

## Therapeutic Potential of *Moringa Oleifera* in Testosterone-Induced Polycystic Ovary Syndrome

**Iram Altaf**

Department of Biological Science, Superior University Lahore, Pakistan

**Asad Munir\***

Department of Biological Science, Superior University Lahore, Pakistan

Email: [asad.munir.sgd@superior.edu.pk](mailto:asad.munir.sgd@superior.edu.pk)

**Talha Akhtar**

Department of Biotechnology, University of Sargodha, Pakistan

**Kashif Ameer**

Department of Food Science and Technology, University of Sargodha, Pakistan

**Syed Zain Ali Raza Shah**

Department of Biological Science, Superior University Lahore, Pakistan

**Sami Ullah**

Department of Biological Science, Superior University Lahore, Pakistan

### Abstract

Polycystic ovary syndrome (PCOS) is a complex endocrine and metabolic disorder affecting women of reproductive age and is a leading cause of infertility worldwide. It is characterized by hyperandrogenism, ovulatory dysfunction, insulin resistance, and metabolic abnormalities that increase the risk of long-term complications. Conventional treatments primarily focus on symptom management and often produce undesirable side effects, highlighting the need for alternative therapeutic strategies. The present study aimed to investigate the therapeutic potential of *Moringa oleifera* aqueous extract (MOE) in alleviating reproductive and metabolic disturbances associated with PCOS using an experimental rat model. PCOS was induced in female rats through testosterone propionate administration combined with a high-fat diet, mimicking key pathological features of the syndrome. Following induction, animals were treated with varying doses of MOE over defined time periods. Reproductive, metabolic, and hematological parameters were evaluated, along with ovarian morphology and renal characteristics, to assess treatment outcomes. The PCOS model exhibited hallmark features of the condition, including ovarian cyst formation, anovulation, hyperandrogenism, insulin resistance, and altered lipid metabolism. Treatment with MOE resulted in notable improvement in ovarian

ovarian morphology and renal characteristics, to assess treatment outcomes. The PCOS model exhibited hallmark features of the condition, including ovarian cyst formation, anovulation, hyperandrogenism, insulin resistance, and altered lipid metabolism. Treatment with MOE resulted in notable improvement in ovarian

### Author Details

Keywords: Polycystic Ovary Syndrome, *Moringa Oleifera*, Hyperandrogenism, Insulin Resistance, Rat Model

Received on 15 Nov 2025

Accepted on 15 Dec 2025

Published on 30 Dec 2025

Corresponding E-mail & Author\*:

**Asad Munir\***

Department of Biological Science, Superior University Lahore, Pakistan

Email:

[asad.munir.sgd@superior.edu.pk](mailto:asad.munir.sgd@superior.edu.pk)

morphology, reduction in PCOS severity, and partial restoration of metabolic balance, with more pronounced effects observed at higher doses and longer treatment durations. Although dyslipidemia was not fully replicated in the model, MOE demonstrated beneficial hypoglycemic and insulin-sensitizing effects. No significant renal damage was observed despite increased kidney size in PCOS-induced rats. In conclusion, *Moringa oleifera* shows promising therapeutic potential in mitigating key reproductive and metabolic features of PCOS. Further studies are warranted to elucidate its mechanisms of action and explore its clinical applicability in PCOS management.

## **Introduction**

A lot of people all over the world can't have kids, which can be bad for their mental and emotional health. One-third of infertility instances are linked to women only. Tubal diseases, uterine abnormalities, endocrine disorders, and ovulatory disorders are the most prevalent reasons why women can't get pregnant. Polycystic ovarian syndrome (PCOS) and other ovulatory abnormalities are responsible for as much as 25% of cases of female infertility [1].

PCOS is one of the most prevalent reasons why women can't get pregnant. It causes 70% of anovulatory cycles in adult women [2]. The CDC (2022) says that this mixed-up syndrome affects up to 5 million women in the United States. The prevalence of PCOS among women of reproductive age ranges from 6% to 20%, contingent upon the diagnostic definition employed [3]. This syndrome is marked by irregularities in both the endocrine and metabolic systems. The clinical manifestations encompass amenorrhea, oligomenorrhea, anovulation, hirsutism, acne, obesity, insulin resistance, and infertility [4]. PCOS increases the chances of getting type II diabetes, high blood pressure, heart attacks, high cholesterol, and other health problems [5].

In patients with PCOS, disturbances in the hypothalamic-pituitary-gonadal (HPG) axis result in symptoms related to infertility. The HPG axis is very important in normal mammal reproduction. Neurons in the hypothalamus emit Gonadotropin Releasing Hormone (GnRH) along this axis. This hormone is the fundamental thing that makes the reproductive system work. The anterior pituitary makes gonadotropins, like Follicle Stimulating Hormone (FSH) and Luteinizing Hormone (LH), based on how often and how strong GnRH is released [5].

GnRH is released in bursts when estrogen and progesterone provide positive or negative signals. In healthy women of reproductive age, the pulsatile release pattern of GnRH alters in response to steroid feedback, facilitating appropriate follicular growth and ovulation. Estrogen levels are low at the start of the follicular phase and rise midway through this period of the cycle. During the mid-follicular phase, estradiol positive feedback raises the frequency of GnRH pulses, which in turn raises the release of LH, which starts ovulation. Following the ovulatory phase, elevated progesterone levels from the corpus luteum diminish GnRH pulse frequency via progesterone-negative feedback [6].

This hormonal imbalance leads to the characteristic condition of anovulation or oligoovulation in women with PCOS. Anovulation, characterized by the lack of normal ovulatory cycles, or oligoovulation, marked by irregular ovulation, are common indications of the reproductive dysfunction linked to PCOS. The increased amounts of androgens, including testosterone, plus the fact that the body is resistant to insulin make the imbalance in the Hypothalamus-Pituitary-Gonad (HPG) axis even worse. These abnormalities affect the regulation of GnRH pulsatility, which in turn disrupts the coordinated release of FSH and LH. Consequently, the typical cyclical alterations in ovarian follicular growth and the subsequent release of mature oocytes

are obstructed, leading to the ovulatory difficulties encountered by women with PCOS [7].

Because of these problems, women with PCOS often have menstrual periods that are not regular or normal. Other models of PCOS, including non-primate mammals, exhibit disturbed cycles known as estrous cycle disturbances. These disturbances are symptomatic of atypical ovulation as observed in women with PCOS. To tackle the pathophysiology of PCOS, contemporary therapeutic approaches focus on mitigating the syndrome's symptoms through the use of insulin sensitizers, anti-androgen medicines, and hormonal contraceptives. *Moringa oleifera* (HU) is a plant frequently utilized in traditional medicine to enhance insulin sensitivity [8]. Insulin sensitizers like metformin are used to treat PCOS. An ethnobotanical that lowers blood sugar levels might have effects similar to those of insulin sensitizers used to treat PCOS. The objective of this study is to examine the potential therapeutic effects of *Moringa oleifera* aqueous extract (MOE) in alleviating PCOS in rats [8].

A woman may be more likely to get PCOS if she has a number of risk factors. These include having a first-degree relative with the condition, being overweight before puberty, having a congenital virilizing disorder, and being born with a birth weight that is higher or lower than typical for the gestational age. Other risk factors are early adrenarche (when adrenal hormones start working) and using valproic acid, an antiepileptic drug that is known to change androgen levels [9].

Epidemiological studies have also shown that the frequency and symptoms of PCOS vary by ethnicity and location. For example, Mexican American women have been found to have a higher prevalence than non-Hispanic White and African American women. These differences show how complicated the relationship is between genetics, environment, and lifestyle in deciding how PCOS shows up in different groups of people. Longitudinal studies indicate that the phenotypic manifestation of PCOS may evolve with time. In a five-year follow-up research, numerous women who initially exhibited the typical form of PCOS (Type A phenotype) no longer satisfied the whole diagnostic criteria after several years, indicating that symptoms may vary or decrease due to hormonal, metabolic, or lifestyle modifications [10].

In general, our results show that PCOS is a dynamic and diverse condition, which emphasizes the need for personalized assessment and management techniques that consider genetic background, environmental factors, and differences in diagnosis. The pathogenesis of polycystic ovarian syndrome (PCOS) is exceedingly intricate, encompassing various overlapping hormonal, metabolic, and molecular disruptions. It is now commonly recognized that PCOS is not merely a reproductive issue but also a metabolic condition, significantly associated with insulin resistance and chronic inflammation [11].

The core of this disorder is a malfunction of the hypothalamic–pituitary–ovarian (HPO) axis, which causes important hormones including luteinizing hormone (LH), follicle-stimulating hormone (FSH), and antimüllerian hormone (AMH) to be out of balance. These hormonal imbalances interfere with ovarian steroidogenesis, the mechanism of sex hormone synthesis, leading to irregular ovulation and excessive androgen production [12].

The bidirectional link between insulin resistance and hyperandrogenism is a key characteristic of PCOS. Around 70% of women with PCOS have some form of insulin resistance, which leads to high insulin levels (hyperinsulinemia). High insulin not only messes up regular glucose metabolism, but it also directly stimulates ovarian theca cells, which makes more androgens. Insulin also makes the ovary more sensitive to LH by messing up its regular process of becoming less sensitive during the ovulatory cycle. This makes a loop that keeps going: high insulin levels cause too

many androgens, and too many androgens make insulin signaling worse, making the metabolic imbalance worse [13].

Within the ovary, theca cells in PCOS patients overexpress many steroidogenic enzymes, including P450c17, which plays a crucial role in androgen production. Granulosa cells, conversely, experience premature luteinization attributed to elevated androgen and insulin levels, hence impeding follicular growth and maturation. Insulin also lowers the amount of sex hormone-binding globulin (SHBG) made in the liver, which raises the amounts of free circulating androgens. It also changes the signaling of insulin-like growth factor (IGF), which is very important for the proliferation of follicles. Low amounts of IGF-1 binding protein and variations in IGF-1 activity make it hard for follicles to mature normally, which can lead to oligo-ovulation or anovulation. Insulin resistance in PCOS can manifest irrespective of body weight, as even slim women with PCOS frequently exhibit indications of metabolic dysfunction. In these patients, high levels of C-reactive protein (CRP) show that they have low-grade chronic inflammation, which is now recognized to be a significant part of the development of PCOS [14].

Approximately two-thirds of women with PCOS present with functional ovarian hyperandrogenism (FOH), which is marked by an excessive synthesis of androgens, including testosterone and 17-hydroxyprogesterone (17-OHP), in response to gonadotropin stimulation. A smaller group of women have functional adrenal hyperandrogenism, which means that their adrenal glands make too many androgens instead of their ovaries. While such testing has limited clinical utility currently, it elucidates the hormonal heterogeneity of PCOS [15].

An excess of androgens also has an effect on the growth of ovarian follicles. It enhances the recruitment of primordial follicles while inhibiting the selection of a dominant follicle, resulting in the buildup of immature follicles, a characteristic feature of polycystic ovarian morphology (PCOM). In PCOS, LH levels are generally too high. LH is necessary for the proper production of steroid hormones, but too much LH causes ovarian theca cells to keep making androgens. This hormonal imbalance keeps the cycle of hyperandrogenism and anovulation going. The imbalance between LH and FSH stops the hypothalamus from generating gonadotropin-releasing hormone (GnRH), which makes the hormonal environment even more aberrant. Elevated LH levels promote androgen synthesis, whereas diminished FSH levels inhibit the conversion of androgens into estrogens via aromatase activity in granulosa cells. This causes too many androgens to be made and not enough estradiol to be made, which leads to a non-cyclic hormonal rhythm [16].

In obese women with PCOS, surplus androgens are transformed into estrogens (predominantly estrone) within adipose tissue, resulting in a perpetual state of estrogen exposure. This unopposed estrogen stimulation can lead to endometrial hyperplasia, which raises the risk of endometrial abnormalities. New studies also suggest that immunological reactions and long-term inflammation may be involved in the ovarian problems that come with PCOS [17].

#### Pathogenesis of PCOS

Hypothalamic neuroendocrine dysfunctions characterize the etiology of PCOS. Individuals diagnosed with PCOS exhibit elevated or accelerated GnRH pulse frequency. This then makes LH levels go up and FSH levels go down. The unequal amounts of LH and FSH being made is because cells that make LH are more sensitive to a faster GnRH pulsatile frequency [18]. Due to the low levels of FSH in women with PCOS, follicles do not mature, which means that ovulation does not happen. These immature follicles then come together on the ovaries to form what are known as cysts in PCOS. Decreased FSH levels impede the standard conversion of androgens to estrogen, while raised LH levels promote the excessive production of androgens,

leading to heightened testosterone levels in affected women. Hyperandrogenism, the clinical characteristic of PCOS, is present in up to 90% of people with the condition [19].

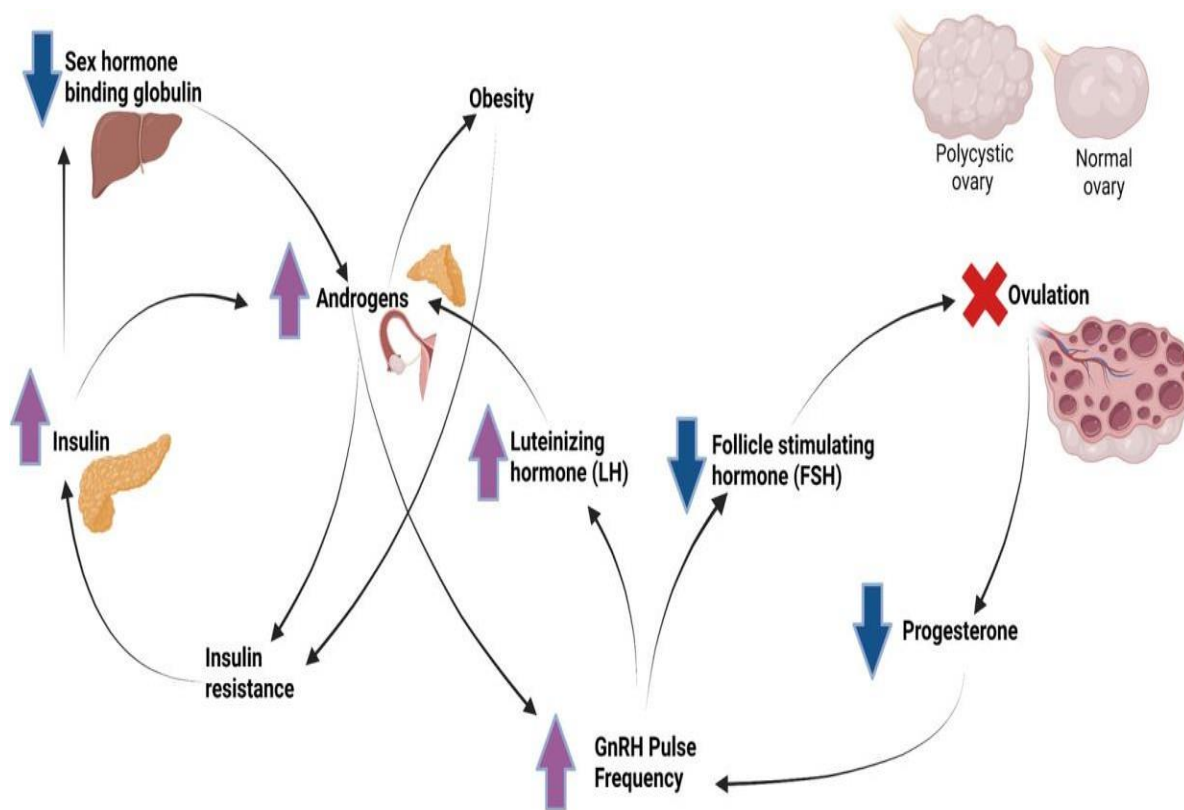
#### Metabolic dysfunction in PCOS

PCOS is linked to metabolic problems as well as neuroendocrine dysfunctions. These metabolic dysfunctions arise from excessive androgen production, which is the primary characteristic of PCOS. Hyperandrogenism has been demonstrated to affect multiple metabolic tissues, including adipose tissue, liver, pancreas, and muscle. Research indicates that hyperandrogenism contributes to metabolic disorders, such as obesity and insulin resistance, frequently observed in PCOS. Insulin resistance is the most common metabolic problem in PCOS, affecting up to 70% of all patients. This underscores the critical influence of hyperandrogenism in the manifestation of metabolic disorders in individuals with PCOS [19].

Women with hyperandrogenism tend to accumulate more visceral fat than women with normal androgen levels, who mostly store fat in subcutaneous adipose tissues. This disease also causes adipocyte hypertrophy, which is seen in both women and rodents with high androgen levels. This could cause problems with adipose tissue. This malfunction, marked by compromised adipocyte activity, may lead to metabolic disorders, including insulin resistance [20]. Furthermore, adipose tissue is vital for fat storage and controlling the release of adipokines like leptin and adiponectin, which are necessary for keeping insulin sensitivity and glucose levels stable. Dysfunction or absence of adipose tissue, as seen in women with hyperandrogenism, might increase circulating triglycerides and fatty acids, hence worsening insulin resistance and disturbing metabolic equilibrium. Moreover, hyperandrogenism has been linked to reduced concentrations of insulin-sensitizing adipokines, including adiponectin and omentin-1, in patients and animal models of PCOS [21]

Prior research has established a correlation between hyperandrogenism and non-alcoholic fatty liver disease (NAFLD) as well as other hepatic disorders. Elevated levels of Alanine aminotransferase (ALT), a marker of hepatic injury, have been demonstrated to correlate positively with heightened androgen levels. Other research that caused hyperandrogenism in mice led to increased hepatic fat storage. Hyperandrogenism impacts skeletal muscles, chiefly influencing insulin sensitivity and glucose uptake. Women with PCOS exhibit decreased insulin-stimulated glucose uptake, which is linked to skeletal muscle insulin resistance. Changes in insulin receptors and the phosphorylation patterns of intracellular insulin pathways have been demonstrated to play a role in skeletal muscle insulin resistance. Furthermore, insulin-facilitated glucose transport in muscles has been demonstrated to be diminished, resulting in compensatory hyperinsulinemia, which is another sign of PCOS. Excessive androgens are believed to directly and indirectly cause  $\beta$ -cell dysfunction in the pancreas since these cells have androgen receptors. Hyperandrogenism leads to the excessive activation of androgen receptors in  $\beta$  pancreatic cells, potentially inducing mitochondrial dysfunction, oxidative damage, and insulin hypersecretion [17].

Consequently, individuals or animal models of PCOS exhibit clinical characteristics of metabolic syndrome, including but not limited to obesity, dyslipidemia, and insulin resistance. Polycystic ovary syndrome (PCOS) increases the risk of type II diabetes mellitus (T2DM), hypertension, myocardial infarction, and various other disorders. Insulin resistance and hyperandrogenism are interlinked in the etiology of PCOS and both facilitate the emergence of additional symptoms associated with the condition. Current data indicates that heightened levels of compensatory insulin further enhance ovarian steroidogenesis, hence affecting the endocrine system [18].



**Multiple systems and comorbidities interact in PCOS, showing how varied it is and how hard it is to figure out what causes it (Baillargeon & Nestler, 2006).**

The numerous metabolic changes linked to this condition elevate the chance of acquiring additional problems, including chronic kidney disease (CKD). Insulin resistance, specifically, is a major factor in the onset of metabolic issues in PCOS, including those that impact the kidneys. Insulin resistance and oxidative stress have been identified as potential contributors to renal damage. Insulin resistance is frequently linked to persistent low-grade inflammation and heightened oxidative stress, which may impair kidney cells and facilitate the onset of kidney disease [19]. Elevated synthesis of proinflammatory cytokines, connective tissue growth factors, and profibrotic factors, coupled with microvascular injury and renal ischemia, may constitute the underlying causes of severe renal impairment (Prasad, 2014). Also, high levels of insulin (another condition that often goes along with PCOS) can directly affect the kidneys by making them hold onto sodium and boosting the production of several growth hormones that can harm the kidneys. Androgen excess in PCOS exacerbates renal impairment by promoting increased visceral fat accumulation. Hyperandrogenism is also associated with endothelial dysfunction, which can worsen kidney injury [20].

PCOS is a metabolic and reproductive endocrine condition, and its treatments focus on the symptoms of these systems either alone or in combination. Presently recognized therapy utilizes insulin sensitizers, such as metformin or clomiphene citrate. In a prior clinical investigation, metformin demonstrated efficacy in restoring and inducing ovulation in women with PCOS, whilst reducing androgen production. Other therapy options are estrogen-progestin drugs, anti-androgen receptor blockers, and modifications to one's lifestyle, like exercising to lose weight if someone is overweight [21].

## Phenotypes of PCOS

PCOS is a complex condition, and its tendency has been linked to specific genes, gene-gene interactions, and gene-environment interactions. Twin studies corroborated these findings by demonstrating that 72% of the variance in PCOS is attributable to genetic factors. There are four forms of PCOS: A, B, C, and D. Phenotype A includes women who have cystic ovaries, don't ovulate or only ovulate a little, and have high levels of androgens. Phenotype B includes women who have normal ovaries, do not ovulate regularly, and have too much testosterone. Phenotype C encompasses women exhibiting cystic ovaries, regular ovulatory cycles, and hyperandrogenism. Phenotype D includes women who have cystic ovaries, don't ovulate very often or at all, and have normal amounts of androgens. Phenotypes A and B are categorized as typical PCOS, characterized by the most severe symptoms, including heightened risks of metabolic syndrome, insulin resistance, and compensatory hyperinsulinemia, accompanied by higher menstrual disruptions [21].

Phenotype C is known as the ovulatory PCOS. It causes higher amounts of insulin resistance and androgens, which leads to increased hirsutism. This phenotype is predominantly seen in affluent socioeconomic groups relative to other phenotypes. Phenotype D, or non-hyperandrogenic PCOS, leads to the least metabolic problems and ovulatory cycles that are better or almost normal. The prevalence of PCOS phenotypes varies, with Phenotype A being the most prevalent, comprising 45-66% of cases, followed by Phenotype B at 8-33%, Phenotype C at 3-29%, and Phenotype D at merely 0-23% [22].

## Genotypes associated with PCOS

Because PCOS changes the normal shape and function of the ovaries and the production of androgens, various genes that are involved in ovarian and adrenal steroidogenesis can be linked to PCOS. The Cyp11A gene makes an enzyme that the ovaries need to turn cholesterol into progesterone. Cyp21 is another gene that codes for an enzyme that helps the ovaries make steroids and is linked to PCOS. Cyp17, a gene encoding an enzyme involved in steroidogenesis, is linked to increased androgen levels, while polymorphisms in the promoter region of this gene are connected with patients with PCOS. CYP19 encodes the aromatase enzyme, which is responsible for estrogen conversion and has reduced activity in women with PCOS.

The Androgen Receptor (AR) Gene and the Sex Hormone Binding Globulin (SHBG) gene are two other genes that can be linked to PCOS. The AR gene, located on the arm of an X chromosome, has been associated with PCOS. It has been demonstrated that any mutation in the AR gene, along with the inactivation of the X chromosome, can lead to PCOS. The SHBG gene makes the SHBG protein in the liver. SHBG is much lower in women with PCOS, which causes hyperandrogenism because SHBG regulates blood androgen levels by attaching to androgens. Research indicates that diminished SHBG levels correlate with single nucleotide polymorphisms in the SHBG gene [22].

Genes implicated in the control and function of gonadotropins, including the LH receptor gene, the follicle-stimulating hormone receptor (FSHR) gene, and the AMH gene, are connected with the etiology of PCOS. Researchers have discovered that variations in the LH gene are linked to PCOS. The AMH gene typically encodes a protein associated with infertility. Subsequent exon sequencing and genome-wide association studies (GWAS) identified numerous polymorphisms in the AMH gene as indicators or predictors of polycystic ovary syndrome (PCOS) development in women [23].

Insulin plays a role in producing androgens, and many women with PCOS have insulin resistance. This is why genes that affect insulin synthesis and activity are

linked to PCOS. Women with PCOS have higher levels of insulin receptor substrate genes that make insulin receptors. Calpain10 is another gene that is linked to both Type I diabetes and PCOS. Changes in the Calpain10 gene have been linked to PCOS. Other genes associated with diseases such as obesity are also connected to PCOS. The Fat mass obesity gene is linked to type II diabetes, and variations of this gene are also associated with PCOS [23].

### **Moringa oleifera**

In many regions of the world, people still use ethnobotanicals as medicine. Numerous medicinal plants exhibit pharmacological effects akin to those of medicines. In many countries, folk medicine uses ethnobotanicals to treat a number of metabolic illnesses. *Moringa oleifera* (HU), (K. Schum.) is a medicinal plant that is utilized as such. The family Apocynaceae includes the tropical rainforest tree HU, which is prevalent in the western sub-Saharan regions of Africa. The ethnomedicinal applications of MOE is used to treat diabetic mellitus, sexually transmitted illnesses, dysmenorrhea obesity, and anemia [24].

Previous studies demonstrate that dose-dependent administration of MOE solution has hypoglycemic effects. Other research indicates the antihyperglycemic properties of MOE in rats, proposing that these benefits arise from the inhibition of intestinal glucose absorption, a mechanism recognized in plants with elevated saponin content such as MOE. MOE may potentially facilitate its effects via other mechanisms to enhance hepatic glucose uptake. Research examined the phytochemical content of MOE, revealing that the crude plant extract had saponins, glycosides, steroids, tannins, phenols, and alkaloids. Erinidine, a bisindole alkaloid extracted from HU, may serve as the antihyperglycemic agent responsible for the hypoglycemic effects of HU by blocking intestinal glucose absorption [25].

### **Moringa oleifera and polycystic ovary syndrome**

As previously noted, contemporary therapy strategies for PCOS include insulin sensitizers, like metformin, to manage insulin resistance and alleviate other symptoms of PCOS, hence facilitating ovulation. Insulin sensitizers or euglycemic medicines lower blood sugar levels by making the body's tissues more sensitive to insulin and stopping the liver from making glucose [26]. Recently, herbal therapy has become a viable way to treat PCOS by focusing on these symptoms. For example, *Cinnamomum verum* has been shown to help in PCOS by lowering insulin resistance. Other research has demonstrated that a mixture of flaxseed and spearmint enhanced ovarian and endocrine characteristics in rat models of PCOS [27]. Another herbal method for treating PCOS, using Berberine, which is an active part of *Rhizoma coptidis*, was found to work better. Compared to the widely utilized insulin sensitizer metformin by improving insulin sensitivity and lowering testosterone levels and LH/FSH ratios [28-29]. While the application of MOE in diabetic treatment has been previously examined, no research have been conducted to explore the impact of HU on PCOS. This study aims to examine the metabolic and hematological changes generated by oral administration of MOE in testosterone propionate-induced PCOS mice, to evaluate the efficacy of its hypoglycemic effects in the therapy of PCOS [30].

### **CONCLUSIONS**

In summary, our research elucidates the intricate characteristics of polycystic ovarian syndrome (PCOS) and the prospective therapeutic benefits of *Moringa oleifera* in a rat model. Our PCOS model displayed clinical symptoms of the illness, including ovarian cysts, hyperandrogenism, ovulatory dysfunction, and metabolic abnormalities, mimicking characteristics of the real condition. Our results indicate that MOE

treatment may result in a decrease in PCOS morphology, with the most significant therapeutic effects noted at extended treatment periods and elevated dosages.

Although it produced metabolic characteristics akin to those observed in PCOS patients, including insulin resistance and modified lipid profiles, our model did not entirely recreate the dyslipidemia commonly associated with PCOS. This inconsistency highlights the necessity for additional research to clarify the processes driving these metabolic alterations in the context of PCOS. Our study also sheds light on the renal consequences of PCOS induction, revealing larger kidneys in rats subjected to PCOS. Nonetheless, no substantial renal impairment was seen, indicating that the provided testosterone dosages and high-fat diet did not cause kidney damage. In general, our work adds to what is already known about the pathophysiology of PCOS and shows that MOE could be a useful addition to other treatments for regulating symptoms and the severity of the disease. Further investigation is necessary to clarify the specific mechanisms of action of MOE and its prospective clinical implications in the management of PCOS.

## REFERENCES

- Adeneye Adejuwon, A., Crooks, P. A., Fadhel-Albayati, Z., Miller, A. F., Zito, S. TENA- SEMPERE., Adeyemi, O. O., & Agbaje, E. O. (2012). Antihyperglycemic profile of erinidine isolated from *Moringa oleifera* seed. *African journal of traditional, complementary, and alternative medicines* : AJTCAM, 10(2), 189–202.
- Iftikhar, A., Iqbal, A., Naveed, N., Akbar, I., Fatima, U., & Bilal, A. (2021). An overview of harmful effects of polycystic ovary syndrome. *Journal of Oncology Research Review & Reports*, 156, 2-5.
- Rizwan, M., Mushtaq, M., Bilal, A., Nawaz, T., Riaz, K., Hussain, M., ... & Basharat, M. (2024). Trace Out the Improvement Level and Awareness of Polycystic Ovary Syndrome (Pcos) among General People and Educational Institute of Developed and Developing Countries. *Journal of Bioresource Management*, 11(3), 5.
- Bilal, A., Ahmad, S., Tanvir, F., Tariq, M., Ramzan, K., Saleem, M., & Saleem, H. G. M. (2022). Predictive modeling of N-acetyl transferase 2 single nucleotide polymorphisms and breast cancer risk using in-silico approaches. *The Journal of Microbiology and Molecular Genetics*, 3(2), 105-121.
- Jawad, M., Bilal, A., Khan, S., Rizwan, M., & Arshad, M. (2023). Prevalence and awareness survey of tuberculosis in the suspected population of Bajaur Agency in Fata, Pakistan: Prevalence and awareness survey of tuberculosis. *Pakistan Journal of Health Sciences*, 56-61.
- Bilal, A., Bibi, R., Umar, M., Sajjad, A., Kharal, S., Noor, E., ... & Munir, A. (2025). The relationship between obesity and breast cancer among women of Punjab, Pakistan. *The Research of Medical Science Review*, 3(2), 668-84.
- Adeneye, A. A., & Adeyemi, O. O. (2009). Further evaluation of antihyperglycaemic activity of *Moringa oleifera* (K. Schum) Hallier f. seed extract in experimental diabetes. *Journal of ethnopharmacology*, 126(2), 238–243. <https://doi.org/10.1016/j.jep.2009.08.037>
- Adeneye, A. A., & Crooks, P. A. (2015). Weight losing, antihyperlipidemic and cardioprotective effects of the alkaloid fraction of *Moringa oleifera* seed extract on normal and triton- induced hyperlipidemic rats. *Asian Pacific Journal of Tropical Biomedicine*, 5(5), 387- 394.
- Adeneye, A. A., Adeyemi, O. O., Agbaje, E. O., & Sofidiya, M. O. (2011). The novel antihyperglycaemic action of *Moringa oleifera* seed fractions mediated via intestinal glucose uptake inhibition. *African journal of traditional,*

- complementary, and alternative medicines : AJTCAM, 9(1), 17–24. <https://doi.org/10.4314/ajtcam.v9i1.3>
- Ajiboye, T. O., Hussaini, A. A., Nafiu, B. TENA-SEMPERE., & Ibitoye, O. B. (2017). Aqueous seed extract of *Moringa oleifera* (K. Schum.) Hallier f. (Apocynaceae) palliates hyperglycemia, insulin resistance, dyslipidemia, inflammation and oxidative stress in high-fructose diet-induced metabolic syndrome in rats. *Journal of ethnopharmacology*, 198, 184–193.
- Bilal, A. (2021). Clinical diagnosis and treatment of absence seizures: Case study. *MAR Ophthalmology*, 2(1).
- Sajjad, M. K., Bilal, A., Iftikhar, A., Awais, M., Asif, I., Shaheen, F., & Zahoor, G. (2024). Examining the Association Between Pesticide Exposures and Chronic Diseases in Agricultural Workers. *Remittances Review*, 9(2), 2153–2176.
- Ajiboye, T. O., Hussaini, A. A., Nafiu, B. TENA-SEMPERE., & Ibitoye, O. B. (2017). Aqueous seed extract of *Moringa oleifera* (K. Schum.) Hallier f. (Apocynaceae) palliates hyperglycemia, insulin resistance, dyslipidemia, inflammation, and oxidative stress in high-fructose diet-induced metabolic syndrome in rats. *Journal of ethnopharmacology*, 198, 184–193. <https://doi.org/10.1016/j.jep.2016.11.043>
- Akre, S., Sharma, K., Chakole, S., & Wanjari, M. B. (2022). Recent Advances in the Management of Polycystic Ovary Syndrome: A Review Article. *Cureus*, 14(8), e27689. <https://doi.org/10.7759/cureus.27689>
- ALhabardi, N. A., Al-Wutayd, O., Eltayieb, K. M., Shiha, TENA-SEMPERE. S., Al-Shafei, A. I., & Adam, I. (2020). Peripheral hematological parameters in women with polycystic ovary syndrome. *Journal of International Medical Research*, 48(9), 0300060520952282.
- Arora, S., Kaur, P., Garg, R., Kaur, M., & Rani, S. (2022). Assessment Of Vitamin D And Calcium Levels In Women With Pcos: An Observational Study. *Assessment*, 15(12).
- Barber, T. M., Hanson, P., Weickert, M. O., & Franks, S. (2019). Obesity and Polycystic Ovary Syndrome: Implications for Pathogenesis and Novel Management Strategies. *Clinical medicine insights. Reproductive health*, 13, 1179558119874042. <https://doi.org/10.1177/1179558119874042>
- Blair, J. A., McGee, TENA-SEMPERE., Bhatta, S., Palm, R., & Casadesus, G. (2015).
- Blank, S. K., McCartney, C. R., & Marshall, J. C. (2006). The origins and sequelae of abnormal neuroendocrine function in polycystic ovary syndrome. *Human reproduction update*, 12(4), 351–361. <https://doi.org/10.1093/humupd/dml017>
- Dubale, S., Kebebe, D., Zeynudin, A., Abdissa, N., & Suleman, S. (2023). Phytochemical Screening and Antimicrobial Activity Evaluation of Selected Medicinal Plants in Ethiopia. *Journal of experimental pharmacology*, 15, 51–62. <https://doi.org/10.2147/JEP.S379805>
- Akbar, B., Tanvir, F., Irfan, A., Bilal, A., Nawaz, A., Minhas, H., & Basharat, M. (2025). ANALYSIS OF NON-SYNONYMOUS SNPS IN THE SET ONCOGENE AND THEIR IMPACT ON LEUKEMIA. *Journal of Medical & Health Sciences Review*, 2(2).
- Bilal, A., Anjum, M. I., Naveed, N., Saif-ur-Rehman, M., Ali, U., & Iftikhar, A. (2021). Impacts of abusing drugs on our society. *J Med Res Surg*, 2(3), 1–3.
- Dunaif A. (1999). Insulin action in the polycystic ovary syndrome. *Endocrinology and metabolism clinics of North America*, 28(2), 341–359. [https://doi.org/10.1016/s0889-8529\(05\)70073-6](https://doi.org/10.1016/s0889-8529(05)70073-6)
- Pasquali, R., & Gambineri, A. (2013). Insulin sensitizers in polycystic ovary syndrome. *Frontiers of hormone research*, 40, 83–102. <https://doi.org/10.1159/000341837>

- Patil, C. N., Racusen, L. C., & Reckelhoff, J. F. (2017). Consequences of advanced aging on renal function in chronic hyperandrogenemic female rat model: implications for aging women with polycystic ovary syndrome. *Physiological reports*, 5(20), e13461. <https://doi.org/10.14814/phy2.13461>
- Roland, A. V., & Moenter, S. M. (2014). Reproductive neuroendocrine dysfunction in polycystic ovary syndrome: insight from animal models. *Frontiers in neuroendocrinology*, 35(4), 494–511. <https://doi.org/10.1016/j.yfrne.2014.04.002>
- Sadeghi, TENA-SEMPERE. M., Adeli, I., Calina, D., Docea, A. O., Mousavi, T., Daniali, M., Nikfar, S., Tsatsakis, A., & Abdollahi, M. (2022). Polycystic Ovary Syndrome: A Comprehensive Review of Pathogenesis, Management, and Drug Repurposing. *International journal of molecular sciences*, 23(2), 583. <https://doi.org/10.3390/ijms23020583>
- Sørensen, A. E., Udesen, P. B., Maciag, G., Geiger, J., Salianni, N., Januszewski, A. S., Jiang, G., Ma, R. C., Hardikar, A. A., Wissing, M. L. M., Englund, A. L. M., & Dalgaard, L. T. (2019). Hyperandrogenism and Metabolic Syndrome Are Associated With Changes in Serum-Derived microRNAs in Women With Polycystic Ovary Syndrome. *Frontiers in medicine*, 6, 242. <https://doi.org/10.3389/fmed.2019.00242>
- Thys-Jacobs, S., Donovan, D., Papadopoulos, A., Sarrel, P., & Bilezikian, J. P. (1999). Vitamin D and calcium dysregulation in the polycystic ovarian syndrome. *Steroids*, 64(6), 430-435.
- Unluhizarci, K., Karaca, Z., & Kelestimur, F. (2021). Role of insulin and insulin resistance in androgen excess disorders. *World journal of diabetes*, 12(5), 616–629. <https://doi.org/10.4239/wjd.v12.i5.616>