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THE INFLUENCE OF SEX HORMONES ON DIABETES: COMPARATIVE ANALYSIS OF WOMEN AND MEN

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ABSTRACT

Diabetes is a complex metabolic disorder whose development and complications are influenced not only by genetic, environmental and lifestyle factors, but also by sex hormones. Women and men differ in regulation of glucose metabolism and insulin sensitivity, which leads to specific patterns in diabetes risk and clinical presentation. Among women, estrogen plays a protective metabolic role before menopause, while hormonal fluctuations during the menstrual cycle, pregnancy and menopause significantly affect glycemic control and may increase the risk of diabetes type 2.
In men, testosterone deficiency is strongly associated with insulin resistance, obesity and an increased risk of developing type 2 diabetes, while diabetes itself may contribute to reduced testosterone levels and reproductive dysfunction. These hormonal differences also contribute to variations in diabetes complications, cardiovascular risk and treatment responses between sexes. This article presents a review of current literature on the relation between sex hormones and diabetes, highlighting biological mechanisms, clinical implications and the importance of considering sex-specific factors in prevention, diagnosis and management strategies. Understanding these distinctions may support more personalized and effective approaches to diabetes care.
Materials and methods: The review was based on an analysis articles published in the PubMed and Google Scholar databases between 2015 and 2025, using the following keywords: sex hormones, diabetes, sex differences, women and men, glucose metabolism, insulin resistance, diabetes risk.
Results: The analysis revealed significant sex-specific differences in the association between sex hormones and glucose metabolism. Women demonstrated a stronger protective influence of estrogens, reflected in better glycemic control and lower insulin resistance before menopause, whereas postmenopausal women showed a marked aggravation in metabolic parameters. In men, higher androgen levels are associated with improved insulin sensitivity, while androgen deficiency correlates with increased diabetes risk. Across both sexes, hormonal imbalance was strongly linked with impaired glucose tolerance, higher prevalence of type 2 diabetes and altered insulin secretion.
Conclusions: This study highlights the crucial role of sex hormones in modulating glucose metabolism and showing the risk and progression of diabetes in women and men. Estrogens exert a protective effect in women, particulary before menopause, while their decline is associated with worsening metabolic control. In men, androgen levels appear beneficial for insulin sensitivity, whereas androgen deficiency increases metabolic disturbances. Overall, the findings emphasize that hormonal status should be considered an important determinant in diabetes risk assessment, prevention strategies and therapeutic approaches. Incorporating sex-specific hormonal factors into clinical practice may improve early identification, management and outcomes in diabetes.

KEYWORDS

Sex Hormones, Diabetes, Sex Differences, Women and Men, Glucose Metabolism, Insulin Resistance

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Introduction

Sex hormones and their binding proteins have emerged as significant modulators of glucose metabolism and the risk of type 2 diabetes mellitus, with accumulating evidence indicating profound sex-specific effects. Circulating concentrations of testosterone, estradiol and sex hormone-binding globulin (SHBG) differ between women and men and these differences appear to influence insulin resistance and glycemic control distinct ways across sexes. [1,2,3] Low SHBG levels have been consistently associated with increased risk of diabetes in both sexes, but the strength of the association and the interplay with other hormones may differ between men and women. [1,2,3,4]. In men lower serum testosterone has been shown to correlate with higher incidence of T2DM, while higher testosterone levels are generally associated with improved metabolic outcomes and decreased diabetes risk. [2]. In contrast, in women elevated testosterone, particularly in states of hyperandrogenism, correlates with greater insulin resistance and increased T2DM risk, especially after menopause. [1,2]. SHBG, which regulates the bioavailability of circulating sex steroids, has inverse relationships with measures of glycemia and diabetes risk in both sexes but appears to experience a more protective effect in women. [1,4]. Furthermore lifestyle factors such as weight loss can alter sex hormone levels and SHBG with differential effects observed in women and men living with T2DM suggesting that metabolic interventions may need to be sex-specific. [5]. These findings underscore the complexity of hormonal influences on diabetes pathophysiology and highlight the importance of considering biological sex as a key variable in both research and clinical practice. Collectively, current evidence supports the concept that sex hormone profiles and SHBG levels contribute to sex-specific pathways in the development and progression of T2DM. Understanding these mechanisms is essential for advancing precision medicine approaches tailored to sex differences in diabetes risk and management.

1.1 Estrogens - Metabolic Mechanisms of Action

Estrogens, primarily 17β-estradiol (E2), are steroid hormones synthesized mainly in the ovaries in premenopausal women and in peripheral tissues in both sexes. Beyond reproductive functions, estrogens exert, metabolic effects through estrogen receptors (ERα, ERβ, and G-protein-coupled estrogen receptor - GPER), which are expressed in liver, adipose tissue, skeletal muscle, pancreas in glucose and energy metabolism. [7,11]

Mechanistically, estrogen enhances insulin sensitivity and protects pancreatic β-cell function by maintaining insulin secretion and resisting β-cell apoptosis. Estrogen receptor activation stimulates signaling cascades such as PI3K-Akt, which improve glucose uptake and suppress hepatic gluconeogenesis. Estrogen also modulates tissue energy partitioning, favoring lipid utilization over carbohydrate storage and attenuates inflammation, which contributes to improved metabolic homeostasis.[7]

Sex differences in metabolic disease incidence aligned with estrogen status support the hormone's protective role. Premenopausal women generally exhibit better insulin sensitivity and lower risk of T2DM compared to men of similar age, a difference that diminishes after menopause when estrogen levels decline. [6,10]

At the cellular level, estrogens increase expression of glucose transporters (e.g. GLUT4) and enhance glucose uptake in adipose and muscle tissues. Estrogen deficiency or altered ER expression can impair these effects, contributing to insulin resistance and metabolic dysregulation. [7]

1.2 Androgens - Effects on Glucose Homeostasis and Insulin

Androgens such as testosterone influence metabolic processes by interacting with the androgen receptor (AR) in various tissues, including skeletal muscle, liver, adipose tissue, pancreatic β cells and the hypothalamus. AR activation has sexually dimorphic effects on metabolic health.

In males, physiological testosterone levels are associated with enhanced insulin sensitivity, improved muscle glucose uptake and reduced adiposity. Testosterone stimulates AR in muscle and hepatic tissues, increasing insulin receptor expression, glycogen synthesis and energy expenditure, which collectively support glucose homeostasis. [8,9]

Conversely, androgen deficiency (e.g. in aging men or prostate cancer patients undergoing androgen deprivation therapy) is linked to increased risk of insulin resistance, central adiposity and progression to T2DM. Evidence from clinical studies suggests that testosterone therapy may improve glucose tolerance and metabolic parameters, but long - term benefits and safety remain under investigation. [8,9]

In females, excessive androgen levels - as seen in polycystic ovary syndrome (PCOS) are associated with metabolic dysfunction, including insulin resistance, visceral fat accumulation and heightened T2DM risk. AR overactivation in women appears to promote adipose tissue dysfunction and antagonize the metabolic benefits typically conferred by estrogen signaling. [8]

1.3 Hormonal Dysregulation and Glucose Metabolism

Disruptions in sex hormone levels contribute significantly to metabolic dysregulation and the development of glucose intolerance and T2DM. In women, the transition through menopause characterized by a decline in circulating estrogen, correlates with increased insulin resistance, visceral adiposity and adverse lipid profiles. These changes elevate the risk of metabolic syndrome and T2DM in postmenopausal women compared with premenopausal women. [6,7]

In men, hypogonadism marked by low testosterone levels is consistently associated with insulin resistance, dyslipidemia, increased fat mass and higher incidence of T2DM. Observational and intervention, although factors such as obesity and aging confound this relationship. [8,9]

Additionally, sex hormone binding globulin (SHBG) levels are inversely associated with insulin resistance in both sexes and alterations in SHBG may further compound metabolic risk by affecting bioavailable estrogen and testosterone. [6]

1.4 Summary of the Importance of Sex Hormones in Metabolic Regulation

Estrogens and androgens significantly impact metabolic regulation through complex mechanisms involving receptor-mediated signaling in peripheral tissues and central metabolic centers. Estrogen generally enhances insulin sensitivity and protects against metabolic disease, whereas androgen effects are context dependent - promoting metabolic health at physiological levels in men but contributing to dysfunction when dysregulated in women. Hormonal imbalances, such as menopause or hypogonadism are closely linked to impaired glucose homeostasis and increased risk of metabolic disorders, underlining the importance of sex hormones in metabolic health across the lifespan.

2. Sex Differences in the Influence of Hormones on Diabetes

Biological sex is a critical determinant of metabolic regulation and diabetes susceptibility, partly mediated by sex hormones such as estrogens and androgens. These hormones modulate insulin sensitivity, body fat distribution, β-cell function and metabolic homeostasis, which results in distinct diabetes risk between women and men. [12,14]

2.1 Protective Effects of Estrogens Before Menopause

In premenopausal women, endogenous estrogens - particularly estradiol - are associated with improved insulin sensitivity and energy homeostasis. Estrogen signaling enhances glucose uptake, modulates adipose tissue distribution toward subcutaneous fat and supports β-cell function, collectively contributing to a lower risk of type 2 diabetes compared to age-matched men. [12,14]

The decline in estrogen levels following menopause coincides with increased visceral adiposity, dyslipidemia and higher insulin resistance, Randomized trials and epidemiological studies have shown that menopausal hormone therapy (MHT) with estrogens may delay the onset of type 2 diabetes in postmenopausal women by improving insulin sensitivity, glucose effectiveness and adipose tissue distribution, even though MHT application requires careful risk - benet evaluation. [13]

2.2 noIncreased Metabolic Risk After Menopause

The menopausal transition is associated with substantial metabolic changes that increase susceptibility to insulin resistance and type 2 diabetes mellitus. The decline in endogenous estrogen levels promotes unfavorable shifts in body fat distribution particularly increased visceral adiposity and impairs glucose homeostasis. These alterations are accompanied by reduced insulin sensitivity, β-cell dysfunction and a higher prevalence of metabolic syndrome traits in postmenopausal women. [15]

Furthermore, a recent systematic review and meta-analysis demonstrated that earlier age at menopause, reflecting prolonged exposure to low estrogen levels, is significantly associated with an increased risk of developing T2DM. This finding supports a direct link between estrogen deficiency during the menopausal transition and deterioration of metabolic control in women. [16]

2.3 Role of Androgens in Insulin Resistance

In men, androgens such as testosterone play a pivotal role in regulating body composition, insulin sensitivity and energy metabolism. Physiological testosterone levels are associated with reduced visceral adiposity, increased lean muscle mass and enhanced insulin action, which can protect against type 2 diabetes and metabolic dysregulation. [12,14]

Conversely, androgen deficiency is strongly linked to insulin resistance, visceral fat accumulation and increased type 2 diabetes risk. Hypogonal men frequently exhibit impaired insulin sensitivity and a higher incidence of metabolic syndrome components, suggesting a key role of testosterone in maintaining glucose homeostasis. [17]

2.4 Low Testosterone and Diabetes Risk

Low endogenous testosterone in men has been associated with increased adiposity, inflammation and reduced insulin sensitivity, which elevate diabetes risk. Clinical interventions with testosterone replacement in hypogonadal men may improve insulin sensitivity and glycemic control, although mechanisms remain under active investigation. [18]

3. Clinical Consequences

Sex-specific differences in hormonal regulation significantly affect the risk, presentation and management of type 2 diabetes mellitus. These biological disparities influence not only disease onset but also complication patterns and responses to prevention and treatment strategies. Recognizing sex-based mechanisms to diabetes care. [19,20]

3.1 Impact on Risk of Type 2 Diabetes

Men and women differ in the trajectory leading to T2DM. Men often develop diabetes at a younger age and at a lower body mass index, whereas women usually present later but with a higher burden of adiposity and cardiometabolic risk factors, particularly after menopause when estrogen protection declines. [19] A large meta - analysis demonstrated that women with diabetes lose much of their premenopausal cardiovascular protection and experience a disproportionately higher relative risk of coronary heart disease and stroke compared with men with diabetes. [22] These findings suggest that hormonal changes modify risk accumulation differently between sexes, making postmenopausal women particularly vulnerable to adverse outcomes. [22] Furthermore, endocrine disturbances such a low testosterone in men and estrogen deficiency in women exacerbate insulin resistance and visceral adiposity, accelerating progression from prediabetes to overt T2DM. [21]

3.2 Differences in Clinical Presentation and Complications

Sex differences are evident in both macrovascular and microvascular complications. Women with T2DM have a higher relative risk of cardiovascular disease compared with men, despite often receiving similar glycemic control. This excess risk is partly attributed to delayed diagnosis, greater clustering of risk factors and loss of estrogen - mediated vascular protection. [19]

Conversely, men appear more prone to certain microvascular complications, including nephropathy and neuropathy, whereas women report higher symptom burden and poorer quality of life related to diabetic complications. [19] Hormonal influences on inflammation, endothelial function and fat distribution may partly explain these sex-specific complication profiles. Additionally, sex hormones influence body composition and lipid metabolism, which further modulate disease severity and complication development in diabetes. [21]

3.3 Implications for Prevention and Treatment

Sex - aware prevention strategies are increasingly recommended in diabetes care. Evidence indicates that women are less likely to receive intensive cardiovascular risk management, including statins and antihypertensive therapy, despite having a higher relative risk of cardiovascular events once diabetes develops. [19]

Lifestyle and pharmacological interventions may also show sex - specific effectiveness. Men tend to achieve greater improvements in weight and insulin sensitivity during standardized lifestyle programs, while women may benefit more from tailored interventions that address hormonal status, menopause changes and psychosocial determinants. [19] Moreover considering hormonal profiles - such as testosterone deficiency in men or estrogen loss in postmenopausal women - may improve individualized risk assessment and therapeutic strategies. Integrating sex - specyfic biology into prevention and treatment models represents an important step toward precision medicine in diabetes management. [20]

4. Conclusions

Sex differences in glucose metabolism and type 2 diabetes are strongly influenced by the action of sex hormones, particularly estrogens, androgens and sex hormone - binding globulin (SHBG). Evidence from recent clinical and population - based studies indicates that these hormones contribute to distinct metabolic profiles in women and men and partly explain differences in diabetes risk, progression and complications. [23]

In women, estrogens exert protective metabolic effects before menopause by improving insulin sensitivity and regulating fat distribution, whereas estrogen deficiency after menopause is associated with increased insulin resistance and higher incidence of type 2 diabetes. [24] In contrast, in men low testosterone concentrations are consistently linked to higher adiposity, impaired glucose regulation and increased diabetes risk, suggesting an important role of androgens in maintaining metabolic health. [25] Furthermore, SHBG has emerged as a key mediator of sex-specific metabolic risk. Higher SHBG levels are associated with lower incidence of type 2 diabetes in both sexes, with stronger effects observed in women, indicating its relevance for risk stratification and prevention strategies. [26] These observations support the concept that biological sex and hormonal status should be incorporated into clinical evaluation, prevention programs and therapeutic decision - making in diabetes care. Overall, the available evidence emphasizes the importance of sex - specific approaches in diabetes research and clinical practice. Future studies should focus on longitudinal and interventional designs to better define causal mechanisms and evaluate whether hormone - targeted strategies can improve personalized prevention and treatment of type 2 diabetes.

Author's Contribution

Conceptualization: Karolina Dubiel, Weronika Białowąs; methodology: Sandra Balon, Michał Maćkowski; software: Monika Białowąs, Natalia Sojka, Jakub Magnowski; check: Kacper Urban, Piotr Szwed; formal analysis: Adam Śmietana, Kacper Urban; investigation: Natalia Sojka, Piotr Szwed; resources: Monika Białowąs, Natalia Sojka; data curation: Michał Maćkowski, Sandra Balon; writing-reviewand: Karolina Dubiel, Piotr Szwed, Jakub Magnowski; writing-rough preparation: Weronika Białowąs, Natalia Sojka, Sandra Balon; visualization: Karolina Dubiel, Kacper Urban; supervision: Adam Śmietana, Michał Maćkowski; project administration Monika Białowąs, Weronika Białowąs

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All authors have read and agreed with the published version of the manuscript. Declaration of the use of generative AI and AI-assisted technologies in the writing process. In preparing this work, the authors used ChatGPT for the purpose of improving language and readability. After using this tool the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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