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Authors:	Inas Ali Hussein[1]*, Rana R Al-Saadi[1], Nahla A Al-Bakri[2]
Affiliations:	[1]High Institute of Infertility Diagnosis and Assisted Reproductive Technologies, Al-Nahrain University, Baghdad, Iraq; [2]College of Education for Pure Sciences, Ibn Al-Haitham, University of Baghdad, Baghdad, Iraq.
Corresponding author:	Inas Ali Hussein High Institute of Infertility Diagnosis and Assisted Reproductive Technologies, Al-Nahrain University P.O. Box 70043, Baghdad, Iraq Phone: +964 773 459 9000 E-mail: enas.ali@ierit.nahrainuniv.edu.iq
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ORIGINAL ARTICLE

Association of seminal plasma coenzyme Q10 with sperm chromatin abnormality and semen parameters: a cross-sectional study

Inas Ali Hussein[1*], Rana R Al-Saadi[1], Nahla A Al-Bakri[2]

[1]High Institute of Infertility Diagnosis and Assisted Reproductive Technologies, Al-Nahrain University, Baghdad, Iraq; [2]College of Education for Pure Sciences, Ibn Al-Haitham, University of Baghdad, Baghdad, Iraq

Running title: Hussein et al. Seminal CoQ10 and chromatin abnormality

Corresponding author:

Inas Ali Hussein

High Institute of Infertility Diagnosis and Assisted Reproductive Technologies, Al-Nahrain University

P.O. Box 70043, Baghdad, Iraq

Phone: +964 773 459 9000

E-mail: enas.ali@ierit.nahrainuniv.edu.iq

ORCID ID: 0009-0007-7223-0138

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ABSTRACT

Aim: To assess whether seminal plasma coenzyme Q10 (CoQ10) concentration is associated with sperm chromatin abnormality and conventional semen parameters across four recorded semen-profile groups.

Methods: This cross-sectional study included 79 infertile men classified in the original clinical dataset as normozoospermic (n = 18), oligozoospermic (n = 25), asthenozoospermic (n = 14), or teratozoospermic (n = 22). Semen was analysed according to World Health Organization 2010 recommendations. Sperm chromatin maturity/condensation was assessed by aniline blue staining and seminal plasma CoQ10 by enzyme-linked immunosorbent assay.

Results: CoQ10 concentration did not differ among the four groups (Kruskal–Wallis p = 0.329). Chromatin abnormality differed among groups ($p < 0.001$), principally between normozoospermic men and the oligozoospermic and asthenozoospermic groups. CoQ10 was not associated with chromatin abnormality (r = -0.110, 95% confidence interval (CI) -0.323 to 0.114; raw p = 0.335; FDR-adjusted p = 0.569), and remained non-significant after adjustment for age, body mass index, and sperm concentration (unstandardised coefficient (B) = -0.458, 95% CI -1.398 to 0.483; standardised $\beta = -0.102$; p = 0.335). Chromatin abnormality was moderately associated with poorer semen parameters after FDR correction.

Conclusion: Seminal plasma CoQ10 was not independently associated with sperm chromatin abnormality in this sample. Associations between chromatin abnormality and conventional semen parameters were exploratory and require validation in larger prospective studies with fertile controls.

Keywords: infertility; oxidative stress; semen; spermatozoa; ubiquinone

INTRODUCTION

Male infertility contributes substantially to infertility worldwide (1). It may arise from diverse genetic, endocrine, anatomical, infectious, and lifestyle-related factors and may also reflect broader aspects of male health (2). Conventional semen assessment is based primarily on sperm concentration, motility, and morphology, but these measures do not fully characterise sperm nuclear integrity. Abnormal chromatin packaging may compromise sperm nuclear integrity (3), while sperm deoxyribonucleic acid (DNA) fragmentation has been associated with impaired fertilisation and adverse reproductive outcomes (4).

Coenzyme Q10 (CoQ10) is a lipid-soluble component of the mitochondrial electron-transport chain with antioxidant activity (5). In male infertility, CoQ10 has been investigated for its potential role in sperm bioenergetics and motility (6). Reactive oxygen species can damage sperm membranes, mitochondria, and nuclear material (7), while oxidative stress is associated with impaired semen quality and abnormal sperm chromatin (8). CoQ10 supplementation has been evaluated in infertile men (9). Its effects on semen parameters have also been examined in a dedicated review (10) and a meta-analysis (11). Less is known about the association between naturally occurring seminal plasma CoQ10 concentrations and sperm chromatin abnormality across distinct semen-profile categories.

The aim of this study was to assess the association of seminal plasma CoQ10 concentration with sperm chromatin abnormality and conventional semen parameters across the four recorded semen-profile groups.

MATERIAL AND METHODS

Patients and study design

This cross-sectional study was conducted from December 2021 to June 2022 at the High Institute of Infertility Diagnosis and Assisted Reproductive Technologies, Al-Nahrain University, Baghdad, Iraq. Following data review and exclusion of two records, the final

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reanalysed dataset comprised 79 infertile men aged 20–54 years. Participants were analysed according to the mutually exclusive semen-profile category recorded in the original clinical dataset: normozoospermia (n=18), oligozoospermia (n=25), asthenozoospermia (n=14), or teratozoospermia (n=22). Semen parameters were measured and reported with reference to the World Health Organization (WHO) 2010 laboratory manual (12). Because individual semen abnormalities overlapped and the original laboratory rule used to assign a single category was not retained, the groups were not retrospectively reconstructed from individual semen variables. Accordingly, they should be interpreted as recorded clinical semen-profile categories rather than independently reconstructed WHO phenotypes.

Men with primary or secondary infertility who had abstained from ejaculation for 3–5 days before sample collection were eligible. Exclusion criteria were azoospermia, systemic illness including diabetes or cancer, febrile illness within the preceding three months, genitourinary infection, current antioxidant supplementation or hormonal therapy, occupational exposure to reproductive toxicants, or incomplete semen-analysis data.

Methods

Semen was collected by masturbation into sterile wide-mouthed containers and allowed to liquefy at 37 °C for up to 30 minutes. Semen volume, sperm concentration, motility, and morphology were assessed according to WHO 2010 recommendations using a Makler counting chamber and phase-contrast microscopy (12).

Sperm chromatin abnormality was assessed by acidic aniline blue staining, which identifies histone-rich, incompletely condensed sperm chromatin rather than directly measuring DNA strand fragmentation (13). Air-dried semen smears were fixed in 3% buffered glutaraldehyde for 30 minutes and stained with 5% aqueous aniline blue solution (pH 3.5) for 5 minutes. Two hundred spermatozoa per slide were evaluated at ×1000 magnification under oil

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immersion. Dark-blue heads were classified as chromatin abnormal, and pale-blue or unstained heads as chromatin normal.

After semen analysis, 1 mL of each semen sample was centrifuged at approximately 865 ×g (3000 revolutions per minute) for 10 minutes at ambient temperature using a Hettich EBA 20 benchtop centrifuge (Andreas Hettich GmbH, Tuttlingen, Germany) equipped with the standard eight-place fixed-angle rotor (effective radius, 86 mm). The separated seminal plasma supernatant was transferred into a sterile microcentrifuge tube and stored at −80 °C for no longer than 50 days. Samples were thawed once before analysis. CoQ10 concentration was measured by enzyme-linked immunosorbent assay (ELISA) using a Human Coenzyme Q10 ELISA kit (BT LAB Bioassay Technology Laboratory; catalogue No. E3164Hu; lot No. 202112002) according to the manufacturer's instructions. Optical absorbance was measured at 450 nm using an RT-2100C microplate reader (Rayto Life and Analytical Sciences Co., Ltd., Shenzhen, China).

The available study records did not contain a seminal-plasma matrix validation report or study-specific precision estimates for the assay; this limitation was considered when interpreting the findings.

Statistical analysis

Continuous variables were summarised as mean ± standard deviation (SD) when approximately normally distributed and as median (interquartile range, IQR) otherwise. Group differences were evaluated using one-way analysis of variance or the Kruskal–Wallis test, as appropriate. Significant Kruskal–Wallis results were followed by Dunn pairwise comparisons with Holm adjustment. Pearson correlations were reported with 95% confidence intervals (CIs) and retained as exploratory estimates of linear association; because several variables were non-normally distributed, correlation results were interpreted cautiously. The 17 correlations displayed in Table 3 were treated as one family and adjusted using the

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Benjamini–Hochberg false discovery rate (FDR) procedure. Multiple linear regression assessed chromatin abnormality in relation to CoQ10 after adjustment for age, body mass index (BMI), and sperm concentration. Unstandardised coefficients (B), standard errors, 95% CIs, standardised β coefficients, variance inflation factors (VIFs), R^2, adjusted R^2, the model F test, and residual diagnostics were reported. Raw or adjusted two-sided p values <0.05 were considered statistically significant. Regression diagnostics included the Shapiro–Wilk test of residual normality, Breusch–Pagan assessment of heteroscedasticity, Durbin–Watson statistic, Cook's distance, and VIF. Because the residuals departed from normality, coefficient-level inference from the ordinary least squares model was interpreted cautiously.

RESULTS

The analysis included 79 men in the recorded semen-profile categories: 18 normozoospermic, 25 oligozoospermic, 14 asthenozoospermic, and 22 teratozoospermic. Overall mean age was 33.2 ± 7.3 years and mean BMI was 27.6 ± 4.9 kg/m². Age and BMI did not differ among groups (p=0.321 and p=0.114, respectively). Median sperm concentration was 22.50, 6.00, 15.50, and 17.50 $\times 10^6$/mL; progressive motility was 39.50%, 30.00%, 5.00%, and 29.00%; and normal morphology was 30.50%, 15.00%, 10.00%, and 10.00% in the normozoospermia, oligozoospermia, asthenozoospermia, and teratozoospermia groups, respectively (all p<0.001) (Table 1).

Median seminal plasma CoQ10 concentration was 2.73, 2.71, 3.09, and 2.75 ng/mL in the normozoospermia, oligozoospermia, asthenozoospermia, and teratozoospermia groups, respectively, without a significant difference (p=0.329). Median sperm chromatin abnormality was 8.25%, 18.00%, 15.50%, and 11.75%, respectively (p<0.001). Dunn–Holm comparisons showed differences between normozoospermia and oligozoospermia (adjusted p<0.001) and between normozoospermia and asthenozoospermia (adjusted p=0.004); all other pairwise comparisons were non-significant (Table 2).

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Seminal plasma CoQ10 was not correlated with age, BMI, sperm concentration, total sperm count, progressive motility, total progressive motile count, immotile sperm percentage, normal morphology, or chromatin abnormality after Benjamini–Hochberg correction (all FDR-adjusted $p>0.05$). Chromatin abnormality correlated inversely with sperm concentration ($r=-0.427$), total sperm count ($r=-0.451$), progressive motility ($r=-0.444$), total progressive motile count ($r=-0.457$), and normal morphology ($r=-0.445$), and positively with immotile sperm percentage ($r=0.522$) (all FDR-adjusted $p<0.001$) (Table 3).

The correlation between seminal plasma CoQ10 and chromatin abnormality was weak and non-significant ($r=-0.110$, 95% CI -0.323 to 0.114; raw $p=0.335$; FDR-adjusted $p=0.569$). In multiple regression, CoQ10 remained non-significant after adjustment for age, BMI, and sperm concentration ($B=-0.458$, 95% CI -1.398 to 0.483; standardised $\beta=-0.102$; $p=0.335$), whereas sperm concentration was inversely associated with chromatin abnormality ($B=-0.274$, 95% CI -0.410 to -0.138; standardised $\beta=-0.426$; $p<0.001$). The model explained 20.5% of outcome variance (adjusted $R^2=0.162$; $F=4.76$; $p=0.002$). VIFs ranged from 1.03 to 1.20. Residuals were non-normal (Shapiro–Wilk $p<0.001$), whereas the Breusch–Pagan test did not indicate heteroscedasticity ($p=0.857$); the Durbin–Watson statistic was 2.01, and no Cook's distance exceeded 1.0 (maximum 0.299) (Table 4).

DISCUSSION

In this study, seminal plasma CoQ10 concentration neither differed across the recorded semen-profile groups nor showed an association with sperm chromatin abnormality, whereas chromatin abnormality was related to several conventional semen parameters. These findings do not support seminal CoQ10 as a clinical biomarker in this sample, although prior meta-analytic evidence has suggested beneficial effects of CoQ10 supplementation on semen parameters (11).

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CoQ10 has a plausible role in male reproductive physiology because of its antioxidant and bioenergetic functions (6). However, reported effects on semen parameters have varied across studies and clinical contexts (10). The present null participant-level association therefore suggests that seminal CoQ10 concentration alone did not explain variation in chromatin condensation in this cohort.

Reactive oxygen species can impair sperm mitochondrial function, promote lipid peroxidation, and damage nuclear material (7). Oxidative stress is also implicated in male infertility and impaired sperm quality (8). Biological plausibility, however, cannot substitute for the null association observed here. Moreover, aniline blue staining reflects histone retention and chromatin condensation rather than direct DNA fragmentation (13); related work also supports the distinction between chromatin condensation abnormalities and DNA fragmentation (14).

Sperm chromatin abnormality was inversely associated with sperm concentration, progressive motility, and normal morphology and positively associated with immotile sperm. Similar associations between aniline blue-defined chromatin abnormalities and conventional semen parameters have been reported in infertile men (13). A previous study linked impaired chromatin condensation with increased DNA fragmentation (14), while sperm DNA fragmentation has more broadly been associated with adverse reproductive outcomes (4). Sperm concentration, rather than CoQ10, remained associated with chromatin abnormality in the adjusted model. Nevertheless, the model explained only a limited proportion of outcome variance, its residuals were non-normal, and clinically relevant confounders were unavailable. Accordingly, the regression findings are exploratory and should not be used to infer an independent effect of seminal CoQ10, particularly given the recognised influence of reactive oxygen species on sperm structure and function (7).

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Normozoospermic participants were infertile men and therefore represent an internal clinical reference group rather than proven fertile controls. This distinction is important because infertility is a clinical condition that cannot be inferred solely from conventional semen parameters (1). Male infertility may also be associated with broader health factors not captured by semen phenotype alone (2).

Because seminal CoQ10 was not associated with chromatin abnormality in this cohort, the present findings do not support its use as a biomarker of sperm nuclear quality. This null association should not, however, be interpreted as evidence against a potential therapeutic effect of CoQ10 supplementation. An intervention study in idiopathic oligoasthenozoospermia reported improvements in oxidative-stress markers and sperm DNA fragmentation (9). A meta-analysis also reported improvements in selected semen parameters following CoQ10 treatment (11). Future studies should therefore combine validated seminal-plasma assays with direct DNA-damage methods and reproductive outcomes, while randomised trials are required to determine whether CoQ10 supplementation can modify sperm nuclear integrity.

This study has several limitations. Its cross-sectional design precludes causal inference, the sample was modest, and the asthenozoospermia subgroup contained only 14 men. Proven fertile controls were absent, and aniline blue staining assesses chromatin maturity/condensation rather than DNA strand fragmentation (13). The four semen-profile categories were inherited from the original clinical dataset; because abnormalities overlapped and the original single-category assignment rule was not retained, these labels could not be independently reconstructed from individual WHO 2010 variables and some degree of clinical-category misclassification cannot be excluded. The regression did not include smoking, varicocele, abstinence duration, infection, lifestyle factors, or oxidative-stress

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markers, and its residuals were non-normal. In addition, the retained records did not include study-specific seminal-plasma validation or precision estimates for the ELISA assay.

CONCLUSION

Seminal plasma CoQ10 was not associated with sperm chromatin abnormality in this sample of infertile men. Chromatin abnormality was related to several conventional semen parameters, but the cross-sectional design, absence of fertile controls, limited covariate adjustment, and regression-diagnostic limitations preclude causal, biomarker, or therapeutic claims.

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FUNDING

No specific funding was received for this study.

CONFLICTS OF INTEREST

None to declare.

AUTHOR CONTRIBUTIONS (CRediT)

Conceptualization, I.A.H.; Investigation, I.A.H. and R.R.A.; Formal analysis, R.R.A.; Writing – original draft, I.A.H.; Writing – review and editing, N.A.A.B. All authors read and approved the final manuscript.

ETHICS STATEMENT

The study was approved by the Ethics Committee of the High Institute of Infertility Diagnosis and Assisted Reproductive Technologies, Al-Nahrain University, Baghdad, Iraq (approval letter dated 10 October 2021; approval number: 0702-MF-2025I63). Written informed consent was obtained from all participants before sample collection in accordance with the Declaration of Helsinki.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author on reasonable request.

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TABLES

Table 1. Demographic characteristics and conventional semen parameters across the four recorded semen-profile groups.

Characteristic	Normozoospermia (n=18)	Oligozoospermia (n=25)	Asthenozoospermia (n=14)	Teratozoospermia (n=22)	p
			Mean ± SD		
Age, years	33.78 ± 7.37	34.28 ± 7.26	34.21 ± 8.89	30.68 ± 6.03	0.321
BMI (kg/m²)	25.80 ± 3.81	27.41 ± 4.81	29.99 ± 4.45	27.85 ± 5.56	0.114
			Median (IQR)		
Sperm concentration (×10⁶/mL)	22.50 (17.13–38.75)	6.00 (4.00–8.00)	15.50 (10.00–19.94)	17.50 (12.75–20.00)	<0.001
Progressive motility (%)	39.50 (35.75–50.00)	30.00 (20.00–40.00)	5.00 (1.00–18.75)	29.00 (20.00–37.25)	<0.001
Normal morphology (%)	30.50 (30.00–34.50)	15.00 (5.00–25.00)	10.00 (2.00–14.50)	10.00 (6.25–15.00)	<0.001

BMI, body mass index; IQR, interquartile range; SD, standard deviation.

Table 2. Seminal plasma CoQ10 concentration and sperm chromatin abnormality across the four recorded semen-profile groups.

Outcome	Normozoospermia (n=18)	Oligozoospermia (n=25)	Asthenozoospermia (n=14)	Teratozoospermia (n=22)	p
			Median (IQR)		
Seminal plasma CoQ10 (ng/mL)	2.73 (2.39–3.73)	2.71 (2.38–3.05)	3.09 (2.93–3.33)	2.75 (2.52–3.25)	0.329
Sperm chromatin abnormality (%)	8.25 (6.25–12.50)	18.00 (13.00–22.50)	15.50 (14.50–16.88)	11.75 (8.63–18.75)	<0.001

Significant pairwise comparisons for chromatin abnormality after Dunn–Holm correction were normozoospermia versus oligozoospermia (adjusted p<0.001) and normozoospermia versus asthenozoospermia (adjusted p=0.004); all other adjusted pairwise p values were >0.05.

CoQ10, coenzyme Q10; IQR, interquartile range.

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Table 3. Correlations of seminal plasma CoQ10 and sperm chromatin abnormality with demographic and semen parameters.

Parameter	Seminal plasma CoQ10			Sperm chromatin abnormality		
	r (95% CI)	p†	p‡	r (95% CI)	p†	p‡
Age (years)	0.038 (−0.185−0.258)	0.738	0.923	−0.006 (−0.227−0.216)	0.961	0.961
BMI (kg/m²)	−0.149 (−0.359−0.075)	0.190	0.456	0.141 (−0.083−0.352)	0.214	0.456
Sperm concentration (×10⁶/mL)	−0.017 (−0.237−0.205)	0.885	0.940	−0.427 (−0.592−−0.227)	<0.001	<0.001
Total sperm count (×10⁶)	−0.029 (−0.249−0.193)	0.797	0.923	−0.451 (−0.611−−0.255)	<0.001	<0.001
Progressive motility (%)	0.113 (−0.110−0.326)	0.320	0.569	−0.444 (−0.606−−0.247)	<0.001	<0.001
Total progressive motile count	−0.027 (−0.247−0.195)	0.814	0.923	−0.457 (−0.616−−0.262)	<0.001	<0.001
Immotile sperm (%)	−0.088 (−0.303−0.136)	0.441	0.681	0.522 (0.341−0.667)	<0.001	<0.001
Morphologically normal sperm, (%)	−0.065 (−0.282−0.159)	0.572	0.810	−0.445 (−0.606−−0.248)	<0.001	<0.001
Seminal plasma CoQ10 (ng/mL)	—	—	—	−0.110 (−0.323−0.114)	0.335	0.569

†Raw p value.
‡Benjamini–Hochberg false discovery rate-adjusted p value.
CI, confidence interval; CoQ10, coenzyme Q10; FDR, false discovery rate.

Table 4. Multiple linear regression of factors associated with sperm chromatin abnormality.

Predictor	B	SE	95% CI for B	Standardised β	VIF	p value
Seminal plasma CoQ10 (ng/mL)	-0.458	0.472	-1.398 to 0.483	-0.102	1.03	0.335
Age (years)	-0.091	0.115	-0.319 to 0.138	-0.088	1.16	0.432
BMI (kg/m²)	0.118	0.176	-0.232 to 0.469	0.077	1.20	0.503
Sperm concentration (×10⁶/mL)	-0.274	0.068	-0.410 to -0.138	-0.426	1.05	<0.001

Model R^2=0.205; adjusted R^2=0.162; F=4.76; p=0.002. Residual Shapiro–Wilk p<0.001; Breusch–Pagan p=0.857; Durbin–Watson=2.01; maximum Cook's distance=0.299.
B, unstandardised coefficient; β, standardised coefficient; BMI, body mass index; CI, confidence interval; CoQ10, coenzyme Q10; SE, standard error; VIF, variance inflation factor.

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