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Age-Related changes in the quality of semen and sperm DNA among a sample of men in Iraq-Kurdistan

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Abstract

To evaluate the impact of advancing paternal age on conventional semen parameters and sperm DNA integrity among men in the Kurdistan Region of Iraq. A cross-sectional study was conducted from September 2024 to April 2025 in private fertility centers across Erbil and Duhok. A total of 203 men aged 20–60 years were enrolled after excluding those with chronic illness, reproductive abnormalities, or heavy smoking. Semen samples were analyzed according to WHO (2010) guidelines, and 76 were assessed for DNA fragmentation index (DFI). Advancing age significantly reduced semen volume (3.24 ± 1.40 mL vs 2.07 ± 1.29 mL; $p < 0.001$), sperm concentration (62.21 ± 48.87 vs 31.25 ± 43.26 million/mL; $p = 0.003$), and total motility (60.21 ± 18.48 vs 39.67 ± 20.96%; $p < 0.001$). Progressive motility and morphology showed no significant differences. Sperm DNA fragmentation increased significantly with age, rising from 20.95 ± 13.81 in men <30 years to 35.18 ± 15.24 in those ≥40 years ($p = 0.008$). Men aged ≥40 years exhibited marked reductions in semen volume, concentration, and motility, alongside higher sperm DNA fragmentation, indicating that paternal aging adversely affects both conventional and molecular sperm quality.

Keywords: Paternal age, Semen quality, Sperm DNA fragmentation, Male fertility, Kurdistan-Iraq

Introduction

Infertility represents a significant health concern in the world. It affects 20% of couples of reproductive age, with male factors contributing to nearly half of the cases.[1] Alterations in semen parameters, such as reduced sperm motility, morphology, and concentration are key determinants of male infertility. In recent decades, a marked global decline in sperm quality has been documented, with environmental, occupational, and lifestyle factors proposed as major contributors.[2] Despite these observations, the mechanisms underlying this decline and the influence of advancing paternal age remain incompletely understood, particularly in populations with distinctive demographic and environmental profiles such as those in the Middle East.

The progressive increase in paternal age at the time of conception, largely due to socioeconomic and cultural shifts, has prompted growing interest in its reproductive consequences. While advanced maternal age is a well-established risk factor for adverse reproductive outcomes, the effects of paternal aging are only recently being recognized.[3,24] Several investigations have demonstrated that men above the age of 35–40 years exhibit measurable deterioration in conventional semen parameters, including lower semen volume, motility, and morphological normality.[2,4] However, other studies have reported inconsistent or negligible associations, underscoring the complexity of the relationship between male aging and fertility potential.[3]

Beyond standard semen parameters, increasing evidence indicates that paternal age adversely affects sperm chromatin integrity and DNA stability.[1] The sperm DNA Fragmentation Index (DFI) has emerged as a valuable biomarker of sperm genomic integrity, closely associated with fertilization capacity, embryonic development, and pregnancy outcomes.[5] Elevated sperm DFI levels are linked to early embryonic arrest, implantation failure, recurrent miscarriage, and a higher incidence of genetic and neurodevelopmental disorders in offspring.[1,5] Reactive Oxygen Species (ROS) accumulation, defective chromatin packaging, and impaired DNA repair mechanisms are thought to contribute to these age-related molecular alterations.[3]

Despite extensive global research, data from Iraq and the Kurdish population remain limited. The Kurdish

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community has a unique demographic and environmental profile characterized by comparatively high consanguinity rates, region-specific occupational exposures such as oil-industry–related pollutants and agricultural chemicals, and lifestyle patterns that include higher smoking prevalence among men. In addition, delayed fatherhood has become increasingly common due to socioeconomic factors. These characteristics may uniquely influence semen quality and DNA integrity. This study, therefore, aimed to assess the impact of advancing paternal age on semen quality and sperm DNA fragmentation among men in Kurdistan-Iraq.[6,25s]

Methodology

A cross-sectional, observational research was carried out over eight months, from September 1, 2024, to April 30, 2025. Data were obtained from five private fertility centers and andrology laboratories located in Erbil and Duhok, within the Kurdistan Region of Iraq.

A total of 245 men aged between 20 and 60 years were initially recruited. Participants either presented for routine fertility assessment or were partners of infertile women attending the centers due to female-factor infertility. Men with known reproductive system abnormalities—such as undescended testes or varicocele—or chronic medical conditions including diabetes mellitus, hypertension, metabolic syndrome, or chronic kidney disease were excluded. In addition, chronic smokers and heavy alcohol consumers were also excluded. After applying these exclusion criteria, 203 participants were eligible for final analysis.

Semen samples were obtained by masturbation into sterile containers following a period of 2–5 days of sexual abstinence. All samples were analyzed in accordance with the World Health Organization (WHO) laboratory manual for the examination and processing of human semen (2010). Parameters assessed included ejaculate volume (mL), sperm concentration (million/mL), total sperm count (million), total motility (progressive + non-progressive, %), progressive motility (%), and normal morphology (%). Participants were categorized into four age groups: <30, 30–34, 35–39, and ≥40 years.[7,23]

A sperm DNA fragmentation test was performed for a subset of 76 participants. The results were interpreted using standard thresholds for the DNA Fragmentation Index (DFI): <15% (low), 15–25% (intermediate), and >25% (high), and categorical comparisons were made accordingly.[8]

The study protocol was reviewed and approved by the Ethics Committee of the Kurdistan Board of Medical Specialties (KHCMS) Meeting Code 2719 granted on December 16, 2024. Written informed consent was obtained from all participants, and only de-identified data were used for analysis. Aggregated results are reported to maintain participant confidentiality.

Statistical analysis

Statistical analyses were performed using SPSS version 29 (IBM Corp., Armonk, NY, USA) with a significance level of $\alpha = 0.05$. A G*Power 3.1 indicated a minimum sample of 172 for one-way ANOVA ($f = 0.25$, power = 0.95); the study's 203 participants exceeded this. Normality was tested using the Kolmogorov–Smirnov test. One-way ANOVA assessed differences in semen parameters and DNA fragmentation across age groups, followed by Dunnett's post hoc test using the ≥40-year group as the reference. Additional analyses compared DFI across categorical levels.

Results

Sperm parameters

ANOVA results (Table 1) revealed that age significantly affected semen volume, sperm concentration, and total motility among men in the Kurdistan Region. Semen volume declined from 3.24 ± 1.40 mL in men under 30 to 2.07 ± 1.29 mL in those aged ≥40 years ($F = 10.919$, $p < 0.001$). Sperm concentration decreased with age ($F = 4.839$, $p = 0.003$), as did total motility ($F = 10.717$, $p < 0.001$). These findings indicate a clear age-related deterioration in key semen parameters that may contribute to reduced male fertility.

Table1: Effect of age on semen parameters (ANOVA Test)

Parameters	G. Age	N	Mean ± SD	F-test (p-value)
Volume	<30	45	3.24± 1.40	10.919 p(0.000**)
	30-34	28	2.22± 1.08	
	35-39	27	3.06± 1.22	
	≥40	103	2.07± 1.29	
Concentration	<30	45	34.78± 37.24	4.839 p(0.003**)
	30-34	28	55.03± 54.24	
	35-39	27	62.21± 48.87	
	≥40	103	31.25± 43.26	
Progressive Motility - PR	<30	45	16.32± 17.05	0.394 p(0.757)
	30-34	28	12.85± 17.32	
	35-39	27	14.55± 21.89	
	≥40	103	13.42± 14.04	
Total Motility - PR+NP	<30	45	45.52± 23.57	10.717 p(0.000**)
	30-34	28	58.24± 16.19	
	35-39	27	60.21± 18.48	
	≥40	103	39.67± 20.96	
Morphology	<30	45	2.69± 2.26	1.909 p(0.129)
	30-34	28	2.54± 2.29	
	35-39	27	2.26± 2.28	
	≥40	103	2.45± 1.92	

**Significant at level (p<0.01)

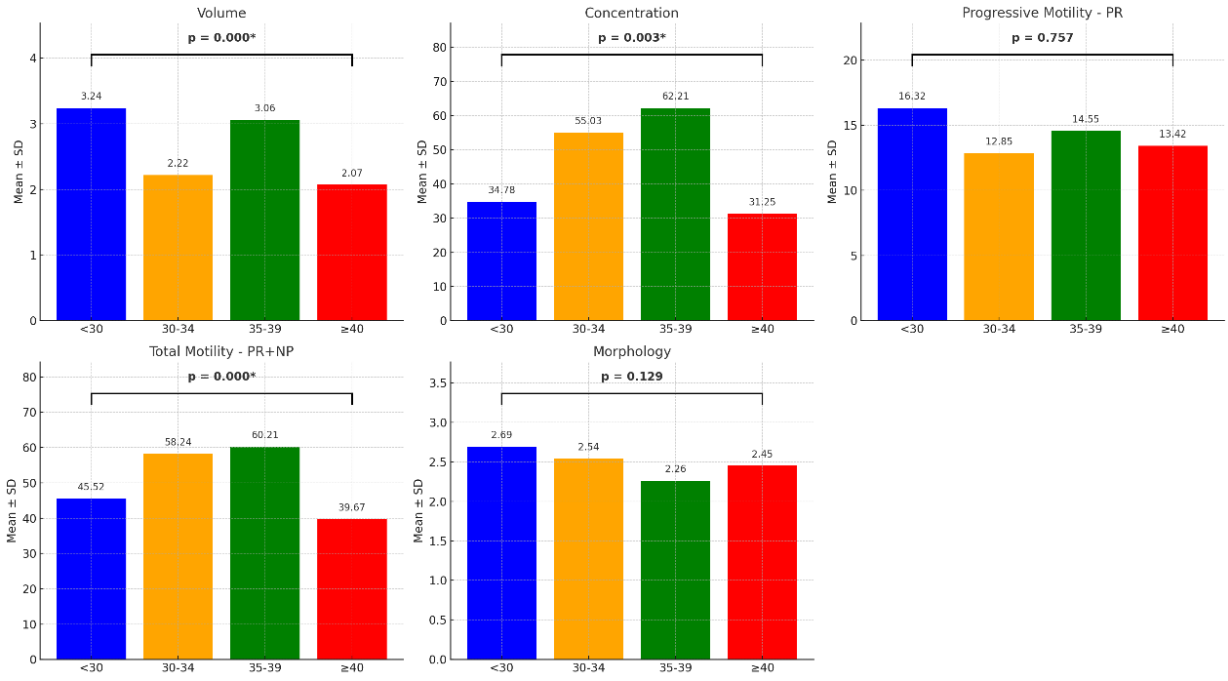

Figure (1) Distribution of age groups with semen parameters

Table (2) shows the Dunnett post hoc comparisons using the ≥40 age group as the reference. Men under

30 and those aged 35–39 had significantly higher semen volumes than men ≥40 years ($p < 0.01$). Sperm concentration was also higher in the 30–34 ($p = 0.037$) and 35–39 ($p = 0.004$) groups. Similarly, total motility (PR+NP) was significantly greater in both the 30–34 and 35–39 groups compared with men ≥40 years ($p < 0.001$).

Table2: Dunnett post hoc test for Age-Related differences in semen parameters

Parameter	Comparison	Mean Difference	Std. Error	p-value	95% CI Lower	95% CI Upper
Volume	<30 vs≥40	1.168	0.229	0.000 **	0.617	1.718
	30-34 vs≥40	0.153	0.274	0.919	-0.509	0.816
	35-39 vs≥40	0.985	0.278	0.001 **	0.319	1.65
Concentration	<30 vs≥40	-5.304	9.45	0.797	-15.533	4.926
	30-34 vs≥40	23.781	9.478	0.037 *	1.153	46.522
	35-39 vs≥40	30.996	10.677	0.004 **	7.922	54.071
Progressive Motility - PR	<30 vs≥40	2.897	2.929	0.677	-4.131	9.924
	30-34 vs≥40	-0.571	3.494	0.998	-8.952	7.811
	35-39 vs≥40	1.124	3.544	0.983	-7.379	9.628
Total Motility – PR+NP	<30 vs≥40	5.846	3.697	0.298	-3.024	14.748
	30-34 vs≥40	18.564	4.41	0.000 **	7.986	29.142
	35-39 vs≥40	20.534	4.473	0.000 **	9.803	31.266
Morphology	<30 vs≥40	0.798	0.344	0.062	-0.03	1.626
	30-34 vs≥40	0.379	0.407	0.717	-0.551	1.309
	35-39 vs≥40	0.459	0.419	0.605	-0.55	1.468

*Significant at level ($p<0.05$), **Significant at level ($p<0.01$)

Table 3: ANOVA test of fragmented with group age

G. Age	Mean± SD	F-value	p-value
<30	20.95± 13.81	4.306	0.008**
30-34	24.51± 10.05		
35-39	26.55± 13.59		
≥40	35.18± 15.24		

** Significant at level ($p<0.01$)

DNA analysis

Table (3) shows a significant rise in sperm DNA fragmentation with advancing age ($p = 0.008$). Mean fragmentation increased steadily from 20.95 ± 13.81 in men under 30 to 35.18 ± 15.24 in those aged ≥40 years, confirming a clear age-related increase in DNA damage.

Table (4) presents Dunnett post hoc results showing that sperm DNA fragmentation in men aged ≥40 years was significantly higher than in younger groups. The greatest difference was between <30 and ≥40 years (mean difference = −14.23, $p = 0.007$), followed by 30–34 vs. ≥40 years (−10.67, $p = 0.030$). No significant difference was observed between the 35–39 and ≥40 groups ($p = 0.120$).

Table (4) Multiple comparisons (Dunnett t-tests)

(I) G.Age	(J) G.Age	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
<30	≥40	-14.23095*	4.53284	.007	-25.2139	-3.2480
30-34	≥40	-10.67312*	4.07503	.030	-20.5468	-.7994
35-39	≥40	-8.63124	4.22926	.120	-18.8786	1.6161

*The mean difference is significant at the 0.05 level.

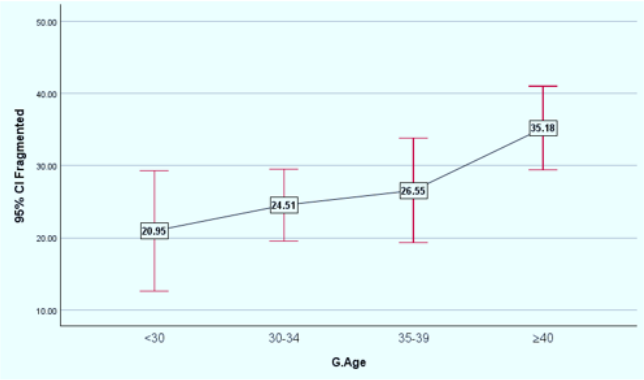

Figure (1) Distribution of age groups with average of DNA fragmentation

Table (5) shows a highly significant difference in DNA fragmentation across DFI categories (F = 94.963, p < 0.001). Mean fragmentation rose sharply from 11.18 ± 3.13 in the <15 DFI group to 43.28 ± 11.63 in the >30 DFI group, confirming that higher DFI categories are associated with substantially greater DNA damage.

Table 5: ANOVA test of fragmented with DFI category

DFI Category	Mean± SD	F-value	p-value
<15	11.18± 3.13	94.963	0.000**
15-30	21.68± 3.63		
>30	43.28± 11.63		

** Significant at level (p<0.01)

Table (6) shows post hoc comparisons revealing significantly higher DNA fragmentation in the >30 DFI group. The largest differences were observed between <15 vs. >30 (mean difference = −32.11, p < 0.001) and 15–30 vs. >30 (−21.61, p < 0.001), confirming a sharp rise in fragmentation at DFI values above 30.

Table (6) Multiple comparisons (dunnett t-tests)

(I) DFI	(J) DFI	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
<15	>30	-32.10516*	2.73296	<.001	-38.3004	-25.9100
15-30	>30	-21.60552*	1.92578	<.001	-25.9709	-17.2401

*The mean difference is significant at the 0.05 level

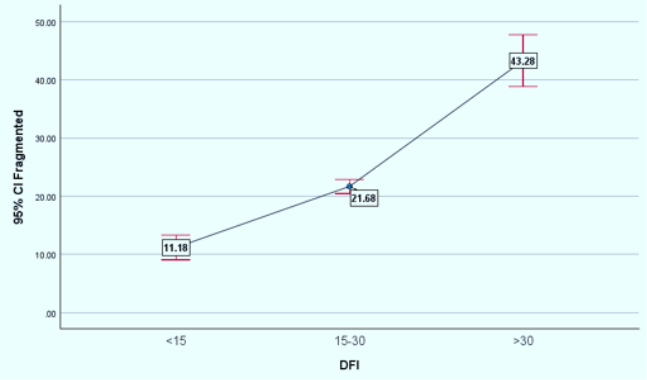

Figure (2) Distribution of DFI with DNA fragmentation

Discussion

This study examined age-related changes in semen quality and sperm DNA integrity among men aged 20–60 years in the Kurdistan Region of Iraq. Semen volume, concentration, and total motility declined significantly with age, while progressive motility and morphology remained largely unchanged. DNA fragmentation rose sharply among men aged ≥40 years, highlighting the dual structural and molecular effects of aging on male fertility. These findings indicate that aging affects both the functional output of spermatogenesis and the genomic stability of spermatozoa, suggesting that chronological age may influence fertility potential long before overt clinical subfertility becomes apparent. Our findings align with multiple international studies. Castellini et al.[9] reported a significant decline in motility beyond 40 years, while Sancı et al.,[4] observed lower sperm count, motility, and morphology among men aged 41–50 compared to younger groups.

Similarly, Pakmanesh et al. noted reductions in semen volume, motility, and morphology with age.[2] The relatively preserved sperm concentration in our cohort parallels their observation that this parameter remains stable until later decades, possibly due to lifestyle or environmental factors.

This stability may reflect compensatory testicular

mechanisms—such as sustained spermatogonial proliferation in early midlife—that temporarily maintain concentration despite declining tissue efficiency. Meta-analyses by Conti and Eisenberg,[10] and Sharma et al.[11] confirm that motility and morphology are the most age-sensitive parameters. Other studies by Pino et al.[12] and Lahimer et al.[13] also show increased DNA fragmentation in older men, reinforcing that sperm integrity declines even when conventional parameters appear normal. This pattern suggests that routine semen analysis may underestimate age-related reproductive risk, as molecular deterioration often precedes or outpaces visible changes in classical parameters. Our observed rise in DNA fragmentation mirrors Schmid et al.'s findings of age-dependent DNA damage in healthy non-smokers.[14] Peng et al. further demonstrated that high DNA fragmentation (>25%) adversely affects fertilization and IVF outcomes.[7] Mechanistically, age-related DNA damage arises from oxidative stress, reduced antioxidant defenses, and impaired DNA repair.[15,16] Mitochondrial dysfunction and reactive oxygen species (ROS) accumulation induce strand breaks and base oxidation, while epigenetic dysregulation and abnormal DNA methylation increase with age, as shown by Kotková and Drábek.[17] Taken together, these molecular alterations indicate that sperm aging is not a passive process but rather a multifactorial phenomenon driven by cumulative oxidative injury, destabilized chromatin packaging, and diminished genomic maintenance pathways. Several biological pathways contribute to these effects. Testicular aging involves seminiferous tubule sclerosis, reduced Leydig and Sertoli cell efficiency, and diminished spermatogenesis.[18] Hormonal changes—lower testosterone and higher FSH—further impair sperm production.[19] Oxidative stress and reduced antioxidant enzyme activity cause lipid peroxidation, damaging membranes and DNA integrity.[20] Cumulative exposures to toxins and inflammation exacerbates DNA fragmentation and epigenetic instability. These changes interact synergistically: endocrine decline weakens the spermatogenic milieu, oxidative imbalance damages maturing germ cells, and reduced Sertoli cell support impairs chromatin remodeling—all converging to worsen sperm quality with age. Comparable studies from Iran and Turkey reveal similar trends, with significant deterioration occurring after 40 years, suggesting a regional threshold.2,4 Western data, such as Stone et al.,21 identified earlier declines (around 34 years), likely reflecting lifestyle or genetic factors. The pattern of preserved morphology but reduced motility in our population is consistent with Castellini et al.[8] This difference in inflection points between populations may reflect sociocultural patterns, dietary habits, or environmental exposures such as heat, pesticides, and air pollution, which are known modulators of sperm function. The marked increase in DNA fragmentation aligns with reports by Rosiak-Gill et al.[1] and Xie et al.,[5] showing elevated DFI in men over 40, even with normal semen parameters. This underscores the clinical importance of DFI testing in fertility evaluation. Increased DNA fragmentation is strongly associated with reduced fertilization, higher miscarriage risk, and greater incidence of genetic and neurodevelopmental disorders in offspring.14 As shown by Sharma et al.11 and Caliskan et al.,[14] aging sperm contributes to de novo mutations, emphasizing male age as a key factor in fertility counseling and assisted reproduction. These findings reinforce the emerging concept that paternal age carries not only reproductive implications but also long-term health consequences for offspring, mediated through genomic instability, altered methylation patterns, and impaired DNA repair capacity.

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