

Clinical Significance of Seminal Plasma Superoxide Dismutase 3 (SOD3) in Relation to FSH, LH, Levels in Men with Oligozoospermia

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ABSTRACT

Introduction: male infertility is frequently linked to oxidative stress and endocrine dysregulation which may impair clinically relevant semen parameters such as progressive motility (a+b%). This study evaluated seminal plasma superoxide dismutase 3 (SOD3) and serum gonadotropins (LH, FSH) in men with primary infertility and examined their relationships with progressive motility. **Methodology:** A case-control design included 63 men with primary infertility (sperm concentration < 15 million/mL: age 20-45: no chronic disease, autoimmune disease, or varicocele) and 25 fertile controls (sperm concentration > 50 million/mL). SOD3 was measured in seminal plasma by ELISA, and serum LH, FSH were assessed in blood, progressive motility defined as (a+b)% (rapid and slow progressive) with $\geq 40\%$ considered normal. Nonparametric comparison (Mann-Whitney U), spearman correlations (infertile group), and multiple linear regression were applied. **Results:** progressive motility was markedly lower in infertile man than controls ($U=0$, $p<0.001$). seminal SOD3 also differed strongly between groups ($U=0$, $p<0.001$). and FSH was higher in infertile men ($U=538$, $P=0.020$), while LH and age were not significantly different. With infertile men, SOD3 correlated inversely with progressive motility ($p=-435$, $p<0.001$). in multivariable regression ($n=63$), SOD3 remained the only significant predictor of motility ($B=0.154$, $95\%CI=-0.235$ to -0.037 , $p<0.001$: $R^2=0.239$: adjusted $R^2=0.187$). **Discussion:** findings support a close link between altered seminal redox status and reduced progressive motility in primary infertility, while gonadotropins no independent association after adjustment. Moderate collinearity between LH and FSH was observed. **Conclusion:** seminal plasma SOD3 is strongly associated with impaired progressive motility in primary male infertility: larger smoking-matched studies are warranted to confirm clinical utility.

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1. Introduction

Male infertility is a major component of the global infertility burden and is increasingly recognized as clinical and public health issues rather than a niche reproductive problem. Contemporary estimates suggest infertility affects a substantial proportion of individual's worldwide, and male factors are a primary of contributing cause in a large share of infertile couples (Harris, 2023; Ribas-Maynou & Yeste, 2020; Vander Borcht & Wyns, 2018). Modern clinical evaluation therefore emphasizes a structured work-up that integrates semen analysis with targeted endocrine testing and when indicated, adjunct functional assessments that capture sperm health beyond conventional parameters (Chung et al., 2024; Harris, 2023; Vander Borcht & Wyns, 2018). Semen analysis remains the cornerstone for initial male fertility assessment, yet its interpretation has evolved with update references frameworks and increasing attention to clinically endpoints such as progressive motility (Chung et al., 2024). Progressive motility is important; it reflects forward progression required for sperm transport and fertilization. While routine seamen analysis provides essential phenotyping, it doesn't fully explain a large proportion of idiopathic or unexplained male infertility, motivating the search for biomarkers that reflect underlying pathophysiology (Chung et al., 2024; Harris, 2023; Vander Borcht & Wyns, 2018). Among the leading pathways implicated in male reproductive dysfunction is oxidative stress, defined as an imbalance between reactive oxygen species (ROS) generation and antioxidant defenses capacity. Oxidative stress can impair sperm function through lipid peroxidation of the

sperm plasma membrane, disruption of axonemal/ mitochondrial performance and downstream effect on motility, viability, and fertilizing potential (Gharagozloo & Aitken, 2011; Walczak-Jedrzejowska et al., 2013). Because oxidative stress is not directly captured by standard semen parameters, several approaches have been developed to quantify redox imbalance including-re741'duction potential (ORP), based assays and biochemical measurements of antioxidant enzymes in seminal plasma. ORP has been proposed as practical integrative measure of redox status, with studies describing standardized measurements protocols and clinically useful discrimination between fertile control and men with abnormal semen quality (Agarwal et al., 2016). Independent validation and qualification efforts support analytical reliability and potential clinical utility (Tan et al., 2024; Vassiliou et al., 2021). These data collectively support the border concept that redox imbalance correlate with poorer semen parameters, including motility and can contribute additional explanatory value in male factor infertility evolutions (Agarwal et al., 2016; Tan et al., 2024; Vassiliou et al., 2021). At the biochemical level, seminal plasma antioxidants form a complex defense network that include enzymatic components such as superoxide dismutase (SODs)(Vashisht & Gahlay, 2024). Extracellular superoxide dismutase (SOD3) is a particular of interest because it functions in extracellular compartment and may reflect the antioxidant microenvironment surrounding spermatozoa. Proteomic and mechanistic literature increasingly links oxidative-stress-related-proteins- including distinct SOD isoform- to sperm motility regulation, supporting the plausibility that altered SOD3 status may accompany reduced progressive motility and impaired sperm function (Aitken, 2020; Baker & Aitken, 2005; Karabulut et al., 2021; Kurkowska et al., 2020; Ribeiro et al., 2022). However, evidence is not uniformly consistent across studies and endpoints some meta-analytic data suggest that deference's in seminal SOD activity between infertile men and controls may be modest or not statistically robust in certain comparison, underscoring heterogeneity across infertility phenotypes, assays, and study designs (Huang et al., 2018). Such mixed finding strengthens the rational for focused studies using clearly defined clinical groups and standardized semen endpoints.

In parallel with oxidative pathways, endocrine regulation is central to spermatogenesis and sperm maturation. The hypothalamic-pituitary-gonadal axis, mediated in part by gonadotropins such as follicle-stimulating hormone (FSH) and luteinizing hormone (LH), influences Sertoli and Leydig cell function and thereby sperm production and maturation (Vashisht & Gahlay, 2024). Clinical practice routinely incorporates serum gonadotropin assessment in male infertility evolution, especially when semen parameters are abnormal (Vander Borgh & Wyns, 2018). Yet the relationship between circulating gonadotropin and semen quality remains complex, with population studies reporting variable association between LH/FSH levels and sperm concentration, morphology and motility (Zhao et al., 2020). These inconsistencies likely reflect differences in study populations, confounding factors, and correlation among hormones that can obscure independent effects unless multivariable modeling is applied (Zhao et al., 2020). Lifestyle and environmental exposure add another layer of complexity by modulating both oxidative stress and reproductive endocrine function. Cigarette smoking, for example, has been associated with deterioration of multiple semen parameters in systematic evolutions, plausibly mediated by increased oxidative load and inflammatory signaling (Loch & Fulgham, 2015). Such exposure can confound comparison between infertile cases and fertile controls if exposure distribution differs between groups, and they should be explicitly considered when interpreting biomarker-semen relationships (Shokeralla et al., 2025).

Taken together, the current literature supports an integrated conceptual model in which oxidative stress and hormonal dysregulation may converge on clinically relevant semen outcomes, particularly progressive motility, but also reveals persistent gaps, key needs include studies that measure oxidative stress-related markers in the biologically proximal component of seminal plasma, pair these measurements with serum gonadotropins, and test their independent association with motility using appropriately specified multivariable models, while acknowledging confounding structures common in infertility cohorts. Accordingly, the present study evaluates oxidative stress status, focusing on seminal plasma SOD3 and endocrine status (serum LH and FSH) in men with primary infertility compared with fertile controls and examine how these biomarkers related to sperm progressive motility as a clinically interpretable semen parameter aligned with contemporary seamen analysis guidance (Chung et al., 2024).

2. Methodology

2.1 Study Design

A case-control study was conducted to evaluate the relationship between oxidative stress and reproductive hormonal profiles and their association with sperm quality in men with primary infertility compared with fertile controls.

2.2 Participant and Eligibility Criteria

Participant were classified into 2 groups: infertile group (cases), men were diagnosed with primary infertility defined as no pregnancy in the first year of marriage, were recruited. Eligibility criteria included age 20-45 years, sperm concentration <15 million/mL, and absence of chronic medical condition such as (diabetes and hypertension), absence of autoimmune disease, and no varicocele. All infertility participants were smokers. Fertile group (controls), healthy men with

proven fertility were included as controls. Eligibility criteria included: age within the same general reproductive age range, sperm concentration >50 million/mL, non-smokers, and confirmed fertility defined as pregnancy with the first year of marriage or the wife being currently pregnant. Controls had no reported infertility complaints. Smoking status was used in recruitment (cases; smokers; controls: non-smokers), but smoking was not recorded as variable in the dataset; therefore, it was treated as potential confounder and addressed in the discussion

2.3 Sample Collection and Laboratory Assessments

2.3.1 Seamen Collection and Analysis

Semen samples were collected for standard semen analysis, including assessments of sperm concentration and motility. Progressive motility was reported as the combined percentage of rapid progressive (a) and slow progressive (b) spermatozoa, expressed as (a+b) %. In this study, normal progressive normal progressive motility was defined as $\geq 40\%$, and values < 40% were considered low. Progressive motility (a+b%) was used as the primary semen quality outcome variable for the main correlation and regression analysis

2.3.2 Measurements of Oxidative Stress Marker (SOD3)

The oxidative stress marker superoxide dismutase (SOD3) was measured in seminal plasma. Seminal plasma was separated according to routine laboratory procedures and SOD3 concentration were determined using an enzyme-linked immunosorbent assay (ELISA) method (commercial ELISA kit protocol). The assay, procedure, calibration, and reporting unit were performed according to the kits manufacturer instructions

2.3.3 Analysis of LH and FSH

Peripheral blood samples were collected to measure serum level of the gonadotropins luteinizing hormone (LH) and follicle-stimulating hormone (FSH) using standard clinical laboratory methods by Cobas- E411 (ROCH- Germany).

2.4 Statistical Analysis

Data were analyzed using Jamovi (de Souza et al., 2024; Şahin & Aybek, 2019). Continuous variables were summarized using descriptive statistics (mean, median, and standard deviation) normality was assessed using the Shapiro-wilk test. Between group comparisons case vs controls were performed using the Mann-Whitney U test for non-normality distributed variables, and effect sizes were reported as rank-biserial correlation. Association between biomarkers and hormones with progressive motility were assessed using spearman's rank association (Spearman's rho) to evaluate independent predictors of progressive motility with the infertile group, multiple linear regression was performed with progressive motility (a+b%) as the dependent variable and SOD3 LH, FSH age as predictors. Model fit was reported using R^2 and adjusted R^2 . Multicollinearity was assessed using tolerance and variance inflation factors (VIF).

3. Results

In total, 88 participants were analyzed (Group 1: primary infertile men: Group 0: healthy controls). Age was comparable between groups (median 31 years in both groups: mean 31.8 ± 7.29 in Group 1 vs 32.5 ± 6.44 in Group 0). Marked differences were observed in progressive motility (a+b%), with substantially lower value in Group 1 (median 13.0%, mean 13.3 ± 5.53) compared with Group 0 (median 47.0%, mean 47.5 ± 5.14) than in controls (median 113, mean 107 ± 19.4). for reproductive hormones, Group 1 showed a higher FSH (median 2, mean 6.76) than controls (median 4, mean 4.76), while LH was relatively similar (Group 1 median 3, mean 4.41) vs Group 0 (median 4, mean 3.84). Normality testing showed non-normal distribution for several variables, particularly in the infertile group (Shapiro-Wilk test p-values: age 0.013, SOD3<0.001, LH <0.001, FSH< 0.001), supporting the use of non-parametric comparisons in subsequent analysis (Table1).

Table 1: Descriptive Statistics and Normality Testing for age, Seminal Plasma SOD3, Serum LH and FSH, and Progressive Motility.

Descriptives	Group	Age	SOD3	LH	FSH	Progressive Motility <40 % (a+b)
Mean	1	31.8	29.4	4.41	6.76	13.3
	0	32.5	107	3.84	4.76	47.5
Median	1	31	26	3	2	13.0
	0	31	113	4	4	47.0
Standard Deviation	1	7.29	16.4	5.62	11.6	5.53
	0	6.44	19.4	1.49	2.49	5.14
Shapiro-Wilk W	1	0.950	0.920	0.715	0.555	0.969
	0	0.952	0.947	0.916	0.882	0.953
Shapiro-Wilk P	1	0.013	<.001	<.001	<.001	0.113
	0	0.284	0.218	0.042	0.008	0.287

Visual inspection of the boxplots demonstrated similar age distribution between the two groups. In contrast, progressive motility (a+b) % was lower in the primary infertile group (Group1), with most values clustering well below 40% reference cutoff, whereas control group (Group 0) showed a higher motility centered around the normal range. Seminal plasma SOD3 levels were substantially lower in Group 1 compared with controls. Hormonal profiles showed greater variability and multiple high outliers in the primary infertile group, particularly FSH (and to a lesser extent LH), while controls exhibited tighter distributions (Figure 1)

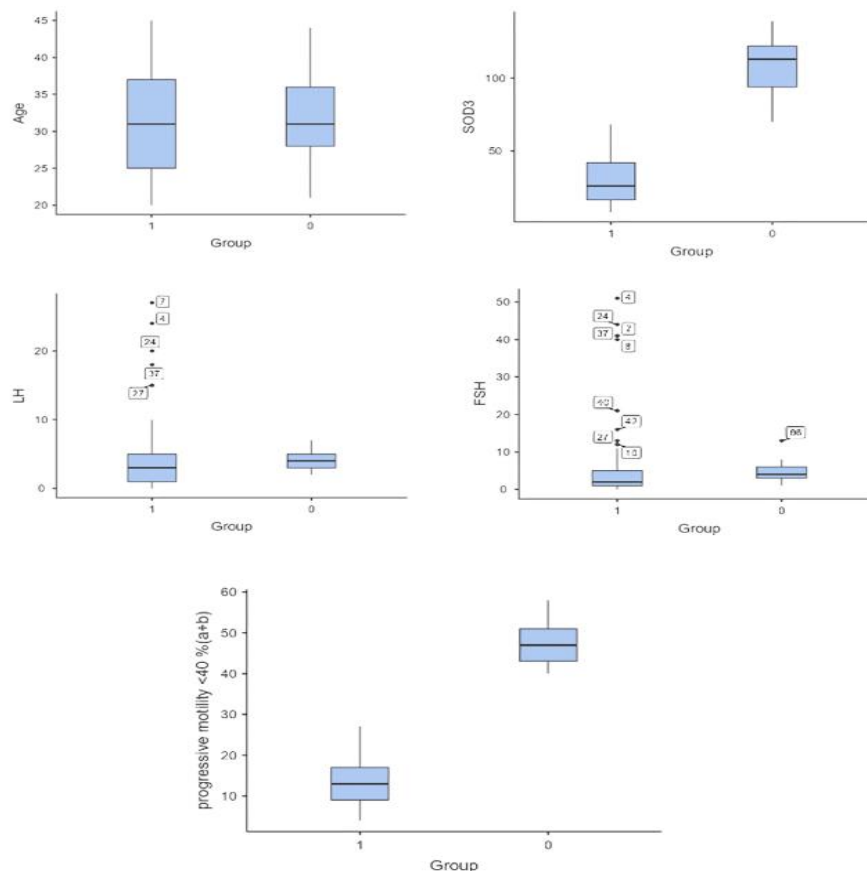


Figure 1: box- and whisker plots comparing age, seminal plasma SOD3, serum LH and FSH, and progressive motility a+b% between study groups (Group1 = primary infertile men: Group 0 = healthy controls). Boxes represent the interquartile range (IQR) with median line: whiskers indicate data spread, and labeled points denote outliers. Progressive motility is expressed as (a+b) % (rapid progressive + slow progressive), with $\geq 40\%$ considered normal and $< 40\%$ indicating reduced progressive motility.

In (Table 2) Mann-Whitney U testing showed no significant differences in age between groups ($U=733$, $p=0.613$, $r_{rb}=0.0698$). SOD3 was significantly different between groups ($U=0$, $P<0.001$, $r_{rb}=1.0000$), and progressive motility (a+b) % also differed markedly ($U=0$, $p<0.001$, $r_{rb}=1.0000$), indicating a very large group separation for both variables. Regarding reproductive hormones LH did not differ significantly ($U=652$, $p=0.207$, $r_{rb}=0.1721$), whereas FSH showed a significant difference between groups ($U=538$, $p=0.020$, $r_{rb}=0.3168$).

Table 2: Between-Group Comparisons of Age, SOD3, Serum LH And FSH, And Progressive Motility (A+B%) Using Mann-Whitney U Test (Group 1= Primary Infertile Men: Group 0= Healthy Controls). Effect Size is Reported as Rank-Biserial Correlation.

Mann-Whitney U	Statistic	P	Effect Size
Age	Mann-Whitney U 733	0.613	Rank biserial correlation 0.0698
SOD3	Mann-Whitney U 0	<.001	Rank biserial correlation 1.0000
LH	Mann-Whitney U 652	0.207	Rank biserial correlation 0.1721
FSH	Mann-Whitney U 538	0.020	Rank biserial correlation 0.3168
Progressive Motility <40 % (a+b)	Mann-Whitney U 0	<.001	Rank biserial correlation 1.0000

Note. $H_a: \mu_1 \neq \mu_0$

Within infertile men, the correlation plot showed a moderate inverse association between SOD3 and progressive motility (a+b%) ($\rho = -0.435$, $p < 0.001$). LH and FSH demonstrate a strong positive correlation ($\rho = 0.780$, $p < 0.001$). Other pairwise correlation with progressive motility were weak and not statistically significant. (Figure 2)

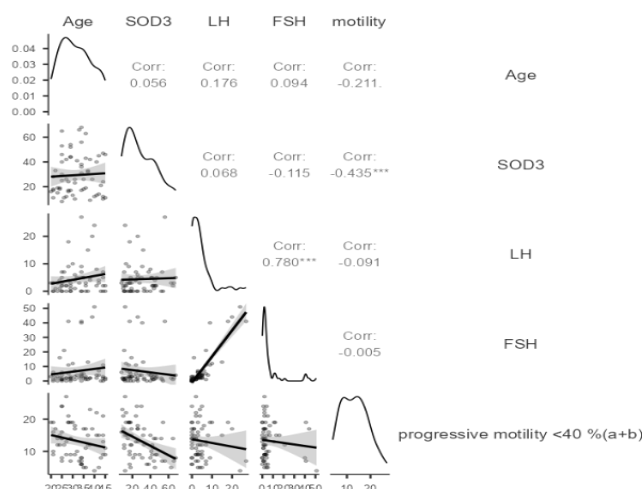


Figure 2: Pairwise scatterplot matrix showed Spearman correlation among age, SOD3, LH, FSH, and progressive motility (a+b%) within the primary infertility group. The upper panels report Spearman correlation coefficients (ρ), the lower panel shows scatterplot with fitted trend lines, and the diagonal panels display variable distributions. Progressive motility represents (a+b%) rapid progressive and slow progressive, with $\geq 40\%$ considered normal and $< 40\%$ indicating reduced progressive motility. Note. * $p < .05$, ** $p < .01$, *** $p < .001$.

In the primary infertility group, a multiple linear regression model was fitted to identify independent predictors of progressive motility (a+b%). The model showed a moderate fit ($R^2 = 0.239$; adjusted $R^2 = 0.187$). SOD3 was the only significant predictor and was inversely associated with progressive motility ($B = -0.154$, 95%CI -0.236 to -0.073 , $p < 0.001$) whereas LH, FSH and age were not a significant in the adjusted model (all $p > 0.05$). (Table 3)

Table 3: Multiple Linear Regression Predicting of Progressive Motility (a+b) % Among Men with Primary Infertility, Using SOD3, LH, FSH and Age as Predictors. Model Fit (R^2 and Adjusted R^2) and Regression Coefficients with 95% Confidence interval are reported.

	B (Estimate)	SE	95% CI (Lower, Upper)	t	P-Value	VIF	Tolerance
Intercept	22.279	3.0360	16.202, 28.3566	7.338	<0.001	—	—
SOD3	-0.154	0.0404	-0.235, -0.0732	-3.816	<0.001	1.09	0.920
LH	0.177	0.2405	-0.304, 0.6586	0.736	0.465	4.56	0.219
FSH	-0.136	0.1162	-0.369, 0.0965	-1.171	0.246	4.53	0.221
Age	-0.137	0.0888	-0.315, 0.0407	-1.543	0.128	1.04	0.959

4. Discussion

In this case-control study oxidative stress and gonadotropins profile were investigated in relation to sperm progressive motility, defined as the combined percentage of rapid and slow progressive spermatozoa (a+b), where values $\geq 40\%$ are considered normal and $< 40\%$ indicate reduced progressive motility. Overall, infertile men (primary infertility) showed markedly lower progressive motility and substantially lower seminal plasma SOD3 compared with fertile controls, while gonadotropins demonstrate a less consistent pattern across comparisons: importantly, within the infertile group, progressive motility exhibited a moderate inverse association with SOD3 and this relationship persisted after simultaneous modeling with age, LH and FSH, where SOD3 emerges as the only independent predictor of progressive motility (negative direction) whereas LH, FSH, and age were not independently associated. This pattern can be biologically interpreted in two complementary ways: first, between-group reduction of antioxidant defenses is commonly reported in male infertility and is broadly consistent with the concept that an impaired redox buffering capacity contributes to poor sperm function and reduced motility (Ayad et al., 2022; Bisht et al., 2017; Kurkowska et al., 2020; Otasevic et al., 2019; Ribas-Maynou & Yeste, 2020; Ritchie & Ko, 2021); second, within affected infertile men, higher SOD3 may reflect a compensatory upregulation in response to an adverse oxidative milieu (i.e. reactive elevation), which can still coincide with poorer motility if oxidative damage ongoing or if the antioxidant response is insufficient to restore physiological redox balance, a framework supported

by clinical oxidative stress testing studies (including oxidation-reduction potential as an integrated marker) linking oxidative dysregulation to abnormal semen quality (Aitken, 2020; Bisht et al., 2017; Ritchie & Ko, 2021; Stagos et al., 2015). The strong correlation observed between LH and FSH highlights shared regulatory control and explains the moderate collinearity detected when both were entered together, which may reduce their apparent independent contribution in multivariable modeling despite known endocrine influences on spermatogenesis (Cannarella et al., 2020). Notably, the broader literature is not fully uniform: some meta analytic evidence suggests that superoxide dismutase- related measures are not always consistently different between infertile and fertile men (Huang et al., 2018), and large randomized of antioxidant supplementations have reported limited or no improvement in key semen outcomes, reinforcing that antioxidant biology in male infertility is heterogenous and not reliably corrected by empirical supplementation alone (de Ligny et al., 2025; Steiner et al., 2020). These discrepancies may relate to differences in infertility phenotypes, assays methodology (isoform-specific measures such as SOD3 activity), timing of sampling, and the degree of which oxidative stress is the dominant driver in each study. The major limitations of our study include its single-center observational design (limiting causal inference), the modest sample size, and the absence of smoking as a recorded covariate in the dataset given that all cases were smokers while controls were non-smokers per inclusion criteria, residual confounding by smoking related oxidative effects is plausible and should be acknowledged explicitly. Future work would benefit from larger multicenter cohorts, standardized isoform-specific oxidative stress profiling, and prospective designs that can disentangle whether elevated SOD3 within infertile man represent adaptive compensation, disease severity, or both, while incorporating lifestyle factors as a measurable covariate (Ayad et al., 2022; Bisht et al., 2017; de Ligny et al., 2025; Ribas-Maynou & Yeste, 2020; Stagos et al., 2015; Steiner et al., 2020).

5. Conclusion

In summary, men with primary infertility showed markedly reduced progressive sperm motility (a+b%) compared with fertile controls, alongside significant alteration in SOD3 and reproductive hormone profiles. Within the infertile group SOD3 demonstrate the most consistent relationship with sperm quality, showing an inverse association with progressive motility and remaining the only independent predictor after adjustment for age, LH, and FSH, indicating that oxidative stress related changes in the seminal microenvironment are closely linked to impaired forward motility. Although endocrine parameters differed between groups, they did not show an independent contribution to motility once modulated together in our study, likely reflecting shared regulation and overlap between gonadotropins. These findings supporting the clinical relevance of integrating oxidative stress assessment particularly SOD3 with routine semen evaluation when investigating male infertility.

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