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Original Research Article

Biotin Supplementation Ameliorated Metabolic and Biochemical Dysfunctions in Female Wistar Rats Induced with Polycystic Ovarian Syndrome

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Abstract: Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder characterised by insulin resistance, oxidative stress, and dyslipidaemia, contributing to long-term metabolic and cardiovascular complications. Despite available therapies, effective and sustainable alternatives remain limited, highlighting the need for novel adjunct interventions. This study investigated the effects of biotin supplementation on glucose metabolism, antioxidant status, and lipid profile in letrozole-induced PCOS female Wistar rats. A controlled experimental design was adopted, with 30 rats randomly assigned to five groups (n = 6): control, letrozole-induced PCOS, letrozole + metformin, letrozole + low-dose biotin (100 mg/kg), and letrozole + high-dose biotin (150 mg/kg). Treatments were administered orally for 15–21 days. Biochemical parameters were analysed using standard laboratory methods, and data were expressed as mean ± SD. One-way ANOVA was used for statistical analysis, with $p < 0.05$ considered significant. Letrozole induction significantly increased blood glucose (95.91 ± 2.35 mg/dl) and reduced antioxidant enzyme activity compared with the control ($p < 0.05$). High-dose biotin significantly reduced glucose levels (85.55 ± 2.46 mg/dl) and improved catalase, SOD, GSH, and GST activities compared with the PCOS group ($p < 0.05$), though metformin achieved superior normalisation (75.01 ± 2.38 mg/dl). Biotin supplementation also improved lipid abnormalities, with high-dose biotin reducing LDL (27.89 ± 1.00 mg/dl) and increasing HDL (155.59 ± 1.74 mg/dl) relative to letrozole ($p < 0.05$). In conclusion, biotin supplementation, particularly at high doses, partially ameliorated hyperglycaemia, oxidative stress, and dyslipidaemia in PCOS-induced rats. Biotin may serve as a supportive therapeutic agent in PCOS management. Further studies are recommended to determine optimal dosing, underlying mechanisms, and long-term clinical relevance.

Keywords: Biotin Supplementation, Polycystic Ovarian Syndrome (PCOS), Insulin Resistance, Antioxidant Enzymes, Dyslipidaemia, Blood Glucose.

1. INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine and metabolic disorders which afflicts women with ovaries worldwide. The aetiology of PCOS "involves an imbalance of hormones, causing reproductive problems such as absent or irregular menstrual cycles and higher or abnormal levels of androgenic hormones such as testosterone, as well as polycystic ovaries (American College of Obstetricians and Gynaecologists (ACOG), 2018). It remains an important public health challenge worldwide, despite improvements in public awareness and management. People living with PCOS complain of a range of problems, including acne, excessive hair growth on their faces and bodies, obesity, and fertility problems. They are likely to experience insulin resistance, type 2 diabetes, and cardiovascular disease, among other consequences, in the future (Teede *et al*., 2018).

The underlying, unifying feature in the case of PCOS is the metabolic dysfunction, primarily arising as a consequence of the inability of the system to effectively utilise insulin. Insulin resistance is commonly observed in the case of women suffering from PCOS, causing an irregularity in glucose metabolism, facilitating weight gain, coupled with an increased risk of glucose intolerance or diabetes, as observed by Teede *et al*., (2018). To compensate for the lack of insulin activity, insulin concentration is increased, further exacerbating hormonal imbalances (Dumesic *et al*., 2015). A

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consequent metabolic imbalance occurs in the system, creating abnormalities in lipid levels, facilitating an increase in the levels of triglycerides, coupled with lower levels of HDL in the system, such as the observed cases of dyslipidemia, causing possible cardiovascular complications in the system, as observed by Dumesic *et al.,* (2015). Excessive levels of total cholesterol, LDL, and triglycerides, along with lower levels of HDL, have been observed in the system in the case of Cardiovascular Complications (Dunaif, 2016)

One of the main reasons for the presence of the co-existing conditions of PCOS is the metabolic imbalance. This is because problems with carbohydrate and fat metabolism, as well as insulin not functioning as it should, may cause an imbalance in the body. This will increase stress, leading to cell dysfunction. However, it is worth noting that people may differ in the levels of biochemical disturbances, which explains the complexity of the syndrome.

Polycystic ovary syndrome (PCOS) is typically treated with a combination of PCOS symptom management and mitigation thereof, with both medications and lifestyle adjustments being employed. For instance, physicians often resort to hormone pills, which have the effect of regulating the cycles and reducing androgen production (Teede *et al.*, 2018). For the unruly androgen-related manifestations such as noticeable body hair and acne, anti-androgen therapies such as spironolactone and finasteride are often the go-to (Teede *et al*., 2018). On the lifestyle side, diet and exercise are the leading therapies and usually involve weight reduction to alleviate symptom manifestations. However, such an approach doesn't always bear fruit. Aside from potential side effects, they are not always sustainable.

Biotin (vitamin B7) is a water-soluble vitamin that helps the body turn food into energy and supports healthy hair, skin, and nails. In this case, it is generated and distributed because it acts as a cofactor for enzymes involved in power generation and metabolic regulation. Therefore, biotin regulates and maintains metabolic balance and various normal body functions, including glucose use, fat metabolism, and skin, hair, and nail health. (National Institutes of Health (NIH), Office of Dietary Supplements, 2021)

In light of the above considerations, the present research work was conducted to assess the impact of biotin supplementation on metabolic and biochemical activities in adult female Wistar rats with induced PCOS. The areas of focus have been glucose metabolism, antioxidant status, and alterations in lipid profile.

2. MATERIALS AND METHODS

2.1 Materials

The experimental set consisted of female Wistar rats and a minimal number of research consumables: standard rat feed pellets, gloves, syringes, a digital scale for weighing, sample bottles, cotton wool, masking tape, and plastic containers with aluminium lids. Letrozole, metformin, and a biotin supplement were used in the experiment. All reagents were dissolved or prepared with distilled water before administration.

2.2 Experimental Animals

The rats used for this experiment were 30 adult female Wistar rats weighing 150–180 g each, obtained from the animal house facility of the Department of Pharmacology, University of Port Harcourt, Rivers State, Nigeria. They were housed in plastic cages covered with aluminium lids in the Faculty of Basic Medical Sciences, College of Medical Sciences animal house at the university. Lighting was natural, and they had free access to tap water and a standard pellet diet ad libitum. The rats underwent 2 weeks of acclimatisation before the experiment began.

2.3 Study Design and Grouping

A controlled experimental design was adopted. After acclimatisation, the 30 rats were randomly assigned to 5 groups (n = 6 per group). Treatments were administered once daily, based on body weight, for the specified duration.

1. **Group I (Biotin Low Dose):** Rats received 100 mg/kg body weight of biotin for 15 days.
2. **Group II (Biotin High Dose):** Rats received 150 mg/kg body weight of biotin for 15 days.
3. **Group III (Metformin):** Rats received metformin at a dose of 2 mg/100 g/day for 15 days.
4. **Group IV (Letrozole):** Rats received letrozole at a dose of 1 mg/kg body weight for 21 days to induce polycystic ovarian syndrome (PCOS).
5. **Group V (Control):** Rats received feed and water only.

All administrations were carried out orally using a syringe.

2.4 Preparation and Dose Calculation

Letrozole was prepared by dissolving 100 mg in 100 ml of distilled water to obtain a concentration of 1 mg/ml. Based on a mean body weight of 0.15 kg, each rat received 0.15 ml daily for 21 days to induce PCOS.

For the low-dose biotin group, 100 mg of biotin was dissolved in 10 mL of distilled water to yield a 10 mg/mL solution. Each rat (mean weight 0.15 kg) received 1.5 ml daily for 15 days. For the high-dose group, 150 mg of biotin was dissolved in 10 ml of distilled water to obtain a 15 mg/ml solution, and each rat received 2.25 ml daily for 15 days.

Metformin was prepared by dissolving 100 mg in 10 ml of distilled water. Each rat received 0.3 ml daily for 15 days, as calculated.

2.5 Statistical Analysis

Data obtained from the study were expressed as mean ± standard deviation (SD). Statistical analysis was performed using one-way analysis of variance (ANOVA) where appropriate, and differences were considered statistically significant at $p < 0.05$.

3. RESULTS

Table 1: Effect of biotin on Blood glucose level and Antioxidant system (catalase, SOD, GSH, GST) in female rats induced with PCOS

Group	Blood glucose levels	Antioxidant system			
		CAT	SOD	GSH	GST
Group I (Control)	75.41 ± 2.30	4.82 ± 0.11	5.68 ± 0.19	6.82 ± 0.11	10.61 ± 0.22
Group II (Letrozole)	95.91 ± 2.35	3.28 ± 0.11	2.88 ± 0.05	4.68 ± 0.26	7.96 ± 0.03
Group III (LET + MET)	75.01 ± 2.38	4.61 ± 0.18	5.54 ± 0.09	6.69 ± 0.09	9.73 ± 0.13
Group IV (LET + LOW BIOTIN)	92.40 ± 2.48 [a, c]	4.21 ± 0.08[a, b]	3.27 ± 0.52[a,c]	6.99 ± 0.17[b]	8.44 ± 0.26[a, c]
Group V (LET + HIGH BIOTIN)	85.55 ± 2.46	4.61 ± 0.07[b]	5.84 ± 0.06[b]	6.95 ± 0.07[b]	9.98 ± 0.10[b]

Abbreviations: CAT (catalase), SOD (superoxide dismutase), GSH (glutathione), GST (glutathione S-transferase). Values are expressed as mean ± SEM; n= 4. Alphabets a, b and c denote statistically significant variables of the biotin-treated groups compared with the control group, letrozole group and LET + MET group, respectively. [a] $p< 0.05$- Significant when compared to the control group, [b] $p< 0.05$- Significant when compared to the Letrozole group, [c] $p< 0.05$- Significant when compared to the LET + MET group.

Table 1 shows that letrozole induction increased blood glucose levels and markedly impaired antioxidant status in female rats when compared with the control group. Specifically, the letrozole group showed higher glucose (95.91 ± 2.35) and lower CAT (3.28 ± 0.11), SOD (2.88 ± 0.05), GSH (4.68 ± 0.26), and GST (7.96 ± 0.03), indicating oxidative stress. Metformin co-treatment (LET + MET) largely normalised these indices, producing values comparable to those in the control for glucose (75.01 ± 2.38) and antioxidant enzymes. Biotin supplementation showed dose-dependent improvement: low-dose biotin modestly reduced glucose (92.40 ± 2.48) and improved CAT and GSH, though SOD and GST remained relatively lower; several changes were significant versus control, letrozole, and/or LET + MET groups ($p < 0.05$). High-dose biotin produced better restoration of antioxidant markers and lower glucose (85.55 ± 2.46), with significant improvements compared with the letrozole group ($p < 0.05$).

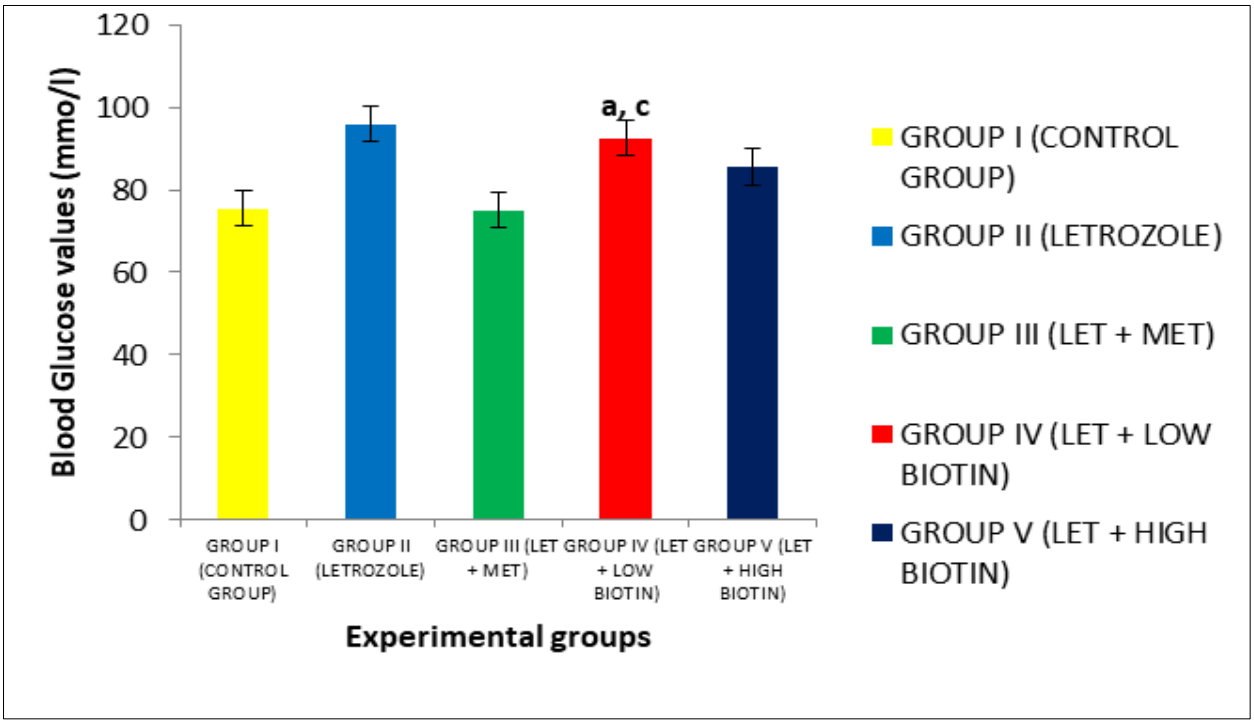

Figure 1: Effect of biotin on blood glucose level in female rats induced with PCOS. [a] $p< 0.05$- Significant when compared to the control group, [c] $p< 0.05$- Significant when compared to the LET + MET group.

Figure 1 shows that letrozole induction markedly increased blood glucose levels in female rats compared with the control group, confirming the hyperglycaemic effect associated with PCOS. The

control group maintained a lower glucose level (approximately 75 mmol/L), whereas the letrozole group showed a pronounced elevation (approximately 96 mmol/L). Co-administration of metformin (LET + MET) effectively restored glucose levels to near control levels, demonstrating its corrective effect. In contrast, low-dose biotin (LET + LOW BIOTIN) produced only a modest reduction in glucose levels, which remained significantly higher than both the control and LET + MET groups ($p < 0.05$), as indicated by annotations a and c. High-dose biotin (LET + HIGH BIOTIN) showed a greater reduction in glucose than the low-dose group, though values remained higher than in the control group. Overall, high-dose biotin showed improved glycaemic control compared with low-dose biotin, suggesting a dose-dependent effect.

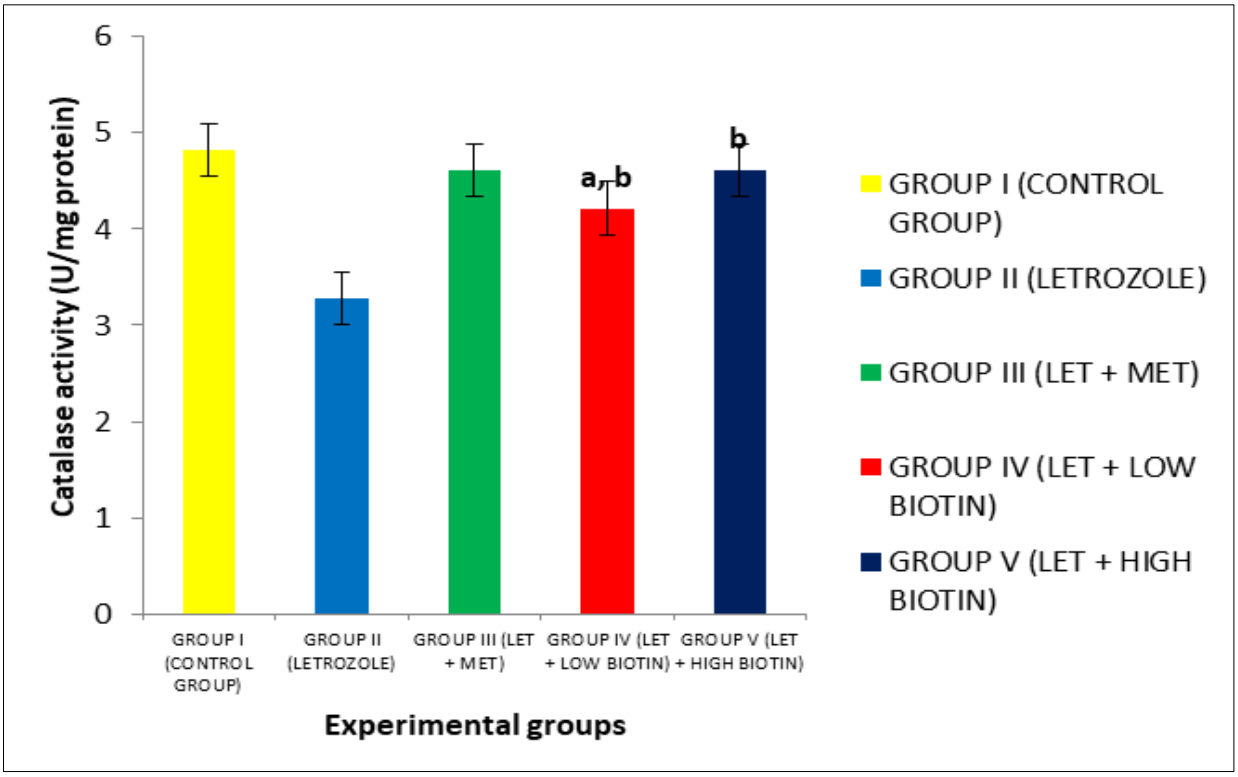

Figure 2: Effect of biotin on the antioxidant system (catalase) in female rats induced with PCOS. [a]p< 0.05- Significant when compared to the control group, [b] p< 0.05- Significant when compared to the Letrozole group.

Figure 2 shows that letrozole induction significantly reduced catalase activity in female rats compared with the control group, indicating impaired antioxidant defence in the PCOS model. The control group exhibited the highest catalase activity (approximately 4.8 U/mg protein), whereas the letrozole group showed a marked decline (approximately 3.3 U/mg protein). Treatment with metformin (LET + MET) restored catalase activity to near control levels, demonstrating a significant improvement relative to the letrozole group. Low-dose biotin (LET + LOW BIOTIN) produced a moderate increase in catalase activity compared with the letrozole group, though values remained significantly lower than those in the control group ($p < 0.05$). High-dose biotin (LET + HIGH BIOTIN) further enhanced catalase activity, with a significant increase compared to the letrozole group ($p < 0.05$) and values approaching those of the control group. Overall, biotin supplementation improved catalase activity in a dose-dependent manner.

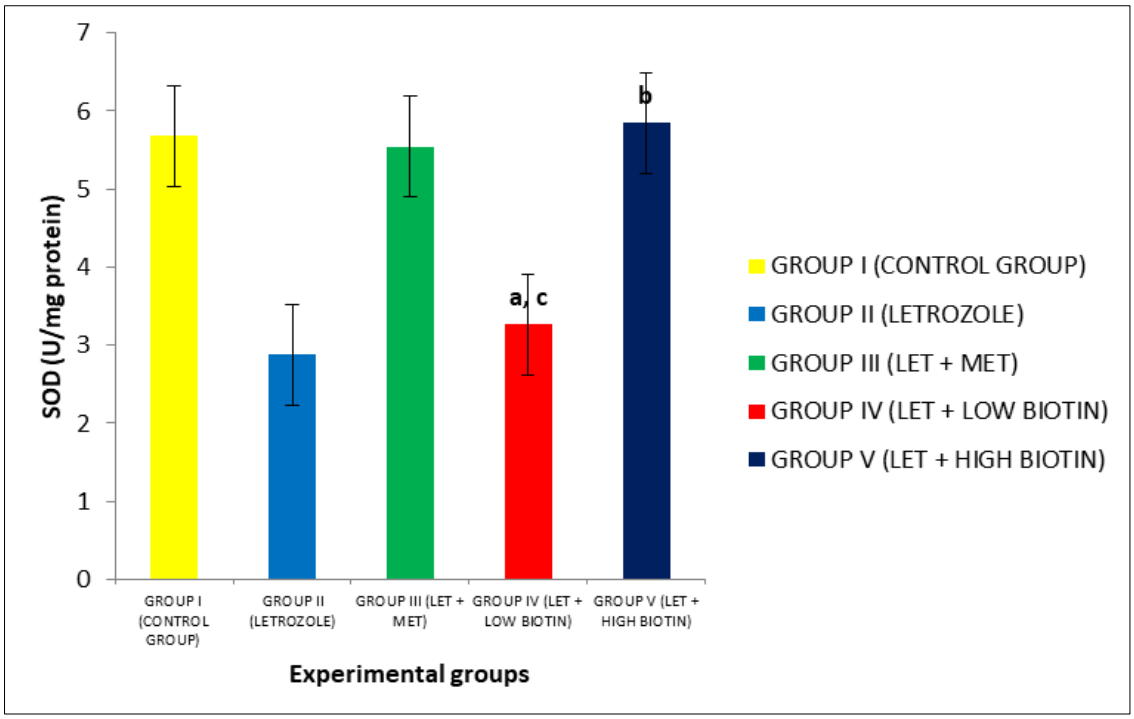

Figure 3: Effect of biotin on the antioxidant system (SOD) in female rats induced with PCOS. [a] p< 0.05- Significant when compared to the control group; [b]p< 0.05- Significant when compared to Letrozole group; [c] p< 0.05- Significant when compared to LET + MET group

Figure 3 shows that letrozole administration significantly reduced superoxide dismutase (SOD) activity in female rats compared with the control group, reflecting heightened oxidative stress in the PCOS model. The control group exhibited high SOD activity (approximately 5.7 U/mg protein), whereas the letrozole group demonstrated a marked decline (approximately 2.9 U/mg protein). Treatment with metformin (LET + MET) restored SOD activity to levels comparable to those of the control group, indicating effective attenuation of oxidative damage. Low-dose biotin (LET + LOW BIOTIN) resulted in only partial improvement, with SOD levels remaining significantly lower than both the control and LET + MET groups ($p < 0.05$). In contrast, high-dose biotin (LET + HIGH BIOTIN) significantly increased SOD activity compared with the letrozole group ($p < 0.05$), achieving values close to normal. These findings suggest that biotin enhances antioxidant defence in a dose-dependent manner.

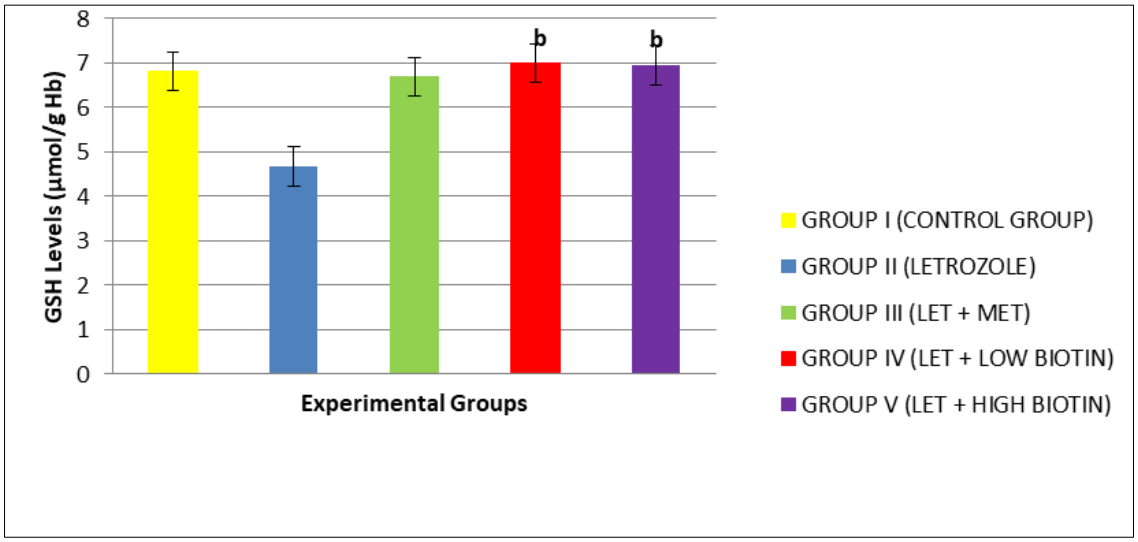

Figure 4: Effect of biotin on the antioxidant system (GSH) in female rats induced with PCOS. [b] p< 0.05-Significant when compared to the Letrozole group. Biotin-treated groups did not differ significantly from the control and LET + MET groups

Figure 4 shows that letrozole administration markedly reduced glutathione (GSH) levels in female rats compared with the control group, indicating depletion of endogenous antioxidant reserves in the PCOS model. The control group maintained higher GSH levels (approximately 6.8 µmol/g Hb), whereas the letrozole group demonstrated a clear decline (approximately 4.7 µmol/g Hb). Treatment with metformin (LET + MET) restored GSH levels close to those of the control group, suggesting effective attenuation of oxidative stress. Both low-dose and high-dose biotin supplementation significantly increased GSH levels compared with the letrozole group ($p < 0.05$). Importantly, the biotin-treated groups did not differ significantly from the control or LET + MET groups, indicating substantial restoration of antioxidant capacity. High-dose biotin produced slightly higher GSH levels than the low-dose group, though both doses were effective in reversing letrozole-induced GSH depletion. Overall, biotin improved GSH status in PCOS-induced rats.

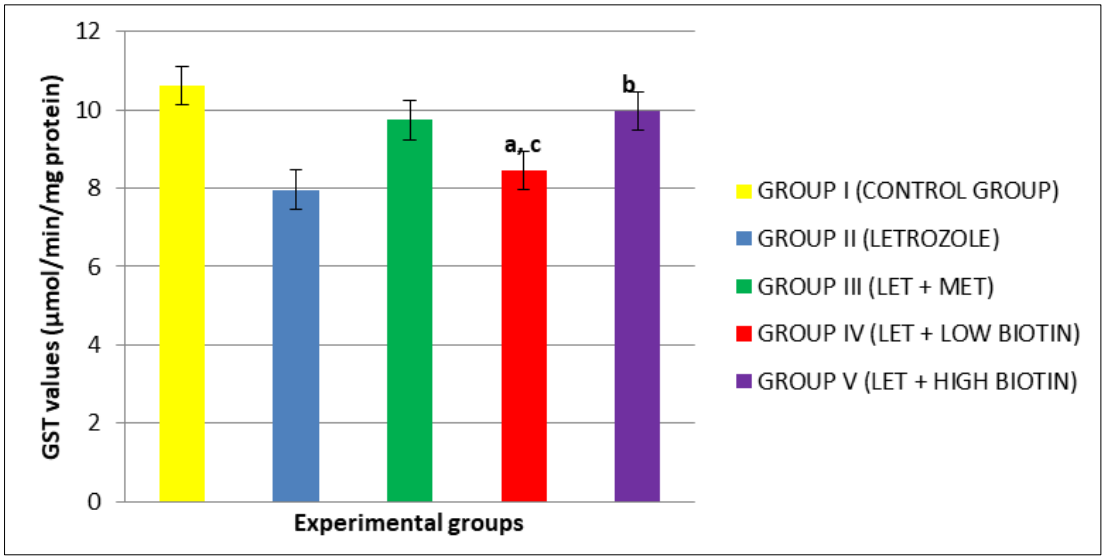

Figure 5: Effect of biotin on the antioxidant system (GST) in female rats induced with PCOS. [a] p< 0.05- Significant when compared to the control group; [b] p< 0.05- Significant when compared to Letrozole group; [c] p< 0.05- Significant when compared to LET + MET group

Figure 5 shows that letrozole administration significantly reduced glutathione S-transferase (GST) activity in female rats compared with the control group, indicating compromised detoxification and antioxidant defence in the PCOS model. The control group exhibited the highest GST activity (approximately 10.6 µmol/min/mg protein), whereas the letrozole group showed a marked decrease (approximately 7.9 µmol/min/mg protein). Treatment with metformin (LET + MET) improved GST levels towards normal values. Low-dose biotin (LET + LOW BIOTIN) resulted in partial restoration; however, GST activity remained significantly lower than the control and LET + MET groups ($p < 0.05$), despite improvement relative to letrozole. In contrast, high-dose biotin (LET + HIGH BIOTIN) significantly increased GST activity compared with the letrozole group ($p < 0.05$), producing values close to control levels. These findings indicate that biotin supplementation enhances GST activity in a dose-dependent manner in letrozole-induced PCOS rats.

Table 2: Effects of biotin supplementation on Biochemical changes (TC, TAG, HDL, LDL)

Group	Biochemical changes			
	TC	TAG	HDL	LDL
Group I (Control)	209.99 ± 3.53	182.91 ± 1.08	159.81 ± 0.87	18.92 ± 0.61
Group II (Letrozole)	241.50 ± 6.84	227.01 ± 7.50	144.99 ± 1.92	47.00 ± 1.72
Group III (LET + MET)	213.70 ± 1.81	195.42 ± 2.59	151.77 ± 1.09	19.09 ± 0.51
Group IV (LET + LOW BIOTIN)	233.71 ± 2.69[a, c]	210.89 ± 4.70	153.38 ± 1.53[b]	37.01 ± 1.44[a, b, c]
Group V (LET + HIGH BIOTIN)	231.30 ± 3.82[a]	146.84 ± 45.54	155.59 ± 1.74[b]	27.89 ± 1.00 [a, b, c]

Abbreviation: TC (Total cholesterol), TAG (Triacylglycerides), HDL (High density lipoprotein), LDL (low density lipoproteins). Values are expressed as mean ± SEM; n= 4. Alphabets a, b and c denote statistically significant variables in the biotin-treated groups compared with the control group, the letrozole group, and the LET + MET group, respectively. [a] p< 0.05 Significant- when compared to the control group, [b] p< 0.05-Significant when compared to the Letrozole group, [c] p< 0.05- Significant when compared to the LET + MET group.

Table 2 shows that letrozole induction disrupted lipid homeostasis in female rats, evidenced by elevations in total cholesterol (TC), triacylglycerides (TAG), and LDL, alongside a reduction in HDL compared with the control group. The letrozole group recorded higher TC (241.50 ± 6.84), TAG (227.01 ± 7.50), and LDL (47.00 ± 1.72), with lower HDL (144.99 ± 1.92), indicating an atherogenic lipid profile. Metformin co-treatment (LET + MET) improved these indices, yielding values close to those of the control group, particularly for TC (213.70 ± 1.81) and LDL (19.09 ± 0.51). Biotin supplementation partially corrected dyslipidaemia. Low-dose biotin significantly reduced HDL impairment and lowered LDL relative to letrozole ($p < 0.05$), although TC and LDL remained significantly higher than control and LET + MET ($p < 0.05$). High-dose biotin showed greater overall improvement, with significant reductions in LDL (27.89 ± 1.00) and higher HDL levels compared with letrozole ($p < 0.05$), suggesting a dose-dependent benefit.

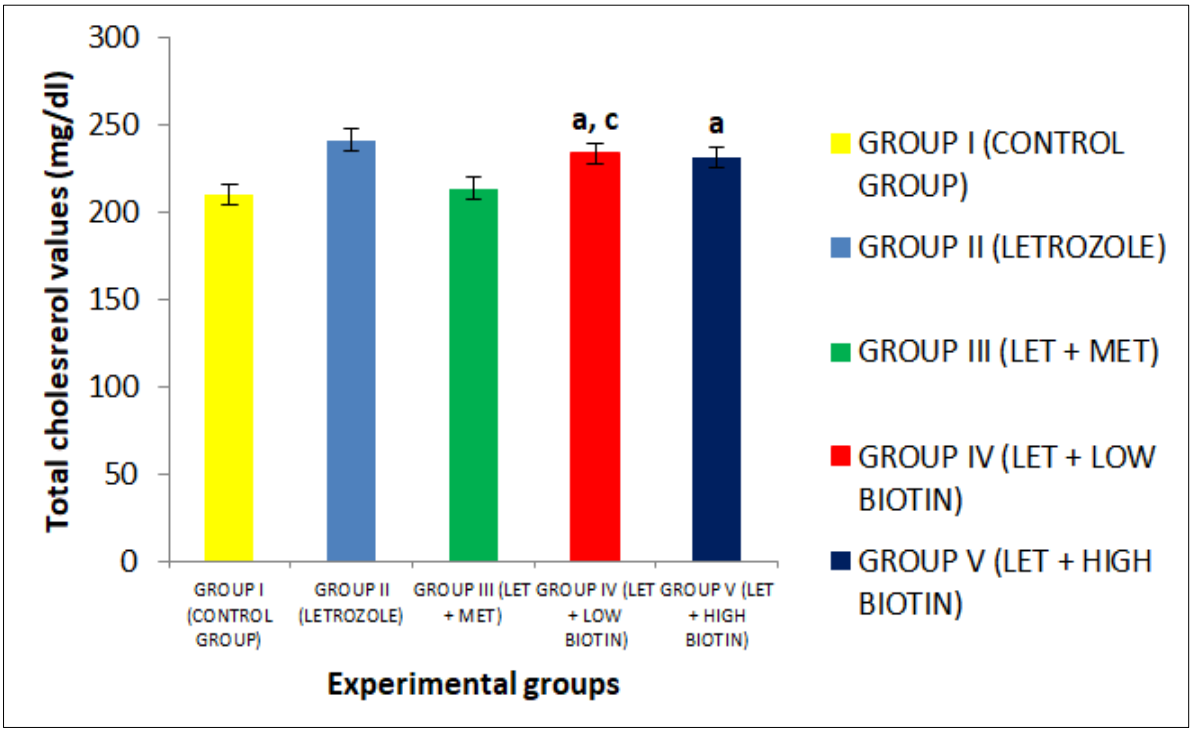

Figure 6: Effects of biotin supplementation on Biochemical changes (TC). [a] p< 0.05- Significant when compared to the control group; [c] p< 0.05- Significant when compared to LET + MET group.

Figure 6 shows that letrozole administration significantly increased total cholesterol (TC) levels in female rats compared with the control group, confirming the induction of dyslipidaemia in the PCOS model. The control group recorded lower TC values (approximately 210 mg/dl), whereas the letrozole group exhibited a marked elevation (approximately 242 mg/dl). Treatment with metformin (LET + MET) effectively reduced TC levels towards normal, with values closely approximating those of the control group. In contrast, both low- and high-dose biotin supplementation resulted in TC levels that remained significantly higher than those in the control group ($p < 0.05$). Additionally, the low-dose biotin group showed TC values that were significantly higher than those of the LET + MET group ($p < 0.05$). Although high-dose biotin produced a slight reduction compared with low-dose biotin, cholesterol levels were not fully restored to control values. Overall, biotin supplementation demonstrated a limited corrective effect on total cholesterol compared with metformin treatment.

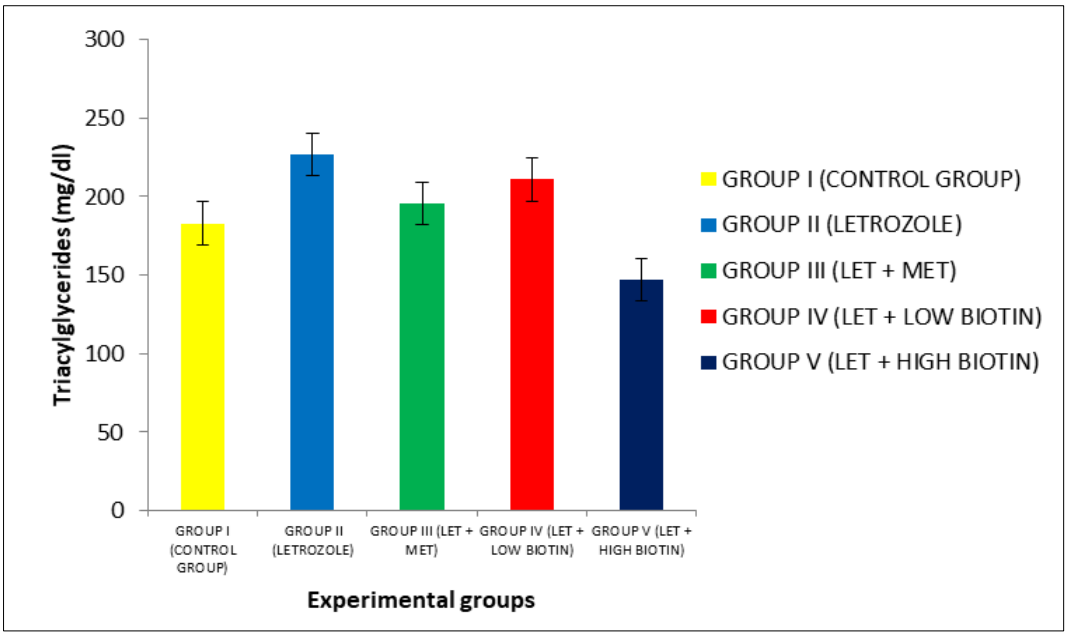

Figure 7: Effects of biotin supplementation on Biochemical changes (TAG). No significant difference exists between the biotin-treated groups compared to the control group, the letrozole group, and the LET + MET group.

Figure 7 shows that letrozole administration increased triacylglyceride (TAG) levels in female rats compared with the control group, indicating altered lipid metabolism in the PCOS model. The control group recorded TAG values of approximately 183 mg/dl, whereas the letrozole group exhibited higher levels (approximately 227 mg/dl). Treatment with metformin (LET + MET) reduced TAG concentrations towards control values, demonstrating partial improvement. Low-dose biotin supplementation maintained elevated TAG levels relative to the control and LET + MET groups. In contrast, high-dose biotin showed a noticeable reduction, with values lower than those of the letrozole and control groups. However, statistical analysis showed no significant difference between the biotin-treated groups and the control, letrozole, or LET + MET groups. Overall, although numerical variations were observed across groups, biotin supplementation did not produce statistically significant changes in TAG levels in this study.

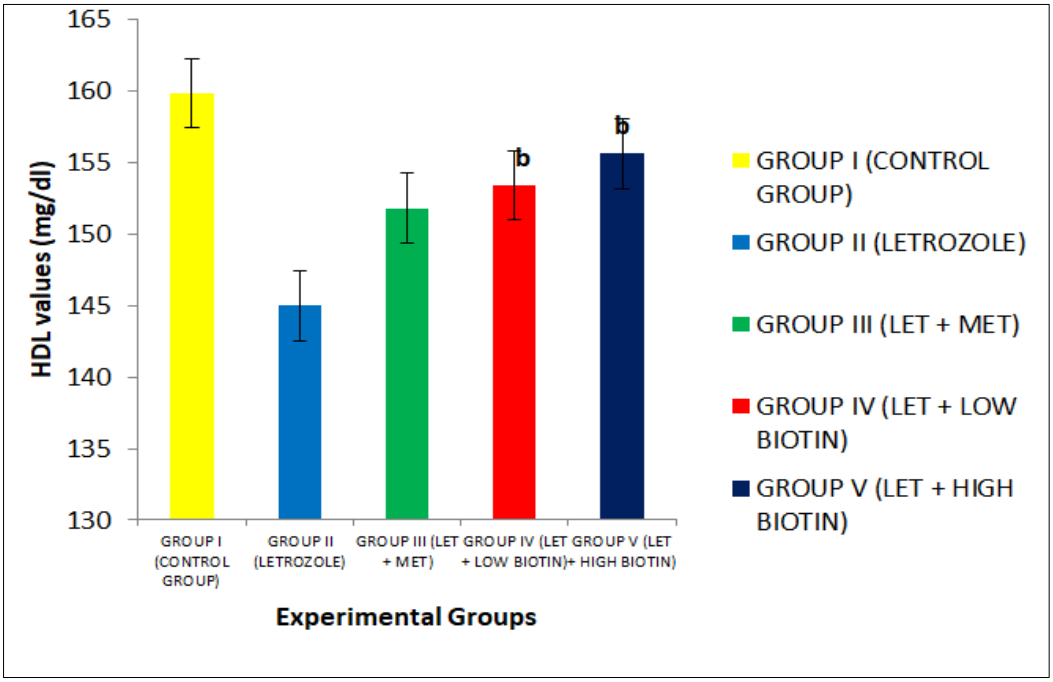

Figure 8: Effects of biotin supplementation on Biochemical changes (HDL). [b] p< 0.05- Significant when compared to Letrozole group, [c] p< 0.05 Significant when compared to LET + MET group. No significant difference exists between the biotin-treated groups and the control and LET + MET groups.

Figure 8 shows that letrozole administration reduced high-density lipoprotein (HDL) levels in female rats compared with the control group, indicating deterioration in protective lipid fractions in the PCOS model. The control group recorded the highest HDL levels (approximately 160 mg/dL), whereas the letrozole group showed a marked decline (approximately 145 mg/dL). Treatment with metformin (LET + MET) improved HDL levels towards normal. Both low-dose and high-dose biotin supplementation significantly increased HDL levels compared with the letrozole group ($p < 0.05$), demonstrating improvement in lipid profile. Furthermore, no significant difference was observed between the biotin-treated groups and the control or LET + MET groups, indicating substantial restoration of HDL concentrations. High-dose biotin produced slightly higher HDL values than low-dose biotin, suggesting a mild dose-related effect. Overall, biotin supplementation effectively counteracted letrozole-induced reduction in HDL levels in this study.

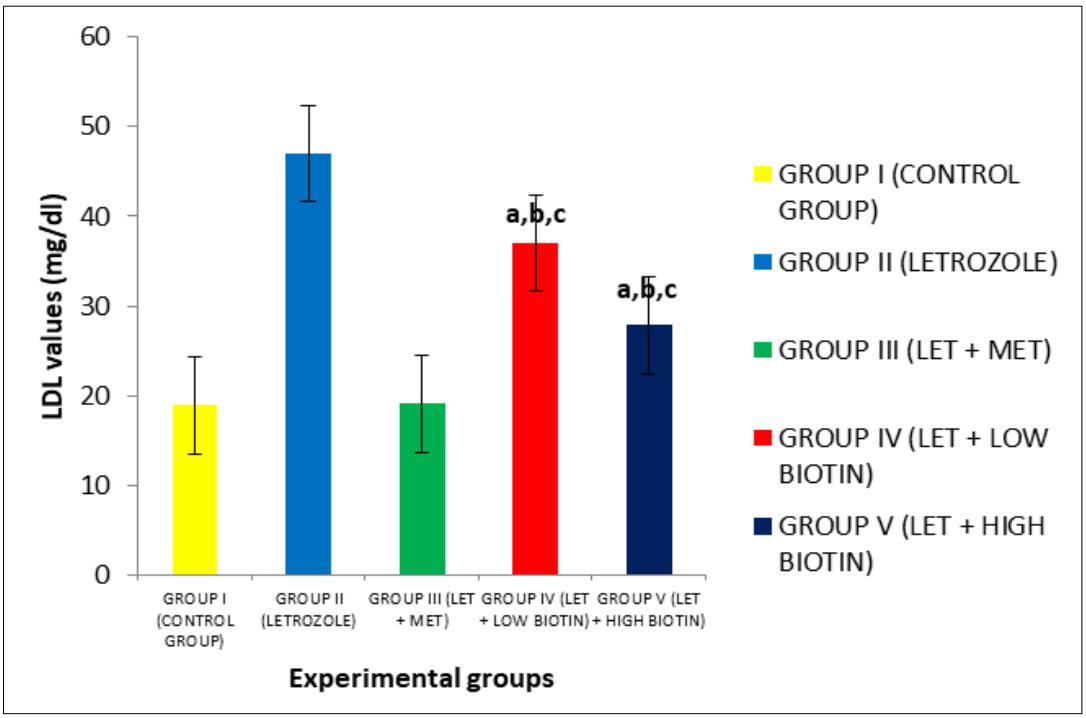

Figure 9: Effects of biotin supplementation on Biochemical changes (LDL). [a] p< 0.05. Significant when compared to the control group; [b] p< 0.05- Significant when compared to the Letrozole group; [c] p< 0.05- Significant when compared to LET + MET group.

Figure 9 shows that letrozole administration markedly increased low-density lipoprotein (LDL) levels in female rats compared with the control group, confirming the induction of an atherogenic lipid profile in the PCOS model. The control group recorded low LDL values (approximately 19 mg/dl), whereas the letrozole group exhibited a pronounced elevation (approximately 47 mg/dl). Treatment with metformin (LET + MET) effectively reduced LDL levels to levels comparable to those of the control group. Low-dose biotin supplementation significantly reduced LDL levels compared with the letrozole group ($p < 0.05$); however, levels remained significantly higher than those in both the control and LET + MET groups ($p < 0.05$). High-dose biotin produced a greater reduction in LDL than the low-dose group and was significantly different from control, letrozole, and LET + MET groups ($p < 0.05$). Overall, biotin demonstrated a dose-dependent improvement in LDL levels, though metformin showed superior normalisation.

4. DISCUSSION

This study has shown that induction of PCOS using letrozole in female Wistar rats results in metabolic complications and a reduced antioxidant defence capacity. Indeed, these rats exhibit elevated blood glucose, reduced antioxidant enzyme activity, and a lipid profile that predisposes to atherosclerosis. In total, biotin helps alleviate some problems, especially at higher doses, although the best results are obtained with metformin.

Also noteworthy was the dramatic increase in blood glucose levels that was observed in the letrozole-only group. Letrozole throws the hormonal levels out of balance by raising the levels of androgens. As a result, insulin is no longer functioning properly, and glucose levels will worsen. Hence, hyperglycemia results from problems with insulin and glucose uptake. As expected, blood glucose levels decreased in the metformin-treated groups compared with the PCOS group. Metformin is an insulin-sensitising agent that increases insulin-stimulated glucose uptake in peripheral tissues. Biotin

supplementation at low doses did not have an appreciable effect on glucose levels compared with the PCOS group; however, a high dose reduced glucose levels.

In terms of antioxidants, oxidative stress is strongly implicated as a key finding in this letrozole-induced PCOS animal study, as reflected by lower levels of catalase, superoxide dismutase, reduced glutathione, and glutathione S-transferase in the PCOS group compared to their control peers. Oxidative stress, as a condition, is characterised by an imbalance between ROS production and antioxidant defences, resulting in disruption of normal cellular function. Increased oxidative stress in PCOS is strongly associated with insulin resistance, non-resolving inflammation, and worsened ovulatory function, partly due to tissue damage. The decline in antioxidant levels in the PCOS group is consistent with oxidative stress as a key finding in PCOS for letrozole.

Biotin appears to support antioxidant defences, with the effect increasing with higher doses, as indicated by antioxidant measurement outcomes among biotin participants. Although it is not yet understood exactly how it works, the pattern suggests that biotin may actually enhance the body's natural antioxidant capacity and/or reduce stress to a point where antioxidant systems can recover. But what's even more surprising is that the antioxidant system seems to recover more in the high-dose group than in the low-dose group. This actually makes sense, since antioxidant defences do not rely solely on one mechanism; it's about enzymes, substrate availability, and redox cycling. Therefore, how much it recovers depends on how many of the antioxidant systems it interacts with.

These results are consistent with the broader body of evidence indicating the role of micronutrient status in regulating oxidative stress in PCOS. Szczuko *et al*., (2020), in their review, highlighted the role of water-soluble vitamins in the treatment of women with PCOS because of their inability to be stored in the body. They highlighted the role of these vitamins in treating PCOS by mitigating oxidative stress and inflammation. They suggested that the antioxidant potential may be decreased in women with PCOS due to their current state of water-soluble vitamins. The positive effects of antioxidants from biotin supplementation also make sense from a general standpoint.

Aside from the oxidative glitches, letrozole-induced PCOS presented notable dyslipidaemia. Indeed, the PCOS group showed elevated levels of cholesterol, triglycerides, and LDL, and lower levels of HDL, compared to the control group. Metformin treatment increased these lipid levels to near-normal levels, which may be related to its possible beneficial effects on lipid metabolism and stability. In contrast, when biotin was administered at high doses, the levels of triglycerides and cholesterol did not change significantly. Still, HDL was increased, and LDL was decreased significantly compared with the PCOS group, suggesting a possible selective beneficial effect of biotin on lipid metabolism, favouring increases in HDL and LDL but failing to affect overall lipid levels.

The specific action regarding increased HDL and decreased LDL cholesterol may be an indirect effect of metabolic benefits mediated by enhanced redox status and corrected insulin resistance. Since oxidative stress can disrupt lipid metabolism by altering hepatic fat metabolism, increasing lipid peroxidation, and activating inflammatory pathways that affect lipid production and clearance, improving antioxidant capacity could improve lipid profiles even if key parameters remain unresponsive. The lack of significant changes in triglyceride and total cholesterol levels, despite clear action on HDL and LDL cholesterol, indicates that a single action cannot fix lipid metabolism; instead, it is a multi-tiered system.

These observations are consistent with clinical studies that suggest the use of a combination of non-hormonal therapeutic approaches for the treatment of PCOS cases. Advances in the treatment of PCOS have been reported by researchers who believe that insulin sensitisers, antioxidants, vitamins, and biotin are safe and effective for both obese and non-obese patients with PCOS (Advani *et al*., 2019). Similarly, researchers Singh *et al*., (2021) are of the view that the combination of inositols, antioxidants, and vitamins for the treatment of PCOS is effective and safe. It is important to note that although the current study is based on an animal model, the improvements achieved with biotin support the use of vitamins for the treatment of PCOS.

Some key findings from the study include that metformin provided more complete and stable normalisation of hyperglycemia compared with biotin treatment. In contrast, high-dose biotin provided more noticeable benefits in restoring antioxidant capacity and improving the lipoprotein profile. This may imply that metformin has a direct effect on glucose metabolism in insulin resistance, whereas biotin's action may be mediated by decreased oxidative stress and subsequent metabolic consequences. It is noteworthy that the results obtained with the low-dose biotin treatment were moderate across the various parameters. This may imply that insufficient vitamin levels may not provide therapeutic benefits and may lead to inconsistent results. This shows the need for careful optimisation of the doses in the study of vitamin deficiencies in experimental PCOS.

Despite the positive aspects of the study, it is necessary to consider the following limitations that may have been encountered in the study: the mechanisms of action of biotin, which may have been at work in the study, were not identified, and no further investigation of the pathways that may have been at work in the

restoration of antioxidant capacity and the changes in the lipoprotein profile was done. The study may have been restricted to biochemical parameters alone, without histopathology of the tissues, which could have provided direct interpretations of the results. The supplementation period may have been too short to allow full expression of effects on triglycerides and total cholesterol levels, as the changes in these parameters may have been slow or required more widespread metabolic remodelling. The combined use of vitamins, antioxidants, and insulin sensitisers, which may have been the case in studies of vitamin deficiencies in experimental PCOS, may have been neglected in the study. Such combinations have been used in previous studies of vitamin deficiencies in experimental PCOS (Advani *et al*., 2019; Singh *et al*., 2021).

Future research can address these gaps by conducting histologic and molecular analyses of the target tissues. It would also be useful for future research to focus on the varying doses administered to ascertain the exact amount needed by the body and to determine whether the benefits of biotin reach a point beyond which they do not increase further. It may also take longer for biotin to affect overall cholesterol and triglycerides in the body. Moreover, animal studies can be conducted to verify the additive effects of the combination of biotin and other drugs, with its benefits, which would align with the new therapeutic direction observed in human studies (Szczuko *et al*., 2020; Advani *et al.,* 2019; Singh *et al*., 2021).

In conclusion, the induction of PCOS with letrozole resulted in hyperglycemia, oxidative stress, and changes in lipid profiles in female rats. Metformin effectively alleviated hyperglycemia, while a high dose of biotin significantly improved glucose metabolism and corrected disturbances in antioxidant and lipid profiles. Therefore, the potential role of biotin in the management of PCOS appears to be clear, especially at high doses. However, the mechanisms and the optimal doses need to be clearly elucidated in further investigations.

5. CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

This study was carried out to determine the potential of biotin to improve metabolic and biochemical parameters in PCOS-induced female Wistar rats. The results obtained showed that biotin supplementation may be a potential therapeutic option for improving metabolic and biochemical function in PCOS.

5.2 Recommendations

For Researchers and Academic Institutions

Researchers should conduct further investigations to determine the most effective dose of biotin in the treatment of PCOS, as the current study evaluated biotin's effects but did not establish an optimum dose. This will yield better outcomes in clarifying dosing requirements for improved metabolic and biochemical function in PCOS.

Researchers should also conduct studies to identify the biotin constituents responsible for its effects on PCOS, as the present findings indicate potential benefits without specifying the active contributors. This will yield a better understanding of the basis of biotin's observed effects.

For Policy Makers and Funding Bodies

Grants should be provided to support additional research on the effect of biotin in PCOS. This will yield better outcomes by expanding the evidence on biotin's potential therapeutic value and by strengthening the research base for future applications.

For Healthcare and Patient Education

Consuming biotin may be encouraged in PCOS patients based on the observed potential benefits for metabolic and biochemical function. This may yield better outcomes in supporting PCOS management through supplementation.

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