We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,300

Open access books available

172,000

International authors and editors

190M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities

WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com

Effects of Exercise Intervention on the Improvement of Polycystic Ovary Syndrome

Shaobing Wang, Zhenghong Zhang and Yiping Liu

Abstract

Polycystic ovary syndrome (PCOS) is a complex endocrine and metabolic disease characterized by hyperandrogenism and insulin resistance. It results in irregular menstruation, obesity and even infertility, which influences badly physical and mental health of the patients. Now, there is no effective drug for treating PCOS and the preferred program is lifestyle modification, including diet control, regular exercise and behavior therapy. Therefore, in the review, we summarize the study progress concerning the effects of lifestyle intervention on the metabolic, reproductive and psychological dysfunctions of PCOS patients and analyze the corresponding mechanisms in these processes. It can radically reduce the factors of PCOS occurrence and development, while providing valuable information for the prevention and treatment of PCOS and is helpful for further research.

Keywords: polycystic ovary syndrome, physiopathology, exercise, lifestyle intervention

1. Introduction

PCOS was an endocrine disorder with complex pathogenesis and diverse clinical phenotypes and was also the most common cause of infertility and abnormal menstrual cycle in women of childbearing age. Current epidemiological investigation showed that PCOS affects 5–10% of women of reproductive age [1]. The effects run through the patient's entire reproductive cycle, from puberty until after menopause, even involving the offspring. PCOS affected patients' reproductive functions, caused of glucose and lipid metabolism disorders, increased the risk of type 2 diabetes and cardiovascular diseases, but also caused anxiety, depression and other psychosocial disorders. Therefore, PCOS was a clinical problem and also a public health problem that needs to be solved urgently [2, 3]. In addition to traditional psychological and drug therapy, exercise had obvious curative effect as a simple and economical adjunctive therapy. In the review, to treat PCOS patients with metabolic, reproductive and psychological dysfunctions, we explored how exercise or combination with other methods could effectively intervene PCOS patients. It provided theoretical basis for clinical treatment of PCOS patients.

2. Exercise or combination with other methods improve reproductive dysfunction in PCOS patients

PCOS accompanied with various clinical manifestations such as obesity, irregular ovulation or anovulation, infertility, menstrual disorder and excessive androgen, insulin resistance and so on. Obese PCOS women showed decreasing reproductive function and increasing risk of metabolic syndrome, but also their mental health and life quality were greatly affected. Before assisted reproduction, weight loss through diet or exercise therapy and other lifestyle interventions could help restore spontaneous ovulation, increase natural pregnancy rate, improve pregnancy rate and live birth rate of assisted pregnancy, reduce the risk of pregnancy complications, and improve pregnancy outcome.

Current clinical statistics showed that nearly 50% of PCOS patients were associated with obesity [4]. The subcutaneous fat distribution of triceps brachii and subscapular in PCOS patients was significantly different from that in the healthy control group, and the waist-hip ratio (WHR) was significantly higher than that in the control group [5]. PCOS patients were often accompanied by abdominal obesity and visceral fat accumulation. Case control analysis showed that the overall fat content and visceral fat content of PCOS women were significantly higher than that of healthy women, especially abdominal and mesenteric fat [6]. Obesity had a great impact on reproductive health, such as ovulation disorders, decreased pregnancy rate and increased abortion rate, increased risk of pregnancy complications including preeclampsia and gestational diabetes, in addition, when assisted reproductive technology (ART) assisted pregnancy, the response of obese PCOS patients to assisted pregnancy drugs reduced, and the success rate of assisted pregnancy decreased [7].

Lin and Lujan [8] reviewed the diet and exercise of PCOS women and found that there was no significant difference in exercise amount between PCOS women and healthy women, but PCOS women were characterized by high-calorie diet, excessive intake of saturated fatty acid and lack of dietary fiber. After receiving ART assisted pregnancy therapy, the clinical pregnancy rate and live birth rate of obese PCOS women were significantly lower than those of normal weight PCOS patients, and the risk of miscarriage was increased [9, 10]. Therefore, it was very important to control BMI in the optimal range before pregnancy assistance to improve the success rate of ART. For PCOS women aged 25–35 years, BMI should be controlled below 24 kg/m². Adjusting the BMI of older women should be more strict and for those over 35 years old, it should be less than 20 kg/m² [11]. Obese PCOS patients could lose weight through lifestyle adjustment such as diet, exercise and behavioral intervention to achieve better pregnancy outcome.

2.1 Effects of regular exercise on the pregnancy outcome of PCOS patients

Although the pathogenesis of PCOS was not clear and clinical phenotypes were diverse, PCOS patients with any phenotype may have insulin resistance, with an incidence of 50–80% [12]. More and more evidences showed that insulin resistance was closely related to PCOS. Endocrine disorders caused by genetic environment and obesity were regarded as the cornerstone of PCOS, and insulin resistance was likely to be the initiation factor and central link of PCOS development [13]. Insulin resistance referred to the decrease of the sensitivity of peripheral tissues to insulin, which leading to the decrease of the biological function of insulin and further caused hyperinsulinemia in peripheral blood circulation. Hyperinsulinemia and insulin resistance played various roles in different tissues in the body and promoted the occurrence of hyperandrogenemia. Firstly, on the surface of ovary,

acted on insulin-like growth factor binding protein and up-regulated the activity of cytochrome P450c17 alpha enzyme in follicular membrane cells, accelerated the biosynthesis of androgens and promoted the proliferation of follicular membrane cells. Secondly, enhanced the effect of adrenocortical hormone and promoted the production of adrenogenic androgen. Thirdly, in the liver, inhibited the activity of sex hormone-binding globulin (SHBG) and led to increase the levels of free androgen in the blood [14]. Under the action of aromatase in fat cells, excessive androgen converted into a high level of estrogen in the blood and positive feedback to the hypothalamus, kept LH at a high level for a long time. Insulin resistance, hyper-androgen and hypothalamic-pituitary-gonadal axis disorders eventually affected follicular maturation, leading to ovulation disorders, which cause infertility and menstrual cycle disorders.

At present, the international Androgen Excess and PCOS Society (AEPCOS) suggested that physical exercise of more than 150 min per week was recommended to maintain a healthy body quality and condition, including at least 90 min of moderate-high intensity exercise (heart rate 150 times/min), such as football, basketball and other team sports or brisk walking, running, rowing, etc. It is proved that physical exercise could effectively improve menstrual disorders and thin ovulation of obese PCOS patients. It had even been reported that exercise therapy was more beneficial to patients' reproductive function than low-calorie diet therapy. In fact, for obese PCOS patients without ovulation, exercise therapy could restore the regular menstrual cycle in 60% of patients, ovulation in 50% of patients and spontaneously conceive in 35% of patients [15]. Before or in conjunction with medication, exercise intervention could effectively improve health-related life quality of overweight or obese PCOS patients, which can improve the insulin resistance of the patients and help them to increase ovulation rate and decrease hormone level for increasing pregnancy rate [16, 17].

Regular exercise may alleviate insulin resistance of PCOS patients by the two ways. First of all, regular physical exercise could lose weight and significantly reduce visceral fat of PCOS patients. Visceral fat had stronger metabolic activity and was closely related to insulin resistance. Secondly, physical exercise could regulate the expression of insulin signaling protein in skeletal muscle, so as to improve the metabolic level of muscle cells and increased the sensitivity of insulin in PCOS patients.

2.2 Lifestyle adjustment improve the pregnancy outcome of obese PCOS women

Lifestyle and behavioral interventions for weight control required weight loss with regular exercise and diet interventions, as well as long-term target weight maintenance after weight loss. AEPCOS recommended at least 150 min of exercise per week for obese PCOS patients, the sport types included fast walking, jogging, aerobics, swimming and other whole-body exercises involving more than 2/3 of the muscles, the exercise intensity reached medium and high intensity. Heymsfield and Wadden [18] reviewed the mechanism of obesity, pathophysiology and weight loss management and concluded that lifestyle intervention to reduce weight through diet and exercise combined with cognitive behavioral therapy could improve health and quality of life by 5–10% in overweight/obese patients. Cognitive behavioral intervention required professional guidance and could effectively guide diet and exercise by providing encouragement, setting weight loss goals and guiding problems encountered during lifestyle interventions. Regular cognitive behavioral intervention was the most effective way to achieve weight loss and long-term weight maintenance. However, the current problem is that it is difficult to achieve long-term behavioral intervention counseling, resulting in easy weight regain after

weight loss. Evidence-based medicine supported PCOS patients to adjust their lifestyle to lose weight, which was helpful to prevent overweight and obesity and improve the long-term quality of life. For PCOS patients of normal weight, there was still a risk of long-term weight gain, so lifestyle intervention was also required. PCOS patients to conduct regular self-monitoring, combine lifestyle intervention with psychological strategies, and developed clinically feasible and economical intervention strategies. Patients with normal weight PCOS received low-intensity interventions to maintain weight and prevent weight gain.

Weight loss therapy included diet, exercise and other lifestyle interventions, weight loss medication and weight loss surgery. As a non-drug intervention, low-calorie diet and exercise with weight loss had strong feasibility, relatively low cost and large benefits. When the weight of obese PCOS women drops by 5%, spontaneous ovulation could be improved and the natural pregnancy rate could be increased [7]. In addition, lifestyle adjustment was beneficial to improve endocrine function, cardiovascular and mental health [19]. Therefore, it was of great significance for overweight/obese PCOS women to help them adjust their lifestyle to reduce weight before pregnancy. It was reported that rapid weight loss had an adverse effect on the outcome of ART assisted pregnancy [20]. Therefore, it was very important to maintain reasonable and stable weight loss and keep long-term weight control, which is also a big problem of obesity PCOS weight loss.

For obese women with endocrine disorders, menoxenia and infertility, adjusting lifestyle to reduce weight was beneficial to improve endocrine function, restore ovulation and increase pregnancy rate. Only 5–10% of weight loss could benefit infertility patients [21–25]. Obese PCOS patients could reduce concentric obesity and improve insulin resistance by adjusting their lifestyle, which was conducive to the recovery of ovulation [26]. A prospective pilot study suggested that lifestyle intervention for weight loss could improve the ovulation function of obese PCOS women. The researchers recruited 32 obese PCOS women with anovulation and conducted lifestyle intervention for 6 months. Compared with the control group of 18 subjects who did not resume ovulation after weight loss by lifestyle intervention, 14 ones of restoring ovulation lost significant weight and significantly reduced abdominal fat. Further analysis found that the reduction of abdominal fat in the ovulation restoration group was more significant than that in the non-ovulation restoration group after lifestyle intervention for 3 and 6 months. It suggested that weight loss and abdominal fat reduction, especially the early and continuous reduction of intra-abdominal fat, was conducive to the recovery of spontaneous ovulation, thereby ultimately improving the natural pregnancy rate [27]. A prospective study by Salama et al. convened 75 overweight/obese PCOS women and implemented a low-calorie diet and exercise intervention for 12 weeks. A total of 27 of the 43 PCOS women with anovulatory amenorrhea resumed their menstrual cycles and seven of the 58 PCOS women had natural pregnancies [28].

For obese PCOS women with fertility requirements, lifestyle intervention and reasonable weight reduction could help improve pregnancy outcome before assisted pregnancy therapy. PCOS patients were often accompanied by androgen overload, irregular ovulation and menstrual disorders. Patients with indications often need to use Oral contraceptive pills (OCP) to adjust menstrual cycle and endocrine function. Legro et al. [29] designed a randomized controlled trial, which 149 overweight/obese infertile PCOS women were pretreated with lifestyle, oral contraceptives or a combination of both for 16 weeks before entering the clomiphene ovulatory cycle to analyze the impact of lifestyle interventions on pregnancy-assisted outcomes. The results indicated that before assisted reproduction, lifestyle interventions combined with oral contraceptives could eliminate the metabolic side effects of oral contraceptives alone. In addition, the ovulation rate and the live

birth rate were higher in the lifestyle adjustment or combination with OCP groups than in the OCP group, but there was no significant difference in clinical pregnancy rate between the groups. Legro et al. [30] also conducted secondary analysis on two multi-center clinical trials, overweight or obese infertile PCOS women were pre-treated with lifestyle, oral contraceptives or a combination of both before entering the clomiphene ovulation cycle. In this study, subjects who entered the ovulation induction cycle without any preconditioning were used as the control group, and the assisted pregnancy outcome of each group were compared. The results showed that there were no significant differences in ovulation rate, pregnancy rate and live birth rate between the OCP pretreatment group and the control group. The ovulation rate, clinical pregnancy rate and live birth rate in the lifestyle intervention group were higher than those in the control group. The combined lifestyle and OCP intervention group had higher ovulation rate, clinical pregnancy rate and live birth rate than the control group, It was suggested that lifestyle adjustment or combined with OCP preconditioning could improve the success rate of clomiphene ovulation induction cycle.

However, some studies showed that lifestyle changes did not improve the outcome of assisted pregnancies in obese infertile women. In a randomized controlled study conducted by Mutsaerts et al. [31], the average weight of overweight/obese infertile patients decreased by 4.4 kg after 6 months of lifestyle intervention before assisted pregnancy treatment. However, the live birth rate in the intervention group was lower than that in the control group receiving direct assisted pregnancy treatment, and there was no significant difference between the pregnancy rate and the control group. van Oers et al. [32] further analyzed that there was no difference in the live birth rate between the obese and control group when assisted pregnancy treatment after 6 months of lifestyle preconditioning. Domecq et al. [33] conducted a meta-analysis on the pregnancy outcomes of PCOS patients with lifestyle intervention before assisting pregnancy and showed that there was no significant difference in clinical pregnancy rate between the lifestyle intervention group and the control group.

Up to 50–70% of PCOS patients were overweight or obese. Obesity, insulin resistance and secondary hyperinsulinemia were correlated with each other. Excessive insulin stimulated ovary to synthesize androgen and inhibit SHBG, which jointly led to the high androgen status in clinical manifestations and biochemical level of PCOS patients. Weight loss with adjusting lifestyle could improve central obesity, enhance insulin sensitivity, decrease fasting insulin and blood glucose levels, increase SHBG levels and reduce total and free testosterone levels, improve ovulation function and restore regular menstrual cycles [33–37].

Weight loss with adjusting lifestyle improved ovulation and intimal function. Ujvari et al. [38] analyzed for the first time the effects of weight reduction by lifestyle adjustment on intima gene expression and insulin signaling pathway in overweight/obese PCOS patients. The expression of endometrial insulin receptor substrate-1 (IRS-1) gene was significantly lower than that in the control group in proliferating phase of PCOS women. After overweight/obese PCOS patients received lifestyle intervention for 3 months, the average weight decreased by 4.7%, fasting insulin decreased significantly, menstruation returned to normal in 65% of subjects and 35% restored ovulation. Meanwhile, subjects of the intervention group had up-regulated mRNA levels of endometrial IRS-1 gene, which was positively correlated with increased mRNA levels of endometrial glucose transporter protein-1 (GLUT-1) gene. These results concluded that after the lifestyle intervention of obese PCOS patients, expression level of the insulin signaling pathway molecule in the endometrium was up-regulated to improve the endometrial function and promoted menstruation to return to normal.

Diet and exercise were beneficial to improve endocrine and metabolic functions. Jiskoot et al. [39] believed that multi-disciplinary lifestyle intervention through the combination of cognition, nutrition and exercise was more conducive to overweight/obese PCOS patients to develop a healthy lifestyle and achieved long-term weight loss, so as to improve the reproductive function, metabolic state and long-term life quality of overweight/obese PCOS women. A randomized controlled study of overweight/obese PCOS women followed a dietary intervention to lose weight for 4 months. The levels of anti-Müllerian hormone (AMH), total testosterone and free testosterone were significantly lower than those before intervention. Metabolic indicators including insulin growth factor (IGF-1) and insulin growth factor binding protein (IGFBP-1) were significantly higher than those before intervention, and insulin, blood sugar and insulin resistance index (HOMA-IR) were significantly lower than those before intervention [40]. Harrison et al. [15] reviewed the effects of exercise on PCOS patients and demonstrated that exercise therapy could improve ovulation function and insulin resistance and suggested that PCOS patients do moderate aerobic exercise for more than 90 min per week to improve reproductive and cardiovascular health. Dyslipidemia was a common cardiovascular risk factor in PCOS patients. Compared with moderate intensity exercise, PCOS subjects with high intensity exercise had higher levels of SHBG and high-density lipoprotein (HDL-C) and lower incidence of metabolic syndrome [41]. Other opinions held that lifestyle intervention had no significant improvement on dyslipidemia and OCP could improve HDL-C function, but lifestyle intervention combined with OCP drug therapy could improve HDL-C function and reduce the side effects of OCP on lipoprotein [42, 43]. Weight loss through diet and exercise intervention was important treatment strategies for PCOS patients, but studies have shown that the mechanisms of the two were different. Dietary intervention to reduce weight improved reproduction, psychology and metabolism of PCOS patients, which the mechanism may lie in the limitation of total energy intake. Palomba et al. [44] considered that after 24 weeks of exercise intervention, the weight loss of subjects was not as great as that of dietary intervention, but the ovulation rate was higher, which may be due to reduced insulin resistance and increased insulin sensitivity, thus improving the ovarian function. Therefore, dietary and exercise interventions for obese PCOS patients also need to be individualized.

Lifestyle intervention for weight loss regulated adipokine secretion. Fat cytokines synthesized and secreted by the fat tissues included adiponectin and leptin and inflammatory cytokines. Adiponectin was considered to be a preventive protective factor for obesity-related diseases such as diabetes and atherosclerosis, while leptin was a risk factor for cardiovascular disease. Obesity was often accompanied by characteristic high leptin levels and it is believed that obese people may exist leptin resistance [45, 46]. Adipocytokines were involved in regulating insulin sensitivity and affecting ovulation and fertilized egg planting. Adipocytokine secretion levels obese patients were changed, including decreased adiponectin levels, increased leptin and inflammatory of cytokines interleukin (IL)-6 and tumor necrosis factor (TNF- α) levels, which may be the possible mechanism of adverse pregnancy outcomes in obese women [21]. Adiponectin level was significantly correlated with visceral and subcutaneous fat. Subcutaneous and visceral fat decreased and adiponectin levels increased after weight loss by lifestyle intervention [22]. The prospective study of Siegrist et al. [23] showed that after 4–6 weeks of lifestyle intervention, the average weight of 402 obese subjects decreased by 8.9 kg, the serum adiponectin level increased, leptin and insulin levels decreased. Weight reduction with lifestyle intervention helped to regulate the secretion of adipokine. It may be one of the important mechanisms to improve PCOS dysfunction by weight loss.

3. Effects of exercise or combination with other methods on metabolic abnormalities in PCOS patients

PCOS was an endocrine disorder syndrome which coexists with reproductive dysfunction and metabolic abnormality. The clinical symptoms and long-term complications of PCOS patients were inseparable from their metabolic dysfunction. Studies have shown that excessive androgen and abnormal lipid metabolism were the main factors leading to skin characteristics (such as acne, hairy, male alopecia, etc.) in PCOS patients. The abdominal subcutaneous adipose tissue of obese PCOS patients was closely related to insulin resistance and androgen. Overweight could lead to lower levels of sex hormone binding globulin and increased androgen and insulin secretion and insulin resistance. This vicious circle would aggravate the clinical symptoms and signs of PCOS patients [47].

Brown et al. [48] showed that body composition (BMI, WHR and body fat rate) was significantly reduced after 12 weeks of aerobic exercise, and exercise and physical activity could reduce the storage of estrogen in body fat and promote the production of steroid hormones. Aerobic exercise significantly reduced the levels of SHBG and LH [49]. In the ovary, decreasing insulin levels reduced the level of androgen, WHR was the abdominal obesity index, and there was a relationship between WHR changes, body fat percentage and insulin sensitivity [50]. Therefore, reducing WHR and body fat percentage through aerobic exercise could increase the body's sensitivity to insulin and promote the production of SHBG. Androgen production was reduced and ovulation was promoted by increasing SHBG [49]. In the study conducted by Brown et al. [48], after 20 weeks of exercise, cholesterol, HDL and LDL levels were significantly improved. Regular endurance exercise could reduce TC, TG, LDL levels and increase HDL. PCOS patients had an increased risk of type 2 diabetes, dyslipidemia and cardiovascular disease. Moderate intensity exercise improved the composition of lipoprotein [51]. A prospective study by Salama et al. [28] convened 75 overweight/obese PCOS women and implemented a low-calorie diet and exercise intervention for 12 weeks. The average body weight of the subjects decreased by 6.3 kg, the body fat rate and visceral fat area decreased by 9.2 and 21.7% respectively, and the average blood pressure decreased from 124/82 mmHg to 120/80 mmHg, with statistically significant differences. In terms of metabolism, fasting blood glucose decreased by 5.15%, fasting insulin decreased by 27.86%, total cholesterol and triglyceride decreased by 8.9 and 18.02% respectively. In terms of endocrine metabolism, free testosterone index and SHBG decreased by 31 and 65.6% respectively. In addition, levels of inflammatory cytokines decreased after lifestyle intervention. Domecq et al. [33] conducted a meta-analysis on the pregnancy outcomes of PCOS patients with lifestyle intervention before assisting pregnancy and showed that weight loss with adjusting lifestyle could reduce fasting insulin and fasting glucose, BMI decline was associated with reduced fasting blood glucose, these would help improve glucose metabolism. Based on previous research data, high-protein diet combined with regular exercise was adopted as the first-line treatment for obese PCOS patients and the results showed that after treatment, the patient's BMI was significantly reduced and the levels of LH, T, fasting blood glucose and insulin, IR and TG were decreased, but HDL was increased. Mehrabani et al. [52] demonstrated that the high-protein diet could significantly reduce the levels of BMI, T, insulin and IR. There were no significant changes in lipid and LH levels except LDL. Studies showed that after 8–12 weeks of moderate intensity exercise, the relevant indexes of lipid metabolism in PCOS patients could be significantly improved [48].

At present, modern medicine believed that the mechanism of exercise regulating metabolism may be related to the hormones involved in energy metabolism. Exercise could strengthen resting energy consumption (REE) of PCOS patients and reduce

the content of adipose tissue in PCOS patients. REE of PCOS patients decreased compared with healthy women of the same age and BMI. Studies have shown that REE in obese people was significantly lower than that in non-obese one [53]. Therefore, REE may be declined in PCOS patients. Georgopoulos et al. [54] have also shown that there was a reduction of REE in PCOS patients, which be associated with increased fat content in the patients. On the other hand, exercise had a profound impact on the neuro-endocrine system, especially on the hormones involved in the regulation of energy metabolism, such as insulin, androgen, lactone and leptin. First, insulin resistance was the pathological basis of PCOS. It was generally agreed that one of the causes of insulin resistance was the abnormality of insulin signal pathway. Exercise or exercise training could affect the insulin action pathway from the three levels of pre-receptor, receptor and post-receptor in the pancreas islet [55]. Secondly, leptin played an important role in PCOS metabolism. Existing research showed that long-term exercise could reduce serum leptin level, improve leptin resistance and the sensitivity of the body to leptin. After sensitization, leptin could further inhibit the excessive secretion of insulin and the occurrence of hyperinsulinism, thus improve IR [56]. Thirdly, prolonged and intense exercise caused a decrease in blood testosterone, which may be caused by the body's hormones and some trace elements. For example, long time exercise or exercise training had obvious influence on zinc and selenium, zinc and selenium deficiency caused serum testosterone to drop [57]. Finally, some scholars have found that exercise could improve the expression of visfatin and promote the differentiation of visceral fat cells, and the differentiated fat cells contained a large number of insulin receptors, thus improving the sensitivity of insulin. Exercise may also promote necrosis and apoptosis of large adipocytes differentiated in visceral tissue, thereby reducing visceral adipocytes, improving insulin sensitivity and lipid metabolism disorder in the body [58].

4. The influence of exercise or combination with other methods on negative emotions of PCOS patients

In contemporary society, fierce competition, heavy work and study tasks, stay up late, lack of exercise and bad eating habits tended to cause stress reactions such as mental tension and emotional fluctuations in women which lead to endocrine imbalance in the hypothalamic-pituitary-ovarian gonadal axis, thus inducing PCOS. At the same time, PCOS patients were more likely to have severe inferiority complex, anxiety, depression and other abnormal psychological reactions than normal women [59]. It was reported that 17.19% patients with PCOS had anxiety, 32.81% had depression, and 10.15% had both depression and anxiety [60, 61]. Sudden or long-term mental depression, tension, anxiety, fear, depression, etc. could lead to mental endocrine disorders, menstrual and ovulation dysfunction, which aggravate the condition of PCOS patients and ultimately not conducive to the treatment of PCOS patients [62]. Therefore, it is increasingly important to treat PCOS patients' psychological diseases and relieve their psychological pressure.

Professor from the British nutrition foundation said doing more exercise could go a long way in boosting your self-esteem, improving your mood and coping with stress. It may even help you sleep and prevent depression. Studies have shown that exercise had obvious effect on the improvement of people's mental health level. Morgan suggested that intense exercise reduced anxiety which lasts for 2–5 h and regular exercise decreased anxiety and depression and boosted self-esteem [63]. Exercise could improve the state of mind, reduce the level of anxiety and have a therapeutic effect on anxiety. Regular exercise could effectively improve the psychological state of PCOS patients, ensure the effect of life intervention, and improve their

quality of life [44]. Large, medium and small exercise loads could effectively improve the anxiety level of patients with severe generalized anxiety disorder, but large and medium exercise could more effectively improve the anxiety level of patients with mental anxiety [64]. Studies have found that a combination of physical activity and dietary intervention in PCOS patients could reduce depression and improve their life quality [65]. Teede et al. showed that PCOS patients lost 5–10% of body weight, their reproductive, metabolic and psychological characteristics were improved, and their sexual satisfaction and life quality were also increased [66].

For the regulation mechanism of negative emotions, exercise could increase the oxygen demand, improve the oxygen consumption and the cardiopulmonary function in human body. On the other hand, it could distract attention, transfer the experience of bad emotions, to achieve a comfortable body and mind and regulate emotions. Sports could produce a wealth of emotional experience and obtain more sports pleasure, comfort, satisfaction and fulfillment, showing an overall psychological state of good.

5. Conclusions

PCOS is a common reproductive endocrine disease in women of reproductive age. The clinical manifestations of PCOS patients are heterogeneous, it mainly affects reproductive, metabolic function and mental health. Its biochemical characteristics include insulin resistance, hyperinsulinemia, androgen overload and imbalance of LH/FSH ratio. Clinical manifestations include obesity, hirsute, acne and other high androgen symptoms, irregular ovulation or anovulation, menstrual disorders and infertility, bilateral polycystic changes of ovaries under ultrasound, and increased ovarian volume.

As one of the important treatment strategies for PCOS patients, adjusting lifestyle to lose weight was helpful to improve endocrine disorders and reduce the risk of metabolic syndrome, which is widely used in clinical practice. For obese PCOS patients who receive assisted pregnancy treatment, dietary and exercise intervention before assisted pregnancy could help restore spontaneous ovulation and enhance natural pregnancy rate, improve the outcome of assisted pregnancy, increase the success rate of assisted pregnancy and reduce the risk of pregnancy complications. Compared with weight-loss surgery, ovulatory drugs and IVF assisted pregnancy treatment, lifestyle adjustment for weight loss had unique advantages such as strong feasibility, low cost and large benefits. However, how to maintain reasonable, stable and long-term weight loss and keep target weight was still a big problem. In recent years, multidisciplinary cooperation involving diet, exercise and psychosocial intervention to guide obesity PCOS weight loss had attracted more and more attention, which is of great significance to guide clinicians to formulate more scientific and effective individual weight loss programs, improve the outcome of assisted pregnancy and long-term life quality of obese PCOS patients.

Acknowledgements

The study was supported by the Fujian Provincial Natural Science Foundation (2017 J01626 and 2017 J01627).

Conflict of interest

None.

IntechOpen

Author details

Shaobing Wang*, Zhenghong Zhang and Yiping Liu
Provincial University Key Laboratory of Sport and Health Science, School of
Physical Education and Sport Sciences, State Key Laboratory for Evaluation of
Exercise Physiological Functions, General Administration of Sport of China, Fujian
Normal University, Fuzhou, China

*Address all correspondence to: wshbingcn@fjnu.edu.cn

IntechOpen

© 2019 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

References

- [1] Li R, Zhang Q, Yang D, Li S, Lu S, Wu X, et al. Prevalence of polycystic ovary syndrome in women in China: A large community-based study. *Human Reproduction*. 2013;**28**(9):2562-2569. DOI: 10.1093/humrep/det262
- [2] Daan NM, Louwers YV, Koster MP, Eijkemans MJ, de Rijke YB, Lentjes EW, et al. Cardiovascular and metabolic profiles amongst different polycystic ovary syndrome phenotypes: Who is really at risk? *Fertility and Sterility*. 2014;**102**(5):1444-1451.e3. DOI: 10.1016/j.fertnstert.2014.08.001
- [3] Mahalingaiah S, Diamanti-Kandarakis E. Targets to treat metabolic syndrome in polycystic ovary syndrome. *Expert Opinion on Therapeutic Targets*. 2015;**19**(11):1561-1574. DOI: 10.1517/14728222.2015.1101067
- [4] Messinis IE, Messini CI, Anifandis G, Dafopoulos K. Polycystic ovaries and obesity. *Best Practice & Research. Clinical Obstetrics & Gynaecology*. 2015;**29**(4):479-488. DOI: 10.1016/j.bpobgyn.2014.11.001
- [5] Cosar E, Uçok K, Akgün L, Köken G, Sahin FK, Arioğlu DT, et al. Body fat composition and distribution in women with polycystic ovary syndrome. *Gynecological Endocrinology*. 2008;**24**(8):428-432. DOI: 10.1080/09513590802234253
- [6] Borrueal S, Fernández-Durán E, Alpañés M, Martí D, Alvarez-Blasco F, Luque-Ramírez M, et al. Global adiposity and thickness of intraperitoneal and mesenteric adipose tissue depots are increased in women with polycystic ovary syndrome (PCOS). *The Journal of Clinical Endocrinology and Metabolism*. 2013;**98**(3):1254-1263. DOI: 10.1210/jc.2012-3698
- [7] Motta AB. The role of obesity in the development of polycystic ovary syndrome. *Current Pharmaceutical Design*. 2012;**18**(17):2482-2491
- [8] Lin AW, Lujan ME. Comparison of dietary intake and physical activity between women with and without polycystic ovary syndrome: A review. *Advances in Nutrition*. 2014;**5**(5):486-496
- [9] Cui N, Wang H, Wang W, Zhang J, Xu Y, Jiang L, et al. Impact of body mass index on outcomes of In vitro fertilization/intracytoplasmic sperm injection among polycystic ovarian syndrome patients. *Cellular Physiology and Biochemistry*. 2016;**39**(5):1723-1734. DOI: 10.1159/000447873
- [10] Koning AM, Kuchenbecker WK, Groen H, Hoek A, Land JA, Khan KS, et al. Economic consequences of overweight and obesity in infertility: A framework for evaluating the costs and outcomes of fertility care. *Human Reproduction Update*. 2010;**16**(3):246-254. DOI: 10.1093/humupd/dmp053
- [11] Wang F, Dai W, Yang XH, Guo YH, Sun YP. Analyses of optimal body mass index for infertile patients with either polycystic or non-polycystic ovary syndrome during assisted reproductive treatment in China. *Scientific Reports*. 2016;**6**:34538. DOI: 10.1038/srep34538
- [12] Stepto NK, Cassar S, Joham AE, Hutchison SK, Harrison CL, Goldstein RF, et al. Women with polycystic ovary syndrome have intrinsic insulin resistance on euglycaemic-hyperinsulinaemic clamp. *Human Reproduction*. 2013;**28**(3):777-784. DOI: 10.1093/humrep/des463. Epub 2013 Jan 12
- [13] Kyaw Tun T, McGowan A, Phelan N, Correia N, Boran G, O'Connor AL, et al. Obesity and insulin resistance are the main determinants of postprandial lipoprotein dysmetabolism in polycystic

ovary syndrome. *International Journal of Endocrinology*. 2016;**2016**:9545239. DOI: 10.1155/2016/9545239

[14] Rojas J, Chávez M, Olivar L, Rojas M, Morillo J, Mejías J, et al. Polycystic ovary syndrome, insulin resistance, and obesity: Navigating the pathophysiologic labyrinth. *International Journal of Reproductive Medicine*. 2014;**2014**:719050. DOI: 10.1155/2014/719050

[15] Harrison CL, Lombard CB, Moran LJ, Teede HJ. Exercise therapy in polycystic ovary syndrome: A systematic review. *Human Reproduction Update*. 2011;**17**(2):171-183. DOI: 10.1093/humupd/dmq045

[16] Palomba S, de Wilde MA, Falbo A, Koster MP, La Sala GB, Fauser BC. Pregnancy complications in women with polycystic ovary syndrome. *Human Reproduction Update*. 2015;**21**(5):575-592. DOI: 10.1093/humupd/dmv029

[17] Spritzer PM, Lecke SB, Satler F, Morsch DM. Adipose tissue dysfunction, adipokines, and low-grade chronic inflammation in polycystic ovary syndrome. *Reproduction*. 2015;**149**(5):R219-R227. DOI: 10.1530/REP-14-0435

[18] Heymsfield SB, Wadden TA. Mechanisms pathphysiology and management of obesity. *The New England Journal of Medicine*. 2017;**376**:254-266

[19] Thomson RL, Buckley JD, Brinkworth GD. Exercise for the treatment and management of overweight women with polycystic ovary syndrome: A review of the literature. *Obesity Reviews*. 2011;**12**(5):e202-e210. DOI: 10.1111/j.1467-789X.2010.00758.x

[20] Tsagareli V, Noakes M, Norman RJ. Effect of a very-low-calorie diet on

in vitro fertilization outcomes. *Fertility and Sterility*. 2006;**86**(1):227-229. DOI: 10.1016/j.fertnstert.2005.12.041

[21] Gosman GG, Katcher HI, Legro RS. Obesity and the role of gut and adipose hormones in female reproduction. *Human Reproduction Update*. 2006;**12**(5):585-601. DOI: 10.1093/humupd/dml024

[22] Zhang C, Luo H, Gao F, Zhang CT, Zhang R. A reduction in both visceral and subcutaneous fats contributes to increased adiponectin by lifestyle intervention in the Diabetes Prevention Program. *Acta Diabetologica*. 2015;**52**(3):625-628. DOI: 10.1007/s00592-014-0655-2

[23] Siegrist M, Rank M, Wolfarth B, Langhof H, Haller B, Koenig W, et al. Leptin, adiponectin, and short-term and long-term weight loss after a lifestyle intervention in obese children. *Nutrition*. 2013;**29**(6):851-857. DOI: 10.1016/j.nut.2012.12.011. Epub 2013 Feb 16

[24] Hollmann M, Runnebaum B, Gerhard I. Effects of weight loss on the hormonal profile in obese, infertile women. *Human Reproduction*. 1996;**11**(9):1884-1891. DOI: 10.1093/oxfordjournals.humrep.a019512

[25] Hoeger KM. Role of lifestyle modification in the management of polycystic ovary syndrome. *Best Practice & Research. Clinical Endocrinology & Metabolism*. 2006;**20**(2):293-310. DOI: 10.1016/j.beem.2006.03.008

[26] Huber-Buchholz MM, Carey DG, Norman RJ. Restoration of reproductive potential by lifestyle modification in obese polycystic ovary syndrome: Role of insulin sensitivity and luteinizing hormone. *The Journal of Clinical Endocrinology and Metabolism*. 1999;**84**(4):1470-1474. DOI: 10.1210/jcem.84.4.5596

- [27] Kuchenbecker WK, Groen H, van Asselt SJ, Bolster JH, Zwerver J, Slart RH, et al. In women with polycystic ovary syndrome and obesity, loss of intra-abdominal fat is associated with resumption of ovulation. *Human Reproduction*. 2011;**26**(9):2505-2512. DOI: 10.1093/humrep/der229
- [28] Salama AA, Amine EK, Salem HA, Abd El Fattah NK. Anti-inflammatory dietary combo in overweight and obese women with polycystic ovary syndrome. *North American Journal of Medical Sciences*. 2015;**7**(7):310-316. DOI: 10.4103/1947-2714.161246
- [29] Legro RS, Dodson WC, Kris-Etherton PM, Kunselman AR, Stetter CM, Williams NI, et al. Randomized controlled trial of preconception interventions in infertile women with polycystic ovary syndrome. *The Journal of Clinical Endocrinology and Metabolism*. 2015;**100**(11):4048-4058. DOI: 10.1210/jc.2015-2778
- [30] Legro RS, Dodson WC, Kunselman AR, Stetter CM, Kris-Etherton PM, Williams NI, et al. Benefit of delayed fertility therapy with preconception weight loss over immediate therapy in obese women with PCOS. *The Journal of Clinical Endocrinology and Metabolism*. 2016;**101**(7):2658-2666. DOI: 10.1210/jc.2016-1659
- [31] Mutsaerts MA, van Oers AM, Groen H, Burggraaff JM, Kuchenbecker WK, Perquin DA, et al. Randomized trial of a lifestyle program in obese infertile women. *The New England Journal of Medicine*. 2016;**374**(20):1942-1953. DOI: 10.1056/NEJMoa1505297
- [32] van Oers AM, Groen H, Mutsaerts MA, Burggraaff JM, Kuchenbecker WK, Perquin DA, et al., LIFEstyle study group. Effectiveness of lifestyle intervention in subgroups of obese infertile women: A subgroup analysis of a RCT. *Human Reproduction*. 2016;**31**(12):2704-2713. DOI: 10.1093/humrep/dew252
- [33] Domecq JP, Prutsky G, Mullan RJ, Hazem A, Sundaresh V, Elamin MB, et al. Lifestyle modification programs in polycystic ovary syndrome: Systematic review and meta-analysis. *The Journal of Clinical Endocrinology and Metabolism*. 2013;**98**(12):4655-4663. DOI: 10.1210/jc.2013-2385
- [34] Tannus S, Burke YZ, Kol S. Treatment strategies for the infertile polycystic ovary syndrome patient. *Women's Health (London, England)*. 2015;**11**(6):901-912. DOI: 10.2217/whe.15.40
- [35] Moran LJ, Hutchison SK, Norman RJ, Teede HJ. Lifestyle changes in women with polycystic ovary syndrome. *Cochrane Database of Systematic Reviews*. 2011;**7**:CD007506. DOI: 10.1002/14651858.CD007506.pub3
- [36] Mahoney D. Lifestyle modification intervention among infertile overweight and obese women with polycystic ovary syndrome. *Journal of the American Association of Nurse Practitioners*. 2014;**26**(6):301-308. DOI: 10.1002/2327-6924.12073
- [37] Phy JL, Pohlmeier AM, Cooper JA, Watkins P, Spallholz J, Harris KS, et al. Low starch/low dairy diet results in successful treatment of obesity and co-morbidities linked to polycystic ovary syndrome (PCOS). *Journal of Obesity and Weight Loss Therapy*. 2015;**5**(2). pii: 259. DOI: 10.4172/2165-7904.1000259
- [38] Ujvari D, Hulchiy M, Calaby A, Nybacka Å, Byström B, Hirschberg AL. Lifestyle intervention up-regulates gene and protein levels of molecules involved in insulin signaling in the endometrium of overweight/obese women with polycystic ovary

syndrome. *Human Reproduction*. 2014;**29**(7):1526-1535

[39] Jiskoot G, Benneheij SH, Beerthuizen A, de Niet JE, de Klerk C, Timman R, et al. A three-component cognitive behavioural lifestyle program for preconceptional weight-loss in women with polycystic ovary syndrome (PCOS): A protocol for a randomized controlled trial. *Reproductive Health*. 2017;**14**(1):34. DOI: 10.1186/s12978-017-0295-4

[40] Nybacka Å, Carlström K, Fabri F, Hellström PM, Hirschberg AL. Serum antimüllerian hormone in response to dietary management and/or physical exercise in overweight/obese women with polycystic ovary syndrome: Secondary analysis of a randomized controlled trial. *Fertility and Sterility*. 2013;**100**(4):1096-1102. DOI: 10.1016/j.fertnstert.2013.06.030

[41] Greenwood EA, Noel MW, Kao CN, Shinkai K, Pasch LA, Cedars MI, et al. Vigorous exercise is associated with superior metabolic profiles in polycystic ovary syndrome independent of total exercise expenditure. *Fertility and Sterility*. 2016;**105**(2):486-493. DOI: 10.1016/j.fertnstert.2015.10.020

[42] Macut D, Bjekić-Macut J, Savić-Radojević A. Dyslipidemia and oxidative stress in PCOS. *Frontiers of Hormone Research*. 2013;**40**:51-63. DOI: 10.1159/000341683

[43] Dokras A, Playford M, Kris-Etherton PM, Kunselman AR, Stetter CM, Williams NI, et al. Impact of hormonal contraception and weight loss on high-density lipoprotein cholesterol efflux and lipoprotein particles in women with polycystic ovary syndrome. *Clinical Endocrinology*. 2017;**86**(5):739-746. DOI: 10.1111/cen.13310

[44] Palomba S, Giallauria F, Falbo A, Russo T, Oppedisano R, Tolino A, et al.

Structured exercise training programme versus hypocaloric hyperproteic diet in obese polycystic ovary syndrome patients with anovulatory infertility: A 24-week pilot study. *Human Reproduction*. 2008;**23**(3):642-650. DOI: 10.1093/humrep/dem391

[45] Smekal A, Vaclavik J. Adipokines and cardiovascular disease: A comprehensive review. *Biomedical Papers of the Medical Faculty of the University Palacky, Olomouc, Czech Republic*. 2017;**161**(1):31-40. DOI: 10.5507/bp.2017.002

[46] López-Jaramillo P, Gómez-Arbeláez D, López-López J, López-López C, Martínez-Ortega J, Gómez-Rodríguez A, et al. The role of leptin/adiponectin ratio in metabolic syndrome and diabetes. *Hormone Molecular Biology and Clinical Investigation*. 2014;**18**(1):37-45. DOI: 10.1515/hmbci-2013-0053

[47] Wehr E, Möller R, Horejsi R, Giuliani A, Kopera D, Schweighofer N, et al. Subcutaneous adipose tissue topography and metabolic disturbances in polycystic ovary syndrome. *Wiener Klinische Wochenschrift*. 2009;**121**(7-8):262-269. DOI: 10.1007/s00508-009-1162-2

[48] Brown AJ, Setji TL, Sanders LL, Lowry KP, Otvos JD, Kraus WE, et al. Effects of exercise on lipoprotein particles in women with polycystic ovary syndrome. *Medicine and Science in Sports and Exercise*. 2009;**41**(3):497-504. DOI: 10.1249/MSS.0b013e31818c6c0c

[49] Lord JM, Flight IH, Norman RJ. Metformin in polycystic ovary syndrome: Systematic review and meta-analysis. *BMJ*. 2003;**327**(7421):951-953. DOI: 10.1136/bmj.327.7421.951

[50] Tymchuk CN, Tessler SB, Barnard RJ. Changes in sex hormone-binding globulin, insulin, and serum

lipids in postmenopausal women on a low-fat, high-fiber diet combined with exercise. *Nutrition and Cancer*. 2000;**38**(2):158-162. DOI: 10.1207/S15327914NC382_3

[51] Chan JL, Kar S, Vanky E, Morin-Papunen L, Piltonen T, Puurunen J, et al. Racial and ethnic differences in the prevalence of metabolic syndrome and its components of metabolic syndrome in women with polycystic ovary syndrome: A regional cross-sectional study. *American Journal of Obstetrics and Gynecology*. 2017;**217**(2):189.e1-189.e8. DOI: 10.1016/j.ajog.2017.04.007

[52] Mehrabani HH, Salehpour S, Amiri Z, Farahani SJ, Meyer BJ, Tahbaz F. Beneficial effects of a high-protein, low-glycemic-load hypocaloric diet in overweight and obese women with polycystic ovary syndrome: A randomized controlled intervention study. *Journal of the American College of Nutrition*. 2012;**31**(2):117-125

[53] Jia WP, Yang M, Shao XY, Bao YQ, Lu JX, Xiang KS. Resting energy expenditure and its relationship with patterns of obesity and visceral fat area in Chinese adults. *Biomedical and Environmental Sciences*. 2005;**18**(2):103-107

[54] Georgopoulos NA, Saltamavros AD, Vervita V, Karkoulas K, Adonakis G, Decavalas G, et al. Basal metabolic rate is decreased in women with polycystic ovary syndrome and biochemical hyperandrogenemia and is associated with insulin resistance. *Fertility and Sterility*. 2009;**92**(1):250-255. DOI: 10.1016/j.fertnstert.2008.04.067

[55] Moghetti P. Insulin resistance and polycystic ovary syndrome. *Current Pharmaceutical Design*. 2016;**22**(36):5526-5534

[56] Pehlivanov B, Mitkov M. Serum leptin levels correlate with clinical

and biochemical indices of insulin resistance in women with polycystic ovary syndrome. *The European Journal of Contraception & Reproductive Health Care*. 2009;**14**(2):153-159. DOI: 10.1080/13625180802549962

[57] Shafiei Neek L, Gaeini AA, Choobineh S. Effect of zinc and selenium supplementation on serum testosterone and plasma lactate in cyclist after an exhaustive exercise bout. *Biological Trace Element Research*. 2011;**144**(1-3):454-462. DOI: 10.1007/s12011-011-9138-2

[58] Qi X, Zhang B, Zhao Y, Li R, Chang HM, Pang Y, et al. Hyperhomocysteinemia promotes insulin resistance and adipose tissue inflammation in PCOS mice through modulating M2 macrophage polarization via estrogen suppression. *Endocrinology*. 2017;**158**(5):1181-1193. DOI: 10.1210/en.2017-00039

[59] Coffey S, Bano G, Mason HD. Health-related quality of life in women with polycystic ovary syndrome: A comparison with the general population using the polycystic ovary syndrome questionnaire (PCOSQ) and the short Form-36 (SF-36). *Gynecological Endocrinology*. 2006;**22**(2):80-86. DOI: 10.1080/09513590600604541

[60] Bhattacharya SM, Jha A. Prevalence and risk of depressive disorders in women with polycystic ovary syndrome (PCOS). *Fertility and Sterility*. 2010;**94**(1):357-359. DOI: 10.1016/j.fertnstert.2009.09.025

[61] Deeks AA, Gibson-Helm ME, Teede HJ. Anxiety and depression in polycystic ovary syndrome: A comprehensive investigation. *Fertility and Sterility*. 2010;**93**(7):2421-2423. DOI: 10.1016/j.fertnstert.2009.09.018

[62] Bazarganipour F, Ziaei S, Montazeri A, Foroozanfard F, Kazemnejad A, Faghihzadeh S. Psycho-

logical investigation in patients with polycystic ovary syndrome. *Health and Quality of Life Outcomes*. 2013;**11**:141. DOI: 10.1186/1477-7525-11-141

[63] Morgan WP. Affective beneficence of vigorous physical activity. *Medicine and Science in Sports and Exercise*. 1985;**17**(1):94-100

[64] Conte F, Banting L, Teede HJ, Stepto NK. Mental health and physical activity in women with polycystic ovary syndrome: A brief review. *Sports Medicine*. 2015;**45**(4):497-504. DOI: 10.1007/s40279-014-0291-6

[65] Thomson RL, Buckley JD, Lim SS, Noakes M, Clifton PM, Norman RJ, et al. Lifestyle management improves quality of life and depression in overweight and obese women with polycystic ovary syndrome. *Fertility and Sterility*. 2010;**94**(5):1812-1816. DOI: 10.1016/j.fertnstert.2009.11.001

[66] Teede H, Deeks A, Moran L. Polycystic ovary syndrome: A complex condition with psychological, reproductive and metabolic manifestations that impacts on health across the lifespan. *BMC Medicine*. 2010;**8**:41. DOI: 10.1186/1741-7015-8-41