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Fertility challenges: Interplay between visfatin and silent information regulator 1 (SIRT1); A cross-sectional study

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Abstract: **Background**: Maintenance of a normal redox balance is essential for optimal ovarian function and fertility. Reduction in silent information regulator1 (SIRT1) can potentiate inflammatory pathways, mitochondrial dysfunction and ultimately programmed cell death via "hypothalamic-pituitary-ovarian axis", developing 'low-grade oocytes', potentiating obesity related problems. Current hypothesis that a reduction in antioxidant levels (measured by SIRT1, visfatin, Manganese Superoxide dismutase (MnSOD), and Glutathione reductase (GR) with increased BMI is associated with decreased fertility. **Objectives**: The study aimed to evaluate the relationship between obesity and OS markers and to compare their levels between fertile and infertile women, thereby elucidating the potential role of redox imbalance in obesity-associated subfertility. **Methods**: A cross-sectional study comprised of 207 fertile and 135 infertile females were recruited. Body mass index (BMI) determines obesity with the formula: weight in kilograms (kg)/height in meters (m^2). Women's stratification was according to the South-Asian criteria (normal=18-22; overweight =23-24; obese >25 kg/m^2). The enzyme-linked immunosorbent assay (ELISA) kits measured serum levels of MnSOD, GR, visfatin and SIRT1. Pearson chi-square and ANOVA were used; study variable association was calculated by Spearman's correlation, where $p<0.05$ was significant. **Results**: The mean BMI was 25.94 ± 5.1 kg/m^2; normal weight females were 27.5%, overweight 22.8% and obese 40.9% and infertile females had significantly increased BMI ($p<0.001$). The antioxidant levels of MnSOD, GR, SIRT1 and visfatin were significantly lower in infertile females (p value =0.049, 0.027, 0.027 and 0.034, respectively). Infertile women had decreased MnSOD and visfatin, decreasing from normal weight to obese (p=0.00 and 0.04). BMI was inversely related to SIRT1 and studied antioxidant concentrations. **Conclusion**: Continuous reduction in antioxidant levels in association with increased BMI indicates that obesity contributes to oxidative stress attributed by reducing antioxidant levels, which may lead to female infertility.

Keywords: Antioxidants; Body mass index; Female infertility; Obesity; Oxidative stress; SIRT1; Visfatin

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INTRODUCTION

Obesity poses a significant threat to ovarian functions and female fecundity (Zhang *et al*., 2025). Maintenance of a normal redox balance is essential for optimal ovarian function and fertility. Advanced maternal age, obesity and unexplained infertility have been strongly associated with an imbalance between oxidant production and antioxidant defense mechanisms (Aitken *et al*., 2022). Hence, oxidative stress (OS) disrupts hormonal homeostasis, resulting in impaired oocyte maturation, reduced fertilization potential, decreased embryonic cleavage rates and an increased risk of pregnancy loss (Agarwal *et al.*, 2012: Wang *et al*., 2021).

Sirtuins (SIRTs) (NAD-dependent deacylase), SIRT1 is known to catalyze the reaction between nicotinamide and the substrate's acetyl group, producing metabolite O-acetyl adenosine diphosphate ribose, which aids ovarian cells' growth. Sirtuins are closely associated with ageing, OS, metabolic homeostasis, attenuation of inflammation, DNA repair and mitochondrial function through the regulation of specific gene pathways. Additionally, sirtuins play a critical role in the regulation of programmed cell death in granulosa cells, contribute to follicular preservation and extend ovarian lifespan (Alam *et al*., 2021). The literature suggests that, in response to OS induced by endocrine dysfunction, SIRT1 expression may be upregulated as a compensatory mechanism to offset the decline in SIRT1 activity (Li *et al*., 2025). Therefore, SIRT1 deficiency activates inflammatory pathways, induces mitochondrial dysfunction and promotes programmed cell death through dysregulation of the hypothalamic–pituitary–ovarian (HPO) axis, leading to poor-quality oocyte development, subfertility and the progression of obesity-associated metabolic disorders (Alam *et al*., 2023).

Visfatin, also known as nicotinamide phosphoribosyl transferase (NAMPT), is an intracellular enzyme that plays a pivotal role in the NAD^+ salvage pathway.

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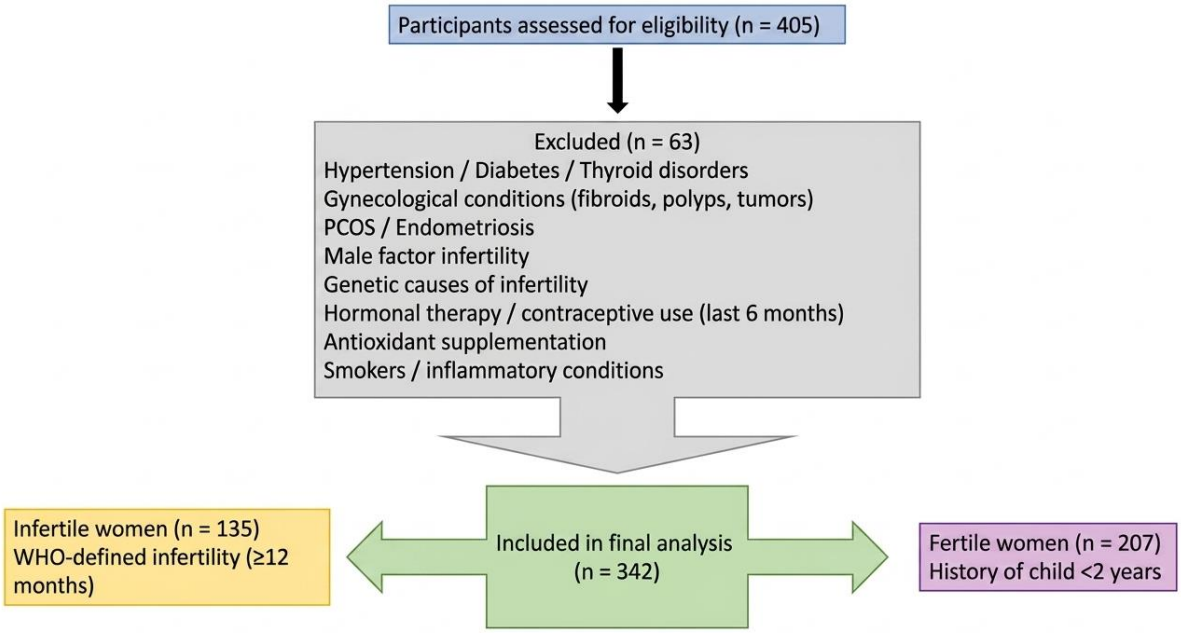

Fig. 1: Flow diagram of participant recruitment and selection.

By regulating NAD^+ availability, visfatin influences the activity of NAD^+-dependent enzymes essential for cellular survival, including SIRT1, a key negative regulator of proinflammatory cytokine production. Dysregulated visfatin expression is inversely associated with antioxidant levels and disrupts glucose metabolism, thereby contributing to OS, obesity and related metabolic disturbances that exacerbate SIRT1-mediated reproductive dysfunction (Koutsoura *et al*., 2025).

Given the recognized interplay between obesity, OS and reproductive dysfunction, this study was designed to investigate the association between increased body mass index (BMI) and alterations in key antioxidant and oxidative stress–related markers, including SIRT1, visfatin, manganese superoxide dismutase (MnSOD) and glutathione reductase (GR). The current study hypothesized that obesity-related reductions in these antioxidant defenses contribute to impaired fertility in women. Accordingly, the present study aimed to evaluate the relationship between obesity and OS markers and to compare their levels between fertile and infertile women, thereby elucidating the potential role of redox imbalance in obesity-associated subfertility.

MATERIALS AND METHODS

Study design & setting

The cross-sectional study was conducted from March 2017 to June 2018 after approval from institutional ethical review board of Aga Khan University (ERC#4426-BBS-ERC-). The convenient random sampling technique was employed to recruit subjects of all ethnic groups from the Australian Concept Infertility Medical Centre (ACIMC), Karachi, as a collaborative project with Aga Khan Hospital University, Karachi.

Study size

All subjects (207 fertile and 135 infertile) were informed and signed written consent. All experiments were conducted according to the ethical standards of Helsinki. The sample size was computed to be 334 to achieve a 97% confidence interval with a 5% confidence limit, using a design effect, assuming a 23% prevalence of infertility.

Participants

Inclusion criteria (fertile subjects): Subjects had a history of a child less than two years of age. *Inclusion criteria (infertile subjects)*: WHO recommendations were followed: unable to reproduce following twelve months of frequent and non-contraceptive interaction, with a normal uterus identified on hysterosalpingogram, or hysteroscopy.

Exclusion criteria (all subjects): Subjects with hypertension, diabetes, thyroid disorders (T3, T4, TSH), gynaecological tumours (fibroids, lumps, polyps, etc.), or serious general health issues or any inflammatory condition were excluded. Moreover, females suffering infertility due to a male partner and any diagnosed genetic causes of infertility, those who had opted for assisted contraception in their previous pregnancies, had pre-eclampsia, ‘gestational diabetes’ recurrent miscarriages, endometriosis, consuming oral contraceptives and antioxidants in last 6 months or utilized any hormonal support, or smokers were also eliminated from the study.

Methods to overcome bias

A total of 405 subjects were initially included in the study. Patients with PCOS and endometriosis were excluded to avoid confounding, as these conditions independently influence oxidative stress markers and fertility outcomes, thereby allowing a more focused assessment of the association between obesity, oxidative stress and infertility. There was no missing data as it was acquired from the hospital records.

Clinical data recording and measurement of body mass index

Clinical data included detailed history (family history, gynecological/obstetric history, eating preferences, etc.) and general physical examination (age, height, weight, blood pressure). Standing body height was assessed by using a floor-type height scale (ZT-120 EVERICH, China; inches converted to meters), while a digital weighing scale measured weight to compute BMI. Employing the 'South Asian criteria of body mass index (BMI)' (normal weight=18.0-22.9 kg/m2; overweight = 23-24.9 kg/m2; obese >25.0 kg/m2) (Muthukrishnan *et al*., 2025)

Sample collection and storage

Blood samples (6 millilitres) were collected from each subject using an appropriate pain-free procedure and assigned a separate ID to be followed throughout the study. Serum was extracted after centrifugation and promptly stored at –80°C until evaluation.

Table 1: List of variables

Category	Variables
Outcome	Fertility status (fertile vs infertile)
Exposure	BMI (continuous + categorical)
Predictors	SIRT1, visfatin, MnSOD, GR
Confounders	Age, lifestyle, reproductive history, comorbidities
Effect Modifiers	BMI category, obesity status
Diagnostic Criteria	WHO infertility definition; fertility = recent childbirth

Variable Estimation of MnSOD, GR, SIRT1 and visfatin levels

Oxidative stress markers (SIRT1, visfatin, MnSOD, and glutathione reductase) were measured using ELISA kits under standardized conditions.. MnSOD (Kit Cat. No. SG-10731 Ltd.); GR (Kit Cat. No SG-00523; SIRT1 (Kit Cat. No. SG-10458) were assessed by manufactured enzyme-linked immunosorbent assay (ELISA) kits by Sino Gene Clone Biotech Co., Ltd), while visfatin enzyme immunoassay was performed by the kits supplied from the same company (Kit Cat. No. 10381).

Statistical analysis

Statistical analyses were performed using SPSS version 19 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD). Normality of data distribution was assessed prior to analysis. Differences among BMI-based groups were analyzed using one-way analysis of variance (ANOVA), followed by Tukey's post hoc test for multiple comparisons. Associations between body mass index (BMI) and serum levels of MnSOD, GR, visfatin and SIRT1 were evaluated using Spearman's rank correlation coefficient due to non-normal data distribution. All tests were two-tailed and a p-value < 0.001 was considered statistically significant.

Quantitative variables were handled using both continuous and categorical approaches. Continuous variables, including BMI and serum levels of SIRT1, visfatin, manganese superoxide dismutase, and glutathione reductase, were expressed as mean ± standard deviation, and normality was assessed prior to analysis. Between-group comparisons were performed using one-way ANOVA with Tukey's post hoc test for multiple comparisons. Due to non-normal distribution of key variables, associations between BMI and oxidative stress markers were evaluated using Spearman's rank correlation coefficient. For analytical purposes, BMI was additionally categorized into normal, overweight, and obese groups using South Asian cut-offs (18.0–22.9, 23.0–24.9, and ≥25.0 kg/m²), allowing stratified analysis to examine trends in oxidative stress markers across adiposity levels. This grouping was chosen as it is more appropriate for the studied population and improves sensitivity in detecting obesity-related risk compared to conventional WHO criteria.

RESULTS

Female subjects had an average age range of 31 ± 6 years and BMI of 25.94 ± 5.1 kg/m^2. The normal weight females were 27.5%, overweight 22.8% and obese 40.9%. Out of 207 fertile females with the same stratification, 72(34.8%) were normal weight and 77 (37.2%) were obese. However, 20 (14.8%) of infertile females were of normal weight compared to 73 (46.7%) females being obese. Fig. 2 shows a stable decreasing trend of MnSOD, SIRT1, visfatin and glutathione reductase as the BMI increases. The most significant low levels are of MnSOD levels in obese females when compared with normal-weight females.

Table 2 represents the comparison of infertile subjects with fertile counterparts. It was observed that infertile women had a significantly higher BMI compared to fertile subjects ($p<0.01$); however, the antioxidants and SIRT1 were lower in infertile females.

Fig. 3 represents the combined effects of inflammation and oxidative stress, which impair reproductive function, contributing to obesity-related infertility. suggests a connection between increased BMI and decreased antioxidant levels.

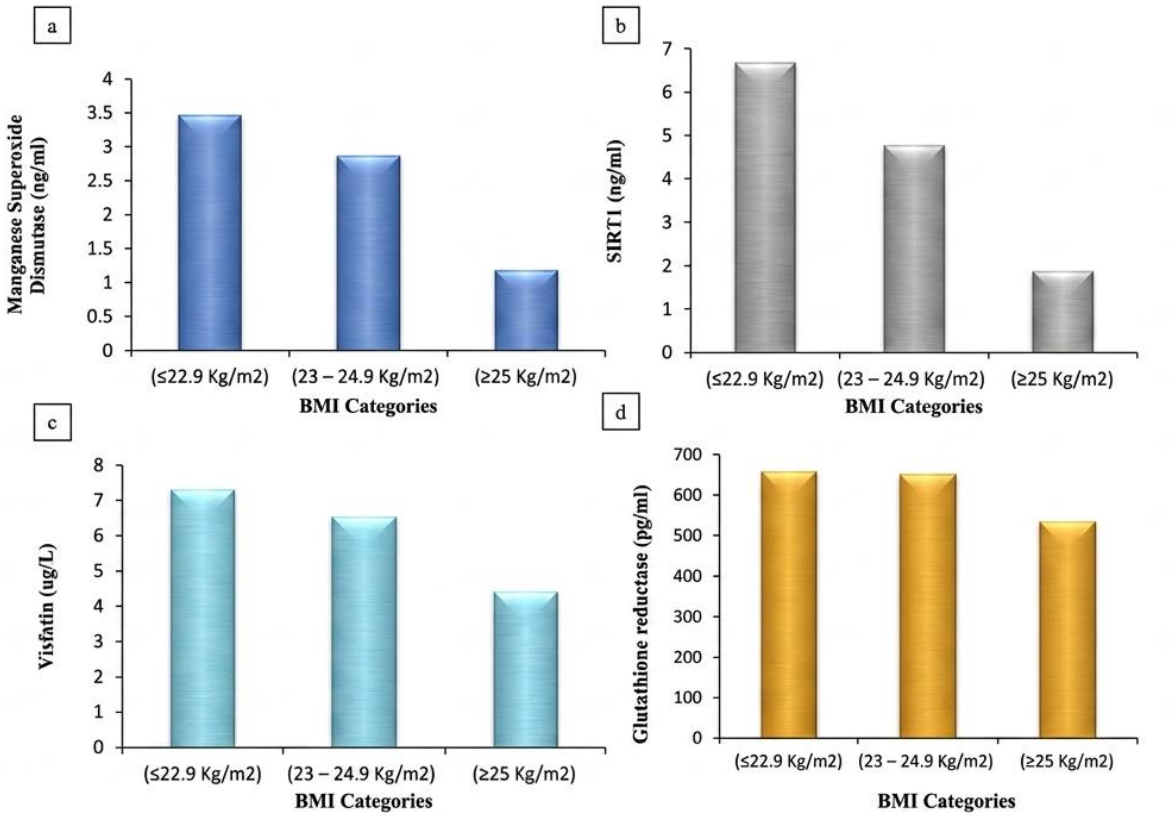

Fig. 2: (a – d): Representation of MnSoD, SIRT1, visfatin and glutathione reductase in different BMI categories

Table 2: Demographic variables and antioxidant levels of the study subjects

Variables	Fertile n=207 Mean ± SD	Infertile n=135 Mean ± SD	p-value
Age (years)	31.22 ± 6.02	31.13 ± 5.7	0.896
BMI (Kg/m^2)	24.48 ± 3.31	27.4 ± 3.69	0.001
Manganese superoxide dismutase (ng/ml)	2.32 ± 1.58	0.89 ± 0.8	0.049
SIRT1 (ng/ml)	5.25 ± 2.83	1.82 ± 1.93	0.027
Visfatin (ug/L)	7.16 ± 2.79	3.97 ± 2.77	0.034
Glutathione reductase (pg/ml)	876.15 ± 339.87	175.71 ± 198.5	0.009

Table 3 represents the frequency of fertile and infertile females based on the BMI classification. It further characterizes the comparison of oxidative stress markers when stratified according to the same BMI classification. On stratifying the OS marker levels according to the BMI groupings, MnSOD, visfatin and GR showed a reverse tendency. The Spearman correlation showed a strong negative relationship of BMI with SIRT 1($r=-0.599$, $p<0.001$).

DISCUSSION

Obesity, a large-scale health problem, exerts a sizable burden on health and the economy (Roh and Kim, 2020). It is a complex multifactorial disease in which the accumulation of excess body fat has adverse health effects, increasing the risk of several problems, including reproductive failure/infertility (Uddandrao *et al*., 2024). In this study, the mean BMI was significantly higher in infertile subjects, and markers such as MnSOD and GR decreased from normal weight to obese categories. This supports that obesity contributes to OS, which may lead to infertility by affecting oocyte quality and metabolic parameters (Koutsoura *et al*., 2025). Another study on infertile females undergoing IVF treatment declared that BMI was associated with elevated OS, supporting its link with obesity and infertility (Pentek *et al*., 2025).

As far as the impact of BMI on reproductive potential is concerned, cut off value of less than 26 kg/m^2 has been documented to improve the pregnancy outcome in our population (Uddandrao *et al*., 2024).

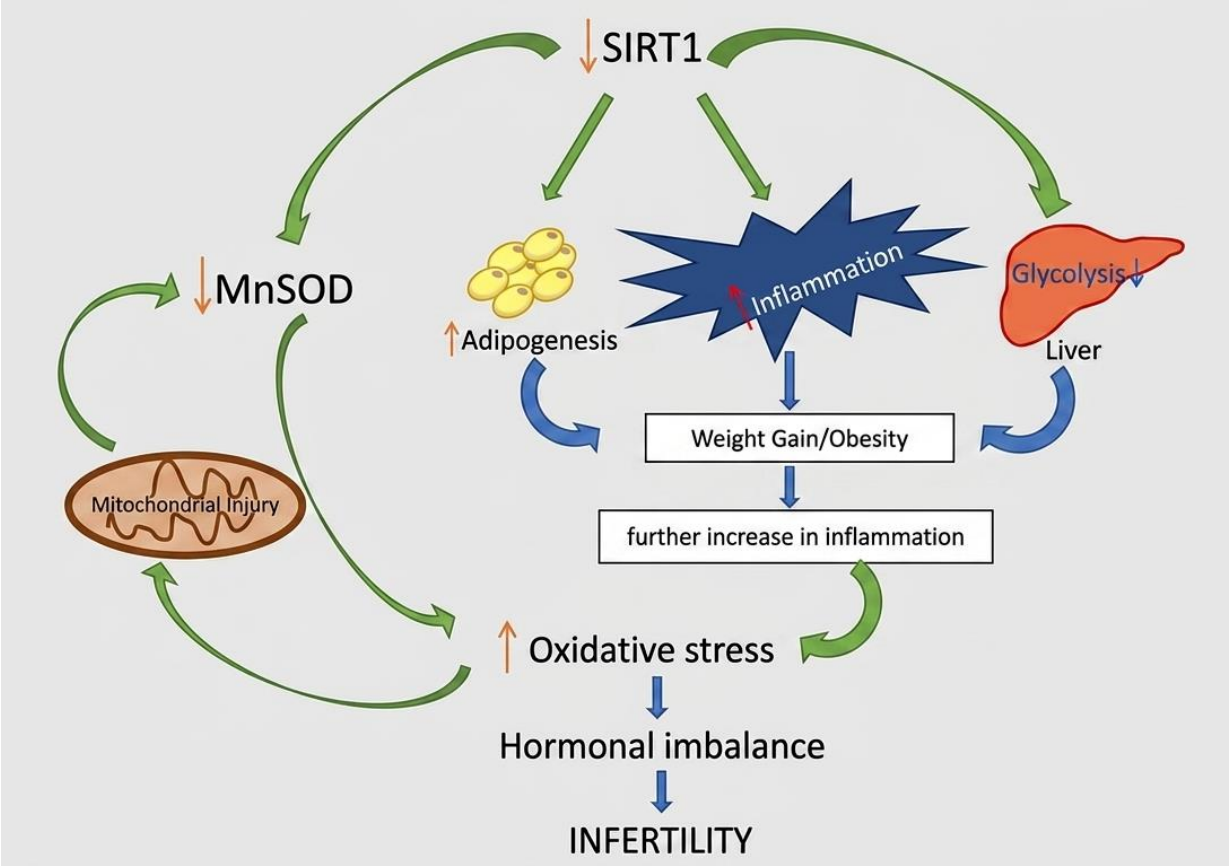

Fig. 3: Hypothetical mechanism of SIRT1-mediated infertility in obesity.

Table 3: Stratification of fertility and oxidative stress markers based on BMI groups:

Variables	BMI <23 Kg/m^2 n=124	BMI<23 – 24.9 Kg/m^2 n= 78	BMI >25 Kg/m^2 n= 140	p-value
	n(%)	n(%)	n(%)	
Fertile (n =207)	72 (34.8%)	58 (28%)	77(37.2%)	*0.015*
Infertile (n = 135)	52 (38.5%)	20 (14.8%)	63(46.7%)	
	Mean ± SD	Mean ± SD	Mean ± SD	p-value
Manganese SOD (ng/ml)	1.651 ± 1.531	2.252 ± 1.532	1.583 ± 1.41	0.00
SIRT1 (ng/ml)	3.997 ± 3.271	4.157 ± 2.723	3.683 ± 2.966	0.48
Visfatin (ug/L)	6.03 ± 3.519	6.568 ± 2.769	5.464 ± 3.079	0.04
Glutathione reductase (pg/ml)	654.875 ± 481.155	649.579 ± 408.748	531.741 ± 440.449	0.05

The impact of high BMI on reproductive failure can be attributed to enhanced production of stress hormones and decreased antioxidant activity (Aitken *et al*., 2019). This study is unique in the sense that BMI is categorized on South Asian criteria (Normal = 18–22.9 kg/m²) (Muthukrishnan *et al*., 2025) rather than the standard WHO criteria, which is not applicable for local population.

The female reproductive system is susceptible to OS, which can interfere with ovulation, menstrual cycles, egg quality and tubal function, ultimately leading to infertility. (Rehman *et al*., 2024; Daraghmeh *et al*., 2025) The high levels of OS markers thus can impair oocyte quality and reproductive potential in women of the reproductive age group (Alam *et al*., 2023). Fertile subjects had higher levels of Manganese SOD, SIRT1 and visfatin compared to infertile subjects. The low levels of antioxidants in infertile female subjects support the fact that an imbalance between reactive oxygen species (ROS) production and the body's antioxidant defense mechanisms can contribute towards reproductive failure (Xia *et al*., 2025). Increased OS and reduced antioxidant defence mechanisms have been shown to play a significant role in the pathophysiology of PCOS (Uckan *et al*., 2022). SIRT1 activates the defensive-signal

cascade as the frontline defence against ROS in human reproductive organs; downregulation of SIRT1 causes an increase in the OS, inflammation and metabolic dysregulation via the SIRT-associated pathway (Alam *et al*., 2021; Walter *et al*., 2025). The presence of SIRT1 genetic variants (rs10509291 and rs12778366) appears to alter SIRT1 expression, leading to impaired mitochondrial antioxidant function within oocytes. This dysfunction may disrupt oocyte maturation and thereby contribute to female infertility (Li *et al*.,2020, Walter *et al*.,2025), as was observed by low levels of SIRT1 in infertile subjects, but the results were not significant. However, significant strong negative correlation of SIRT 1 with BMI can be explained by a reduction in statistical power following stratification, as subdividing the cohort decreases the sample size within each BMI group and limits the ability to detect moderate correlations. In addition, categorization of a continuous variable such as BMI may obscure underlying variability and attenuate true associations due to loss of information. These findings suggest that the observed negative correlation is strong at the population level but may not be sufficiently strong or uniform within narrower BMI categories to achieve statistical significance.

Reduced levels of MnSOD in infertile females are supported by the polymorphism of low glutathione levels observed in a study, which corroborates its critical role in maintaining oocyte quality and protecting oocytes from oxidative stress–induced damage. (Adeoye *et al*., 2018). While the current study did not demonstrate elevated levels in obese females, several comorbidities commonly associated with obesity, including type II diabetes mellitus and cardiovascular disease, have been documented in the literature. (Picklo *et al*., 2015).

The generalizability of these findings is limited by the single-centre design and convenience sampling, which may not represent broader populations. Strict exclusion criteria (e.g., PCOS, endometriosis, comorbidities) create a controlled sample that differs from routine clinical settings. Additionally, the use of South Asian BMI cut-offs may limit applicability to other populations; therefore, findings should be interpreted with caution.

Visfatin is an adipocytokine secreted from multiple sources, including visceral adipose tissue and macrophages. It exhibits insulin-mimetic activity, influences glucose metabolism and promotes inflammatory responses (Abdalla, 2022). Although the role of visfatin in the hypothalamo–pituitary–ovarian axis remains largely unexplored, the reduced levels observed in infertile females support the hypothesis linking oxidative stress to infertility (Koutsoura *et al*., 2025), OS marker associated with the risk of unexplained infertility (Hussien, 2020).

Addressing obesity is crucial in managing female infertility, and an integrative approach combining prevention, treatment and advancements in research is essential for improving reproductive health and optimize fertility outcomes for obese women. (Frank *et al*., 2025).

Limitations

Though the hypothetical mechanism suggests that obesity causes the reduction in SIRT1, yet due to the cross-sectional nature of the study, cause effect relationship cannot be proved. A small sample size was another limitation due to inadequate funds. The body fat percentage of these subjects was not calculated, which is a more reliable indicator of the measurement of obesity. In addition to that, females were not stratified on the basis of the cause of infertility. Yet this relationship has not been explored previously in the Pakistani population; the current study results will pave a pathway to link obesity, metabolic syndrome and infertility.

CONCLUSION

Progressive decline in antioxidant levels with increasing BMI highlights the contributory role of obesity in enhancing oxidative stress. This obesity-associated reduction in antioxidant defences may impair reproductive homeostasis and represents a potential mechanistic pathway linking increased adiposity to female infertility.

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Authors' contributions

FA and RR conceived the project; MB, IN, FS and RA; Recruited all the fertile patients and collected all the samples and data. All authors contributed equally to writing and finalizing the manuscript.

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Data availability statement

All data generated or analysed during this study are included in this published article.

Ethical approval

The ethical approval was obtained from the institutional ethical review board of Aga Khan University (ERC#4426-BBS-ERC-16).

Conflict of interest

This manuscript is a part of the PhD thesis of Dr Faiza Alam, submitted to the Higher Education Commission, Pakistan, to be added to the repository.

Supplementary data

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