Review

Sex hormones, appetite and eating behaviour in women

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ABSTRACT

Sex hormones play essential roles in the regulation of appetite, eating behaviour and energy metabolism and have been implicated in several major clinical disorders in women. Estrogen inhibits food intake, whereas progesterone and testosterone may stimulate appetite. This review describes recent findings concerning interactions between sex hormones and neuroendocrinological mechanisms in the control of appetite and eating in women. Furthermore, we are gaining insights into the roles played by sex hormones in the development of eating disorders and obesity. For instance, androgens may promote bulimia by stimulating appetite and reducing impulse control, a proposal supported by the observation that antiandrogenic treatment attenuates bulimic behaviour. Androgens are also involved in the pathophysiology of abdominal obesity in women. On the other hand, hormone replacement therapy with estrogen counteracts the weight gain and accumulation of abdominal fat associated with the menopausal transition. In conclusion, sex hormones and/or agents that exhibit similar activities may provide novel strategies for the treatment of eating disorders and android obesity, two of the most serious health problems for women today.

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Contents

1. Introduction .......................................................................................................................... 249
2. Hormonal control of appetite and food intake ................................................................. 249
   2.1. Central regulation of feeding ...................................................................................... 249
   2.2. Signals produced in response to a meal ....................................................................... 249
   2.3. Adiposity signals ......................................................................................................... 249
   2.4. Sex hormones ............................................................................................................ 249
3. Regulation of normal eating in women by sex hormones .................................................. 250
   3.1. During the various phases of the menstrual cycle .................................................... 250
   3.2. Menopause ............................................................................................................... 250
   3.3. Pregnancy and lactation ............................................................................................ 251
4. The involvement of sex hormones in eating disorders and obesity ................................. 252
   4.1. Anorexia nervosa ....................................................................................................... 252
   4.2. Bulimia nervosa ........................................................................................................ 252
   4.3. Obesity ...................................................................................................................... 252
5. Effects of treatment with sex hormones on appetite and body weight ......................... 253
   5.1. Oral contraceptives .................................................................................................... 253
   5.2. Hormone replacement therapy .................................................................................. 254
6. Conclusions ........................................................................................................................ 255
Contributors ............................................................................................................................ 255
Competing interests ............................................................................................................... 255
Provenance and peer review ................................................................................................. 255
Acknowledgement ................................................................................................................ 255
References ................................................................................................................................ 255

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1. Introduction

The sex hormones estrogen, progesterone and androgens are involved in the complex regulation of appetite, eating and energy metabolism. In most species, including man, food intake and reproductive functions are closely linked. Thus, during the different hormonal phases of the menstrual cycle, daily food intake varies and, moreover, remarkable physiological adaptations of appetite and body composition occur during pregnancy and lactation. In addition, regulation of eating behaviour and metabolic functions by sex hormones is of considerable general importance for women’s health, as indicated by the disturbances in this regulation associated with a number of clinical disorders. In this context eating disorders and obesity, which are becoming more and more widespread and are coupled to severe morbidity and mortality, are of particular concern. Recent research designed to elucidate mechanisms and provide strategies for the prevention and treatment of these major health problems has revealed new insights into the regulation of appetite and eating behaviour by sex hormones.

The present overview of the role of sex hormones in the control of appetite and regulation of normal, as well as pathological eating behaviour in women focuses on recent findings in the following areas:

1. Interactions between sex hormones and neuroendocrinological factors
2. Regulation of eating behaviour by sex hormones during the different phases of the menstrual cycle, menopause, pregnancy and lactation
3. The potential role of sex hormones in eating disorders and obesity
4. The effects of treatment with estrogens (i.e., oral contraception and menopausal hormone therapy) on appetite and body weight

2. Hormonal control of appetite and food intake

In response to hunger or the desire for some specific item of food, we normally eat until satiated. In this connection, various factors such as the size of meals and rate and frequency of food intake may vary greatly in response to various environmental, psychological, social and cultural influences.

2.1. Central regulation of feeding

The hypothalamus plays a major role in the regulation of appetite and food intake, both on a meal-to-meal and longer-term basis. As part of the central feeding system, this organ coordinates a number of hormonal and neural inputs and also responds to blood levels of nutrients, in turn generating behavioural and autonomic outputs designed to meet caloric needs and maintain energy balance [1,2]. In this regard a key role is played by the hypothalamic arcuate nucleus (ARC), which extends projections to the paraventricular nucleus (PVN) and the lateral hypothalamic area (LHA), as well as to other nuclei involved in appetite regulation, such as the dorsomedial hypothalamus and ventromedial hypothalamic nucleus (VMH) (Fig. 1).

Two major populations of ARC neurons influence food intake and, thereby, energy homeostasis: those that extend projections to the LHA and express neuropeptide Y (NPY) and Agouti-related peptide (AgRP) stimulate food intake and decrease energy expenditure and are therefore referred to as orexigenic effectors (Fig. 1). Other ARC neurons extend projections to the PVN and express proopiomelanocortin (POMC) and cocaine-amphetamine-regulated transcript (CART), which inhibit food intake and increase energy expenditure, i.e., anorexigenic effectors [2,3]. In addition, several other factors produced in the hypothalamus also regulate appetite, including the anorectic peptide corticotrophin-releasing hormone and the orexigenic hormones melanin concentrating hormone and orexins [2,3].

There is also some evidence that food intake by humans is under the control of cortical and subcortical areas of the brain associated with reward and cognition [Fig. 1] [4]. The mesolimbic dopamine system, including the ventral tegmental area of the midbrain and the nucleus accumbens in the striatum, is considered to be the primary neural reward system. In response to environmental and psychological stimuli this network may enhance food intake independent of caloric need, the so-called hedonic feeding system.

2.2. Signals produced in response to a meal

The peripheral feeding system regulates short-term food intake in response to a meal by releasing a cascade of peptides from the gastrointestinal tract, where they interact with dietary components and act peripherally to modulate digestion and absorption of nutrients (Fig. 2). By activating vagal afferent nerve fibers, these peptides also signal via the nucleus of the solitary tract in the brain stem that the stomach is full. In addition, gastric distention evokes a feeling of satiety, also through activation of the vagal nerve, causing us to stop eating and reduce the size of our meals [1–4]. The gastrointestinal peptides proposed to produce satiation include cholecystokinin (CCK), glucagon-like peptide-1 and peptide YY [2–4]. On the other hand, the appetite-stimulating hormone ghrelin is secreted from the gastric mucosa during fasting and reaches the brain via the bloodstream to signal hunger (Figs. 1 and 2) [5].

2.3. Adiposity signals

Circulating adiposity signals produced in amounts directly proportional to body fat depots are also involved in the feedback regulation of energy homeostasis [1–3]. For instance, leptin and insulin have been implicated in long-term control of food intake and energy expenditure (Fig. 2). Produced by adipocytes, leptin inhibits food intake and stimulates the metabolic rate in experimental animals by acting directly on specific leptin receptors in the hypothalamus [6], thereby inhibiting orexigenic NPY/AgRP neurons and stimulating anorexigenic POMC/CART neurons in the ARC, as well as activating satiety neurons in the VMH (Fig. 1) [3]. However, the reported effects of administration of leptin to obese humans have been modest and variable [7]. These observations have led to the concept of leptin resistance in obese individuals [8].

Insulin released from the pancreas elevates leptin production and, at the same time, like leptin, acts directly on the hypothalamus and other areas of the brain to exert anorexigenic effects [9]. However, systemic administration of insulin elicits hypoglycaemia, which in itself enhances food intake [2].

2.4. Sex hormones

The sex hormones estrogen, progesterone and androgens are important modulators of food intake and energy balance in mammals (Fig. 2) [10], where, as in most species, food intake and reproductive function are closely linked. Sex hormones both interact with gastrointestinal peptides and neurotransmitters to achieve central control of appetite and energy expenditure, while also exerting direct peripheral action on adipocytes [10].

Ovariectomy of rats increases food intake and, concomitantly, body weight [11] and these effects can be reversed by restoring physiological levels of estradiol [11]. There is evidence that the effects of estradiol on food intake are mediated via estrogen receptors in the hypothalamus (e.g., the ARC and the PVN) and in the nucleus of the solitary tract in the brain stem [11]. However, it
remains unclear whether estrogen receptor (ER) α or β is involved [11–13]. Moreover, these effects of estradiol appear to involve several different mechanisms. For instance, estradiol potentiates the effect of the satiating CCK peptide released from the small intestine in response to food intake [14,15], while attenuating the appetite-stimulating potency of the gastric hormone ghrelin [16]. Furthermore, estradiol stimulates anorexigenic POMC/CART activity and inhibits orexigenic NPY/AgRP neurons in the ARC [17,18].

In contrast to estrogen, progesterone itself does not significantly influence feeding behaviour in ovariectomized rats, except when administered in non-physiological, pharmacological doses [11]. However, in the presence of estrogen, progesterone does stimulate appetite and promote weight gain [19]. Testosterone stimulates appetite and eating in a manner thought to be mediated centrally [10], selectively increasing the number of meals, but not the size of each individual meal in rats [20]. Recently, neonatal exposure of female mice to testosterone was found to enhance food intake and attenuate the expression of anorexigenic POMC/CART neurons in the ARC of these same animals as adults [21].

3. Regulation of normal eating in women by sex hormones

3.1. During the various phases of the menstrual cycle

In the female of numerous species, eating behaviour is closely linked to the functioning of the hypothalamic–pituitary–gonadal (HPG) axis. For example, both female rodents and primates eat less during the estrus phase prior to and following ovulation, when they are more sexually receptive and active [10]. It has been proposed that specific central neural mechanisms promote either feeding or sexual behaviour at the appropriate times [22].

Similarly in the case of women, food intake during the different phases of the menstrual cycle varies (Fig. 3). Thus, a meta-analysis revealed that mean food intake is lowest during the periovulatory phase of the menstrual cycle, when estradiol levels are high [23]. In contrast, a peak in food intake occurs during the premenstrual period, when progesterone levels are high [23–28].

These cyclic variations have been documented in several studies, particularly in women exhibiting the premenstrual syndrome [23–28]. Moreover, binge-eating may be more pronounced during the premenstrual period [29], a process which may involve low levels of serotonin in the brain [25]. In addition, alterations in energy expenditure during the course of the menstrual cycle are related to variations in eating. For instance, during the postovulatory phase elevated energy expenditure is associated with the higher levels of progesterone [30].

Although the relevant reports have been somewhat contradictory, intake of specific macronutrients may also vary during the menstrual cycle [24]. Some investigators have observed enhanced intake of fat [27,31], carbohydrate [26] or both [25] during the premenstrual period; whereas others have not seen any change in food preferences during the course of the menstrual cycle [24].

3.2. Menopause

During the menopausal transition, cyclic secretion of estradiol and progesterone disappears and production of the female sex hormones declines rapidly. However, circulating levels of androgens
decline as a consequence of age-related reductions in secretion by both the adrenal gland and ovaries. Weight gain is common during these years and longitudinal studies have demonstrated an increase in total and abdominal fat and a decrease in lean body mass [32,33]. These changes have been observed as early as 3–4 years prior to the onset of menopause and to remain relatively stable for at least 1–2 years after menopause [33].

At present, little is known about the relationship between eating and physical activity, on the one hand, and estrogen depletion and subsequent cessation of ovarian function in women, on the other. However, menopause is associated with reductions in energy expenditure and oxidation of fat that can predispose to weight gain [33,34]. The attenuated resting metabolic rate is probably a consequence of the loss in lean body mass, while the alterations in fat distribution appear to reflect modified metabolism in adipose tissue. Estradiol stimulates the activity of lipoprotein lipase (LPL) in femoral adipocytes and lipolysis in abdominal adipocytes [35], thereby promoting accumulation of gluteo-femoral fat. On the other hand, estrogen deficiency is associated with enhanced accumulation of abdominal fat [35].

3.3. Pregnancy and lactation

The major physiological changes in appetite and body composition that occur during pregnancy and lactation represent functional adaptations. Both female animals (especially when they give birth to large litters) and women eat more during pregnancy to ensure normal growth and development of the fetus. In the rat food intake is doubled, whereas in humans the increase is 10–15% [19]. At about the twelfth week of pregnancy women begin to eat progressively more until somewhere around midgestation, when physical activity and, subsequently, food intake both decline [36].

Although the mechanisms underlying this enhancement of appetite during pregnancy remain to be elucidated, it is likely that sex hormones, and in particular progesterone are involved [37]. Progesterone may exert this effect via specific receptors on neurons in the VMH and ARC [37]. Furthermore, the levels of adiposity signals and of factors produced in response to a meal are altered as a consequence of positive energy balance. For example, the level of satiating CCK peptide is elevated [38], whereas basal levels of the hormone ghrelin are lower [39]. In addition, circulating concentrations of leptin rise during a normal human pregnancy, an effect which may be secondary to augmented leptin resistance [40].

As expected, maternal fat deposition during gestation reflects maternal food consumption. Under the influence of estrogen and progesterone, fat is deposited primarily in gluteofemoral regions to provide an energy reserve for lactation [41]. During lactation, energy requirements are even higher than during pregnancy, with breastfeeding demanding approximately 500 extra kcal per day [42]. This elevated need is met by eating 20–25% more, along with mobilization of femoral fat through enhanced lipolysis and reduced LPL activity in response to prolactin, insulin and growth hormone [19]. At the same time, the woman tends to be less physically active [42].

The mechanisms underlying stimulation of food intake during lactation have not yet been clarified, but prolactin has been proposed to play a major role in this connection [19]. In the lactating rat, hyperphagia is a response to the suckling stimulus and the metabolic demands associated with milk production per se [19]. These animals may also be less sensitive to satiating factors such as CCK [43].
The potential role of gonadal hormones in the pathogenesis of AN is not yet known. Recently, an association between paternal polymorphisms in the gene encoding ERα receptors and restrictive AN was reported [46], indicating the involvement of estrogen. In addition, this disease is associated with alterations in the levels of appetite-regulating hormones and neuropeptides, including corticotrophin-releasing hormone, NPY, serotonin metabolites, and leptin, in the cerebrospinal fluid [47]. However, recovery tends to eliminate these abnormalities, suggesting that they may be a consequence of starvation rather than a primary cause of the disease [47].

4.2. Bulimia nervosa

Bulimia nervosa is characterized by frequent episodes of binge eating, followed by attempts to compensate by vomiting, abuse of laxatives, excessive exercise and/or other purging behaviours [44]. Although its etiology has yet to be elucidated in detail, a genetic predisposition, biological disturbances (including dysregulation of the serotonin system) and socio-psychological factors all appear to be involved [47].

Even though body weight remains normal, the menstrual cycle of bulimic women is often disturbed, probably as a consequence of several different processes. As in the case of AN, low circulating levels of estradiol and gonadotropins, indicative of hypothalamic inhibition of the HPG axis, are observed in bulimic women [48]. In addition, BN may be connected with the polycystic ovary syndrome (PCOS) [49,50], which has a prevalence of 5–10% in the general population and is thus the most common endocrinological disorder in women of fertile age. This syndrome is associated with disturbance of the menstrual cycle, hyperandrogenism and polycystic ovaries (as detected by ultrasound) [51] and is also related to insulin resistance and abdominal obesity. There is evidence that PCOS has a strong genetic component, although environmental factors appear to be important as well.

Women with hyperandrogenism and PCOS exhibit dysregulation of appetite, together with a craving for carbohydrates and fat, as also observed in bulimics [52–54]. Similarly, elevated incidences of polycystic ovaries, acne and hirsutism, along with abnormally high serum levels of androgens have been observed in bulimic women [49,50]. Female mice in which the gene encoding estrogen receptor β has been knocked out (BERKO mice) display attenuated fertility due to ovarian dysfunction and dysregulation of androgen receptors [55]. Interestingly, an association between polymorphisms in the ERβ gene and bulimia has been demonstrated in humans as well [56], an observation which may have important consequences for the development and treatment of both menstrual disorders and abnormal eating behaviour in bulimic women.

Testosterone stimulates appetite [10] and high circulating levels of this androgen in women have been associated with impaired impulse control, irritability and depression [57], i.e., common features of women with bulimia [57]. Accordingly, it has been proposed that elevated levels of androgens may promote bulimic behaviour by influencing craving for food and/or impulse control. Hypothetically, bulimia may, in some cases, have a hormonal, rather than a psychiatric etiology, a suggestion supported by the observation that antiandrogenic treatment reduces bulimic behaviour [58,59]. This may turn out to be a novel and valuable approach to treating women with BN, particularly those with hyperandrogenic symptoms.

4.3. Obesity

Obesity (body mass index (BMI) > 30 kg/m²) is a growing health problem that has reached epidemic proportions worldwide. The
etiology involves both environmental and genetic factors, with
the latter exerting an impact of 40–70% [60]. Only in rare cases, such
as congenital leptin deficiency, does a disturbance in appetite and
weight regulation appear to play a primary role [61]. In other obese
individuals the hedonic response to food, particularly enjoyment
of high-fat foods, seems to be greater than in lean individuals [62].
Obesity is also associated with several endocrinological abnormal-
ities, most of which are secondary, but some of which may actually
be involved in its etiology and development.

Interactions between glucocorticoids and sex hormones partici-
пate in the pathophysiology of abdominal obesity [63], which is
associated with stressful life events leading to over-stimulation
of the hypothalamic–pituitary–adrenal axis (HPA) [64]. Such
hyperactivity elevates levels of cortisol, increases lipolysis and
stimulates the conversion of free fatty acids to glucose and stor-
age of fat in abdominal deposits. Central fat depots predispose an
individual to insulin resistance and secondary hyperinsulinemia,
which, in turn, stimulates ovarian biosynthesis of testosterone
and inhibits hepatic production of sex-hormone binding globu-
lin (SHBG), thereby enhancing levels of free testosterone (e.g.,
in women with PCOS). High levels of testosterone may then
induce accumulation of abdominal fat and insulin resistance
(Figs. 4 and 5) [65].

It has been proposed that testosterone might cause hepatic
insulin resistance by facilitating catecholamine-stimulated lipol-
ysis in visceral fat tissue, thus exposing the liver to a surge of free
fatty acids [65]. In addition, testosterone may promote insulin resis-
tance by decreasing the density of capillaries in peripheral muscles.
Such a vicious circle could aggravate metabolic complications and
thus enhance the long-term risk of developing type 2 diabetes and
cardiovascular disease (Fig. 4).

Whereas testosterone may influence fat distribution in women
adversely, it is noteworthy that the situation appears to be exactly
the opposite in men. Larger depots of abdominal fat in men are
associated with lower testosterone levels because gonadotropin
secretion is reduced [63], while upon weight loss testosterone lev-
els and insulin sensitivity return to normal. Furthermore, long-term
treatment of obese men with testosterone causes them to burn fat
and enhances lean body mass [63]. This apparent sex difference has
not yet been clarified.

5. Effects of treatment with sex hormones on appetite and
body weight

5.1. Oral contraceptives

Oral contraceptives (OCs) containing both estrogen and pro-
estgen are used by countless women for birth control, as well as for
medical treatment of dysmenorrhea, heavy bleeding, endometrio-
sis and menstrual disorders. Such treatment is generally tolerated
well, with few side-effects and only a small risk for thromboem-
bolitic events. Nonetheless, many women, especially adolescents,
stop taking OCs because of weight gain, mood swings and sexual
dissatisfaction [66]. An American survey found that more than 50%
of women believe that OCs cause weight gain and 20% said that
this was the reason they had stopped taking them [67]. Although
some women do experience alterations in appetite and weight
when using such combination OCs, several investigations have all
concluded that these do not significantly affect body weight or com-
position [68,69]. At the same time, individual women may respond
differently and, moreover, different types of OCs may differ in this
respect.

Combination OCs usually contain the synthetic estrogen
ethinylestradiol or, more recently, the natural estradiol, whereas
the type of progestogen present varies. The major progestins
employed are derivatives of either 19-nortestosterone or 17-
α-hydroxyprogesterone. The former exhibits varying degrees of
androgenic activity, whereas the latter, which are related struc-
turally to progesterone, lack androgenic effects and may also
demonstrate antiandrogenic activity. Combinations of androgenic
progestins with ethinylestradiol appear to be associated with a
lower risk for vascular thromboembolism and are therefore the
recommended choice in a number of countries [70]. On the other
hand, side-effects such as acne, mood swings, and weight gain may
be more common with androgenic OCs [71].

Progestins are known to stimulate food intake (Fig. 5) [72].
For example, cancer-related cachexia and anorexia and other
forms of malnutrition can be effectively treated with high doses
of megestrol acetate. Such treatment enhances serum levels of
insulin and insulin-like growth factor (IGF)-I markedly [72]. More-
over, an androgenic OC can interfere with regulation of appetite,
suppressing secretion of the satiating peptide CCK and increasing body fat [73,74], which may be one mechanism underlying the weight gain experienced by certain women.

In contrast, treatment of women with bulimia nervosa with an antiandrogenic OC containing ethinylestradiol and drospirenone not only reduces androgen levels, but also attenuates binge eating and appetite in connection with meals [59]. Furthermore, users of this particular OC exhibit a small reduction in body weight [75]. However, despite extensive clinical experience with OC treatment, little is presently known concerning the effects of different types of OCs on appetite and body weight and much more research in this area is clearly motivated.

5.2. Hormone replacement therapy

For decades now, hormone replacement therapy (HRT) has been employed to relieve menopausal symptoms such as flushing, sweating and sleep disturbances, as well as for to prevent osteoporosis and related bone fractures. However, more recent randomized, as well as, epidemiological investigations have revealed that HRT of postmenopausal women with estrogen and progestin increases the risk for breast cancer, as well as for cardiovascular disease and thrombosis. Consequently, the benefits versus risks of such treatment have been discussed extensively and new guidelines proposed. Importantly, a women’s age or the period that has elapsed since her menopause appears to influence the benefit–risk ratio in this connection. Recently, the Endocrine Society Scientific Statement concluded that HRT provides highly effective alleviation of climacteric symptoms and that the risk–benefit ratio is favourable if treatment is initiated shortly after onset of menopause and limited to approximately five years [76].

Many women are concerned about gaining weight during menopause and believe that HRT may increase their appetite and, thereby, weight. However, studies have shown that energy intake is not affected by HRT [77,78] and, furthermore, numerous randomized trials have failed to reveal any evidence that HRT, either with estrogen alone or in combination with progestin, elevates body weight or BMI [76,79]. In contrast, most such investigations conclude that HRT actually lowers weight gain and body fat [76,80,81]. In addition, HRT prevents the shift in fat deposition from the normal female condition to the more unhealthy central fat depots associated with the menopausal transition (Fig. 5) [76,82,83].

The mechanism(s) in this case probably involves metabolic effects of HRT on adipose tissue. Thus, treatment of postmenopausal women with estrogen enhances LPL activity in the femoral region and at the same time lipolysis in the abdominal region, which might promote fat accumulation in the former region and fat loss from the abdomen [84]. In addition, as demonstrated by the large Women’s Health Initiative Study [83], lean body mass may be preserved by combined HRT. Interestingly, the route of estrogen administration may be an important factor in this context, since one crossover study found less fat gain with transdermal than with oral administration [85]. Oral administration of estrogen stimulates the secretion of growth hormone, reduces circulating levels of IGF-I and raises circulating levels of triglycerides; whereas none of these changes appears to occur following transdermal administration [76].

In recent years, there has been increasing interest in treating postmenopausal women with androgens because of the beneficial effects, including improved sexual functioning, energy and overall quality of life [86]. Testosterone treatment also enhances bone mineral density, lean body mass and muscle strength [86]. However, there remain concerns about potential adverse effects
of androgens on body weight, as well as carbohydrate and lipid metabolism similar to those observed in individuals with excess endogenous androgens, e.g., women with PCOS. For instance, short-term oral treatment of postmenopausal women with testosterone induces insulin resistance, produces an adverse lipid profile and alters the expression of lipolytic signaling proteins, which may promote the accumulation of fat [87,88]. Transdermal preparation of testosterone is now available for treatment in women, and this kind of administration may be associated with less risk of adverse metabolic effects [86]. Furthermore, the safety of long-term testosterone administration with respect to the endometrium and breast has not yet been elucidated.

6. Conclusions

The present review highlights the complex involvement of sex hormones in the regulation of appetite and eating behaviour in women. In both experimental animals and humans, estrogen reduces food intake; whereas testosterone, as well as progestosterone in combination with estrogen, may enhance food intake. Sex hormones modulate appetite and energy expenditure during the menstrual cycle, pregnancy, lactation and menopause. Moreover, alterations in sex hormones may play a role in disturbed eating behaviour. For example, elevated levels of androgens in women have been associated with impaired impulse control and bulimic behaviour. In addition, antiandrogenic oral contraceptives appear to reduce meal-related appetite and bulimic behaviour. Accordingly, at least hypothetically, bulimia may in some cases be a hormonal rather than primarily a psychiatric illness.

The development of obesity, and particularly the visceral type thereof, may also be influenced by sex hormones. Thus, interactions between glucocorticoids and elevated levels of androgens play a role in the pathophysiology of abdominal obesity and insulin resistance in women. In contrast, abdominal obesity in men is associated with lower testosterone levels. Furthermore, treatment with estrogen and progestin counteracts weight gain and the accumulation of abdominal fat associated with the menopausal transition. In conclusion, sex hormones and/or agents that exhibit similar activities may provide novel strategies for the treatment of eating disorders and android obesity, two of the most serious health problems for women today.

Contributors

All authors contributed equally.

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