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Evaluation of Nutritional Status and Testosterone of Smokers

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ABSTRACT

Smoking is a global public health problem associated with excessive morbidity and mortality. We designed this study to estimate the nutritional status and testosterone levels among smokers in Ekpoma, Edo State. We recruited 100 smokers after obtaining informed consent and also included 50 seemingly healthy non-smokers as a control group. The study recruited both male and female subjects who had been smoking at least one stick of cigarettes per day for the past six months. We collected five milliliters (5 ml) of blood from the antecubital vein of both smokers and non-smokers and then transferred it into sterile, anticoagulant-free sample containers (plain tubes). We performed the determination of total protein, albumin, globulin, total cholesterol and testosterone using standard laboratory procedures. The results showed that total protein levels were significantly higher ($p < 0.05$) in smokers (7.02 ± 0.87 g/dl) when compared with the control (6.43 ± 1.06 mg/dl). Albumin levels were significantly higher ($p < 0.05$) in smokers (3.70 ± 0.71 g/dl) when compared with the control (3.32 ± 0.78 g/dl). Globulin levels were significantly higher ($p < 0.05$) in smokers (3.32 ± 0.78 g/dl) when compared with the control (3.11 ± 0.92 g/dl). On the contrary, total cholesterol levels were not significantly different ($p > 0.05$) in smokers (176.10 ± 45.04 mg/dl) when compared with the control (184.94 ± 46.39 mg/dl). Testosterone levels were significantly higher ($p < 0.05$) in smokers (4.49 ± 1.55 ng/ml) when compared with the control (3.44 ±1.25 ng/ml). These results showed that smoking affected the nutritional parameters and testosterone. This could be attributed to increased synthesis by the liver. Additionally, smokers showed an increase in serum testosterone, suggesting a potential compromise in its function.

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Introduction

Smoking, the act of inhaling and exhaling fumes from burning tobacco products such as cigars, cigarettes, and pipes, remains a significant global health concern. Historically, smoking practices can be traced back to Native Americans in the Western Hemisphere, who utilized it for therapeutic and religious purposes [1]. In modern times, smoking is categorized based on frequency and intensity. An individual is typically classified as a smoker if they have smoked at least 100 cigarettes in their lifetime and report smoking daily or occasionally [2]. Regular smoking, defined by Perkins and Leone, involves daily tobacco use, even in minimal amounts. The World Health Organization (WHO) provides further classification, identifying heavy smokers as those who consume more than 20 cigarettes per day. The age at which an individual begins smoking regularly, often referred to as the "Starting Age,"

plays a critical role in determining long-term smoking habits and associated health risks [2]. Understanding these classifications and patterns of smoking behavior is essential for evaluating its impact on public health and developing effective interventions.

Proteins are among the most vital biomolecules in human serum, playing a pivotal role in maintaining the body's delicate acid-base balance and supporting various physiological functions. When carbohydrate intake is insufficient, the body relies on proteins as an alternative energy source, underscoring their multifaceted importance. Smoking, however, has been shown to disrupt serum protein homeostasis, leading to significant alterations in protein levels [3,4]. Serum proteins are primarily composed of albumin and globulins, which together constitute total serum protein [3]. Quantitative determination of these proteins is typically conducted using automated analyzers, ensuring precision in clinical diagnostics [5]. Albumin, the predominant serum protein, accounts for 50-60% of the total protein and is crucial for maintaining oncotic pressure and serving as a transport molecule [6]. Studies have consistently reported a notable decrease in serum albumin levels among smokers, which may reflect underlying inflammation or metabolic disturbances. Globulins, a heterogeneous group of proteins with slower solubility compared to albumin, play critical roles in immune function. The decreased serum albumin levels in smokers are often accompanied by a compensatory increase in globulin levels, particularly immunoglobulins synthesized by lymphocytes [6]. This adaptive response to smoking-induced oxidative stress highlights the body's attempt to counteract free radicals generated by cigarette smoke. However, when globulin synthesis is impaired, it can lead to severe pathological conditions characterized by diminished protein synthesis and heightened protein catabolism [7].

Cigarette smoke exerts systemic effects, impacting even organs not directly exposed to it by altering enzymatic activity critical for anabolic protein metabolism [8, 9]. Albumin, known for its antioxidant properties and acute-phase response during inflammation, is particularly affected by smoking [8]. Simultaneously, free radicals in cigarette smoke stimulate the production of globulins, especially gamma globulins, which are synthesized by plasma cells or B lymphocytes. These proteins not only serve enzymatic functions but also provide innate and adaptive immunity against pathogens [10]. These findings underscore the profound impact of smoking on serum protein dynamics, revealing a complex interplay between oxidative stress, immune responses, and protein metabolism. Understanding these mechanisms is essential for developing targeted interventions to mitigate the health risks associated with smoking.

Stavridis highlights the profound global health implications of smoking, linking it to elevated rates of morbidity and mortality [11]. Alarming statistics underscore this connection, with smoking estimated to contribute to 4.84 million premature deaths worldwide. The World Health Organization (WHO) found a link between smoking and 3.83 million deaths in men and 1.0 million deaths in women. 4.83 million fatalities worldwide were attributed to tobacco use in 2000; this number rose to 6.4 million in 2015 and 8.3 million in 2030. 150 million of the 300 million young smokers worldwide will die from smoking- related causes in their later years [4]. Makwana predict a rise in these deaths from 3.4 million to 6.8 million in low- and middle-income countries between 2002 and 2030 [12]. According to Nabila smoking cigarettes is a significant risk factor for peripheral, atherosclerotic, and coronary vascular disease [13]. It modifies the cardiac muscle's cell division process. Furthermore, Nabila found a dose-dependent relationship between the number of cigarettes smoked and cardiovascular morbidity and death. According to Qulander smoking increases the absorption and inhalation of many harmful metabolites and chemicals. Despite its detrimental effects on human health, tobacco consumption remains widespread worldwide [14]. Zhong implicate tobacco as a cause of fatal illnesses such as cancer, respiratory conditions, and many infections, as well as cardiac conditions [15].

Cigarette smoke, a complex mixture of over 4,000 compounds including oxidants, free radicals, and lipid- soluble polycyclic aromatic hydrocarbons, significantly impacts various bodily systems and biochemical markers [4,15]. These toxic substances, known to accumulate gradually in the liver, disrupt enzymatic and metabolic functions, leading to alterations in biochemical parameters linked to reproductive and liver health [16]. Testosterone, a key hormone for reproductive and metabolic processes, is particularly affected by these disruptions. The oxidative stress caused by free radicals in cigarette smoke is a primary mechanism influencing testosterone levels. Free radicals damage Leydig cells in the testes, responsible for testosterone synthesis, leading to dysregulation of hormonal balance. This can result in either elevated or reduced testosterone levels, depending on the extent of oxidative damage and the body's adaptive response [8,9]. Elevated testosterone levels observed in smokers may indicate an overactive hypothalamic-pituitary- gonadal axis as a compensatory mechanism to counteract smoke-induced stress. However, this elevation may mask underlying impairments in the hormone's bioavailability or receptor functionality. The lipid-soluble nature of many toxicants, such as polycyclic aromatic hydrocarbons, allows for their accumulation in the liver, where they interfere with steroid hormone metabolism. The liver, critical for hormone regulation, including testosterone degradation and clearance, is adversely affected by cigarette smoke. This disruption can result in altered testosterone levels in circulation, further influencing processes dependent on normal testosterone function, such as muscle mass maintenance and secondary sexual characteristics [16].

Cigarette smoke-induced damage also extends to other fertility hormones, such as Luteinizing Hormone (LH) and Follicle-Stimulating Hormone (FSH), which regulate reproductive processes. The oxidative stress and toxicant accumulation associated with smoking can dysregulate these hormones, creating a cascading effect that impacts testosterone synthesis and overall reproductive health [17,18]. These findings highlight the profound effects of cigarette smoke on testosterone and other fertility-related biochemical parameters. By elucidating the mechanisms underlying these disruptions— ranging from oxidative stress to liver dysfunction—it becomes possible to design targeted interventions to mitigate the adverse health consequences of smoking, particularly in relation to reproductive health.

To evaluate the extent of nutritional and reproductive deficiencies associated with smoking, it is essential to assess both the nutritional status and testosterone levels of individuals who smoke. This study aimed to investigate these parameters among smokers in Ekpoma, Edo State, providing insights into the biochemical and hormonal disruptions caused by cigarette smoking.

Materials and Methods

Study Area

The study was carried out in Ekpoma, Esan-west local government, Edo state, Nigeria. Ekpoma is located in the geographical coordinates 6045 N 08 E with a population of one hundred and twenty-five thousand, eight hundred and forty-two (125,842).

Ekpoma, after its designation as headquarters and as the host of state-owned university (Ambrose Alli University), the town has grown into an urban center [19].

Study Population

We recruited a total of 100 smokers for this study after obtaining verbal informed consent. We also included 50 apparently healthy non-smokers as a control group. Participants comprised both males and females, with smokers defined as individuals who had been consuming at least one cigarette per day for the past six months. The age range of smokers was 15 to 50 years, while the control group comprised individuals aged 18 to 40 years.

Inclusion and Exclusion Criteria

Only apparently healthy smokers were included in this study, while individuals exhibiting any signs of illness were excluded. Similarly, non-smokers with evident health conditions were excluded, with only apparently healthy non-smokers serving as the control group.

Research Design

This study employed a cross-sectional research design to evaluate serum levels of total protein, albumin, globulin, total cholesterol, and testosterone among cigarette smokers in Ekpoma, using non-smokers as the control group. In addition to the biochemical parameters, demographic information, including age, was also collected from both smokers and non-smokers. Blood samples were obtained from both groups for laboratory analysis, and the results were compared between the smoker and control groups.

Sample Collection and Preparation

About 5 ml of blood from the antecubital vein was collected from subjects (smokers and non-smokers). The samples were transferred into sterile, anticoagulant-free sample containers (plain tubes). The blood samples were allowed to stand for an hour to clot and then centrifuged with a laboratory centrifuge. The serum was collected and analyzed for total protein, albumin, globulin, total cholesterol, and testosterone.

Sample Analysis Estimation of Total Protein

The total protein content in the sample was determined using the method described by Gomall [20]. The principle of the biuret reaction involves the formation of a chelate between copper ions (Cu2+) and the peptide bonds present in proteins, which occurs in an alkaline solution. This results in the formation of a violet-colored complex, the intensity of which is directly proportional to the protein concentration in the sample. The reaction occurs when the copper ions bind with the serum proteins under specific conditions, forming a stable copper-protein complex that can be measured colorimetrically. To perform the analysis, 1.0 mL of biuret reagent was added to test tubes labeled as blank, standard, and sample. A 20 µL portion of the sample and standard were introduced into their respective tubes, mixed thoroughly, and incubated for 10 minutes at 37°C. Following incubation, the absorbance was measured at 540 nm against a reagent blank. The concentration of total protein in the sample was then calculated by comparing the absorbance of the sample to that of the standard.

Estimation of Albumin

The albumin content in the sample was determined using the method outlined by Doumas [21]. This method is based on the specific binding interaction between Bromo-Cresol Green (BCG), an anion dye, and albumin at an acidic pH. The binding leads to a shift in the absorption wavelength of the complex, with the intensity of the color formed being directly proportional to the concentration of albumin in the sample. For the procedure, 2.0 mL of bromo-cresol reagent was added to test tubes labeled as blank, standard, and sample. A 10 µL portion of both the sample and the standard was introduced into their respective tubes. The mixture was thoroughly mixed and allowed to stand for 1 minute at room temperature. The absorbance was then measured at 630 nm against a reagent blank. The concentration of albumin in the sample was determined by comparing the absorbance of the sample to that of the standard.

Estimation of Globulin

We indirectly estimated the plasma globulin concentration by subtracting the albumin concentration from the total protein concentration.

Determination of Total Cholesterol

Total cholesterol was quantified using the enzymatic endpoint method (CHOD-PAP) as described by Richmond [22]. The principle of this method involves the enzymatic hydrolysis of esterified cholesterol by cholesterol esterase, which releases free cholesterol. The free cholesterol is then oxidized in the presence of cholesterol oxidase, producing hydrogen peroxide. This hydrogen peroxide further reacts with phenol and 4-aminoantipyrine in the presence of peroxidase to form a red quinoneimine dye complex. The intensity of the resulting color is directly proportional to the amount of cholesterol present in the sample. The assay provides an accurate and reliable measurement of total cholesterol levels by measuring the absorbance of the quinoneimine dye formed, which correlates with cholesterol concentration in the sample. The procedure involves dispensing precise amounts of standard, sample, and distilled water into labeled test tubes, followed by the addition of cholesterol reagent. After mixing, the tubes are incubated at 37°C for five minutes, after which the absorbance of the standard and sample is measured at a wavelength of 500 nm using a spectrophotometer. The concentration of total cholesterol is then calculated based on the absorbance readings, providing a clear and efficient method for cholesterol determination.

Estimation of Testosterone

The DRG Testosterone ELISA Kit is a Solid-Phase Enzyme-Linked Immunosorbent Assay (ELISA) based on the principle of competitive binding. The microtiter wells are coated with a monoclonal (mouse) antibody directed towards a unique antigenic site on the testosterone molecule. Endogenous testosterone of a patient sample competes with a testosterone horseradish peroxidase conjugate for binding to the coated antibody. After incubation, the unbound conjugate is washed off. The amount of bound peroxidase conjugate is reversely proportional to the concentration of testosterone in the sample. After the addition of the substrate solution, the intensity of color developed is reversely proportional to the concentration of testosterone in the patient sample. For the procedure, each run included a standard curve. The desired number of microtiter wells was secured in the holder. 25 µL of each standard, control, and sample with new disposable tips were dispensed into appropriate wells. 200 µL enzyme conjugate was dispensed into each well. It was thoroughly mixed for 10 seconds. It is important to have a complete mixing in this step. Incubated for 60 minutes at room temperature (without covering the plate) The contents of wells were briskly shook out. The wells were rinsed 3 times with diluted wash solution (400 µL per well). The wells were stroked sharply onto absorbent paper or paper towels to remove all residual water droplets. 200 µL of substrate solution was added to each well. It was incubated for 15 minutes

at room temperature. The enzymatic reaction was stopped by adding 100 µL of stop solution to each well. The absorbance (OD) of each well was determined at 450 ± 10 nm with a microtiter plate reader. (Within 10 minutes after adding the stop solution).

Data Analysis

The results obtained were subjected to statistical analysis using SPSS (version 21). The test groups' values were compared with the values of the control group using ANOVA (LSD) and Student's t-test at a 95% level of confidence. The mean and standard deviation (SD) were also calculated in each case.

Results

The results in table 1 showed that total protein levels were significantly higher ($p < 0.05$) in smokers (7.02± 0.87 g/dl) when compared with the control (6.43 ± 1.06 mg/dl). Albumin levels were significantly higher ($p < 0.05$) in smokers (3.70 ± 0.71 g/dl) when compared with the control (3.32 ± 0.78 g/dl). Globulin levels were significantly higher ($p < 0.05$) in smokers (3.32 ± 0.78 g/dl) when compared with the control (3.11 ±0.92 g/dl). On the contrary, total cholesterol levels were not significantly different ($p > 0.05$) in smokers (176.10 ± 45.04 mg/dl) when compared with the control (184.94 ± 46.39 mg/dl). Testosterone levels were significantly higher ($p < 0.05$) in smokers (4.49 ± 1.55 ng/ml) when compared with the control (3.44 ± 1.25 ng/ml).

The results in table 2 showed that total protein levels were significantly higher ($p < 0.05$) in male smokers (7.03 ± 0.90 g/dl) when compared with the control (6.47 ± 1.12 g/dl). Albumin levels were significantly higher ($p < 0.05$) in male smokers (3.73 ± 0.72 g/dl) when compared with the control (3.28 ± 0.78 g/dl). Globulin levels were significantly higher ($p < 0.05$) in male smokers (3.30 ± 0.92 g/dl) when compared with the control (3.19 ± 0.95 g/dl). On the contrary, total cholesterol levels were not significantly lower ($p> 0.05$) in male smokers (178.38 ± 45.14 mg/dl) when compared with the control (189.94 ± 51.86 mg/dl). Testosterone levels were significantly higher ($p < 0.05$) in male smokers (4.50 ± 1.50 ng/ml) when compared with the control (3.24 ± 1.10 ng/ml).

The results in table 3 showed that total protein levels were significantly higher ($p < 0.05$) in female smokers (6.96 ± 0.70 g/dl) when compared with the control (6.34 ± 0.97 g/dl). On the contrary, albumin levels were not significantly higher ($p > 0.05$) in female smokers (3.58 ± 0.66 g/dl) when compared with the control (3.39 ± 0.79 g/dl). Globulin levels were not significantly different ($p > 0.05$) in female smokers (3.38 ± 0.68 g/dl) when compared with the control (2.95 ± 0.88 g/dl). Also, total cholesterol levels were not significantly lower ($p > 0.05$) in female smokers (164.13 ± 43.97 mg/dl) when compared with the control (176.06 ± 34.18 mg/dl). Testosterone levels were not significantly higher ($p > 0.05$) in female smokers (4.41 ± 1.85 ng/ml) when compared with the control (3.80 ± 1.44 ng/ml).

The results in table 4 showed that there was a non-significant increase in total protein levels in male smokers (7.03±0.90 g/dl) when compared with the female smokers (6.96±0.70 g/dl). Albumin levels were higher in male smokers (3.73±0.72 g/dl) when compared with the female smokers (3.58±0.66 g/dl). Total cholesterol levels were also higher in male smokers (178.38±45.14 mg/dl) when compared with the female smokers (164.13±43.97 mg/dl). Also, testosterone levels were higher in male smokers (4.50±1.50 ng/ml) when compared with the female smokers (4.41±1.85 ng/ml).

Table 5 presents a comparison of total protein, albumin, total cholesterol, and testosterone levels among smokers categorized by age. The results showed that total protein levels were higher in the age range of 20-23 years (7.09±0.74 g/dl) when compared with the age range of 16-19 years (7.02±0.97 g/dl) and 24 years and above (6.85±0.91 g/dl). Albumin levels were higher in the age range of 20-23 years (3.71±0.73 g/dl) when compared with the age range of 16-19 years (3.70±0.72 g/dl) and 24 years and above (3.70±0.66 g/dl). Total cholesterol levels were higher in the age range of 20-23 years (185.84±41.60 mg/dl) when compared to the age range of 24 years and above (174.82±45.50 mg/dl) and 16-19 years (168.36±47.04 mg/dl). Testosterone levels were higher in the age range of 16-19 years (4.66±1.61 ng/ml) when compared with the age range of 24 years and above (4.37±1.40 ng/ml) and 20-23 years (4.33±1.57 ng/ml).

Table 1: Serum Levels of Total Protein, Albumin, Total Cholesterol and Testosterone in Smokers and Control

Parameters	Control (n=50)	Smokers (n=100)	t Value	P Value
Total Protein (g/dl)	6.43±1.06	7.02±0.87	3.628	0.000
Albumin (g/dl)	3.32±0.78	3.70±0.71	3.020	0.003
Globulin (g/dl)	3.11±0.92	3.32±0.78	2.341	0.013
Total Cholesterol (mg/dl)	184.94±6.39	176.10±5.04	1.122	0.264
Testosterone (ng/ml)	3.44±1.25	4.49±1.55	4.146	0.000

Key: n=Sample size, p>0.05= Not significant, p<0.05= Significant

Table 2: Serum Levels of Total Protein, Albumin, Total Cholesterol and Testosterone in Male Smokers and Control

Parameters	Male Control (n=32)	Male Smokers (n=84)	t-Value	p-Value
Total Protein (g/dl)	6.47±1.12	7.03±0.90	2.755	0.007
Albumin (g/dl)	3.28±0.78	3.73±0.72	2.941	0.004
Globulin (g/dl)	3.19±0.95	3.30±0.81	2.319	0.027
Total Cholesterol (mg/dl)	189.94±5.86	178.38±5.14	1.182	0.240
Testosterone (ng/ml)	3.24±1.10	4.50±1.50	4.339	0.000

Key: n=Sample size, p>0.05= Not significant, p<0.05= Significant

Table 3: Serum Levels of Total Protein, Albumin, Total Cholesterol and Testosterone in Female Smokers and Control

Parameters	Female Control (n=18)	Female Smokers (n=16)	t Value	p Value
Total Protein (g/dl)	6.34±0.97	6.96±0.70	2.110	0.043
Albumin (g/dl)	3.39±0.79	3.58±0.66	0.716	0.479
Globulin (g/dl)	2.95±0.88	3.38±0.68	0.239	0.671
T Chol (mg/dl)	176.06±4.18	164.13±3.97	0.889	0.381
Testosterone (ng/ml)	3.80±1.44	4.41±1.85	1.072	0.292

Key: n=Sample size, p>0.05= Not significant, p<0.05= Significant

Table 4: Serum Levels of Total Protein, Albumin, Total Cholesterol and Testosterone in Male and Female Smokers

Parameters	Male Smokers (n=84)	Female Smokers (n=16)	t-Value	P Value
Total Protein (g/dl)	7.03±0.90	6.96±0.70	0.267	0.790
Albumin (g/dl)	3.73±0.72	3.58±0.66	0.786	0.434
Globulin (g/dl)	3.30±0.81	3.38±0.68	0.522	0.694
T Chol (mg/dl)	178.38±5.14	164.13±3.97	1.162	0.248
Testosterone (ng/ml)	4.50±1.50	4.41±1.85	0.229	0.819

Key: n=Sample size, p>0.05= Not significant, p<0.05= Significant

Table 5: Serum Levels of Total Protein, Albumin, Total Cholesterol and Testosterone in Smokers According to Age

Parameters	16-19 Years (n=45)	20-23 years (n=38)	24 & above Years (n=17)	t Value	P Value
Total Protein (g/dl)	7.02±0.97 [a]	7.09±0.74 [a]	6.85±0.91 [a]	0.440	0.645
Albumin (g/dl)	3.70±0.72 [a]	3.71±0.73 [a]	3.70±0.66 [a]	0.006	0.994
Globulin (g/dl)	3.32±0.85[a]	3.38±0.71 [a]	3.15±0.79 [a]	0.134	0.848
T.Chol (mg/dl)	168.36±7.04 [a]	185.84±4.60 [a]	174.82±4.50[a]	1.579	0.211
Testosterone ng/ml)	4.66±1.61 [a]	4.33±1.57 [a]	4.37±1.40 [a]	0.523	0.594

Key: n=Sample size, p>0.05= Not significant, p<0.05= Significant Values in a Row with the Same Superscript are not Significantly Different at p<0.05

Discussion

Smoking has increasingly emerged as a significant public health concern, particularly in developing nations such as Nigeria. It is well-established that cigarette smoking contributes to the development of atherosclerotic vascular diseases, including those affecting the peripheral, coronary, and cerebral circulations. Smokers are exposed to a range of harmful substances and their metabolites, which are released during the combustion of tobacco [4]. Given the widespread impact of smoking on various physiological systems, this study aims to assess the effects of smoking on blood testosterone levels and key nutritional parameters, shedding light on the broader implications of tobacco use on metabolic and hormonal health.

The results of this study revealed significantly higher levels of total protein, albumin, and globulin in smokers compared to the control group. Albumin, a critical protein in human serum, plays a vital role in binding and transporting drugs, bilirubin, and metal ions, and its levels are often used as an indicator of liver synthetic function. The elevated levels observed in smokers may suggest an enhanced synthesis of these proteins by the liver. This contrasts with the findings of Rothschild who noted that serum protein levels are controlled by hepatic synthesis and are indicative of the liver's capacity to produce proteins [23]. Interestingly, the results of this study conflict with those of Parvez who observed lower serum protein levels in smokers, attributing this decline to emotional or financial stress, or the appetite-suppressing effects of nicotine and other harmful compounds in cigarette smoke [24]. However, our findings does not align with those of Faruque who reported no significant difference in protein and fat intake between smokers and non-smokers [25]. The increased levels of albumin and total protein in this study may be attributed to a compensatory increase in protein production by the liver, potentially in response to the dehydration commonly associated with smoking. This heightened protein synthesis could reflect the body's attempt to counteract the physiological stresses imposed by smoking.

The findings of this study revealed that smokers exhibited higher serum total cholesterol levels compared to the control group. This observation contradicts the results of several studies, including those by Tilwani Sinha and Rastogi which reported a significant increase in serum total cholesterol levels among smokers. Similarly, other investigations have observed similar trends, highlighting the association between smoking and altered lipid profiles [26-28]. Our study contributes to this body of research, showing higher levels of total cholesterol levels between smokers and non-smokers. However, the findings agree with the 2009 study by Dirican, which found no significant difference in lipid profiles between smokers and non-smokers. Despite these discrepancies, numerous studies, including those by Ratnam, have consistently shown that smoking is linked to elevated levels of triglycerides, total cholesterol, LDL-C, and VLDL, along with a reduction in HDL-C levels [29,30]. These lipid imbalances are considered risk factors for cardiovascular diseases. One possible explanation for the higher total cholesterol levels observed in long-term smokers could be a decrease in lipoprotein lipase activity, a condition often associated with chronic smoking. This enzyme plays a crucial role in breaking down triglycerides and regulating lipid metabolism, and its reduced activity may contribute to the accumulation of cholesterol in the bloodstream. This reinforces the need for further investigation into the long-term effects of smoking on lipid metabolism and its implications for cardiovascular health.

This study also demonstrated that smokers had significantly higher testosterone levels compared to the control group. This finding aligns with previous research by Johan. which investigated the relationship between smoking and endogenous testosterone levels in men [31]. Our study's results are further supported by two large cross-sectional studies, one conducted by Field and another smaller study by Svartberg which also observed higher testosterone levels in smokers [32,33]. These findings contribute to a growing body of literature suggesting that smoking may influence hormonal profiles, particularly testosterone. However, there is some inconsistency in the literature on this topic. For instance, Barrett-Connor & Khaw found no statistically significant differences in testosterone levels

between male smokers and non-smokers when adjusting for age and BMI [34]. Out of the sixteen studies reviewed, seven reported higher testosterone levels in male smokers, while five studies, including those by English and Svartberg observed increased free testosterone levels in smokers [35]. Conversely, two studies found no correlation between smoking and free testosterone levels, and two studies reported no significant differences between smokers and non-smokers in testosterone levels [32,35-37]. The exact mechanisms behind the increased testosterone levels in smokers remain unclear. One possibility is that smoking could affect the function of Leydig cells, which are responsible for testosterone production, or alter the hypothalamic-pituitary-gonadal axis, a key hormonal regulation system. Additionally, it has been suggested that the observed differences in testosterone levels might reflect personality traits, with smokers possibly exhibiting higher levels of risk-taking behavior that are linked to elevated testosterone. This relationship could be indirect, mediated by an unrecognized mechanism. Notably, research by Booth suggested that men with higher testosterone levels are more likely to engage in risky behaviors, including smoking, which could explain why smoking is more prevalent among those with elevated testosterone [38]. Interestingly, quitting smoking appears to reverse the effect, with testosterone levels in former smokers returning to levels similar to those of men who have never smoked, further supporting the notion that the influence of smoking on testosterone might be reversible [31]. This suggests that smoking's impact on testosterone is not permanent and could be modified with lifestyle changes, emphasizing the potential for recovery once smoking cessation occurs.

Conclusion

The findings of this study underscore the significant impact of smoking on blood biochemical markers, particularly total cholesterol, albumin, globulin, and testosterone levels. The observed elevation in these parameters may be attributed to increased protein production by the liver, possibly as a compensatory mechanism in response to smoking-related stress. Moreover, the higher serum testosterone levels in smokers suggest potential alterations in endocrine function, which could reflect broader physiological disruptions associated with smoking. Given these findings, further research is crucial to elucidate the biochemical pathways linking smoking to changes in protein metabolism and testosterone regulation. Such studies will provide valuable insights into the underlying mechanisms of these alterations and help deepen our understanding of smoking-related health risks. Additionally, the integration of biochemical indicators into screening protocols is essential for monitoring and mitigating the onset of smoking-related complications, thereby improving public health outcomes.

Conflict of Interest: The authors declare no conflicts of interest. The authors alone are responsible for the content and the writing of the paper.

Ethical Permission: Ethical approval was obtained from the University Ethics Committee and also informed consent was sought from the subjects before collection of blood samples.

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