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EXPERIMENTAL INDUCTION AND EVALUTION OF POLYCYSTIC OVARIAN DISEASE (PCOD) IN RODENT MODELS: MECHANISM, METHODS, AND PHARMACOLOGICAL INSIGHTS

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ABSTRACT

Polycystic Ovarian Disease (PCOD), also widely known as Polycystic Ovary Syndrome is a complex endocrine and metabolic disorder affecting a significant proportion of women of childbearing age worldwide. Characterized by menstrual irregularities, hyperandrogenism, polycystic ovarian morphology, and metabolic abnormalities such as insulin resistance and dyslipidemia, PCOD presents a multifaceted clinical challenge. Understanding its pathophysiology is critical for developing effective therapeutic strategies. Rodent models, contain rats and mice, have emerged as indispensable tools for elucidating the underlying mechanisms of PCOD. These models replicate key features of the disorder, including hormonal imbalance, ovarian cyst formation, anovulation, and metabolic disturbances, under controlled experimental conditions. Several induction methods, including hormonal administration (androgens and Estrogen), pharmacological agents such as letrozole, and diet-induced approaches using high-fat or high-sugar diets, have been employed to create reliable PCOD models in rodents. Evaluation of these models involves comprehensive assessment of ovarian morphology, serum hormonal profiles, metabolic parameters, and molecular markers related to oxidative stress, inflammation, and apoptosis. Furthermore, rodent models serve as effective platforms to evaluate pharmacological interventions, ranging from conventional agents like metformin to herbal and novel bioactive compounds. By bridging preclinical findings with clinical relevance, these models provide valuable insights into disease mechanisms, potential therapeutic targets,

and translational applications for human PCOD. This review consolidates current experimental strategies, evaluation techniques, and therapeutic insights, emphasizing the importance of rodent models as a cornerstone in PCOD research and highlighting their role in advancing our understanding and management of this prevalent disorder.

KEYWORDS: Polycystic Ovarian Disease, PCOD, Rodent Models, Hormonal Imbalance, Insulin Resistance, Ovarian Dysfunction, Experimental Induction, Pharmacological Evaluation.

INTRODUCTION

Polycystic Ovarian Disease (PCOD) stands out as one of the most common hormonal imbalance among women of child bearing age, affecting 6–20% globally. Clinically, it is characterized by irregular menstrual cycles, hyperandrogenism, polycystic ovarian morphology, and metabolic disturbances including insulin resistance and obesity. Despite its prevalence, the precise lifecycle remains complicated and several factors involving genetic, environmental, and lifestyle factors. Rodent models have emerged as crucial tools in understanding PCOD pathophysiology and evaluating therapeutic interventions. These models mimic various aspects of human PCOD, providing controlled environments to explore mechanisms that are otherwise challenging to study in humans. This review focuses on experimental induction methods, evaluation parameters, and translational insights from rodent models.

EXPERMENTAL INDUCTION ON PCOD IN RODENTS

1. Hormonal Induction

Androgen-Induced Models: Administration of testosterone propionate, dihydrotestosterone, or dehydroepiandrosterone (DHEA) induces hyperandrogenism and ovarian cysts. Estrogen-Induced Models: High-dose estradiol disrupts normal follicular development, mimicking cyst formation.

2. Pharmacological Induction

Letrozole Models: Aromatase inhibitors like letrozole reduce estrogen synthesis, leading to hyperandrogenism and polycystic ovarian morphology. Streptozotocin or Nicotinamide Models: Induce insulin resistance, a hallmark of PCOD, often combined with high-fat diets to mimic metabolic aspects.

3. Diet-Induced Models

High-Fat or High-Sugar Diets: Cause obesity, insulin resistance, and subsequent ovarian dysfunction, closely resembling human metabolic PCOD.

Evaluation of PCOD in Rodent Models

1. Ovarian Morphology

Histological examination reveals increased cystic follicles, decreased corpora lutea, and stromal hypertrophy, reflecting disrupted ovulation.

2. Hormonal Assessments

Serum levels of testosterone, luteinizing hormone (LH), follicle-stimulating hormone (FSH), and insulin are measured to confirm endocrine alterations.

3. Metabolic Parameters

Glucose tolerance tests, insulin tolerance tests, and lipid profiling are conducted to evaluate metabolic dysregulation.

4. Molecular and Biochemical Markers

Oxidative stress markers, inflammatory cytokines, and apoptotic proteins are assessed to understand underlying cellular mechanisms.

PHARMACOLOGICAL AND THERAPUTIC INSIGHTS

Rodent PCOD models have been extensively used to test:

Metformin and Thiazolidinediones: Improve insulin sensitivity and ovulatory function.

Herbal and Phytochemicals: Flavonoids, polyphenols, and other bioactive compounds demonstrate anti-androgenic and antioxidative properties.

Novel Drug Candidates: Target hormonal modulation, oxidative stress, and inflammation.

These studies provide a translational bridge, allowing insights into potential interventions for human PCOD.

CONCLUSION

Rodent models of Polycystic Ovarian Disease provide a robust and reliable framework for studying the complex interplay of hormonal, metabolic, and molecular abnormalities characteristic of this disorder. Hormonal induction models using androgens or estrogens, pharmacological models such as letrozole administration, and diet-induced models simulating

obesity and insulin resistance collectively allow researchers to replicate specific features of human PCOD under controlled experimental conditions. Evaluations encompassing ovarian histology, serum hormonal profiles, metabolic assessments, and molecular analyses not only validate these models but also provide a mechanistic understanding of disease progression. Importantly, these preclinical models serve as essential platforms for testing conventional therapeutic agents, including insulin-sensitizing drugs, as well as herbal, phytochemical, and novel pharmacological interventions. By mirroring both reproductive and metabolic disturbances, rodent models offer translational insights that inform clinical research, improve treatment strategies, and guide personalized approaches for managing poly cystic ovarian disease in women. The integration of morphological, endocrine, metabolic, and molecular evaluations ensures comprehensive characterization and reproducibility, fostering more accurate extrapolation to human conditions. In conclusion, rodent models remain pivotal in bridging the gap between experimental research and clinical applications, enhancing our understanding of PCOD pathophysiology, elucidating potential therapeutic targets, and advancing the development of innovative interventions. Continued refinement of these models and adoption of standardized evaluation protocols will further strengthen their relevance, ultimately contributing to improved clinical outcomes and womens life condition is damaged by PCOD worldwide.

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