have direct lipotoxic effects on the underlying myocardium and coronary vasculature [13]. It releases inflammatory cytokines and reactive oxygen species that have paracrine and vasocrine effects creating a proatherogenic milieu. Epicardial fat may also contribute to structural and electrical remodeling leading to atrial fibrillation [3]. In addition, individuals with obesity not only have high levels of fat mass, but also have elevated fat-free mass (FFM), which is thought to be an adaptation to carrying an extra load or weight in their daily activities [10]. Increased FFM increases the circulating blood volume which, in turn, increases the left ventricular (LV) stroke volume and cardiac output, placing extra burden on the heart. This leads to altered cardiac structure and function including ventricular (both left and right) concentric hypertrophy and enlargement, left atrial enlargement, and systolic and diastolic dysfunction which can eventually manifest as obesity cardiomyopathy or congestive heart failure [10].

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### Table 1 Limitations of studies that observe an obesity paradox

| Methodological limitation         | Example                                                                                                                                 |
|----------------------------------|----------------------------------------------------------------------------------------------------------------------------------------|
| Misclassification bias           | BMI may inappropriately assign overweight status to individuals who are normal weight by body composition. This may underestimate mortality in the overweight group |
| Reverse causation                | Weight loss in the normal weight group may be related to underlying illness and loss of fat free mass, leading to a higher relative mortality in that group compared to overweight groups |
| Collider stratification bias     | Smoking may be a significant causal factor, and lower rates of smoking in the overweight group may present as improved survival |

*Banack HR, Stokes A. The ‘obesity paradox’ may not be a paradox at all. *Int J Obes (Lond)*. 2017;41(8):1162–1163. https://doi.org/10.1038/ijo.2017.99*
Both severity of obesity and duration of obesity are associated with cardiac performance and cardiovascular disease [10]. Increased cardiorespiratory fitness has been found to reverse much of the negative impact of obesity on cardiovascular health and mortality. However, only 20% of individuals with obesity are thought to have adequate cardiorespiratory fitness [10].

3 Obesity and Respiratory Disease

Respiratory function is adversely affected by obesity in a number of ways. Excess adiposity on the thoracic wall and in the abdomen limits chest wall movement and decreases lung compliance, heightening the demand on the diaphragm [14]. Although respiratory muscle strength is preserved, diaphragmatic endurance is reduced as much as 45%, which may explain the common occurrence of breathlessness and susceptibility to respiratory failure in patients with obesity in the setting of abdominal surgery, sepsis or metabolic derangements. Lung perfusion is impacted by obesity as well. Perfusion is greatest in the dependent portions of the lung. In obesity, however, shallow breathing leads to basal atelectasis and distributes ventilation to the upper lung zones leading to ventilation-perfusion mismatch and increased vulnerability to hypoxia.

Obesity also leads to reduced airway caliber and increased airway resistance. This may explain in part the relationship between obesity and asthma wherein a weight gain of >5 kg increases risk of asthma in a dose-dependent manner and obesity is associated with symptom severity and increased bronchodilator use [14].

Upper airway function is particularly impacted in obesity by both the mechanical load of excess adiposity on pharyngeal structures and obesity-related inflammatory cytokines that disrupt pharyngeal neuromuscular function [14]. These changes manifest in obstructive sleep apnea (OSA), which has a prevalence of over 70% in the bariatric surgical population. Despite its strong association with obesity, 80% of obstructive sleep apnea remains undiagnosed [14]. Hypopneas and apneas in OSA result in hypoxia, hypercapnia, increased sympathetic activity, increased respiratory effort, cortical arousal, and sleep fragmentation which in turn leads to functional and physiologic impairments [15]. Specifically, OSA causes neuropsychiatric disturbances, cardiac arrhythmias, pulmonary hypertension, cor-pulmonale, systemic hypertension, coronary artery disease, congestive heart failure, polycythemia, stroke and increased mortality [14–15]. These complications are worsened in obesity hypoventilation syndrome (OHS) which is characterized by non-apneic hypoxemia and CO₂ retention. Both mechanical and central mechanisms are thought to play a role in OHS [15].

Obesity is also associated with worse outcomes in respiratory infections, including community acquired pneumonia, H1N1 influenza and coronavirus disease 2019 (Covid-19) [15–17]. Higher rates of hospitalization, intubation and mortality in the setting of Covid-19 are possibly related to multiple mechanisms including the aforementioned alterations in respiratory function predisposing to respiratory failure and/or hypoxia, altered immune responses leading to weakened
host defense and increased chances of cytokine storm, and increased quantities of angiotensin converting enzyme-2 (ACE-2), the transmembrane enzyme that SARS-CoV-2, the virus that causes Covid-19, uses for cell entry [17].

4 Obesity and Cancer

Obesity is associated with 13 types of cancer (Table 2) [18]. Among women in North America, Europe and the Middle East, the obesity-related cancer burden comprises 9% of the total cancer burden. There is increasing evidence of causal links between obesity and cancer that center on obesity-related metabolic and endocrine abnormalities. Specifically, alterations in sex hormone metabolism, insulin and insulin-like growth factor signaling, adipokines, and several inflammatory pathways have been implicated [18]. Despite the higher prevalence of various cancers in patients with obesity, rates of cancer screening have been shown to decrease with increasing BMI [19]. This disparity in care needs to be urgently addressed given the rising rates of both epidemics.

Although there is limited data to show the benefit of weight loss for cancer prevention or prognosis, it has been found that the mortality benefit of surgical weight loss is not only related to a reduction in cardiovascular mortality but also

| Cancer site or type       | Relative risk of highest BMI category evaluated versus normal BMI (95% CI) |
|---------------------------|-------------------------------------------------------------------------|
| Esophagus adenocarcinoma  | 4.8 (3.0–7.7)                                                           |
| Gastric cardia            | 1.8 (1.3–2.5)                                                           |
| Colon and rectum          | 1.3 (1.3–1.4)                                                           |
| Liver                     | 1.8 (1.6–2.1)                                                           |
| Gallbladder               | 1.3 (1.2–1.4)                                                           |
| Pancreas                  | 1.5 (1.2–1.8)                                                           |
| Breast (post-menopausal)  | 1.1 (1.1–1.2) \(^a\)                                                   |
| Corpus uteri              | 7.1 (6.3–8.1)                                                           |
| Ovary                     | 1.1 (1.1–1.2)                                                           |
| Kidney (renal cell)       | 1.8 (1.7–1.9)                                                           |
| Meningioma                | 1.5 (1.3–1.8)                                                           |
| Thyroid                   | 1.1 (1.0–1.1) \(^a\)                                                   |
| Multiple myeloma          | 1.5 (1.2–2.0)                                                           |

Adapted from Lauby-Secretan B, Scoccianti C, Loomis D, et al. Body Fatness and Cancer—Viewpoint of the IARC Working Group. *N Engl J Med*. 2016;375(8):794–798. [https://doi.org/10.1056/NEJMsr1606602](https://doi.org/10.1056/NEJMsr1606602)

\(^a\)Shown is the relative risk per 5 BMI units
a reduction in cancer mortality [20]. Weight loss has also been shown to improve prognosis in breast cancer treatment [18].

5 Other Obesity-Related Conditions

In clinical medicine, there has been a predominating focus on the impact of obesity on cardiovascular health, and more recently, an increased focus on the respiratory and oncologic diseases described thus far. This is due, in part, to the global burden of these specific co-morbidities, the high mortality associated with them and/or, in the case of cardiovascular disease, the well-established relationship between obesity and cardiovascular risk factors such as hypertension, dyslipidemia and type 2 diabetes.

The health effects of obesity, however, span every medical discipline and effect every organ system. Table 3 lists specific obesity-related diseases by system, which have not been discussed in the preceding sections. The range of obesity-related conditions, many of which are under-diagnosed or under-appreciated in routine clinical practice, points to the substantial morbidity and reduced quality of life that can be associated with excess adiposity.

6 Health Effects of Obesity in Special Populations

6.1 Transplant Recipients

Considering that obesity is a risk factor for end stage renal disease (ESRD), heart failure, and cirrhosis, it is not surprising that many transplant recipients have an elevated BMI. Unfortunately, obesity that has contributed to the end organ damage in these patients, also leads to worse post-transplant outcomes. The relationship between obesity and transplant has probably been most studied in the renal transplant field in which obesity has been associated with delayed graft function, graft failure, urine protein and acute rejection, independent of diabetes [29]. In lung transplant recipients, obesity affects short- and long-term survival above BMI \( \geq 30 \text{ kg/m}^2 \), whereas in liver transplant recipients it does not seem to confer added risk until much higher BMIs [30]. Obesity in heart transplant patients is associated with multiple complications related to the heart transplant, left ventricular assist devices, and cardiothoracic surgery more generally. These complications include infection, wound dehiscence, mediastinitis, prolonged mechanical ventilation and intensive care unit stays, thrombosis, premature device failure, cardiac arrhythmias, and early and late mortality [31].

Due to the adverse effect of obesity on transplant outcomes, many transplant centers have implemented BMI thresholds resulting in an increased demand for more effective weight loss options in this population [30].
Table 3  Other obesity-associated conditions

| System                  | Obesity-associated Condition                                                                                                                                 |
|-------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------|
| **Gastrointestinal (21)** |                                                                                                                                                                  |
| Liver                   | • Non-alcoholic fatty liver disease (NAFLD)                                                                                                                   |
|                         |   - Increased cardiovascular mortality and hepatocellular carcinoma (HCC) risk                                                                             |
|                         | • Non-alcoholic steatohepatitis (NASH)                                                                                                                        |
|                         |   - Increased mortality; 20% progress to cirrhosis                                                                                                         |
|                         | • Cirrhosis                                                                                                                                                    |
| Gallbladder             | • Gallstone disease                                                                                                                                             |
| Pancreas                | • Acute pancreatitis                                                                                                                                              |
| Esophagus               | • Esophageal dysmotility                                                                                                                                          |
|                         | • Gastroesophageal reflux disease (GERD)                                                                                                                       |
|                         | • Erosive esophagitis                                                                                                                                              |
|                         | • Barrett’s esophagus                                                                                                                                              |
| Stomach                 | • Erosive gastritis                                                                                                                                               |
| Small intestine         | • Diarrhea                                                                                                                                                      |
| Colon                   | • Diverticular disease                                                                                                                                              |
|                         | • Colonic polyps                                                                                                                                                   |
|                         | • Clostridium difficile infection                                                                                                                                  |
| Anorectum               | • Dyssynergic defecation                                                                                                                                            |
| **Urogenital (22)**     |                                                                                                                                                                  |
| Upper tract             | • Chronic kidney disease (CKD)                                                                                                                                     |
|                         |   - Related to hypertension and/or type 2 diabetes                                                                                                               |
|                         | • End-stage renal disease (ESRD)                                                                                                                                    |
|                         |   - Even when controlling for HTN and T2DM, obesity affects progression of CKD to ESRD                                                                            |
|                         | • Obesity-related glomerulopathy                                                                                                                                   |
|                         | • Kidneys stones                                                                                                                                                   |
|                         |   - Evidence strongest for uric acid stones but likely to increase calcium oxalate stones as well                                                               |
| Lower tract - women     | • Urge incontinence                                                                                                                                                |
|                         | • Stress incontinence                                                                                                                                              |
| Lower tract - men       | • Lower urinary tract symptoms (LUTS)                                                                                                                             |
|                         | • Benign prostatic hypertrophy                                                                                                                                     |
| **Neurologic (23)**     |                                                                                                                                                                  |
| Central Nervous System  | • Idiopathic intracranial hypertension                                                                                                                           |
|                         | • Alzheimer’s dementia                                                                                                                                               |
|                         | • Mild cognitive impairment                                                                                                                                           |
|                         |   - Attention deficits, poor executive function, impaired decision making, decreased verbal learning and memory                                                      |
| Peripheral Nervous System|                                                                                                                                                                  |
| -Autonomic              | • Autonomic dysfunction                                                                                                                                              |
|                         |   - Increased sympathetic outflow                                                                                                                                    |
| -Somatosensory          | • Peripheral polyneuropathy                                                                                                                                          |
|                         |   - Associated with obesity, prediabetes, and dyslipidemia; obesity also an independent risk factor                                                             |
| **Psychiatric (24)**    |                                                                                                                                                                  |
|                         | • Depression                                                                                                                                                      |
|                         |   - Bidirectional relationship                                                                                                                                       |
|                         | • Anxiety                                                                                                                                                          |
| Dermatologic (25)       |                                                                                                                                                                  |
| Physical effects        | • Venous stasis, stasis pigmentation, stasis dermatitis                                                                                                           |
|                         | • Venous ulcers                                                                                                                                                     |
### Table 3 (continued)

| Inflammatory dermatoses | Lymphedema  
|-------------------------|---------------------------------------------------|
|                         | o Lower limbs and abdominal wall                   |
|                         | Recurrent cellulitis                               |
|                         | o Lower limbs and abdominal wall; poor wound healing with prolonged hospitalizations |
|                         | Hidradenitis suppurativa                           |
|                         | Intertrigo and cutaneous infections (candida)      |
| **Inflammatory dermatoses** | Psoriasis                                             |
|                         | o Increases both risk and severity                 |
| **Cancer**              | Melanoma                                           |
|                         | Non-melanoma skin cancer (excluding basal cell carcinoma) |
| **Cosmetic**            | Skin tags                                          |
|                         | Striae                                            |
| **Signs of metabolic disturbance** | Acanthosis nigricans (insulin resistance)     |
|                         | Acne, hirsutism, androgenetic alopecia (hyperandrogenism, polycystic ovarian syndrome) |
| **Hematologic (15)**    | Thromboembolic disease                             |
|                         | o Higher in hospitalized patients and women        |
|                         | o May be related to immobility, endothelial dysfunction, and reduction in fibrinolysis |
| **Rheumatologic/ Musculoskeletal (26, 27)** | Osteoarthritis                               |
|                         | Degenerative joint disease                         |
|                         | Plantar fasciitis                                  |
|                         | Low back pain                                      |
|                         | Carpal tunnel                                      |
|                         | Rheumatoid arthritis                               |
|                         | o Associated with worse disease activity, increased odds of non-remission, worse functional ability and health-related quality of life |
|                         | Psoriatic arthritis                                |
|                         | o Recognized as a risk factor and associated with worse treatment efficiency |
|                         | Ankylosing spondylitis                             |
|                         | o Associated with adverse outcomes                 |
| **Reproductive (28)**   | Decreased testosterone                            |
| **Men**                 | Erectile dysfunction                              |
| **Women**               | Polycystic ovarian syndrome                        |
|                         | Reduced natural fecundity/ increased time to conception (even in ovulatory women) |
|                         | Infertility                                        |
|                         | Anovulation / menstrual irregularities             |
| **Fertility treatment** | Increased gonadotropin requirement                 |
|                         | Lower oocyte yield in severe obesity               |
|                         | Reduced implantation rates, clinical pregnancy rates and live birth rates |
|                         | Increased pregnancy loss rate prior to 24 weeks gestation |
|                         | If using egg donor, live birth rate per cycle is lower |
|                         | Compromised pelvic ultrasound imaging for oocyte retrieval |
Orthopedic Surgery Patients

Obesity is a risk factor for multiple musculoskeletal issues including knee osteoarthritis. There has been an increase in total knee arthroplasties in patients with elevated BMI [32]. In these patients, obesity is associated with a functional recovery similar to those without obesity. However, there is a significant increase in mid- to long-term revision rates in those with severe obesity. Obesity also poses a higher risk of post-operative superficial wound infections and thromboembolism [32]. Many orthopedic surgeons recommend a BMI cut-off for knee replacements. As is the case in transplant medicine, the BMI cut-offs lead to increased demands for effective weight loss options in this population.

Pregnancy

Obesity impacts both maternal and neonatal health. Rates of miscarriage are higher in women with obesity irrespective of spontaneous conception or in vitro fertilization [28]. The rate of gestational diabetes doubles for BMI $\geq 30$ kg/m$^2$ and triples for BMI $\geq 40$ kg/m$^2$. Risk of pre-eclampsia doubles with overweight and triples with obesity. There is also a more than 30% chance of pre-term delivery (before 37 weeks) in women with obesity. The peripartum risks include a prolonged first stage of labor, less success with vaginal birth after cesarean (VBAC), and increased rates of cesarean section delivery. Other obstetrical risks include increased fetal distress, instrumental deliveries, and shoulder dystocia. Wound infection and dehiscence, perinatal hemorrhage, and deep venous thrombosis are also more common in pregnant women with obesity. Neonatal effects of obesity include macrosomia and congenital anomalies, such as neural tube defects, oral clefts, hydrocephaly, anorectal atresia, limb reduction and cardiovascular anomalies [28].

Children and Adolescents

Much of the health effects of obesity in children and adolescents parallel those in adults. The increasing prevalence of obesity in children is therefore accompanied by an increase in type 2 diabetes, dyslipidemia, hypertension, non-alcoholic fatty liver disease (NAFLD), non-alcoholic steatohepatitis (NASH), and OSA [33]. There are, however, additional musculoskeletal and psychological considerations. Obesity during periods of growth can exert biomechanical forces leading to flatfoot, Blount’s disease, and slipped capital femoral epiphysis. Children with obesity also experience significant psychosocial distress thought to be related to lower self-esteem, social isolation, depressive symptoms, and body dissatisfaction [33].
7 Conclusion

Excess adiposity has widespread effects on health and well-being leading to significant morbidity and mortality. Obesity is not only a risk factor for numerous diseases, but it can also exacerbate underlying conditions leading to more severe symptoms, more rapid progression, and worse treatment prognosis. In some cases, obesity is even the primary cause of specific conditions such as obesity cardiomyopathy, NAFLD/NASH, and obesity-related glomerulopathy. The extensive endocrine and physical effects of excess and ectopic fat depots warrant a thoughtful and comprehensive assessment of the patient with obesity in clinical practice. The degree to which a therapy improves upon the many negative health effects of obesity also warrants evaluation, so the full risk–benefit of treatment is understood.

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