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Dehydroepiandrosterone treatment in rats for the study of metabolic disorders in polycystic ovary syndrome

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DESCRIPTION

Polycystic Ovarian Syndrome is characterized by hyperandrogenism (PCOS). Rats treated with DehydroEpiAndrosterone (DHEA) are regarded to be a good animal model for studying PCOS. The severity of reproductive and metabolic problems in DHEA-treated rats was examined in this study. Female Sprague–Dawley rats were separated into two groups: control and DHEA-treated. After sexual maturity, reproductive characteristics such as the estrus cycle and sex hormones were measured. To measure metabolic profiles, researchers looked at adiposity, insulin sensitivity, and plasma lipid profiles. Immune blotting and polymerase chain reaction were used to examine the insulin signaling pathway and lipo genic genes after sacrifice.

PCOS (polycystic ovarian syndrome) is an endocrine-metabolic disorder that affects 5–10% of women of reproductive age. Chronic anovulation, hyperandrogenism, and many tiny sub capsular cystic follicles in the ovary on ultrasonography are all symptoms of PCOS. Obesity, insulin resistance with compensatory hyper insulinemia, dyslipidemia, type 2 diabetes mellitus, and an elevated risk of cardiovascular disease are common in PCOS patients, in addition to reproductive problems. Early-life androgen excess may be the etiology of PCOS, and inducing polycystic ovaries in animals with an excess of testosterone has become the most widely used approach. Androgen therapy can cause serious reproductive problems such anovulation and ovarian cyst growth. Metabolic problems, such as poor glucose tolerance, can because by an excess of androgen.

In 1962, Mahesh and Greenblatt discovered that most PCOS patients had puberty indications and secrete more dehydroepiandrosterone (DHEA) than normal women, and that providing DHEA to normal women enhances their testosterone levels in the blood. By injecting immature rats with DHEA every day and analyzing the major symptoms of clinical PCOS, an animal model of PCOS can be created. In these conditions, prepubescent female rats develop ovarian cysts and become an ovulatory and acyclic. DHEA-treated rats had significantly higher serum concentrations of DHEA sulphate, testosterone, and androstenedione than control rats, which is similar to human PCOS. In a PCOS animal model treated with DHEA, insulin resistance was also seen.

DHEA, on the other hand, has been demonstrated to lower body weight, blood sugar, insulin, and blood pressure. DHEA therapy has also been shown to lower liver triglycerides and glycogen levels in rats. Women with PCOS may have hyper insulinemia and metabolic problems, which makes this different from human PCOS. As a result, we used a DHEA-exposed rat model in this study to look into the impact of DHEA on the severity of reproductive and metabolic repercussions. This rat model was used to examine reproductive function, ovarian characteristics, metabolic parameters, and molecular underpinnings of insulin resistance.

Ovarian Torphology in the DHEA-Exposed Rats

The ovaries of DHEA-exposed rats were smaller than those of control rats, and cysts on the surface of the ovaries were apparent. The ovarian characteristics of the control rats were normal, including all stages of ovarian follicles and the post-ovulatory corpus lutein. The granulose and theca cell layers in the control ovarian sections were also normal under high power field magnification. However, light microscopy investigation revealed an aberrant ovarian structure in the DHEA-exposed rats, including an increased number of big and small cystic follicles and no corpus lutein in the ovaries.

The thickness of the granulose cell layer was smaller in the DHEA-treated rats' ovaries than in the control rats' ovaries under high power field magnification, and in the ovaries of DHEA-treated rats, the theca cell layer was

thicker. In the ovaries of DHEA-treated rats, some collapsed follicles with theca cell layer hypertrophy and vascular invasion were observed, indicating that these follicles were experiencing atresia. We used anti-CD68 antibodies to measure macrophage infiltration in the ovarian follicles to validate the atresia of the follicles in the ovaries of DHEA-treated rats. In the ovarian follicles of control rats, only a few macrophages were seen. The macrophage infiltration in the ovarian follicles of DHEA-exposed rats, particularly atretic follicles, was substantial.

Steroid Genic Gene Expressions in The DHEA-Exposed Rats

RT-PCR was used to examine the level of mRNA expression of steroid genic genes in the ovaries of DHEA-exposed rats. In the ovaries of DHEA-exposed rats, Star protein, 3-hydroxysteroid dehydrogenase (3-HSD), and aromatase expression all decreased significantly.

Body Weight and Adiposity in The DHEA-Exposed Rats

After 12 weeks of treatment, rats exposed to DHEA were significantly heavier than control rats. However, the fat pad of DHEA-exposed rats containing per ovarian and retroperitoneal fat was significantly smaller than that of control rats. The average subcutaneous fat weight was also low, but not statistically significant. Histological analysis of adipose tissue showed no significant changes in adipocyte size. Further measurements of adipocyte size using virtual microscopy software showed no statistical significance and only a slight decrease in adipocyte size.

Plasma Lipid Profiles

Plasma triglyceride and total cholesterol levels were measured. There was a slight increase in plasma triglyceride concentration, but the difference was not statistically significant.