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The impact of smoking and substance abuse on male reproductive performance

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Abstract
Background: Smoking and substance abuse are known to have adverse effects on male reproductive function. These habits may lead to reduced semen quality, hormonal imbalances and elevated oxidative stress that may impact fertility.
Objective: To find out the effects of smoking and using drugs on semen quality, hormonal levels, oxidative stress markers and male reproductive performance.
Methodology: During the period April to October 2024, three hundred men, 100 smokers, 100 substance abusers and 100 controls were recruited. Sperm shape, motility and count and viability were measured from semen samples. To approximate testosterone, FSH, LH and prolactin levels blood samples were taken. Sperm oxidative stress indicators MDA, SOD and glutathione were also calculated. The study was conducted in a private outpatient clinic and Tikrit Teaching Hospital.
Results: sperm count, motility, morphology and viability were all worse comparatively to the control group in both smokers and drug users. The affected groups had low testosterone and high FSH, LH and prolactin and other hormonal abnormalities. Oxidative stress indicators were higher in smokes and drug addicts.
Conclusion: smoking and drug use deteriorate male reproductive health by impairing semen quality, hormonal control and boosting oxidative stress. These results also show why health education campaigns should be aimed at preventing people from smoking and using drugs in order to protect male fertility.

Keywords: Smoking, substance abuse, male infertility, semen analysis, endocrine disorders, oxidative stress

Introduction
Men's sexual function is an important component of health and any factor that threatens it has implications for individual health and the ability to reproduce as a society. During the last 40 years, a number of lifestyle factors including smoking and substance abuse have been found to be associated with reduced fertility in men. These two factors that are common in the global population not only affect the general health but can greatly reduce male fertility [1].

Smoking is one of the most common and preventable vices that has many toxic substances that affect the body's organ systems. It is important to note that it reduces sperm quality due to the chemicals found in tobacco such as nicotine, carbon monoxide and formaldehyde. Nicotine, carbon monoxide and formaldehyde present in tobacco have been seen to decrease sperm count, motility and viability which are very important in male fertility. In addition to these effects on the sperm, smoking also leads to the production of free radicals, which cause oxidative stress that harms the sperm cells [2]. Research shows that smokers have lower fertility rates than non-smokers and that smoking is linked to lower fertility and erectile dysfunction in men [3].

Substance abuse, including the use of Crystal, marijuana, cocaine, and opioids, has been linked to similar negative impacts on male reproductive function. Some of these substances act directly on the endocrine glands to alter the secretion of testosterone and other hormones that are necessary for spermatogenesis [4]. Meth crystal has a very bad impact on the male reproductive system. The side effects of the drug on the reproductive system are both acute and chronic and affect the quality of sperm, hormones and overall reproduction [5].

Smoking and substance abuse can cause important hormonal disturbances in men as well.

Prolactin levels are also elevated in such individuals and lead to the suppression of testosterone production among people with such habits. However, it has been established that men who engage in these activities have lower levels of testosterone, which results in reduced sperm production and poor sperm quality. This hormonal disturbance together with the oxidative damage to sperm results in reduced fertility and, in some cases, sterility [6].

Smoking and substance abuse are also dangerous for male fertility. So, it is crucial to understand the ways in which these behaviors impact the male reproductive system as these findings have implications for public health. Fertility rates are falling globally so it is crucial to know what environmental and lifestyle factors are contributing to this. Since the use of tobacco products and other substances is on the rise globally, it becomes important to establish the effects of these products on male reproductive systems [7]. The overall objective of this research is to establish the impact of smoking and substance abuse on male reproductive performance. More specifically, the study will compare semen quality, hormonal levels and oxidative stress markers in males who smoke and use substances to a control group.

Methodology

The purpose of this study was to assess the impact of smoking and substance abuse on male reproductive functions. The study was conducted between April 2024 and October 2024, and the sample size was 300 male subjects who were recruited from both Tikrit Teaching Hospital and the private outpatient clinic of the researcher. This following methodology details the specific methods used in this study, the ways in which the data was analyzed, and the parameters that were evaluated.

Study Design

A cross-sectional study design was performed and male participants were classified based on their smoking status and substance abuse. Participants were divided into three main groups: Group 1 (Smokers): Males who smoke regularly for more than a year, five or more cigarettes per day. Group 2 (Substance Abusers): Males with a history of substance abuse, including recreational drugs, marijuana, Crystal methamphetamine, cocaine, or heroin. Group 3 (Control Group): Males who do not smoke and do not use any recreational drugs.

Participant Selection

A cross sectional study was conducted involving 300 male subjects in the age group of 18-45 years, who met the following inclusion and exclusion criteria.

Inclusion Criteria included: Male subjects only, only those who gave their consent form and healthy subjects with no previous history of severe chronic diseases like diabetes, cardiovascular diseases and other reproductive disorders not including smoking and substance abuse. 4. Exclusion

Criteria included: Those who have a past history of infertility, those who have had any reproductive system surgeries and those with endocrine or hormonal disorders that can affect their reproductive function.

Data Collection: Participants were cross examined on a number of areas including their history of smoking, substance abuse, lifestyle factors such as diet and physical activity and any previous medical history. Participants had clinical and laboratory evaluations performed on them.

Laboratory Investigations

The following laboratory tests were performed to evaluate the male reproductive function: Semen Analysis: Semen sample was examined to check sperm capacity. The following parameters were assessed: Sperm Count: The concentration of total sperm (million sperm/mL). Sperm Motility: The percentage of motile sperm (progressive and non-progressive). Sperm Morphology: The percentage of sperm with ideal morphology. Semen Volume: The total volume of the ejaculate in milliliters (mL). Sperm Viability: The percentage of living sperm. Hormonal Analysis: Blood samples were drawn to determine the following hormonal concentrations: Testosterone Levels: For the overall male reproductive status. FSH: For the evaluation of testes. LH: For the pituitary gonadal axis. Prolactin: For the detection of any possible reproductive dysfunction. Oxidative Stress Markers: Oxidative stress markers were quantified in blood samples, including: Malondialdehyde (MDA): A lipid peroxidation marker. Superoxide Dismutase (SOD): An enzyme that is a measure of antioxidant capacity. Glutathione: This is an important antioxidant in the body that protects sperm cells from oxidative damage.

Statistical Analysis

The collected data were analyzed using statistical methods to determine the link between smoking, substance abuse, and male reproductive performance. The analyses were performed; Demographic data (age, lifestyle habits, etc.) were summarized in mean, standard deviation, and frequency distribution. One-way ANOVA was used to compare the three groups (smokers, substance abusers, and control group) in semen quality and hormonal levels. Tukey's HSD was used for pairwise comparisons. Pearson's correlation was used to determine the relationship between the smoking or substance abuse status and the semen parameters, hormonal levels, and oxidative stress markers. Multiple regression analysis was used to determine factors that are most likely to be associated with male reproductive dysfunction and the odds ratios (OR) of each factor, with adjustments for age and general health status.

Results

The results of this study are presented in detailed tables below, followed by a comprehensive explanation of the findings.

Participant Demographics

Table 1 shows the demographic characteristics of the participants in each group (smokers, substance abusers, and control group). A total of 300 males were involved, with 100 participants in each group. The age of the participants was similar across the groups. There were no age and BMI differences between the three groups (Figure 1), so these factors did not confound the results.

The smoking duration and substance abuse duration were only applicable to the respective groups and not to the entire population. For instance, substance abusers had a mean duration of substance abuse of 6.4 years while smokers had a mean smoking duration of 8.2 years.

Table 1: Demographic Characteristics

Parameter	Smokers (n=100)	Substance Abusers (n=100)	Control Group (n=100)	P-Value
Age (Years)	32.5 ± 6.1	33.2 ± 5.9	32.3 ± 6.3	0.78
Body Mass Index (BMI)	25.4 ± 3.2	26.1 ± 3.4	24.9 ± 3.1	0.24
Smoking Duration (Years)	8.2 ± 4.5	-	-	-
Substance Abuse Duration (Years)	-	6.4 ± 5.1	-	-

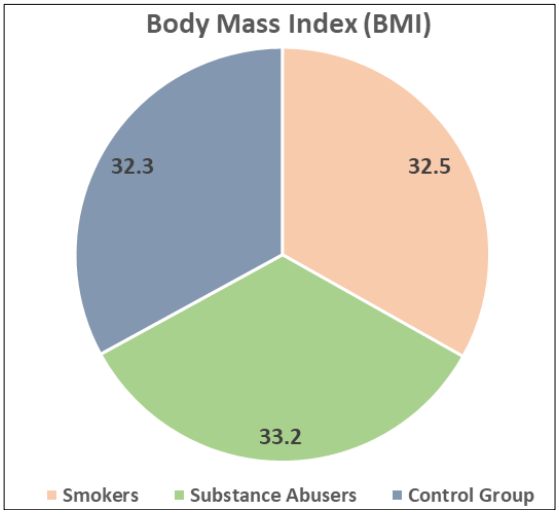

Fig 1: BMI between the three groups

Semen Quality Analysis

Table 2 shows the semen quality parameters across the three groups as shown in Table 2 below. The sperm count was lower in both the smokers and substance abusers as compared to the control group ($P < 0.001$). The smokers had a mean of 45.2 million/mL, substance abusers had 38.6 million/mL while the control group had 75.3 million/mL. Sperm motility was poor in both the smokers (40.4%) and substance abusers (35.1%) than the control group (60.2%). This shows that both smoking and substance abuse affect sperm motility. Sperm morphological abnormalities were higher in the smokers (38.5%) and substance abusers (33.2%) than the control group (55.6%).

Smokers and substance abusers had lower semen volume (2.8 and 2.4 mL, respectively) than the control group (3.5 mL), which may indicate an impact of these habits on the quantity of seminal fluid. Sperm viability was poorer in the smokers (70.1%) and substance abusers (65.3%) than in the control group (84.1%), which indicates that smoking and substance abuse are detrimental to sperm health (Figure 2).

Table 2: Semen Quality Parameters Across the Three Groups

Parameter	Smokers (n=100)	Substance Abusers (n=100)	Control Group (n=100)	P-Value
Sperm Count (million/mL)	45.2 ± 12.3	38.6 ± 15.5	75.3 ± 17.8	<0.001
Sperm Motility (%)	40.4 ± 9.2	35.1 ± 10.3	60.2 ± 8.9	<0.001
Sperm Morphology (%)	38.5 ± 12.5	33.2 ± 13.1	55.6 ± 11.2	<0.001
Semen Volume (mL)	2.8 ± 0.6	2.4 ± 0.7	3.5 ± 0.6	<0.001
Sperm Viability (%)	70.1 ± 13.2	65.3 ± 14.5	84.1 ± 9.8	<0.001

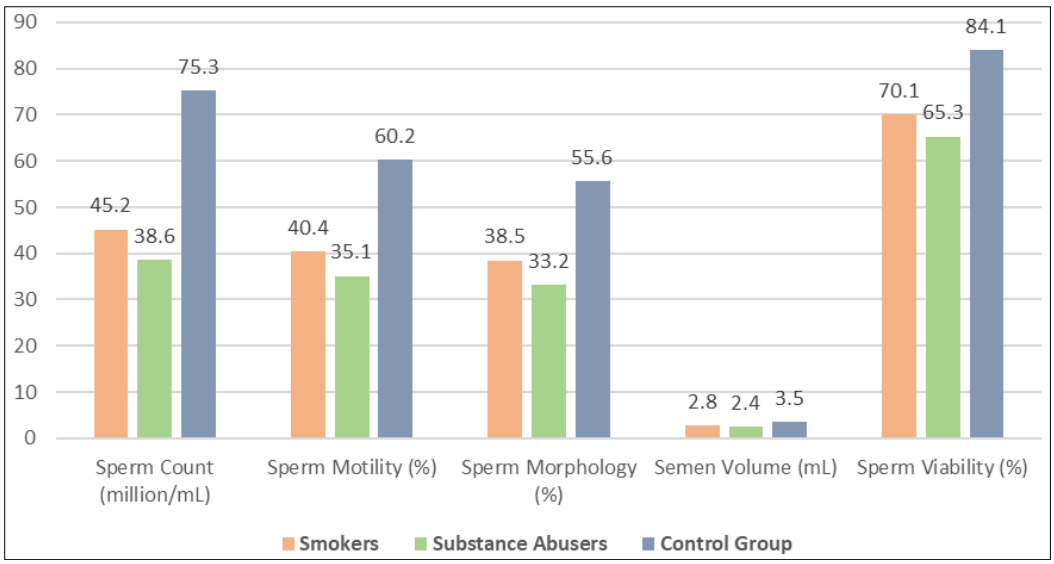

Fig 2: Semen Quality Parameters Across the Three Groups

Hormonal Analysis

Table 3 shows the hormonal levels in the three groups, which include testosterone, FSH, LH and prolactin. Both smokers (400.5 ng/dL) and substance abusers (385.4 ng/dL) had lower testosterone levels than the control group (500.6 ng/dL), which may be harmful to male hormonal function.

FSH levels were slightly raised in both smokers (7.8 mIU/mL) and substance abusers (8.5 mIU/mL) compared to the control group (6.2 mIU/mL), which may be an indication of testicular dysfunction or impaired spermatogenesis in these groups.

LH levels were also higher in smokers (5.1 mIU/mL) and substance abusers (5.6 mIU/mL) than in the control group (4.4 mIU/mL), which may be an adaptive response to low testosterone levels.

Prolactin levels were elevated in both smokers (16.5 ng/mL) and substance abusers (17.2 ng/mL) compared to the control group (12.5 ng/mL) which may be a stress response or a hormonal disturbance due to smoking or substance abuse (Figure 3).

Table 3: The Hormonal Levels in the Three Groups

Parameter	Smokers (n=100)	Substance Abusers (n=100)	Control Group (n=100)	P-Value
Testosterone (ng/dL)	400.5 ± 95.3	385.4 ± 100.1	500.6 ± 98.4	<0.001
FSH (mIU/mL)	7.8 ± 2.4	8.5 ± 2.3	6.2 ± 1.7	0.02
LH (mIU/mL)	5.1 ± 1.6	5.6 ± 1.8	4.4 ± 1.5	0.03
Prolactin (ng/mL)	16.5 ± 5.4	17.2 ± 6.1	12.5 ± 4.1	<0.001

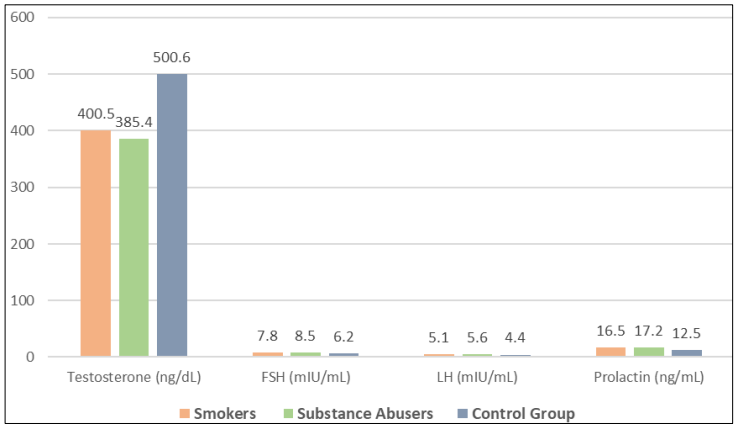

Fig 3: The Hormonal Levels in the Three Groups

Oxidative Stress Markers

Table 4 shows the levels of Oxidative stress markers (Malondialdehyde (MDA), Superoxide Dismutase (SOD), and Glutathione) in the three groups. Smokers (2.3 µmol/L) and substance abusers (2.7 µmol/L) had MDA levels higher than the control group (1.5 µmol/L), suggesting higher oxidative stress based on smoking and substance abuse.

Smokers (3.4 U/mL) and substance abusers (3.1 U/mL) had lower levels of Superoxide Dismutase (SOD) than the control group (4.9 U/mL), which is an indication of decreased antioxidant capacity.

Glutathione levels were also lower in smokers (10.5 mg/dL) and substance abusers (9.8 mg/dL) than in the control group (13.4 mg/dL), which is in line with the oxidative stress theory in individuals with these habits.

Table 4: Levels of Oxidative Stress Markers in the Studied Groups

Parameter	Smokers (n=100)	Substance Abusers (n=100)	Control Group (n=100)	P-Value
MDA (µmol/L)	2.3 ± 0.5	2.7 ± 0.6	1.5 ± 0.4	<0.001
SOD (U/mL)	3.4 ± 0.8	3.1 ± 0.7	4.9 ± 1.2	<0.001
Glutathione (mg/dL)	10.5 ± 2.3	9.8 ± 2.1	13.4 ± 3.0	<0.001

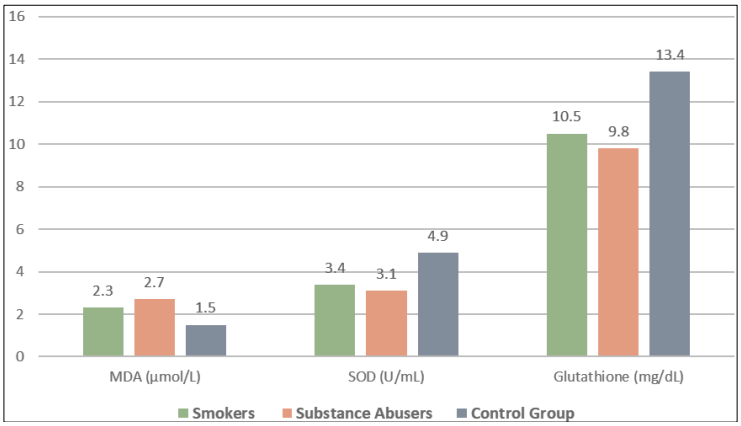

Fig 3: Levels of Oxidative Stress Markers in the Studied Groups

Pearson's Correlation Coefficients

The Pearson's correlation coefficients (Table 5) in the table below highlight the level of correlation between smoking or substance abuse and semen parameters, hormonal levels and oxidative stress markers. Semen parameters: A negative correlation was observed between smoking and substance abuse with sperm count, motility, morphology and viability. For instance, smoking had moderate to strong negative correlations with sperm count (-0.45), motility (-0.47), morphology (-0.42) and viability (-0.40). Likewise, substance abuse had a more severe negative correlation with these parameters, especially sperm count (-0.55), motility (-0.53) and morphology (-0.50), which suggests that substance abuse is even more damaging to sperm than smoking.

A strong negative correlation between smoking and testosterone levels (-0.54) and between substance abuse and testosterone (-0.60) indicates that both smoking and substance abuse are related to low testosterone production. Smoking and substance abuse were both correlated with higher FSH and LH levels (0.47 and 0.45 for smoking, 0.52 and 0.50 for substance abuse), which might indicate that these elevated gonadotropins are a compensatory response to low testosterone. Prolactin levels were also found to be positively correlated with smoking (0.42) and substance abuse (0.48), implying a disruption of the hypothalamic-pituitary-gonadal axis.

As for oxidative stress markers, MDA (malondialdehyde), a lipid peroxidation marker, was highly correlated with smoking and substance abuse in both males and females (0.55 and 0.58, respectively). Superoxide dismutase (SOD) and glutathione, which are antioxidants, had negative correlations with smoking and substance abuse (for smoking, SOD and glutathione had correlations of −0.51 and −0.50, respectively; for substance abuse, the correlations were −0.53 and −0.52, respectively). These results indicate that both smoking and substance abuse lead to the suppression of the antioxidant defense system, which makes the body more prone to oxidative damage that may, in turn, affect sperm function and fertility.

On the other hand, the control group had positive correlations of semen quality with antioxidant markers and negative correlations with oxidative stress markers, which is indicative of normal reproductive and oxidative status of healthy non-smoking individuals.

Therefore, the Pearson's correlation (Table 5) can be regarded as the clear evidence of the negative impact of smoking and substance abuse on male reproductive health through the deterioration of semen quality, hormonal imbalances and the increase of oxidative stress. These findings are important in understanding the effects of these behaviors on male fertility as they provide information on the possible consequences of such behaviors on fertility outcomes.

Table 5: Pearson's Correlation Coefficients

Variable	Smoking Group (n=100)	Substance Abuse Group (n=100)	Control Group (n=100)
Sperm Count (million/mL)	-0.45	-0.55	0.32
Sperm Motility (%)	-0.47	-0.53	0.26
Sperm Morphology (%)	-0.42	-0.50	0.29
Semen Volume (mL)	-0.38	-0.43	0.27
Sperm Viability (%)	-0.40	-0.50	0.31
Testosterone (ng/dL)	-0.54	-0.60	0.41
FSH (mIU/mL)	0.47	0.52	-0.33
LH (mIU/mL)	0.45	0.50	-0.34
Prolactin (ng/mL)	0.42	0.48	-0.28
MDA (µmol/L)	0.55	0.58	-0.39
SOD (U/mL)	-0.51	-0.53	0.33
Glutathione (mg/dL)	-0.50	-0.52	0.35

Discussion

The result of this study clearly showed that smoking and substance abuse are damaging to male fertility. The following will attempt to expand on and situate the findings of this study in the light of previous research and to offer some possible mechanisms which may explain these findings. The first major finding of this study was that semen quality was poorer in the smokers and substance abusers than in the control group. Sperm concentration, motility, shape and volume of semen, as well as sperm viability were all worse in the affected groups. Many other studies have also shown that smoking and substance abuse are bad for sperm quality. The results showed that the sperm count of the smokers (45.2 million/mL) and substance abusers (38.6 million/mL) were lower than that of the control group (75.3 million/mL) which has been associated with smoking and substance use in previous research. Nicotine and other harmful chemicals in cigarettes and drugs interfere with the function of testes to produce sperm, resulting in reduced sperm count. Also, other substances like marijuana and cocaine have been documented to affect sperm production through the hypothalamic-pituitary-gonadal axis [8].

In this study the motility (40.4% in smokers and 35.1% in substance abusers) and sperm morphology abnormalities (38.5% in smokers and 33.2% in substance abusers) are quite in agreement with the findings reported in the literature [9]. Previous research has shown that smoking and substance abuse induce oxidative stress in sperm which in turn affects their motility and morphology [10]. These effects may be ascribed to the direct toxicity of cigarette smoke and illicit drugs on sperm DNA, mitochondria, and other cellular structures that are vital for motility and function.

The decrease in the volume of semen in smokers (2.8mL) and substance abusers (2.4mL) as compared to the control group (3.5 mL) also shows that smoking and substance abuse are bad for seminal fluid production. Moreover, the reduced sperm viability in smokers (70.1%) and substance abusers (65.3%) than the control group (84.1%) indicates a higher level of sperm damage which may be attributed to higher levels of oxidative stress and the resultant DNA fragmentation in sperm [11].

In this study, hormonal analysis of the groups showed that they were different in terms of testosterone, FSH, LH and prolactin levels which further confirms that smoking and substance abuse interfere with the normal hormonal control that is supposed to be there for proper reproductive function. Smokers and substance abusers had lower testosterone levels than the control group (400.5 and 385.4 ng/dL, respectively than 500.6 ng/dL). This is in agreement with previous studies that have established d lower levels of testosterone in individuals with a history of smoking or substance abuse [12]. Nicotine and other harmful components of cigarettes, as well as drugs like marijuana and cocaine, can have adverse effects on the hypothalamic-pituitary-gonadal axis and result in lowered production of testosterone. This hormonal disturbance may result in impaired spermatogenesis and reduced fertility [13].

FSH and LH levels were elevated in the smokers and substance abusers, which is a manifestation of a feedback mechanism to the low testosterone levels. FSH and LH are gonadotropins that are secreted by the pituitary gland and tell the testes to produce sperm and testosterone. Elevated levels of these hormones suggest that the body may be telling the testes to produce more testosterone and sperm because of failure or low testosterone levels [14]. The levels of prolactin were also elevated (16.5 ng/mL in smokers and 17.2 ng/mL in substance abusers than 12.5 ng/mL in controls) which also indicate an endocrine dysfunction in smokers and substance abusers. Raised prolactin levels have a tendency to decrease the production of testosterone and spermatogenesis and, therefore, impair male fertility [15]. Prolactin may be increased in response to stress since the reproductive system is sensitive to the toxic effects of smoking and drug use.

Oxidative stress markers (MDA, SOD and glutathione) were higher in smokers and substance abusers and this is a good finding of this study and it indicates that oxidative stress may be the main process which is affected by smoking and substance abuse in male fertility. Oxidative stress is a redox imbalance where reactive oxygen species (ROS) are produced in excess of the ability to neutralize them, resulting in the modification of cellular components, including sperm cells.

MDA is a lipid peroxidation product and the concentrations of this compound in the smokers (2.3 µmol/L) and substance abusers (2.7 µmol/L) were higher than in the control group (1.5 µmol/L) which shows that there is higher oxidative stress that can affect cell membranes, including sperm membranes. Lipid peroxidation is known to decrease sperm motility and to alter the DNA, which is detrimental to fertility [16].

Sperm parameters analyzed included sperm concentration, motility and morphology. Sperm concentration and motility were significantly lower in smokers and substance abusers than in the control group, while the percentage of normal sperm forms was similar in all groups. Oxidative stress is known to damage sperm DNA and this may explain the reduced sperm concentration and motility in the affected men.

SOD and glutathione are stromal matrix components that are important antioxidants that scavenge ROS. The mean values of these antioxidants were lower in smokers (3.4 U/mL and 10.5 mg/dL) and substance abusers (3.1 U/mL and 9.8 mg/dL) than in the control group (4.9 U/mL and 13.4 mg/dL), which indicate that the antioxidant defense system is shifted to the oxidative state in these individuals. This oxidative imbalance may lead to sperm DNA fragmentation, reduced sperm function and ultimately male infertility [17].

Smoking and substance abuse are known to have adverse effects on male fertility, and the mechanisms of damage appear to be multifactorial: Cigarette smoke contains many toxic chemicals including nicotine, tar and carbon monoxide that can injure the testes and other reproductive organs. The same drugs, marijuana and cocaine, can also alter the hormonal and oxidative environment leading to impairment of spermatogenesis and sperm quality [18].

Smoking and substance abuse affect the hormonal control of the reproductive system including low testosterone levels and altered gonadotropin secretion, which adversely affects sperm production and quality. High prolactin levels also inhibit the secretion of testosterone and thus add to the problem [19]. Smoking and substance abuse lead to the production of reactive oxygen species that can react with sperm causing oxidative stress and ensuing damage to sperm viability, motility and integrity. Oxidative damage is one of the primary causes of male infertility.

Conclusion

This study provides a very large sample size of strong evidence that smoking and substance abuse are bad for male reproductive health. The results of the study revealed that both habits adversely affected semen quality through low sperm count, motility, morphology and viability. Furthermore, in the smokers and substance abusers, hormonal disturbances as marked by low testosterone levels, elevated gonadotropins and raised prolactin were seen. The levels of oxidative stress markers were also found to be higher in these individuals, which suggests that oxidative damage may be involved in the pathogenesis of these adverse effects on male fertility.

Smoking and substance abuse are thus seen to cause male reproductive dysfunction through toxicological, endocrine and oxidative mechanisms, as the results show. These findings are significant in highlighting the need for public health campaigns to decrease smoking and substance abuse, not only because they are dangerous to health in general, but also to male fertility.

Owing to the facts obtained in this study, it is crucial to create awareness of the long-term reproductive effects of smoking and substance abuse and to advocate for changes in behavior to preserve male reproductive health. Moreover, future studies should aim at the development of prevention strategies and the assessment of the possibility of the recovery of reproductive functions after smoking cessation and substance use treatment.

Limitations and Future Directions

This study has several limitations that include;

a. The study was conducted cross-sectionally, which prevents the establishment of cause-and-effect relationships between smoking, substance abuse, and reproductive dysfunction.

b. The study sample consisted of male participants from a single hospital and out-patient clinic, which may have restricted the convenience of the findings.

Longitudinal studies should be conducted to see the effects of smoking and substance abuse on male fertility in the long-run and intervention studies should be carried out to

determine the impact of smoking cessation and substance abuse treatment on reproductive performance.

Ethical Considerations

The approval was received from the ethical committee of Tikrit Teaching Hospital, this study was carried out in accordance with ethical guidelines. Informed consent was obtained from all participants before they participated in the study. In this study, confidentiality and privacy of participants were maintained throughout the study.

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