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# Effects of Alcohol and Tobacco Consumption on Semen Quality in Healthy Men

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**Abstract-** The definition of infertility is the failure of a sexually active, non-contraceptive couple to get pregnant within a year. Semen analysis is the cornerstone of evaluating infertility since it tells us how well the seminiferous tubules, epididymis, and accessory sex glands are working. Given that there is a lack of knowledge about male infertility in India, it is crucial to be aware of the many risk factors linked to infertility.

**Methods:** 177 healthy men's semen samples were obtained. The individuals were divided into groups based on their alcohol intake and smoking habits. The examination of the sperm was done in accordance with WHO standards. Utilizing statistical analysis SPSS-22 Results was used to examine the data.

**Results:** A lower sperm count was linked to heavy smoking (> 20 cigarettes per day). An increase in sperm with morphological abnormalities was shown to be connected with moderate/high alcohol use ( 15.4 g/day). Significant increases in plasma membranes and architecturally aberrant nuclei were seen in alcohol-consuming individuals.

**Conclusions:** Heavy cigarette smoking was linked to lower sperm counts, while drinking alcohol was linked to more sperm with morphological abnormalities.

***Index Terms/ Keywords***- Alcohol consumption; Infertility, Male; Semen Analysis; Smoking; Spermatozoa.

## INTRODUCTION

A meta-analysis of Asian males during the previous 35 years revealed a decline in sperm concentration of around half, and poor semen quality was linked to male subfertility (Sengupta et al., 2017). According to data gathered by Boivin et al. (2007), around 15% of couples who are of reproductive age struggle with infertility. Patients using assisted reproductive technology (ART) in NortheastChina had male-only causes contributing 18% of the time and combined male and female infertility contributing 20% of the time (Fanget al., 2018). The two main categories of infertility patterns are primary and secondary; the former describes an inability to conceive after a year, while the latter describes an inability to conceive after a year after having fathered one or more biological children (Mohamed et al., 2017). According to earlier research, between 2012 and 2016 in China, the percentage of primary infertility fell while the proportion of secondary infertility rose (Fang et al., 2018). Both intrinsic (such as genetic and congenital abnormalities) and extrinsic (such as lifestyle and environmental variables) factors may contribute to male infertility (Wang et al., 2018). Male infertility has received more attention recently due to changeable lifestyle choices made by both patients and infertility professionals. Semen quality in

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males with primary or secondary infertility may be impacted by lifestyle variables such as dietary habits, physical activity levels, occupational traits, smoking, and alcohol use, according to many publications (Jensen et al., 2014; Boeri et al., 2019). Men with secondary infertility may experience an ever-increasing decrease of semen quality due to the detrimental effects of an unhealthy lifestyle, indicating that secondary infertility is more often linked to harmful lifestyle choices than basic infertility (Katib et al., 2014).

Due to the fact that drinking is so prevalent among adult males, research has concentrated on understanding how alcohol use affects spermatogenesis and male fertility (Ricci et al., 2018). According to some writers, drinking negatively affects semen characteristics such semen volume, total sperm count, progressive motility, and normal morphology (Boeri et al., 2019). However, contradictory findings from other research have been reported. For instance, sperm quality and moderate alcohol use (4–7 units/week) were shown to be positively correlated (Ricci et al., 2018).

An vast variety of disorders, ranging from anatomical issues [1] to unfavorable lifestyle and environmental variables [3–7], may cause male infertility. Age, nutrition, exercise, stress, use of illicit and prescription medicines, alcohol use, cigarette smoking, frequent caffeine intake, preventative care, and occupational and environmental exposures may all have an effect on fertility [5]. Men's germ lines experience oxidative stress due to environmental or lifestyle variables, which raises the likelihood that their offspring would develop congenital disorders including cancer and other congenital illnesses [8]. Toxic chemicals are among these lifestyle and environmental variables that affect spermatozoa, although up to this point, research have produced conflicting findings [5,9,10]. Such studies were either carried out on healthy or infertile males [11]. In terms of bad lifestyle habits, drinking alcohol and smoking are often at the top of the list. Numerous prior research noted that smoking has negative effects on sperm quality, hormonal imbalance, larger concentrations of seminal leukocytes, and its relationship with elevated reactive oxygen species (ROS) levels [12,13]. However, other studies that included fertile smoking men with children [14] disprove the harmful impact of smoking on male reproductive health. According to Mostafa, some smokers' reproductive potential may not be impacted by smoking, however quitting smoking would benefit smokers with poor semen quality [13].

In addition, tobacco smoke included a number of mutagens that might have an impact on future generations. The DNA integrity and viability of spermatozoa may be impacted by genotoxic chemicals found in cigarette smoke [15]. It has been noted that binge drinking often (5 episodes in the previous month)[9] has been linked to higher ROS production[16]. When ethanol is metabolized in the body, one of the byproducts is acetaldehyde, which interacts with proteins and lipids to cause peroxidation of lipids and protein adducts, respectively [17]. According to Maneesh et al findings, .'s oxidative stress in alcoholics' testes considerably reduces testosterone and antioxidant capabilities while raising levels of lipid peroxidation (LPO) [18]. Additionally, Varshini et al. came to the conclusion that drinking alcohol had a deleterious impact on sperm DNA damage [19]. Regarding the processes of alcohol-induced oxidative damage to the male reproductive system, there is relatively little information in the literature. Numerous studies have so far shown a negative correlation between infertile men's semen parameters and unbalanced seminal plasma antioxidant or oxidative stress markers [20–23]. The majority of these investigations evaluated and validated the tight association between

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the blood and seminal plasma antioxidant activity and the sperm profile in infertile males [24,25].

Blood antioxidants were shown to be related with semen quality in infertile men as opposed to fertile (healthy) controls, comparable to seminal plasma antioxidants [24]. These results imply that blood antioxidants may serve as a useful diagnostic tool for assessing the possibility for high-quality sperm [26]. Since the relationship between blood antioxidants and sperm quality has received less attention, this research largely focused on that relationship. This study sought to determine a relationship between sperm quality and the level of oxidative damage experienced by smoking and alcohol-exposed infertile men. Sperm quality, male reproductive hormone levels, oxidative stress, malondialdehyde (MDA), nitric oxide (NO), and antioxidant profiles that included superoxide dismutase (SOD) and reduced glutathione (GSHr) were assessed.

Infertility affects over 15% of all couples attempting to conceive, and in nearly 50% of these instances, male infertility is the only or a significant reason [1]. The concern of declining male fertility is not unfounded; data show that sperm concentration has been declining over the last 35 years [2]. In order to reduce the socioeconomic costs of male infertility and the ensuing burdens on public health, it is crucial to discover preventable causes. These studies have renewed interest in the possible influence of environmental variables and lifestyle choices on fertility. A more thorough analysis of the effects of sports and physical exercise on male fertility is covered in this Special Issue. Sedentary behaviour and obesity have both been linked to reduced male fertility [3, 4] and often discussed as possibly preventive variables. Other unhealthy habits, such smoking and drinking, as well as environmental stressors are known to have an impact on overall health, but their consequences on male fertility are less well understood [5].

In fact, male reproductive health may be a sensitive indicator of environmental exposures and pollution [6, 7]. Interventional research on the consequences of tobacco use, passive smoking, recreational drug use, and alcohol use are often not possible in people for ethical reasons. Because of this, most research on these subjects is retrospective [8]. Sadly, this results in a variety of confounders over which there is little control. Animal studies provide a partial answer, but since human exposure is far lower than that in these models, conclusions should be regarded with care. We conducted a full literature study of the available literature and collected all required information in order to give an up-to-date and trustworthy reference about the potential impact of alcohol, cigarettes, and recreational drugs on male fertility.

## CAUSES OF MALE INFERTILITY

Failure to get pregnant after at least a year of regular, unprotected sex is what is meant by couple infertility. On the other hand, definingmale infertility is more challenging since the diagnosis is often dependent on semen analysis findings that are compared to reference levels established by the World Health Organization [9].

Endocrine, paracrine, and autocrine components interact intricately to produce successful spermatogenesis [10]. Unsurprisingly, a number of inherited and congenital disorders may impede the delicate processes involved in spermatogenesis (Table 1). Aazoospermia is often linked to acquired testicular failure, which is frequently seen after testicular torsion, orchitis, or the administration of cytotoxictherapy. Varicocele, testicular injuries, and medicines may all have

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an impact on fertility, however spermatogenesis is often only slightly reduced in these situations. Small nucleotide polymorphisms are being looked into as a potential cause of "idiopathic" oligo-azoospermia, which is a genetic abnormality that typically manifests as azoospermia. These genetic abnormalities include Klinefelter Syndrome and microdeletions in the AZF (azoospermia factor) region on the human male **Y chromosome.**

However, despite recent advances in our understanding of the genetics of male infertility, the majority of the reasons of oligozoospermia remain unidentified as of this writing.

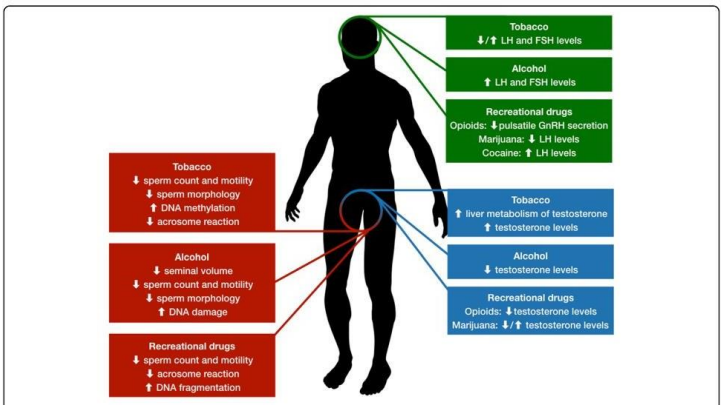


Fig 1: Effects of Smoking Alcohol and Drug Abuse in males

Age is strongly correlated with a reduction in semen quality, which is thought to be caused by the continuing reproduction of spermatogonial stem cells that have undergone mutation [11]. The pathophysiology of male infertility may also include DNA fragmentation and chromatin condensation [12]. The most well-known non-genetic cause of male infertility is oxidative stress, which is brought on by an excessive synthesis of reactive oxygen species (ROS). Capacitation, the acrosome reaction, and eventually fertilization all need ROS; yet, both inadequate clearance and excessive generation of ROS may cause DNA damage and compromised membrane integrity in sperm cells, lowering their reproductive potential [13]. The antioxidant capacity of sperm from fertile men is higher than that of sperm from infertile men, and immature teratozoospermic forms generate comparatively more ROS than normal, mature sperm [13]. Any infertile guy must have a complete assessment to see if any predisposing factors are present, including inflammatory processes and vascular illnesses like varicocele that may enhance ROS generation.

## Tobacco smoking and male fertility

Smoking is one of the risk factors for more than 60% of noncommunicable illnesses, and tobacco use and secondhand smoke are responsible for more than six million deaths annually [14]. Recent reports from the World Health Organization show that smoking is still a common practice despite the growing body of research that supports its harmful consequences. Worldwide, more than one-third of all individuals who are male smoke cigarettes [15]; similarly, around 30% of women who are of reproductive age do so [16]. While smoking rates in the United States have recently progressively decreased, Europe continues to be the most tobacco-use-heavy region [17].

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## CLINICAL STUDIES

Since 1983, smoking has been linked to negative effects on fertility. Olsen and colleagues [18] found tobacco use as one of the factors contributing to more than 1000 ladies' otherwise unexplained infertility. More than 4700 distinct compounds, including heavy metals, polycyclic aromatic hydrocarbons, and carcinogenic substances, have been found in cigarette smoke as of this writing [19]. Smoking is regarded as the most prevalent form of lead and cadmium exposure, and studies have shown a substantial correlation between seminal plasma lead levels and lifetime smoking estimates [20, 21]. Arsenic, along with the previously mentioned cadmium and lead, is a common metal micronutrient that is breathed during the burning of tobacco or cigarette paper and is implicated in the pathophysiology of oxidative stress and male infertility [21]. Despite the fact that there are no appreciable changes in semen volume, concentration, or motility, all of these metals have carcinogenic qualities and are similarly linked to an elevated risk of male infertility [22, 23].

On the other hand, sperm parameters have been shown to be impaired in several investigations over the last few decades [24, 25]; in the majority of them, smokers have been found to have altered morphology and lower concentration, motility, and viability. A meta-analysis of more than 2500 males from five different studies found that current smokers' sperm concentrations were significantly lower than those of men who had never smoked [26]. Similar to this, 2100 males arriving for a reproductive assessment who smoked had lower sperm concentration, according to research by Kunzle and colleagues [27].

Although mechanisms causing poor sperm parameters have been studied, conclusive proof is still lacking. Heavy smokers have been shown to have ultra-structural anomalies, particularly impacting axonemal microtubules and tail modifications. Smoking also affects the acrosome response and capacitation, two procedures ultimately necessary for fertilization. Impaired sperm functioning may be caused by increased oxidative stress, according to certain theories. Smoking-related hypoxia may also contribute to decreased spermatogenesis, and this effect may be particularly pronounced in individuals with varicocele [8, 32]. Numerous toxins may damage the mitochondrial function and chromatin structure of human sperm, adversely reducing their ability to fertilize both in vivo and in vitro[17, 33, 34].

Theoretically, long-term cigarette smoking enhances the liver's ability to process testosterone while also causing Sertoli and Leydig cells to develop secretory dysfunction [24]. There does not seem to be an agreement on the effects of smoking on the production of FSH and LH, however, since some researches have shown lower levels of both gonadotropins in smokers while other studies have found higher concentrations of LH and/or FSH after smoking [35, 36]. Given all the potential confounding variables, measuring testosterone concentration in smokers is remarkably challenging: some studies have found higher levels of dehydroepiandrosterone and serum testosterone in smokers [35, 37], whereas others have found that the mean testosterone levels between smokers and non-smokers are not significantly different [38].

Numerous civilizations regularly use alcoholic beverages, and about 60% of adults in the world who are 15 years of age and older have reported doing so in the last year [1]. According to a poll by the European Commission [2], 76% of people in Europe had drunk alcoholic drinks

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in the year before. When many alcoholic drinks are consumed quickly and the blood alcohol level is close to 0.08 g/dL, this is known as binge drinking [3]. In the United States, around 26% of adults reported binge drinking, and almost 15 million adults over the age of 12 had an alcohol addiction problem, according to the National Institute on Alcohol Abuse and Alcoholism [4]. According to estimates, 4% of adults worldwide are impacted by this practice [5].

Heavy drinking is defined as having more than three or four drinks per day, or more than seven or fourteen drinks per week, for men and women, respectively. It has been shown to have a negative impact on human health, increase the risk of traffic accidents, and change social behaviors, with serious ramifications for personal, social, and professional lives [5]. Clinical studies have shown a link between alcohol use and an increased risk of several cancers [6], cardiovascular ailments [7], liver diseases [8], birth abnormalities [9], and mental problems [10]. Alcohol misuse may lead to alcohol dependency; this is a dynamic and complicated process that is influenced by biological, socioeconomic, and environmental variables [11–13]. Alcohol generally has an effect through reducing the nervous system's activity. This becomes apparent during withdrawal from substance addiction, which is marked by the system's own hyperactivation, leading to tachycardia, hypertension, profuse sweating, tremors, and convulsions [14]. Rapid mood swings, irritability, agitation, anxiety, sleep issues, and an inability to enjoy pleasure (anhedonia), as well as a decrease in pain tolerance, are other prevalent signs [15,16].

To determine the consequences of alcohol misuse on the male reproductive ability, several clinical research on alcoholic men have been carried out [17–20]. The secondary sexual traits might initially change, including the amount of body and facial hair decreasing, the size of the breasts increasing, and the buildup of fat tissue from the abdomen to the hips [17]. Following this, a proinflammatory condition is established, which is shown by increased leukocyte numbers in the semen [18]. Alcoholics often lament sexual problems or infertility from a functional perspective [19,20]. Understanding the factors contributing to alcohol-related male infertility is crucial since many male drinkers are of reproductive age and may be interested in having children [1].

This research seeks to describe alcohol metabolism and the molecular processes through which ethanol (EtOH) might cause cellular damage in order to explore the effects of alcohol use on male fertility. Additionally, we address data from research on humans and animals about the effects of EtOH intake on semen quality, sexual hormonal regulation, and epigenetics, with a focus on the effects on children. Finally, we discuss the evidence's shortcomings and future research directions for locating the molecular indicators of alcoholism-related male infertility.

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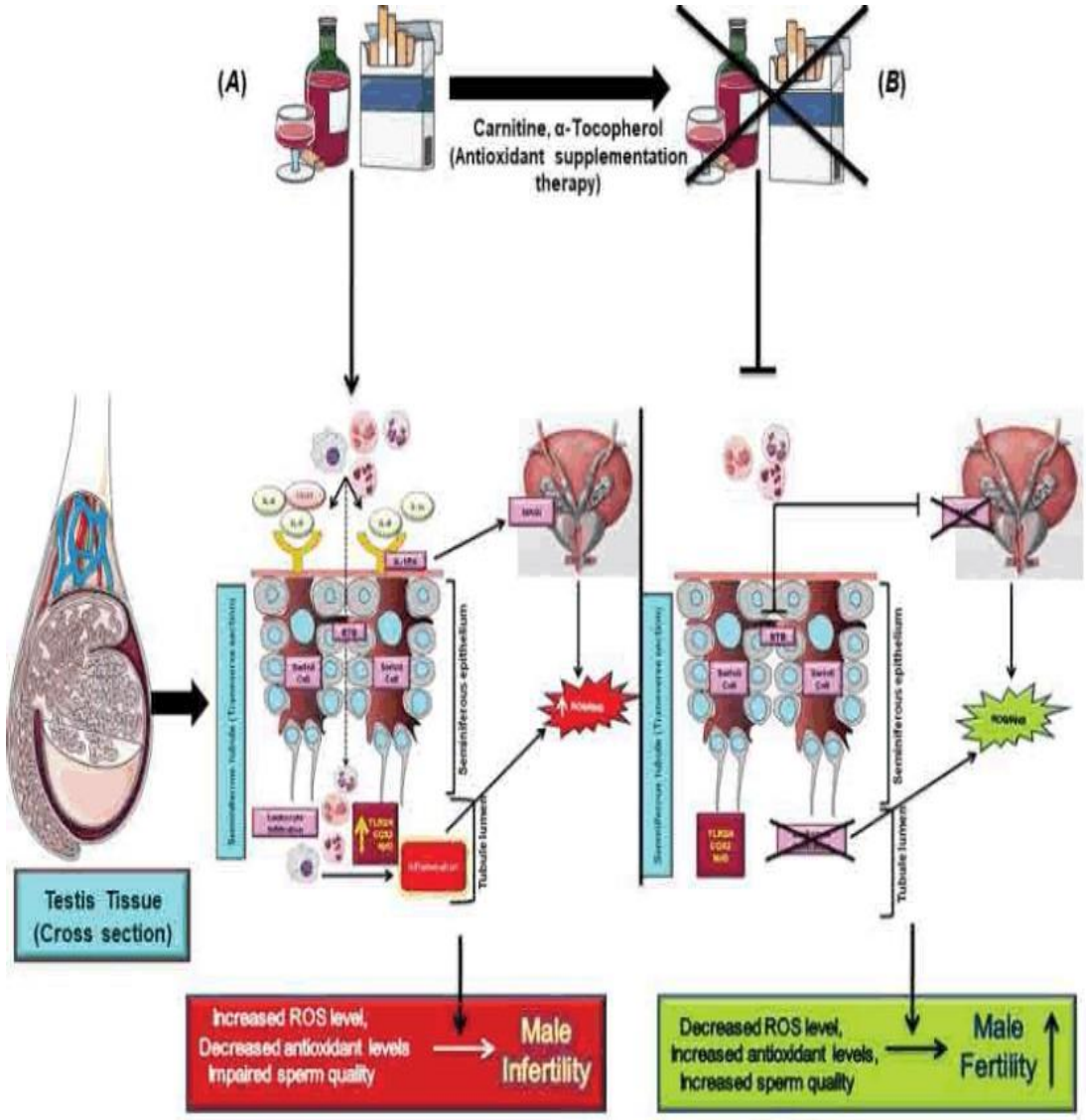


Fig 2: Possible mechanisms of the inflammation mediated ROS in male reproductive organs (A) smoking and alcohol use effects, cigarette and alcohol trigger infiltration of leukocytes through the blood-testis barrier mediated by cytokines, chemokines, and upregulation of TLR-2/4, COX-2, and Nrf-2 expressions. This results in inflammation of testis and male accessory gland infection (MAGI) by increasing ROS generation. (B) effects after antioxidant therapy without smoking and alcohol use- Without smoking and alcohol drinking condition, maintains redox homeostasis with normal leukocyte concentration. IL- interleukin, IL-1Ra- interleukin-1 receptor antagonist, BTB- blood-testis barrier, MAGI- male accessory gland infection, TLR2/4- toll-like receptor 2/4, COX-2- cyclooxygenase-2, Nrf2- nuclear factor erythroid 2 (NFE2)-related factor 2, ROS/RNSreactive oxygen/nitrogen species

## MATERIAL & METHODS

An analytical cross-sectional study was undertaken in Pathology laboratory for a period of 6-month duration from May 2020 to October 2020 at a tertiary care hospital in Gurugram. According to ICMR guidelines patients' data will be delinked.

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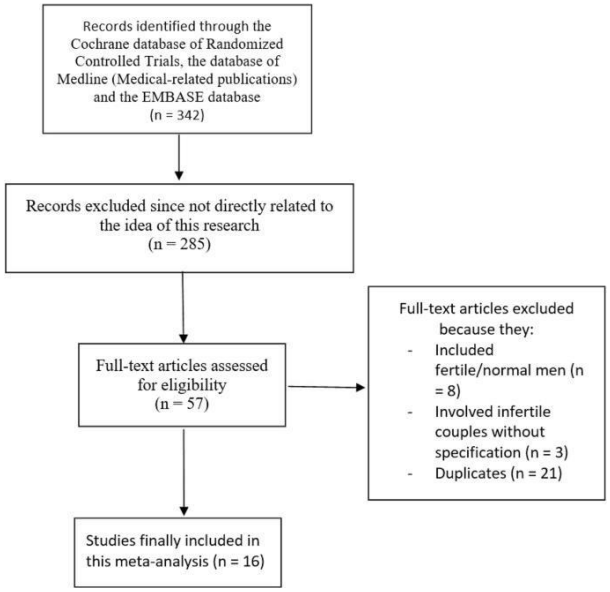


Fig 3: Methodology

## AIMS AND OBJECTIVES

1) To study sperm characteristics among consumers of tobacco and alcohol along with non-consumers undergoing semen evaluationin a tertiary care hospital.
2) To grade tobacco users and alcohol consumers as mild, moderate and severe.
3) To compare the sperm characteristics between tobacco smokers, tobacco chewers and alcohol consumers.
4) To find out the correlation between grade of tobacco usage and alcohol consumption and sperm characteristics.

## COLLECTION OF SAMPLES

After carefully documenting the history of alcohol and cigarette use, a questionnaire was completed. The patients were given an explanation of the study's methodology and their part in it. Before incorporating individuals in the research, a signed informed consent was obtained from them. Patients were given detailed instructions on how to collect semen samples, with an emphasis on the need of 3-5 days of abstinence. The patient was informed of the significance of the sperm-rich first component of semen and how its loss might affect outcomes.

In order to prevent significant temperature fluctuations, the sample was collected in a labelled, sterile container that was maintained at 37°C. Semen appearance, liquefaction, viscosity, volume, and pH were evaluated after samples were allowed to liquefy in accordance with World Health Organization (WHO) guidelines. [9] Using a Neubauer's chamber and light microscopy, the sperm count was performed. The resorcinol technique, in which fructose interacts with resorcinol in concentrated hydrochloric acid (HCl) solution to generate a red chemical, was used to measure the fructose concentration in seminal plasma. At a 560 nm

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wavelength, compare the zincand fructose caloric combination to control samples.

## MICROSCOPY

Semen samples were examined for sperm density, sperm motility, sperm morphology. Sperm morphology was studied on Leishman stained smear, counting a minimum of 200 spermatozoa using high power lens. Sperm morphology and motility was assessed using WHO criteria: A-Progressive motility (PN), B- Non progressive motility (NP), C-Immotility (IM).

Of the 240 cases reported over a period of 6 months, 52 cases were selected for control group (A) and study groups-62 Alcohol users (B), 63 Smokers (C),27 Tobacco chewers (D) and 36 those with all three addiction (E). The control and the case will be selected from patients coming for semen analysis in department of Pathology.

All three addictions are classified into different grades according to the level of consumption.

1. **Group A (Control group):** non-alcohol consumers and non-tobacco chewers or smokers.
2. **Group B:** Exclusively alcohol users (Nonsmokers/ non tobacco chewers)
    a) Mild alcohol consumer – those consuming 40gm or less alcohol per day
    b) Moderate alcohol consumer – consuming 40-80gm alcohol per day
    c) Heavy alcohol consumer – consuming more than 80 gm alcohol per day (40 gm = 60 ml alcohol – 70%)
3. **Group C:** Exclusively Smoker (Non-alcohol consumer and non-tobacco chewer) a. Mild - 1 to 9 Cigarettes per day b.Moderate-10 to 19 Cigarettes per day c. Severe-More than 20 Cigarettes per day
4. **Group D:** Exclusively Tobacco chewer (Nonsmoker and non-alcohol consumer) Mild- Tobacco chewing < 3 times/dayModerate- 3 to 6 times/day Severe- > 6 times/day
5. **Group E: Those** have all three addiction- alcohol and tobacco (smoker) and tobacco(chewing).

**Inclusion criteria:** During the research period, patients in the 20–year age range were sent to the pathology department for the examination of semen. Exclusion standards:

1. cases of abnormal male genital anatomy.
2. Previous conditions or procedures affecting reproductive function, such as vasectomy and vasectomy reversal; varicocele,cryptorchidism, epididymitis, mumps, and azoospermia.
3. Chemical exposure at work
4. persistent renal failure, diabetes, high blood pressure, or TB
5. Situations when the subject was unwilling to agree.

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Analytical Statistics utilising SPSS software (Statistical Package for Social Services) version 26 to interpret and analyse an observation. Quantitative information was expressed as frequency and percentage. Quantitative information expressed as mean, SD, and mean percent. Observation tables were created after statistical analysis of the given data. Microsoft Word, Excel, and the statistical software SPSS-26 were used for the statistical analysis.

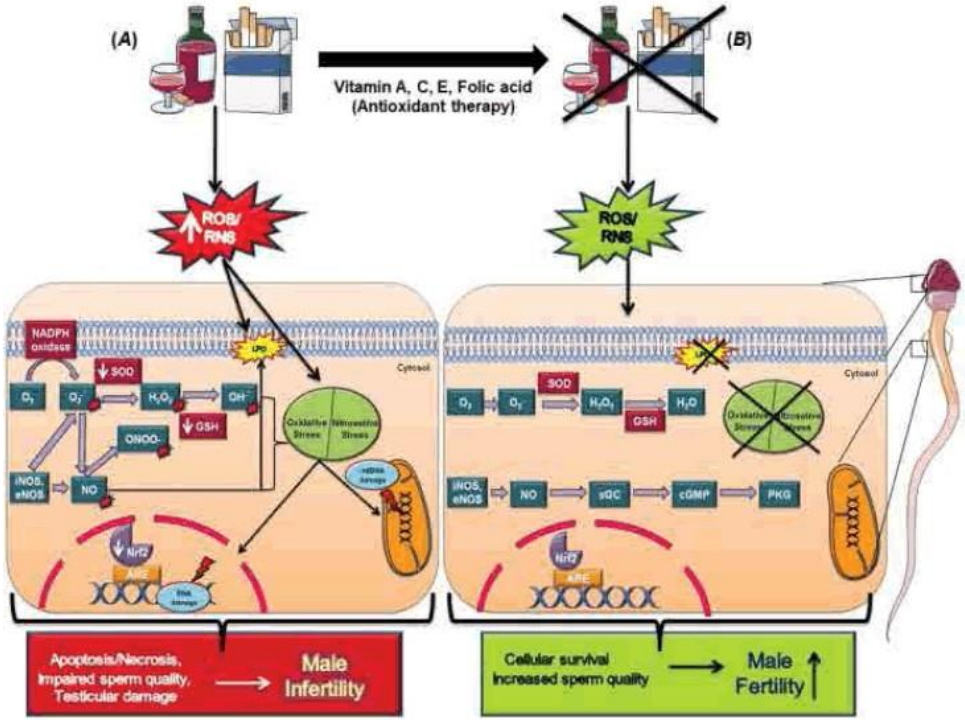


Fig 4: Smoking and alcohol use have been shown to have intracellular oxidative mechanisms in spermatozoa (A); these mechanisms may be produced by increased NADPH oxidase activity, which lowers the levels of SOD, GSH, and Nrf2 by generating reactive oxygen intermediates (O2 -, H2O2 and OH-). eNOS interacts with NO and O2 when decoupled to generate ONOO-. Increased levels of ROS and RNS cause oxidative/nitrosative stress, which triggers sperm membrane peroxidation and causes DNA damage to both mitochondria and the nucleus. (B) Effects of antioxidant treatment in the absence of cigarette smoking or alcohol use. Spermatozoa remove ROS generation in the form of H2O and NO, which in turn triggers sperm capacitation and the acrosome response through the cGMP/PKG pathway. Sperm DNA damage is not possible due to these signalling mechanisms. Reactive oxygen/nitrogen species, NADPH (nicotinamide adenine dinucleotide phosphate) (reduced), oxygen, superoxide, hydrogen peroxide, hydroxide, peroxynitrite, inducible NO synthase, endothelial NOS, superoxide dismutase, glutathione, lipid peroxidation, nuclear factor erythroid 2 (NFE2)-related factor 2, antioxidant response element

## RESULTS

240 guys who had their semen analyses reported at the Department of Pathology during a six-month period are included in this research. The majority of these 240 guys (n=117/240, or 48.7%) were in the age range of 29 to 38 years, followed by those in the 18 to 28 years age range (61/240, or 25.4%). (Table 1) Age groups were examined based on the forms of addiction (Table 2) Smoking addiction accounts for the majority of cases (n=63, 26.25%), followed by alcohol abuse (n=62, 25.83%), while chewing tobacco addiction accounts for the least amount of instances (n=27, 11.25%). According to the amount of intake, each of the three addictions is independently categorised into distinct categories in (Table 3). Depending on the age category,

cases are divided into mild, moderate, and severe categories. (Table 4,5,6) Teratozoospermia (T) was the most common finding as a result (n=72/240, 30.0 percent).

Oligozoospermia (O) was observed in 19.1 percent of cases (n=46/240), whereas asthenozoospermia (A) was the second most prevalent semen characteristic (n=56/240, 23.3 percent). Azoospermics made up 3.7% of the key semen variables overall (n=09/240). Cases who weren't impacted by any exposure (n=57/240), or 23.7%. (Table 7) Teratozoospermia and oligozoospermia were the most common findings in cases with all three addictions (n=24/72) and (n=18/46), respectively. Asthenozoospermia was the most common result in cases with gutka addiction (n=18/56). (Table 8) Asthenospermia and azoospermia had lower seminal fructose concentrations than oliogozoospermia. Norrmozoopermia has a substantially lower concentration of fructose than oligozooseprmia. In oligozoospermia, sperm concentration, vitality, and progressive motility are also much lower than in normozoospermia.

**Table 1: Showing the distribution of total cases according to age groups:**

| Age group | No. of patients(n=240) |
|---|---|
| 18-28 | 60 |
| 29-38 | 117 |
| 39-48 | 30 |
| 49-58 | 15 |
| 59-68 | 13 |
| ≥69 | 05 |

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Fig 5: Distribution of cases according to age

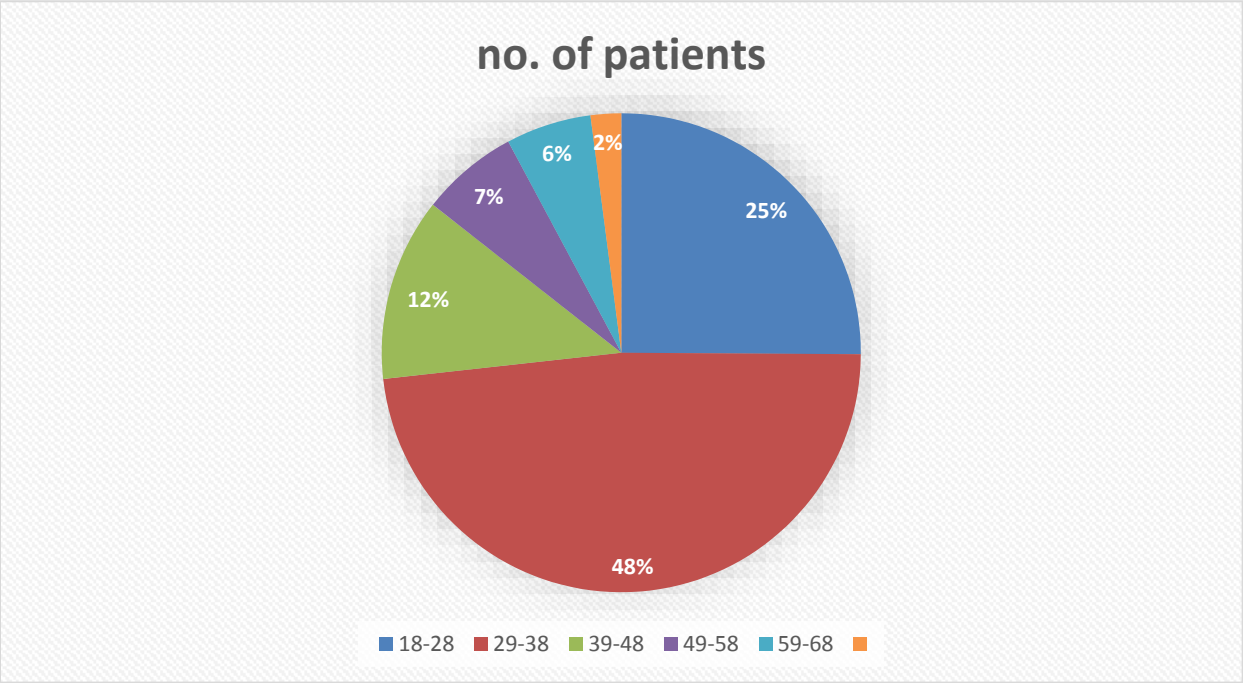


**Table 2: Distribution of different age groups according to types of addiction**

| Age group | A | B | C | D | E |
|---|---|---|---|---|---|
| 18-29 | 13 | 17 | 18 | 05 | 08 |
| 29-38 | 28 | 29 | 31 | 12 | 17 |
| 39-48 | 05 | 07 | 07 | 06 | 05 |
| 49-58 | 03 | 05 | 04 | 02 | 02 |
| 59-68 | 02 | 04 | 03 | 02 | 03 |
| ≥69 | 01 | 01 | 01 | 01` | 01 |

**Table 3: Showing the distribution of total cases according to addiction**

| Total cases(n) | No Addiction | Only alcohol consumers | Only tobacco smokers | Only tobacco chewers |
|---|---|---|---|---|
| 238 | 51 | 62 | 63 | 27 |

**Table 4: Showing the distribution of alcohol consumers**

| AGE GROUP | NO. OF ALCOHOL CONSUMERS | MILD | MODERATE | SEVERE |
|---|---|---|---|---|
| 18-29 | 17 | 06 | 05 | 06 |
| 29-38 | 29 | 11 | 09 | 09 |
| 39-48 | 07 | 04 | 03 | 02 |
| 49-58 | 05 | 01 | 02 | 02 |
| 59-68 | 04 | 02 | 01 | 01 |
| ≥69 | 01 | 01 | 00 | 00 |

**Table 5: Showing grade wise distribution of Tobacco smokers**

| AGE GROUP | NO. OF TOBACCO SMOKERS | MILD | MODERATE | SEVERE |
|---|---|---|---|---|
| 18-29 | 18 | 03 | 05 | 10 |
| 29-38 | 31 | 10 | 08 | 13 |
| 39-48 | 07 | 03 | 02 | 02 |
| 49-58 | 04 | 02 | 01 | 01 |
| 59-68 | 03 | 02 | 01 | 00 |
| ≥69 | 01 | 01 | 00 | 00 |

**Table 6: Showing grade wise distribution of Tobacco chewers**

| AGE GROUP | NO. OF ALCOHOL CONSUMERS | MILD | MODERATE | SEVERE |
|---|---|---|---|---|
| 18-29 | 05 | 03 | 01 | 01 |
| 29-38 | 12 | 05 | 04 | 03 |
| 39-48 | 06 | 03 | 02 | 01 |
| 49-58 | 02 | 01 | 00 | 01 |
| 59-68 | 02 | 01 | 01 | 00 |
| ≥69 | 01 | 00 | 01 | 00 |

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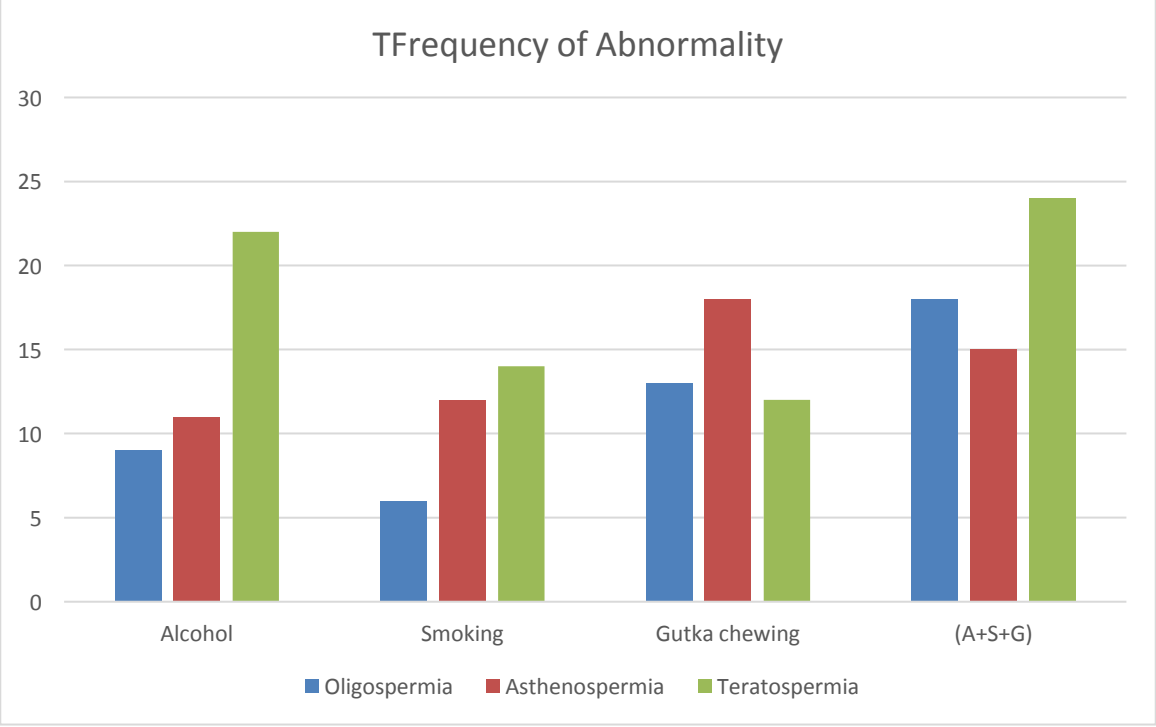
**Table 7: Distribution of cases according to diagnosis**

| Diagnosis | Frequency | Percentage(%) |
|---|---|---|
| Normal | 57 | 23.7% |
| Oligozoospermia | 46 | 19.1% |
| Teratozoospermia | 72 | 30.0% |
| Asthenozoospermia | 56 | 23.3% |
| Azoospermics | 9 | 3.7% |

**Table 8: Frequency of abnormality in relation to types of addiction**

| Types of addiction | Alcohol(A) | Smoking(S) | Gutka chewing(G) | A+S+G | Total |
|---|---|---|---|---|---|
| Oligozoospermia(O) | 09 | 06 | 13 | 18 | 46 |
| Asthenozoospermia(A) | 11 | 12 | 18 | 15 | 56 |
| Teratozoospermia(T) | 22 | 14 | 12 | 24 | 72 |

Figure 7: Frequency of abnormality in relation to types of addiction



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## DISCUSSION

Asthenozoospermia was the most frequent finding in subjects addicted to gutka chewing (n=18/56), teratozoospermia and oligozoospermia were the most frequent findings in cases with all three addictions (n=24/72) and (n=18/46) respectively. These three main semen variables (T, A, and O) were correlated with the type of addiction. There is evidence to suggest that alcohol may damage male reproductive hormones and the quality of semen. Alcohol induces testicular injury and raises endorphin levels, which ultimately leads to sperm death. Nuclear maturation and DNA integrity are affected by alcohol use. [18]

Chronic alcohol use results in high levels of oxidative stress because of increased lipid peroxidation or owing to diminished antioxidantsystems in the testis.

[19] Alcohol interferes with the hypothalamus-pituitary-gonadal (HPG) axis' feedback processes, impairing the release of leutinizing hormone (LH) and follicle stimulating hormone (FSH), which causes sertoli cells to degenerate.

[20] As a result, higher oxidative stress and decreased levels of testosterone, LH, and FSH disrupt spermatozoa's normal morphological growth and maturation and slow down the generation of sperm by testicular germ cells. [21] Toxins from cigarettes make seminal and other accessary gland secretions more viscous, which slows down spermatozoa's ability to develop forward linearly and leads to asthenozoospermia. Due to the excessive generation of reactive oxygen species, cigarette smoking results in oxidative stress (ROS).
[20] Spermatozoa create ROS, which contribute to DNA damage and defects in spermatozoa's membrane integrity as well as their unique tasks such the acrosome response, capitation, and fertilisation. Consequently, spermatozoa's capacity and shape are negatively impacted.
[18] Nicotine is absorbed via the oral mucosa more quickly in tobacco chewers than it is in smokers, and residual toxins circulate for alonger length of time in chewers than in smokers.
The concentration of nicotine and other chemicals in tobacco affects the epididymis' normal function, especially the activity of the enzyme alpha-1,4 glycosidase, which prevents the secondary maturation of spermatozoa and results in teratozoospermia. Additionally, nicotine starts a large amount of ROS generation that results in oxidative stress, which damages DNA and causes oligozoospermia and teratozoospermia. [22] The presence of a normal seminal fructose concentration supports the proper function of the vas deferens, vesicles, and testosterone. [23] The blockage in CBAVD or retrograde ejaculation coincides with the lack of both sperm and fructose. [24,25]

## CONCLUSION

It is undeniable that smoking, drinking, and recreational drug use may affect male fertility in some way, perhaps via synergistic rather than addictive effects, despite the absence of strong evidence from interventional studies, which are often not practicable in people. On the other hand, effects on the endocrine control of reproductive and sexual function have been reported in clinical and experimental studies. Impairments in spermatogenesis and sperm parameters as well as increased DNA methylation and oxidative stress have been observed in humans and animal models alike. Although nothing is known about the length of time required for

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cessation of negative effects, it should be recommended that all patients undergoing examination for infertility discontinue all of these behaviours in order to deliver the best results.

Alcohol use, cigarette smoking, and tobacco chewing are examples of lifestyle choices that have a detrimental influence on the quality of semen. Those receiving infertility treatment should abstain from these harmful lifestyle choices, including alcohol and cigarette use. The normozoospermia group had a much lower seminal fructose content than the oligozoospermia group. Sperm concentration, motility, and fructose seminal concentration all have negative associations with one another.

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