SEXUAL MEDICINE REVIEWS

# Sexual Function, Obesity, and Weight Loss in Men and Women

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## ABSTRACT

**Introduction:** Obesity is a major health concern in the United States and many developed countries. Among its many deleterious effects are those that can affect the sexual response.

**Aim:** To update and evaluate the state of knowledge on the possible link between obesity and sexual dysfunction.

**Methods:** A comprehensive search and review of biomedical, physiologic, and psychological databases were used to integrate findings on obesity, weight loss, and sexual function.

**Main Outcome Measures:** We briefly explain the two variables of interest—sexual functioning and obesity—indicating possible points of linkage. Then, we attempt to (i) describe possible direct links between obesity and sexual dysfunction; (ii) understand potential mediating biological, comorbid, and psychological factors and the interactions among such factors; (iii) discern differences in the mechanism and impact of obesity across the sexes; and (iv) review evidence suggesting that weight loss improves sexual functioning in obese individuals.

**Results:** The link between obesity and sexual function is complex and multivariate, with at least three different pathways likely: direct effects from adipose tissue; effects from pathophysiologic comorbidities; and effects mediated by psychological factors. In addition, effects and pathways appear to be different for men and women.

**Conclusion:** We conclude by identifying some existing challenges for the study of obesity and sexual function, specify areas that warrant further investigation, and reiterate the potential value of encouraging obese patients to consider weight loss as a path toward a healthier and more sexually satisfying life. **Rowland DL, McNabney SM, Mann AR. Sexual Function, Obesity, and Weight Loss in Men and Women. Sex Med Rev 2017;X:XXX–XXX.**




**Key Words:** Obesity; Sexual Function; Metabolic Syndrome; Anxiety; Hormones; Weight Loss

## INTRODUCTION

Obesity is a global epidemic that negatively affects the physical, emotional, and psychosocial well-being of individuals. Approximately 1 billion people worldwide are overweight and 300 million are obese. To meet the criteria for obesity, a person must weigh 35 pounds over their estimated body mass index [BMI] for what is regarded as a healthy average weight for their height-to-weight ratio [BMI > 30]. A BMI higher than 40 is considered extreme obesity.[1]

Obesity rates worldwide and in the United States are increasing, causing alarm among public health and health care professionals (Table 1). In the United States specifically, obesity has seen a slow, significant increase[2] from 13.4% to 35.7% from 1960 to 2010. The percentage of US adults considered overweight, obese, or extremely obese in 2010 had reached 75%: approximately 33% were overweight, 36% were obese, and 6% were extremely obese.[2–4]

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Obesity has been associated with diabetes, heart disease, hypertension, urinary incontinence, and various cancers and—the focus of this review—sexual dysfunction in men and women.[5] The relation between obesity and sexual function is not fully understood—for example, whether obesity itself increases the risk of sexual problems or whether obesity's effects on sexual function are mediated primarily through comorbidities. Because obesity is strongly associated with different pathologies, it also imposes significant medical and societal costs, currently estimated at $147–210 billion in the United States (stateofobesity.org; a joint effort of the Trust for America's Health and the Robert Wood Johnson Foundation).[6,7]

This review attempts to update and evaluate the state of knowledge on the link between obesity and sexual dysfunction. First, we briefly describe the two variables of interest: sexual function and obesity, indicating feasible points of linkage. Second, we (i) examine possible direct links between obesity and sexual response; (ii) discuss potential mediating biological, comorbid, and psychological factors, including sex differences where they exit; and (iii) review studies indicating that weight loss improves sexual functioning in obese individuals. A better understanding of this relation not only might benefit health care

**Table 1.** Obesity rates in men and women in the United States

| | Men | Women |
|---|---|---|
| Overweight or obese (BMI $\geq$ 25), % | 74 | 64 |
| Obese (BMI $\geq$ 30), %* | 36 | 36 |
| Extremely obese (BMI $\geq$ 40), %* | 4 | 8 |

BMI = body mass index.
*Percentages in each category are subsumed under the percentages in the rows above.
Source: National Health and Nutrition Examination Survey, 2009–2010. Available at: https://www.cdc.gov/nchs/data/databriefs/db219.pdf.

professionals who care about the quality of life of their patients but also might serve as a motivator for change toward healthier lifestyles within this population.[8]

## LINKING OBESITY TO SEXUAL DYSFUNCTION

### Understanding the Sexual Response

The sexual response is multidimensional, with many sexual problems affected by an individual's psychological well-being and physical and physiological factors.[1,8] Disturbances in sexual function typically affect at least one of the three phases of the sexual response cycle: desire, arousal, and orgasm.[9,10] In men, the most common problem with the arousal phase is difficulty getting and/or sustaining an erection; common problems with the orgasmic phase include premature ejaculation and delayed or inhibited ejaculation. In women, common problems with the desire-arousal phase (recently combined from two phases into one) include low desire, lack of lubrication, and inadequate psychological arousal[11]; the most common problem with the orgasmic phase is difficulty or inability to reach orgasm.[12–14] Multiple factors can affect sexual functioning, including partner and relationship issues, individual vulnerabilities (eg, low self-esteem or body image), psychiatric problems (eg, depression, anxiety, or stress), and medical issues (eg, diabetes, heart disease, etc).[12]

### Understanding Obesity

Traditional models of obesity have emphasized energy balance, with weight gain defined as caloric intake that exceeds daily expenditure.[15] Although this perspective might accurately describe the development of obesity, particularly in its initial stages, it oversimplifies the dynamic nature of adipose tissue, its underlying influence on physiology, and the interactive nature between the organism and the environment that bestows an evolutionary advantage on individuals capable of storing caloric resources for future use.

Just as sexual problems are associated with (and in some instances caused by) individual vulnerabilities, psychiatric problems, medical problems, and relationship issues, obesity has the potential to affect each of these domains. For example, (i) obesity is associated with depression, anxiety, poor body image, and low self-esteem, all of which can interfere with sexual function. (ii) The biochemical activity of adipose tissue in the obese has been associated with the development of metabolic syndrome, a cluster of pathologies (eg, dyslipidemia, insulin resistance, hypertension, hyperglycemia, increased cardiovascular disease [CVD], and chronic inflammation) known to adversely affect sexual function. (iii) Obesity can negatively affect sexual relationships by diminishing partner attraction and/or sexual engagement. One major challenge in understanding the link between obesity and sexual function lies in identifying relevant individual or multivariate mediating factors—whether biological, pathophysiological, or psychological—and how they interact.

## Physiologic Regulation of Fat Tissue

Adipose tissue or fat is loose connective tissue composed of adipocytes. These adipocytes expand or contract as the result of free fatty acids entering and leaving adipose tissue, controlled by insulin and leptin, respectively. When insulin is stimulated by high blood sugar after consuming carbohydrates or fats, free fatty acids enter the cell, and only when insulin is low do free fatty acids leave adipose tissue. When muscles and other tissues need energy, adipocyte stores are broken down to release fatty acids and glycerol, which are useable energy sources for cells. This process results from the activation of the enzyme lipase, which is stimulated by different hormones, including epinephrine, norepinephrine, glucagon, and adrenocorticotropin, that bind to the adipocytes and generally signal a condition of physiologic stress.

At the molecular level, the progression of pre-adipocytes to their mature counterparts in vivo requires the presence of peroxisome-proliferator activated receptor-$\gamma$ (PPAR-$\gamma$), with homozygous null mutations proving embryonically lethal in mice.[16] Pro-adipogenic transcription factors such as CCAAT/enhancer binding protein-$\beta$ appear to exert their effects by inducing PPAR-$\gamma$ expression.[17] Interestingly, the antidiabetic thiazolidinedione medications are PPAR-$\gamma$ agonists, suggesting that whole-body insulin sensitivity is at least partly dependent on adipose tissue maturation and functionality.[16] Endogenous and synthetic glucocorticoids also have been identified as pro-adipogenic molecules.[17] In contrast to these well-established metabolites, the effects of testosterone (T) and estradiol (E2) on adipose tissue formation remain equivocal, thus warranting further study.

The mobilization of triglyceride stores from adipose tissue also depends on molecular regulation, with contributions from endocrine and paracrine signals. Insulin is one of the most potent inhibitors of lipolysis, instead facilitating plasma glucose uptake and subsequent use by peripheral tissues.[17] Conversely, the catecholamines (epinephrine and norepinephrine) bind to $\beta$-adrenergic receptors on the adipocyte plasma membrane and ultimately stimulate lipolysis. Moreover, stimulation of $\beta$-adrenergic receptors increases non-shivering thermogenesis in brown adipose tissue (BAT) through the recruitment of uncoupling protein-1.[17] Adiponectin, a secretory product of adipocytes, can stimulate lipolysis and fatty acid oxidation in adjacent