

Original Research

Received 20 May 2026

Revised 05 July 2026

Accepted 20 July 2026

Natural Resources for Human Health 2026, Vol. 6 (13s) 594–602

KEYWORDS:

DNA fragmentation, oligospermia, Interleukin-17, Interleukin-33.

DNA Damage and some Interleukins in Oligospermia

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ABSTRACT: Male infertility is greatly exacerbated by oligospermia, which is characterized by a sperm concentration of less than 15 million/mL. It is often associated with an elevated sperm DNA fragmentation index (DFI), which indicates compromised DNA integrity. One major factor contributing to the increase in DFI is oxidative stress (OS), which is caused by an imbalance between reactive oxygen species (ROS) and antioxidants. Oxidative stress and male infertility are linked to inflammatory cytokines, such as interleukin-17 (IL-17) and interleukin-33 (IL-33). The aim of the study is to examine the relationship between sperm DNA integrity and inflammatory cytokines (IL-17 and IL-33) in oligospermic males as opposed to normospermia controls. Between November 2024 and March 2025, a case-control study was conducted in Baghdad to investigate the relationship between DFI and IL-17/IL-33 in 30 fertile controls and 50 oligospermic men. the result of seminal concentrations of IL-17 and IL-33 were markedly diminished in oligospermic males relative to controls ($P \leq 0.01$), The level of IL-17 was decreased highly significantly in oligospermic mean compared to healthy (0.6917 ± 0.146 pg/ml) , (0.8903 ± 0.112 pg/ml) respectively whereas for IL-33 it was (1.09 ± 0.195 pg/ml) , (1.184 ± 0.132 pg/ml) ,for IL-17 there was negative moderate correlation ($r = -0.47$) with sperm DNA quality while there was no discernible relationship for IL-33 ($r = 0.05$).

INTRODUCTION

Infertility is when a couple can't get pregnant despite having unprotected sex for a year (1, 2). About 10–15% of couples around the world are infertile (3, 4), and male factors are to blame for over half of these cases (5). The first test is a conventional semen analysis, which looks at the number, movement, and shape of sperm. However, this test often does not explain infertility because about 40% of infertile men have "normal" WHO-standard semen characteristics. This difference has led to more investigation into additional biomarkers and the causes behind them. (6). Oligospermia is a common kind of male infertility that is marked by a low sperm count (7). The World Health Organization (WHO) says that oligozoospermia is when there are less than 15×10^6 sperm per mL of semen, and less than 5×10^6 sperm per mL is considered severe (8, 9). There are many things that might induce oligospermia, such as varicocele (enlarged veins in the scrotum), genital infections, hormonal or genetic problems, and things in the environment (10, 11). Oligospermia is associated to infertility in the clinic, and people often need help with reproductive technology (12). Immune and inflammatory mechanisms can affect how sperm works. IL-17 is a pro-inflammatory cytokine that is linked to lower sperm quality. Some scientists looked into a negative correlation between IL 17A levels and Sperm DNA fragmentation (SDF). This means that higher IL 17A levels were linked to less DNA fragmentation (13). IL-33 has been shown in other studies to be an important alarmin that causes oxidative stress and lipid peroxidation in seminal fluid, which could harm sperm membranes and the general integrity of the sperm. So, even while high levels of IL-17 are linked to oligospermia and sperm damage, it's still not clear what IL-33 does in male infertility. The DNA Fragmentation Index (DFI) is a new diagnostic marker that shows the percentage of spermatozoa with fragmented (damaged) DNA (14, 15). Infertile patients have often been shown to have higher DFI. In a large study, the average DFI in men with normal sperm was about 15%, while the average DFI in men with significantly low sperm was about 26% (8). In oligospermia, higher DFI means

that the chromatin is less stable, which is linked to lower motility and lower odds of pregnancy success (16–18).

MATERIALS AND METHODS

From November 2024 to March 2025, 80 semen samples were obtained from donors at Al-Mansour and Al-Harthiya laboratories in Baghdad, Iraq. After conducting macroscopic and microscopic assessments, the samples were categorized into two groups: 50 oligospermic men and 30 healthy men. Samples were collected through masturbation following a 72-hour period of abstinence. From each sample, 500 μ l was promptly utilized for DNA fragmentation analysis, while the residual fraction was liquefied at room temperature or 37°C to decrease viscosity. Liquefied samples underwent centrifugation at 4,000 x g for 10 minutes to isolate seminal plasma, which was subsequently either tested immediately or kept under suitable conditions. The semen analysis adhered to WHO recommendations (19). Evaluating factors include sperm quantity, volume, motility, morphology, DNA fragmentation index (DFI), and concentrations of IL-17 and IL-33. The study's objective was explained to each participant, and their verbal consent was secured.

• *Assessment of DNA Fragmentation Index (DFI)*

Sperm chromatin dispersion was done according to (20). Intact unfixed spermatozoa (fresh, frozen/unthawed, diluted, or clean samples) are immersed in an inert agarose microgel on a pretreatment slide. The DNA in sperm cells exhibiting fragmentation is denatured by an initial acid treatment. The predominant nuclear proteins are subsequently removed by the lysis solution, which, in the absence of substantial DNA fragmentation, yields nucleoids characterized by extensive halos of dispersed DNA loops radiating from a central core. Nonetheless, the dispersion halo is either nonexistent or markedly diminished in the nucleoids of spermatozoa with fragmented DNA(36).

• *Assessment of interleukins, interleukin 17, and interleukin 33*

A sandwich ELISA was employed to measure IL-17 and IL-33 concentrations with commercially available microplate strips pre-coated with particular capture antibodies. Standards and samples were introduced into the wells, subsequently followed by HRP-conjugated detection antibodies to create antibody-antigen-antibody complexes. Following the washing process to eliminate unattached substances, tetramethylbenzidine (TMB) substrate was introduced, resulting in a colorimetric shift to yellow following the addition of sulfuric acid. Optical density (OD) was quantified at 450 nm utilizing a microplate reader, with the blank well serving as the reference standard. Cytokine concentrations were quantified by correlating optical density results with a standard curve. All measurements were conducted within 15 minutes following the cessation of the reaction to guarantee accuracy(4, 37) .

Statistical analysis

Statistical analysis was performed using a number of statistical packages includes Python, SPSS, and R package for windows. The probability was calculated using unpaired t-test or ANOVA test for parametric data and Mann-Whitney test or Kruskal-Wallis test for non-parametric data Correlation between parameters was calculated using a Pearson or Spearman correlation test. P-value of less than 0.05 was considered a cut-off for significant statistical difference.

RESULTS

• *Macro/Microscopic Characteristics diagnosis*

Based on macroscopic and microscopic examination 50 of 80 samples were oligospermia while 30 were controls. The ejaculate volume shows no significant difference ($P \geq 0.05$) for both oligospermia and control (2.732 ± 0.467 , 2.81 ± 1.109) ml. respectively, also the color of semen was showed no significant difference ($P \geq 0.05$) for both oligospermia and control (50,29) % as grayish color and (0,1) % as yellowish color respectively, as shown in (Table 1) .

The results of microscope seminal fluid analysis which showed in Table 2. appeared a highly significant difference decrease compared to healthy samples ($P \leq 0.01$) where the sperm count was (8.32 ± 3.934 million) , (66.2 ± 25.12 million) respectively whereas (3.18 ± 2.025 million/ml) , (26.77 ± 13.3 million/ml) for sperm concentration. For the motile sperms was decreased highly significantly in oligospermic mean compared to healthy ($10.82 \pm 8.918\%$) , ($69.17 \pm 10.83\%$) respectively while for immotile sperm it showed the opposite a highly significant increase compare to healthy ($89.18 \pm 8.918\%$) , ($30.83 \pm 10.83\%$) respectively and alongside the abnormal sperm ($82.9 \pm 12.5\%$) for oligospermia , ($23.67 \pm 8.298\%$) for control while the normal sperm showed ($17.1 \pm 12.5\%$) for oligospermia and ($76.33 \pm 8.298\%$) for control.

Table 1. Macroscopic characteristics of seminal fluid of Control and Oligospermia cases.

Characteristics		Control (n=30)	Oligospermia (n=50)	P value
Ejaculate volume (ml), mean $\pm$ SD		2.81 $\pm$ 1.109	2.732 $\pm$ 0.467	0.2454
Color, n (%)	Grayish white	29 (96.67)	50 (100)	0.1939
	Yellowish	1 (3.33)	0 (0)	

P > 0.05 Non Significant or. P $\leq$ 0.05 Significant

Table 2. Microscopic characteristics of seminal fluid of Control and Oligospermia cases

Characteristics		Control (n=30)	Oligospermia (n=50)	P value
Total sperm count (million), mean $\pm$ SD		66.2 $\pm$ 25.12	8.32 $\pm$ 3.934	<0.0001
Sperm conc. (million/ml), mean $\pm$ SD		26.77 $\pm$ 13.3	3.18 $\pm$ 2.025	<0.0001
Motility (%), mean $\pm$ SD	Motile	69.17 $\pm$ 10.83	10.82 $\pm$ 8.918	<0.0001
	Immotile	30.83 $\pm$ 10.83	89.18 $\pm$ 8.918	<0.0001
Morphology (%), mean $\pm$ SD	Normal	76.33 $\pm$ 8.298	17.1 $\pm$ 12.5	<0.0001
	Abnormal	23.67 $\pm$ 8.298	82.9 $\pm$ 12.5	<0.0001

P $\leq$ 0.05 Significant. or. P $\leq$ 0.01 Highly Significant

• DNA fragmentation estimation

the result of all states showed a significant difference between control and oligospermia cases as presented in (Table 3). All states or cases showed a significant difference with p-value < 0.0001 compared to p-value = 0.05, except small halo and big halo, the p-value was 0.0015 and 0.0005, respectively. Also Fig. 1, showing spermatozoa types of DNA fragmentation seen by SCA scope and Fig. 2, shows the Comparison of frequency of DNA fragmented and intact sperm between healthy control and Oligospermia.

Table 3. Comparison of DNA fragmentation characteristics between Control and Oligospermia

Characteristics	Control (n=30)	Oligospermia (n=50)	P value
DNA-fragmented sperm (%), mean $\pm$ SD	10.32 $\pm$ 3.66	57.92 $\pm$ 26.05	<0.0001
DNA-intact sperm (%), mean $\pm$ SD	89.73 $\pm$ 3.67	42.07 $\pm$ 26.07	<0.0001
Without halo sperm (%), mean $\pm$ SD	3.637 $\pm$ 1.399	34.77 $\pm$ 21.55	<0.0001
Small halo sperm (%), mean $\pm$ SD	23.49 $\pm$ 10.85	12.64 $\pm$ 9.38	0.0015
Medium halo sperm (%), mean $\pm$ SD	34.11 $\pm$ 12.29	11.87 $\pm$ 10.48	<0.0001

Big halo sperm (%), mean $\pm$ SD	34.04 $\pm$ 5.283	16.69 $\pm$ 14.38	0.0005
Degraded sperm (%), mean $\pm$ SD	6.077 $\pm$ 2.861	23.75 $\pm$ 15.83	<0.0001

P $\leq$ 0.05 Significant. or. P $\leq$ 0.01 Highly Significant.

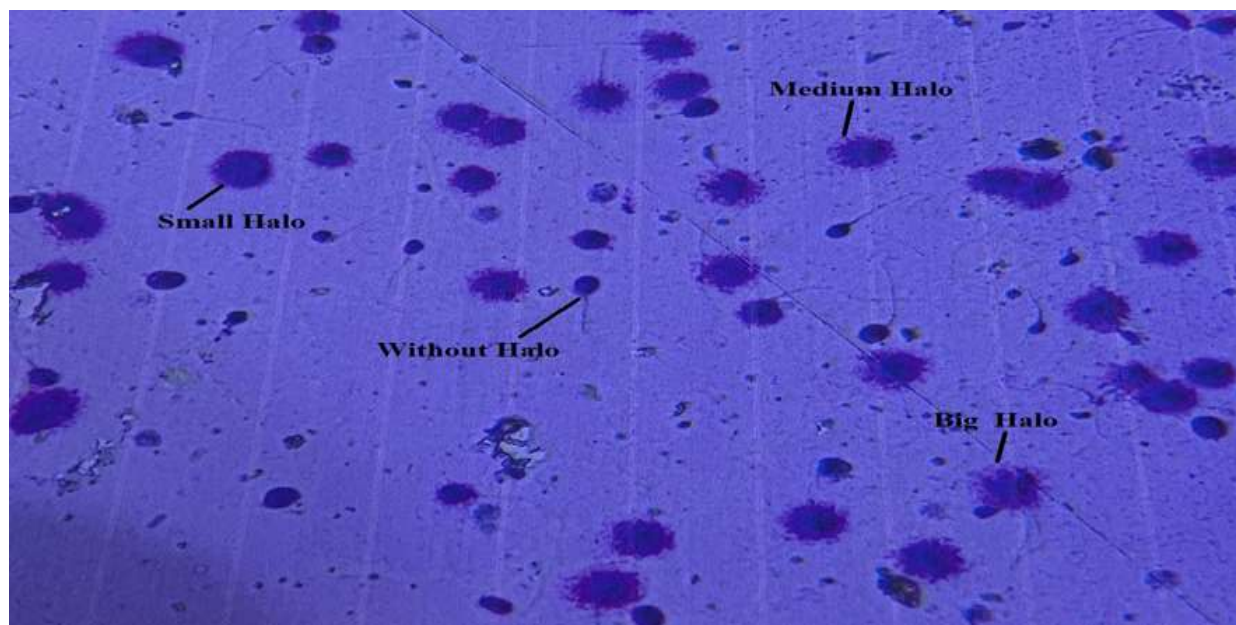


Figure -1 Four dispersion patterns of halo Sperm with big halo: the halo has a 2 times larger width than that of the sperm core, with a darker spot (sperm head) in the middle. Sperm with medium halo: having a halo size between big and small halos. Sperm with small halo: a very small, clear film, that has a halo appearance, surrounds the sperm head. Sperm without halo.

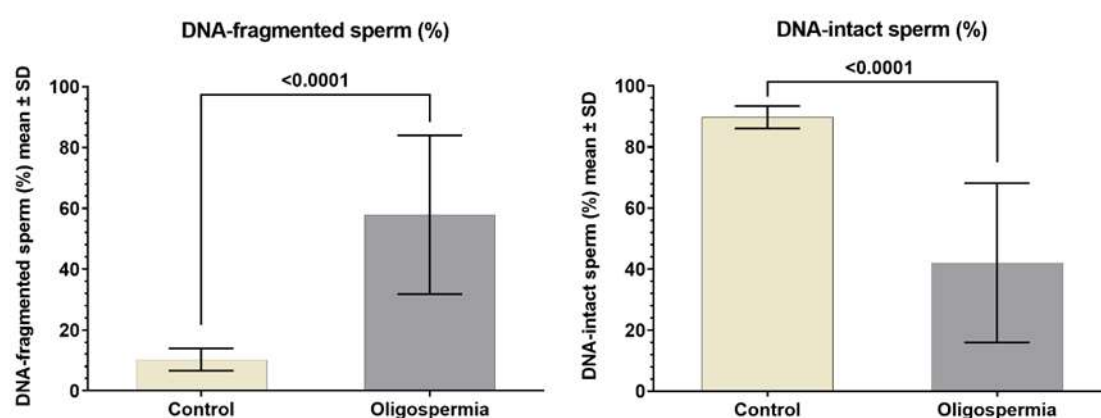


Figure -2 Comparison of frequency of DNA fragmented and intact sperm between healthy control and Oligospermia

• Immunological parameters evaluation

The result of IL-17 and IL-33 levels between control and oligospermia there was a significant difference between control and oligospermia for IL-17 and IL-33 concentrations the p value showed a highly significant difference for both of them with (P $\leq$ 0.01) for IL-17 there was a significant decrease in (0.6917 $\pm$ 0.146 pg/ml) for oligospermia compared to (0.8903 $\pm$ 0.112 pg/ml) for control. Also the result revealed that IL-33 decrease significantly (P $\leq$ 0.01) in oligospermia was (mean 1.09 $\pm$ 0.195 pg/ml) compared to control was (1.184 $\pm$ 0.132pg/ml) as shown in Fig. 3

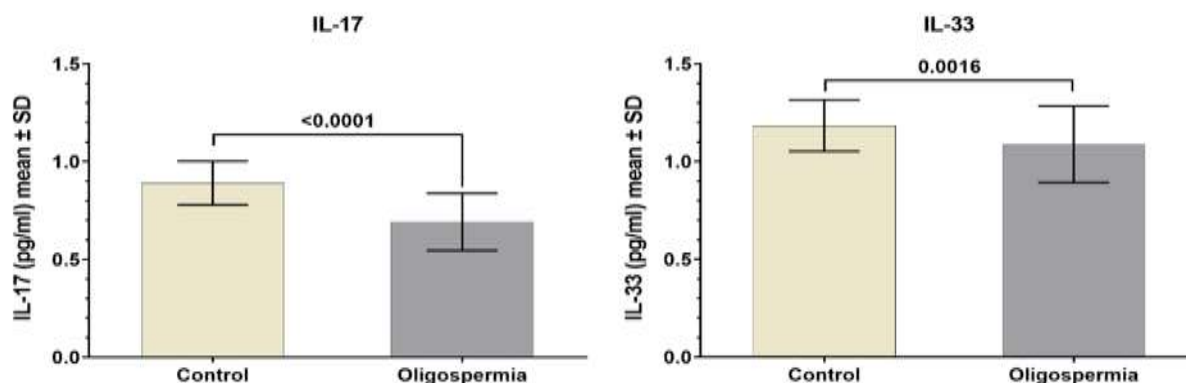


Figure -3 Interleukins concentration in healthy control and Oligospermia.

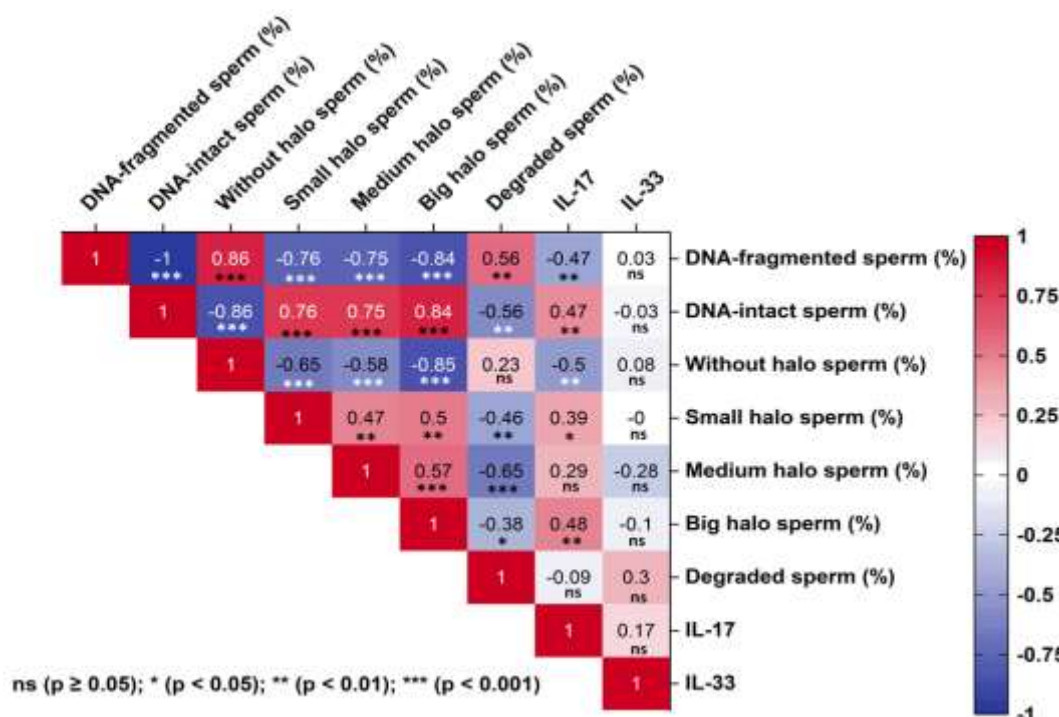


Figure -4 Heatmap shows correlation between DNA fragmentation and interleukins in Oligospermia. Numbers represent (r) value. Positive and negative r values represent positive and negative correlation respectively. R values of 0.9-1 (very high), 0.7-0.9 (high), 0.5-0.7 (moderate), 0.3-0.5 (low), and 0-0.3 (negligible) indicate level of correlation. P value of ≤ 0.05 (*), ≤ 0.01 (**), ≤ 0.001 (***)

Statistical correlation analysis

We connected the interleukins (IL-17 & IL-33) with DNA fragmentation index, the correlation of interleukin-17 with dna fragmented sperm shows negative correlation as shown in (Fig.4) ($r = -0.47$) which mean it's a negative moderate correlation while in interleukin-33 the result was ($r = 0.03$) as shown in (Fig.4) which mean a positive weak correlation. For the intact sperm it was the opposite for both parameters. As for degraded sperm the result revealed for interleukin-17 there is negligible level of correlation with ($r = -0.09$) while for interleukin-33 it showed a positive weak correlation.

DISCUSSION

The study found that the volume and colour of seminal fluid were not significant for either healthy men or men with oligospermia. This is because the usual range for semen volume is very different (1.5–6 mL). This range usually only has small

changes unless accessory glands are involved, so (22) says that there are no affected accessory glands. The colour usually doesn't change in oligospermia, but unusual colour is more likely to mean infection, blood, long periods of abstinence, or other health problems, not sperm count (22) as agreed with (23). The infertile group (oligospermia) had a much more DNA fragmentation than the control group (normospermia), with a p-value of less than 0.01. This is because (as established by many studies) more sperm DNA fragmentation is linked to infertility. DNA fragmentation is a good sign of sperm-count-related infertility even when traditional semen measurements are normal. This is because biological processes that directly damage sperm DNA are often the main causes of oligospermia, such as testicular dysfunction, defects in spermatogenesis, oxidative stress, and other problems (24, 25).

Our research showed that people with oligospermia had much lower sperm counts and concentrations than controls, as well as much lower motility and a higher rate of sperm with abnormal shapes. This profile shows the usual pattern of semen that is linked to male-factor infertility. A standard semen study looks at important things including the total number of sperm, their concentration, motility, and shape. Together, these things can tell you how well a guy can reproduce (26). WHO says that oligozoospermia is when the concentration is less than $15 \times 10^6/\text{mL}$. Our group of oligospermic people did far worse than this criterion in both count and concentration. Clinically significant low levels mean that males who go above the WHO limits ($\geq 15 \times 10^6/\text{mL}$ concentration, $\geq 40\%$ total motility, $\geq 4\%$ normal morphology) have far higher rates of conception. In a large group, males who fulfilled these reference criteria were around 70% more likely to get pregnant than men who did not (27). On the other hand, the sub-threshold traits of our patients show that they have a much lower reproductive viewpoint. In short, the much lower counts and concentrations in oligospermia show that spermatogenesis is much less effective and fertility is lower than in normospermia controls(27).

The oligospermic group had a significant drop in the number of motile sperm and a corresponding rise in the number of immotile sperm. Asthenozoospermia, or impaired motility, often happens at the same time as a lower sperm count. This means that the sperm are not working properly (28). A low sperm count already has negative consequences on reproduction, and decreased motility makes these effects more worse. more if there are sperm present, slow movement makes it far less likely that they will reach and fertilize the egg. Studies on fertility show that better results are linked to higher motility and overall motile sperm counts. Progressive motility showed a 65% higher chance of conception than those below that threshold (27). Because of this, the asthenozoospermia seen in our oligozoospermic people makes it very hard for them to have children. In real life, combined oligo-astheno patterns, which are frequently called "OAT syndrome," mean that a man has very bad infertility (29). The oligospermic group had a lot fewer normal sperm shapes and a lot more abnormal ones. The high rate of teratozoospermia in oligospermia in our group probably means that there is either a sickness in the testicles or something in the environment that is causing it. Studies show that men with several semen abnormalities (such as a lower count, motility, and abnormal morphology) had much worse total semen quality and reproductive potential compared to normozoospermic men (28, 29). There is a statistically significant difference in DNA fragmentation qualities between normospermia (control) and oligospermic men, which shows how oligospermia affects the integrity of sperm chromatin. The oligospermic group had a far higher percentage of DNA-fragmented sperm ($57.92 \pm 26.05\%$) than the control group ($10.32 \pm 3.66\%$; $P < 0.0001$). This big difference shows that oligospermia is linked to a big drop in sperm DNA integrity, which fits with other studies that found a link between high sperm DNA fragmentation and male infertility (30). The percentage of DNA-intact sperm, on the other hand, was much lower in people with oligospermia ($42.07 \pm 26.7\%$) than in the control group ($89.73 \pm 3.67\%$; $P < 0.0001$). This means that oligospermic semen samples have fewer sperm and lower-quality chromatin, which could make it harder for fertilization and embryo development to happen (31). The control group had a lot more small, medium, and big sperm DNA halos, which means that the DNA was more intact or less broken up. On the other hand, oligospermic males exhibited much lower percentages of medium and large halo sperm ($11.87 \pm 10.4\%$ and $16.69 \pm 14.38\%$, respectively). This supports the idea that this group had more chromatin disruption. Compared to controls ($23.49 \pm 10.85\%$; $P < 0.0001$), oligospermic samples had a lot less small halo sperm ($12.64 \pm 9.38\%$). This suggests that the sperm are becoming more fragmented. The studies show that sperm from people with oligospermia have both fewer sperm and sperm that are far less healthy, notably in terms of DNA integrity. These changes are necessary because sperm DNA fragmentation is linked to lower fertilization rates, lower embryo quality, a higher chance of miscarriage, and worse results in assisted reproductive technologies (ART). As a result, measuring DNA fragmentation in oligospermic patients may provide important diagnostic and prognosis information and should be part of the evaluation of male reproductive potential (24).

Higher levels of IL-17 in seminal plasma are usually connected to inflammatory disorders such varicocele or infection. These conditions often cause oxidative stress and make sperm less mobile and viable (32). We saw that IL-17 levels were lower in

oligospermia, which could mean that it has a unique non-inflammatory phenotype. This could mean that Th17 is less active or that there is localized immunodeficiency at the mucosal level. This could mean that the body's natural immune and mucosal defense systems aren't working well enough to support healthy sperm production. If the usual Th17/ $\gamma\delta$ T cell axis is messed up, it could make it harder to get rid of apoptotic germ cells, which would affect sperm production (33). On the other side, the drop in IL-33 levels in oligospermia could mean less alarmin signalling and slower tissue repair. Testosterone controls the production of IL-33 by mast cells, which helps men tolerate their immune systems in different situations (34). This suggests that lower IL-33 levels may mean that the connection between the endocrine and immune systems has changed. Figure 4 of this study reveals that there is a statistically significant link between interleukin-17 (IL-17) levels and the integrity of sperm DNA in men with low sperm counts. IL-17 had a moderate negative link with sperm DNA fragmentation and a moderate positive correlation with the fraction of sperm that had intact DNA. These results suggest that IL-17 may affect the DNA of sperm in men with oligospermia. Our results were likewise in line with (35). The results showed that the inflammatory cytokines IFN γ , IL-17A, and IL-1 β are bad for sperm quality because they generate mitochondrial ROS, lower sperm motility, and kill sperm. The current results suggest that higher levels of IL-17 in men with oligospermia may be linked to less DNA damage and better sperm chromatin integrity. This goes against the common view that pro-inflammatory cytokines are bad for male fertility. This finding is in line with (36). Research suggests that IL-17 may play a big role in the development of male infertility caused by inflammation and infection of the testis and male genital tracts. This means that IL-17 could be a good target for treatment, but more research is needed to find out exactly how IL-17 causes male infertility. The fact that IL-17 and DNA fragmentation are negatively related may mean that IL-17 has a protective or regulatory role in the testicular microenvironment. This could mean that the immune system is trying to protect itself from oxidative stress or DNA damage caused by spermatozoa death. Also, the positive link between IL-17 and DNA-intact sperm shows how important this connection is on a biological level. It suggests that IL-17 may help keep the genome stable during spermatogenesis or sperm maturation. We also looked at the relationship between interleukin-33 (IL-33) levels and the integrity of sperm DNA in oligospermic samples. We focused on DNA fragmentation and DNA integrity. Figure 4 shows that the correlation analysis found a very weak positive association between IL-33 and DNA fragmentation ($r = 0.03$) and a very weak negative link between IL-33 and intact sperm DNA ($r = -0.03$). However, both correlations were not statistically significant ($p > 0.05$), which means that there was no strong linear relationship between IL-33 levels and sperm DNA quality measurements in this group. These results imply that IL-33 doesn't have a big effect on the integrity of sperm DNA in men with oligospermia, which goes against (37). We found that IL-33 was not present in any of the semen samples we looked at. This suggests that a nuclear factor, rather than a released cytokine, may be involved in human seminal plasma, since our results showed that IL-33 was secreted but had no meaningful effect. IL-33 is known to be an alarmin cytokine that has a role in many inflammatory processes, but in this case, it doesn't seem to have a direct effect on sperm DNA fragmentation or protection. This is particularly surprising because inflammation and oxidative stress are known to damage sperm DNA, and IL-33 has been linked to inflammatory responses in the male reproductive system. There could be a number of reasons why no significant connections were found. There are many other ways that sperm DNA can be damaged, and IL-33 is just one of them. Other cytokines and oxidative stress mediators may also play a role. When looked at on its own, IL-33 may have less of an effect on DNA integrity because it may work with other pro-inflammatory or anti-inflammatory molecules. Second, the study's sample size may make it harder to detect subtle connections, therefore more research with larger groups is needed to confirm these results. The effects of IL-33 on male infertility may depend on the situation. For example, it may have more impacts on those with varicocele, infections, or systemic inflammatory illnesses.

CONCLUSIONS

This study shows a strong correlation between IL-17 levels and sperm DNA quality in men with low sperm counts. Higher IL-17 levels are linked to less DNA fragmentation and more intact sperm. The results point to a possible beneficial immunoregulatory effect of IL-17 in male reproductive health. More research is needed to understand its molecular role and how it might be used in clinical settings to assess and treat male infertility. The results do not show a strong link between IL-33 levels and the fragmentation or integrity of sperm DNA in men with low sperm counts. The data suggest that IL-33 might not be a good biomarker for checking the integrity of sperm DNA in this group. More research that looks at a wider range of inflammatory markers and studies how they work is needed to figure out what IL-33 might do for male reproductive health.

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