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Review Article

A Thorough Overview of Polycystic Ovary Syndrome (PCOS) in Female Rats

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ABSTRACT

Among women of reproductive age, Polycystic Ovarian Syndrome (PCOS) is a common endocrine condition marked by polycystic ovaries, hyperandrogenism, and ovulatory failure. Numerous animal models, particularly rodent models like female rats, have been used to comprehend the pathophysiology and create therapeutic approaches. This study covers the features of female rat PCOS, induction techniques, applicability of the rat model to human PCOS, underlying processes, and treatment strategies evaluated in various animals. Hyperandrogenism, ovulatory dysfunction, and metabolic abnormalities are the hallmarks of PCOS, a prevalent and complex endocrine condition affecting women of reproductive age. Owing to the intricacy of PCOS, mouse models in particular have been developed to evaluate prospective treatments and gain a better understanding of the disease's aetiology. In order to replicate the many symptoms of PCOS, rodent models—especially those of rats and mice—are useful. This enables researchers to investigate hormonal, genetic, and metabolic causes. This article offers a thorough examination of the many rodent models of PCOS, including information on their induction techniques, benefits, drawbacks, and applicability to PCOS in humans.

Keywords: Polycystic ovaries; Menstrual abnormalities; Hormones; Polycystic Ovarian Syndrome (PCOS)

INTRODUCTION

PCOS, or polycystic ovarian syndrome, is a complicated condition that, depending on diagnostic criteria, affects 6-20% of women worldwide. Menstrual abnormalities, infertility, hyperandrogenism, insulin resistance, and metabolic disturbances are just a few of the symptoms that are linked to it. Animal models are frequently used by researchers to investigate the underlying causes of PCOS and test novel treatments, while human studies are constrained by ethical considerations and individual variability. Because of their ease of developing PCOS-like symptoms and similar reproductive systems to humans, female rats are one of the most commonly utilised animal models in PCOS research.

PCOS is a complex disorder characterised by problems in both metabolism and reproduction. Our knowledge of PCOS, its molecular causes, and possible treatments has significantly increased thanks to the creation of rodent models that replicate these characteristics. Since rodents have well-defined reproductive systems, are easy to manipulate, and share genetic similarities with humans, they are frequently utilised in biomedical research. Rats and mice in particular are popular choices. The primary rat models used to investigate PCOS are reviewed here, along with information on the phenotypic traits of each model and information on hormonal, genetic, and nutritional induction techniques.

Important features of PCOS

Three primary characteristics define PCOS in humans:

- Hyperandrogenism (excessive androgen production resulting in symptoms such as acne and hirsutism).
- Ovarian dysfunction (polycystic ovaries, oligo or anovulation).
- Metabolic abnormalities (obesity, dyslipidaemia, insulin resistance).

These signs and symptoms are the main criteria used to diagnose PCOS in clinical settings and as the main benchmarks for creating animal models.

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Female rats with induced PCOS

Female rats are induced with PCOS using a variety of techniques, each simulating a distinct facet of the illness:

Induction of hormones:

- **Letrozole (Aromatase inhibitor):** Letrozole prevents androgens from being converted to oestrogens, which causes hyperandrogenism, a major component of PCOS. When letrozole is administered to rats, it replicates the clinical characteristics of polycystic ovaries, hyperandrogenism, and anomalies in the oestrous cycle that are associated with PCOS.
- **Testosterone Propionate (TP):** Giving rats androgens, such as testosterone or DHEA, causes them to become hyperandrogenic immediately. Rats given TP have irregular periods, many cystic follicles, and metabolic abnormalities like insulin resistance that are reminiscent of PCOS in humans.

Androgenisation of the neonatal: In this model, immediately after birth, female rats are exposed to androgens such as Dihydrotestosterone (DHT), which causes long-term reproductive and metabolic problems similar to Polycystic Ovarian Syndrome (PCOS). These rats experience irregular menstrual cycles, develop cystic ovaries, and develop obesity and insulin resistance as metabolic problems.

Induction of oestradiol valerate (EV): The Hypothalamic-Pituitary-Gonadal (HPG) axis is upset by oestradiol valerate, which results in ovarian cyst formation, anovulation, and hormonal imbalances that resemble PCOS. For researching the neuroendocrine components of PCOS, this model is helpful.

Corticosterone chemical induction: A glucocorticoid called corticosterone is utilised to cause stress-related PCOS-like symptoms in rats, including as irregular periods and disruptions in metabolism. This model emphasises the connection between stress and the onset of PCOS.

Every approach simulates distinct aspects of PCOS, enabling researchers to investigate particular mechanisms of the syndrome.

LITERATURE REVIEW

Pathophysiology of PCOS in rats

Ovarian morphology: In PCOS-induced rats, the ovaries often contain many antral follicles that do not burst and release eggs, resulting in cyst development. The ovarian tissue exhibits theca interna hyperplasia, which is a sign of androgen excess.

Endocrine abnormalities:

- PCOS rat models consistently show elevated androgen levels (hyperandrogenism). This disrupts folliculogenesis, resulting in anovulation.
- Some models exhibit an elevated LH/FSH ratio, similar to human PCOS. This hormonal imbalance is necessary for the formation of polycystic ovaries.
- **Insulin resistance and hyperinsulinemia:** Insulin resistance is a prevalent metabolic characteristic in PCOS models, similar to real cases. This is especially noticeable in the letrozole and androgen-induced models. Hyperinsulinemia worsens hyperandrogenism by raising ovarian androgen levels.

Metabolic features:

- **Obesity:** Some PCOS rat models, particularly those with neonatal androgenisation, exhibit increased body weight, similar to metabolic problems reported in human PCOS.
- **Dyslipidaemia:** In PCOS-induced rats, elevated triglyceride, low HDL, and elevated LDL cholesterol levels are frequently reported; these findings mirror the cardiovascular risk observed in PCOS patients in humans.
- **Intolerance to glucose:** PCOS models frequently exhibit reduced insulin sensitivity and glucose tolerance, offering insights into the metabolic dysfunctions linked to the condition.

The rat model's applicability to human PCOS

Rat models are useful for understanding PCOS, but they have drawbacks when applied to human conditions:

- **Comparabilities:** Important aspects of rat physiology and reproduction are similar to those of humans, such as ovulation structure, hormone cycles, and androgen sensitivity. They are therefore appropriate for researching hyperandrogenism and ovarian dysfunction.
- **Variations:** Rats do not suffer from spontaneous ovulatory failure as do people with PCOS, which is one significant distinction. PCOS symptoms need to be brought on by hormone or pharmaceutical means. Furthermore, the human menstrual cycle is longer than the rodent oestrous cycle, which could limit the direct applicability of findings. Notwithstanding these drawbacks, the rat model is still very useful for preclinical research and for understanding the endocrine and metabolic characteristics of PCOS of potential therapies.

Medicinal interventions in rat models of PCOS

To assess their therapeutic potential, a number of pharmacological and non-pharmacological strategies have been investigated in rat models of PCOS.

Treatments with pharmacology metformin: A popular insulin-sensitizing medication used to treat PCOS, metformin has been proven to improve insulin sensitivity, lower hyperandrogenism, and restore regular oestrous cycles in rat models.

- **Thiazolidinediones:** Medications known as, such as pioglitazone, target insulin resistance and have been shown to improve metabolic and reproductive parameters in PCOS rats.
- **Clomiphene citrate:** In letrozole-induced rat models, clomiphene citrate has been demonstrated to enhance ovulation and decrease ovarian cysts. It is a first-line treatment for ovulation induction in women with PCOS.

Hormone treatments:

- **Anti-androgens (such as flutamide):** In androgen-induced PCOS models, anti-androgens are used to lower androgen levels and enhance folliculogenesis. These treatments also lessen hirsutism and acne, two hyperandrogenism-related symptoms.
- **Gonadotropins:** In rat models, exogenous FSH or LH injection has been used to treat ovulatory failure.

Dietary interventions and natural compounds:

- **Inositol:** Research has been done on the insulin-sensitizing qualities of iso-inositol, namely myo-inositol. It has been demonstrated to lessen metabolic abnormalities and improve ovarian function in PCOS rats.
- **Curcumin:** In PCOS models, curcumin's anti-inflammatory and antioxidant qualities have shown protective effects against

Changes to lifestyle:

- **Exercise and diet:** Reversing metabolic abnormalities and enhancing reproductive function in rats has been demonstrated to be a hopeful outcome of exercise and dietary interventions (e.g., calorie restriction, low glycaemic index meals), which is consistent with clinical observations in the management of PCOS in humans.

DISCUSSION

Prospects for PCOS research employing rat models

Even while current rat models have shed light on PCOS, further study is still required to fully comprehend the disorder's intricate pathophysiology:

- **Models generic:** Present models mostly fail to reproduce the genetic components of human PCOS. It may be possible to gain important insights by creating genetically altered rat models that resemble the genetic predispositions seen in PCOS patients.
- **Long-term research:** By following rodents over time, longitudinal studies may be able to shed light on how PCOS develops from adolescence to menopause and the long-term health effects it may have.
- **Multi-organ involvement:** To develop a more thorough understanding of the illness, future research should examine how PCOS affects many organs, including the liver, pancreas, and adipose tissue.

Techniques for bringing on PCOS in mice

Depending on how PCOS is induced, rodent models fall into a number of categories. Certain features of the disease, like hyperandrogenism, ovulatory dysfunction, and metabolic abnormalities, are attempted to be replicated in these models.

Models of hormonal induction: The most popular models are hormonal ones, which aim to replicate the endocrine irregularities associated with PCOS.

PCOS model induced by Letrozole:

- **Mechanism:** Letrozole inhibits the aromatase enzyme, which stops androgens from becoming oestrogens. Hyperandrogenism brought on by prolonged medication resembles the hormonal dysregulation associated with PCOS.
- **Phenotypic features:** Letrozole-treated rats or mice experience insulin resistance, anovulation, polycystic ovaries, and irregular oestrous cycles. The model also mimics characteristics of PCOS in humans, such as increased levels of Luteinizing Hormone (LH) and testosterone.
- **Benefits:** The letrozole model closely resembles the problems in metabolism and reproduction found in PCOS in humans. The letrozole model has limitations as it is based on pharmacological induction and may not adequately represent the intricacy of PCOS progression over time.

PCOS Model induced by Testosterone Propionate (TP):

- **Mechanism:** Increasing androgen levels through the administration of testosterone propionate, a synthetic androgen, causes hyperandrogenism and ovarian dysfunction.
- **Phenotypic features:** Polycystic ovaries, irregular oestrous cycles, and signs of insulin resistance are developed in rats given TP. The hyperandrogenic phenotype observed in PCOS-affected patients is also replicated in this model.
- **Benefits:** The TP model accurately simulates the consequences of ovarian dysfunction and hyperandrogenism.
- **Restrictions:** Compared to other models, the metabolic phenotype (such as insulin resistance) might not always be as noticeable. The model also depicts the symptoms of PCOS, which are mostly brought on by an excess of testosterone.

PCOS induced by Oestradiol Valerate (EV) model:

- **Mechanism:** Ovarian dysfunction and anovulation are caused by oestradiol valerate's disruption of the Hypothalamic-Pituitary-Gonadal (HPG) axis.
- **Phenotypic features:** This model causes prolonged anovulation and the production of ovarian cysts. Comparing hyperandrogenism to other models, it is frequently less pronounced.
- **Benefits:** It is very helpful for researching the neuroendocrine abnormalities linked to PCOS.
- **Limitations:** The EV model, which mainly focusses on ovarian dysfunction, falls short of accurately describing the metabolic abnormalities associated with clinical PCOS.

PCOS model induced by Dehydroepiandrosterone (DHEA):

- **Mechanism:** Oestrogens and testosterone are precursors to DHEA. In rodents, chronic DHEA treatment simulates androgen excess.
- **Phenotypic features:** Polycystic ovaries, erratic oestrous cycles, and increased testosterone levels are all results of the DHEA model. Additionally noted are certain metabolic characteristics like insulin resistance.
- **Benefits:** DHEA mimics the reproductive and endocrine features of PCOS by inducing hyperandrogenism and ovarian dysfunction.
- **Limitations:** The metabolic phenotype might be less noticeable than the reproductive failure in this animal, similar to previous androgen-induced models.

Androgenisation of the neonatal

- **Mechanism:** In this model, androgens (such as testosterone or dihydrotestosterone) are administered to neonatal female rats or mice shortly after birth, resulting in long-term modifications to their metabolic and reproductive systems.
- **Phenotypic features:** These rats have irregular oestrous cycles, polycystic ovaries, persistent hyperandrogenism, and metabolic abnormalities such obesity and insulin resistance. This model imitates PCOS's developmental genesis.

Benefits: The neonatal androgenisation include the ability to explore the impact of early-life androgen exposure on adult phenotype and the ability to capture both the reproductive and metabolic elements of PCOS.

- **Limitations:** Because this model concentrates on the developmental causes of PCOS, it might not be applicable to all women who have the syndrome, particularly those who acquire it later in life [1].

Models genetic

Additionally, genetic models have been created to study the inherited components of PCOS.

Model of GnRH hypersecretion:

- **Mechanism:** Gonadotropin-Releasing Hormone (GnRH) is overexpressed in transgenic rodents, which results in hyperandrogenism and enhanced LH secretion.
- **Phenotypic features:** These rodents closely resemble the neuroendocrine regulation of PCOS, with polycystic ovaries, disturbed oestrous cycles, and increased androgens.
- **Benefits:** The model offers insights into neuroendocrine regulation and directly examines the hypothalamic-pituitary dysfunction associated with PCOS.
- **Restrictions:** The environmental or lifestyle factors that contribute to PCOS in humans may not be completely replicated in rodents through genetic modification.

Models of the environment and diet

Environmental factors can impact PCOS, and these effects are mimicked in rodent models by dietary adjustments.

Model of High-Fat Diet (HFD):

- **Mechanism:** To cause obesity and insulin resistance, two frequent metabolic traits of PCOS, rats are given a high-fat diet.
- **Phenotypic features:** The high-fat diet paradigm causes weight gain, insulin resistance, and frequently problems with ovarian function. It resembles the metabolic syndrome that PCOS is often linked to.
- **Benefits:** This model investigates the metabolic elements of PCOS, namely the connection between obesity and impaired reproduction.
- **Limitations:** The HFD model is more suited for researching metabolic dysfunction than the whole range of PCOS symptoms because it may not cause hyperandrogenism or polycystic ovaries [2].

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Phenotypic features: Insulin resistance, anovulation, polycystic ovaries, and irregular oestrous cycles are all developed in rats or mice given letrozole. The model also mimics characteristics of PCOS in humans, such as increased levels of Luteinizing Hormone (LH) and testosterone.

Benefits: The Letrozole model closely resembles the problems in metabolism and reproduction found in PCOS in humans. The Letrozole model has limitations as it is based on pharmacological induction and may not adequately represent the intricacy of PCOS progression over time.

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- **Limitations:** Because this model concentrates on the developmental causes of PCOS, it might not be applicable to all women who have the syndrome, particularly those who acquire it later in life.

Molecular models: Additionally, genetic models have been created to study the inherited components of PCOS.

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- **Benefits:** The model offers insights into neuroendocrine regulation and directly examines the hypothalamic-pituitary dysfunction associated with PCOS.
- **Limitations:** It's possible that genetic modification in rats won't accurately mimic the environmental or lifestyle elements that lead to PCOS in people [8,9].

Models of the environment and diet

Environmental factors can impact PCOS, and these effects are mimicked in rodent models by dietary adjustments.

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Phenotypic similarities between human PCOS and rodent models

Excessive masculinity: The majority of rodent models accurately replicate hyperandrogenism, a feature of human PCOS, particularly those that use testosterone, DHEA, and letrozole. Researching how androgen excess affects ovarian function and metabolic health depends on this.

Ovarian dysfunction: In most rodent models, especially the letrozole, DHEA, and oestradiol valerate models, polycystic ovaries and anovulation are prominent features. Rats find it challenging to adequately mimic the spontaneous ovulation problems associated with PCOS in humans.

Metabolic abnormalities:

- In the high-fat diet and neonatal androgenisation models, obesity, dyslipidaemia, and insulin resistance are more prevalent. The degree to which insulin resistance is produced differs throughout hormonal models (e.g., letrozole, TP).

Benefits and drawbacks of rat models

Benefits:

- **Reproducibility:** Experiments using rodent models may be controlled and repeated to replicate important features of PCOS.
- **Accessibility:** Rodents are a great choice for extensive research because they are reasonably priced and simple to modify hormonally and genetically.
- **Ethical considerations:** Using animal models to study illness causes and test potential treatments is an ethical substitute for using human subjects in research studies.

Restrictions:

- **Differences across species:** Although rats and humans have comparable reproductive physiologies, there are significant variations in the control of hormones, ovarian cycles, and metabolic responses.
- **Simplified models:** A lot of rodent models concentrate on particular features of PCOS, including hyperandrogenism, and might not fully represent the disorder's complexity, which includes the interaction of genetics, environment, and lifestyle.
- **Short oestrous cycle:** The limited application of findings stems from the fact that rodents' short reproductive cycles (4-5 days) diverge from the menstrual cycle in humans.

Prospects for further research on rodent PCOS

The following areas should be the focus of future research:

- **Genetic studies:** Creating more complex genetic models that replicate the heritable features of PCOS.
- **Longitudinal studies:** Investigating how PCOS develops over time in rat models from adolescence to maturity, simulating the course of a human disease.
- **Multi-organ involvement:** To gain a better understanding of the systemic nature of PCOS, research is being done on how the disorder affects the liver, pancreas, and adipose tissue.

CONCLUSION

PCOS is still a complex condition that affects metabolism, endocrine function, and reproduction. The advancement of our knowledge of the syndrome has been made possible by the use of female rat models. By using these models, scientists have been able to examine the underlying processes, evaluate treatment options, and look at how hormone and environmental factors affect the development of PCOS. Notwithstanding its drawbacks, rat models will always be important for PCOS research and will help provide better therapies for women who have the illness. When it comes to comprehending the pathophysiology of PCOS and creating treatment strategies, rodent models are an invaluable resource. These models mimic

several of the main features of PCOS, including hyperandrogenism, ovarian dysfunction, and metabolic abnormalities, despite certain limitations. Further creation and improvement of rodent models will allow for more accurate investigations of the mechanisms behind PCOS and the testing of new therapies.

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