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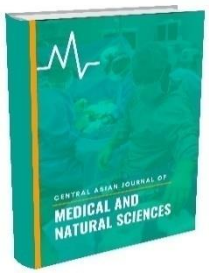

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Effects of Vaping and Water Pipe on Lipid Profiles, Glucose and Liver Enzymes

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Abstract: To evaluate the effect of vaping and water pipe smoking on some physiological parameters in men, the present study has been carried out at college of sciences, Al-Qadisiyah Univerisity in cooperation with central lab of Al-Dewaniyah Hospital in Al-Dewaniyah city during the period extended from June-December 2022. One Hundared fifty samples of blood were collected from men were divided in to 3 groups, first group 50 healthy, second group 50 smoking with Argila and third group 50 smoking with Vape. Venous blood (5ml) were collected from all sample for measuring immunity parameter and physiological parameters. Results shown clarified the AST increase of the smoking groups (Argila and Vape). It has been found that AST recorder a significant increase ($p<0.05$) in smoking groups (Argila and Vape) when compared with Non-Smoking group (25.48±0.71) and (45±0.42; 38.8±0.35) respectively. And ALT increase of the smoking groups (Argila and Vape). It has been found that ALT recorder a significant increase ($p<0.05$) in smoking groups (Argila and Vape) when compared with Non-Smoking group (22.76±1.42) and (45.6±0.53; 38.76±0.63) respectively. Also ALP increase of the smoking groups (Argila and Vape). It has been found that ALP recorder a significant increase ($p<0.05$) in smoking groups (Argila and Vape) when compared with Non-Smoking group (90.84±1.38) and (139.28±0.91; 122.18±1.006) respectively. Also results shown clarified the Cholesterol increase of the smoking groups (Argila and Vape). It has been found that Cholesterol recorder a significant increase ($p<0.05$) in smoking groups (Argila and Vape) when compared with Non-Smoking group (134.4±2.11) and (211.54±1.85; 191.4±1.04) respectively.

Key words: Water Pipe.

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And Triglyceride increase of the smoking groups (Argila and Vape). It has been found that Triglyceride recorder a significant increase ($p<0.05$) in smoking groups (Argila and Vape) when compared with Non-Smoking group (152.98±9.11) and (229.1±6.19; 195.48±2.65) respectively. Also HDL decrease of the smoking groups (Argila and Vape). It has been found that HDL recorder a significant decrease ($p<0.05$) in smoking groups (Argila and Vape) when compared with Non-Smoking group (33.74±0.68) and (22.14±1.39; 25.38±0.45) respectively. The results illustrated showed non-significant differences ($p>0.05$) between all non-Smoking (99.69±0.0042) and smoking (Argila and Vape) groups (99.70±0.0014; 99.71±0.0036) respectively.

Introduction

Smoking dates back to 5,000 BC and has been found in many different cultures around the world. Smoking was necessary in the past for religious ceremonies; such as making offerings to the gods, ritual purification, or to enable shamans and priests to change their minds for the purposes of divination and spiritual enlightenment. With the European exploration and conquest of the Americas, tobacco smoking spread rapidly throughout the world. In regions such as India and sub-Saharan Africa, tobacco smoking has merged with the smoking processes common in these countries, of which cannabis is the most common. In Europe, smoking introduced a new social activity and a previously unknown form of drug use (Jha *et al.*,2002).

Smoking is one of the most common addictions of modern times. It has been implicated as an etiological agent for vari- ous chronic diseases including variety of infections, cancers, heart diseases, and chronic lung diseases which put together are the leading causes of morbidity and mortality in today's society (Liu *et al*.,2008).

Smoking leads to many diseases and health crises, such as heart attack, stroke, respiratory diseases, and cancer (lung cancer in particular), in addition to other health problems, and thus premature death. In addition, the symptoms that result from infection with a disease resulting from smoking lead to an increase in nervous and psychological stress and thus negatively affect the quality of life from an early age (WHO,2005).

The jar at the bottom of the hookah is filled with water sufficient to submerge a few inches of the body tube, which is sealed tightly to it (Shane,2011). Deeper water will only increase the inhalation force needed to use it (Rudolph and Matthee,2005). In February 2020, the Centers for Disease Control and Prevention (CDC) confirmed 2,807 cases of e-cigarette or vaping use-associated lung injury (EVALI) and 68 deaths attributed to that condition (Orellana-Barrios *et al*.,2015). Cigarette smoke has been shown to affect a wide range of host defence mechanisms (Shapiro,2004), findings between studies can be controversial and sometimes contradictory, probably because of differences in smoking history, genetic susceptibility and socioeconomic status (such as exercise, nutrition, occupation and ambient air quality, which can modify disease).

This study was conducted for the purpose of verifying the effect of smoking (hookah and electronic cigarettes) on some of the criteria chosen in the current study.

Material and Methods

Collection of blood samples

Blood samples collection by gel tube, Blood serum samples were separated (by centifugation at 3000 rpm for 5 minuts) and kept at –20 ºC until assessment of liver enzymes Glucose, cholesterol, triglyceride, HDL concentrations.

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Results

Liver Enzymes

AST

Results shown in figure (1) clarified the AST increase of the smoking groups (Argila and Vape). It has been found that AST recorder a significant increase ($p<0.05$) in smoking groups (Argila and Vape) when compared with Non-Smoking group (25.48±0.71) and (45±0.42; 38.8±0.35) respectively.

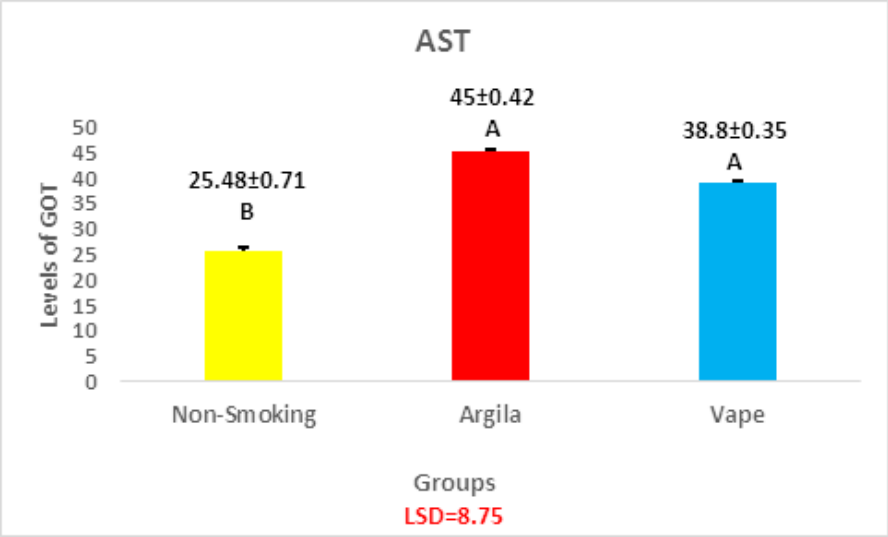

Figure (1): AST levels between non-Smoking and smoking groups (Argila and Vape).

The results represented as mean ± SE.

Different capital letters denotes a significant differences ($P<0.05$) between periods.

Similar capital letters denotes non-significant differences ($P<0.05$) between periods.

ALT

Results shown in figure (2) clarified the ALT increase of the smoking groups (Argila and Vape). It has been found that ALT recorder a significant increase ($p<0.05$) in smoking groups (Argila and Vape) when compared with Non-Smoking group (22.76±1.42) and (45.6±0.53; 38.76±0.63) respectively.

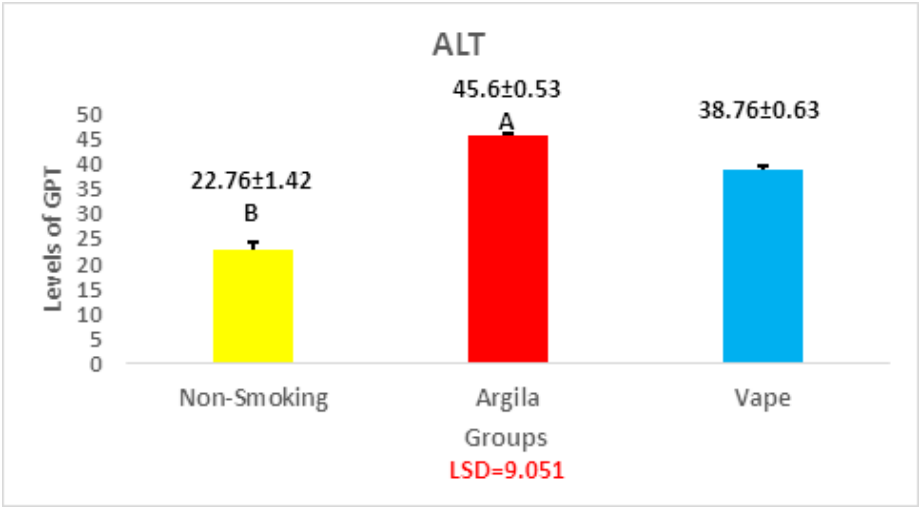

Figure (2): ALT levels between non-Smoking and smoking groups (Argila and Vape).

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The results represented as mean ± SE.

Different capital letters denotes a significant differences (P<0.05) between periods.

Similar capital letters denotes non-significant differences (P<0.05) between periods.

ALP

Results shown in figure (3) clarified the ALP increase of the smoking groups (Argila and Vape). It has been found that ALP recorder a significant increase (p<0.05) in smoking groups (Argila and Vape) when compared with Non-Smoking group (90.84±1.38) and (139.28±0.91; 122.18±1.006) respectively.

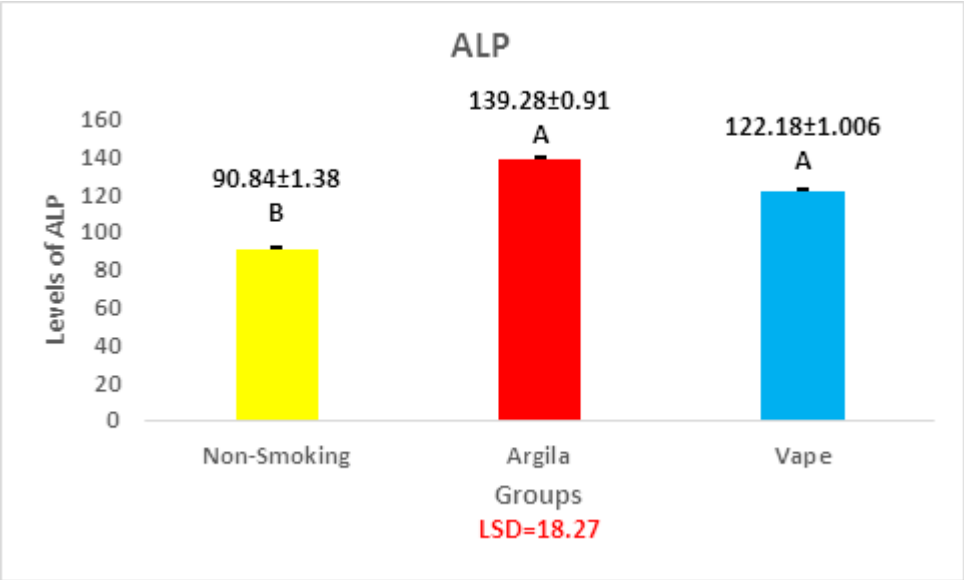

Figure (3): ALP levels between non-Smoking and smoking groups (Argila and Vape).

The results represented as mean ± SE.

Different capital letters denotes a significant differences (P<0.05) between periods.

Similar capital letters denotes non-significant differences (P<0.05) between periods.

Lipid Profile

Cholesterol

Results shown in figure (4) clarified the Cholesterol increase of the smoking groups (Argila and Vape). It has been found that Cholesterol recorder a significant increase (p<0.05) in smoking groups (Argila and Vape) when compared with Non-Smoking group (134.4±2.11) and (211.54±1.85; 191.4±1.04) respectively.

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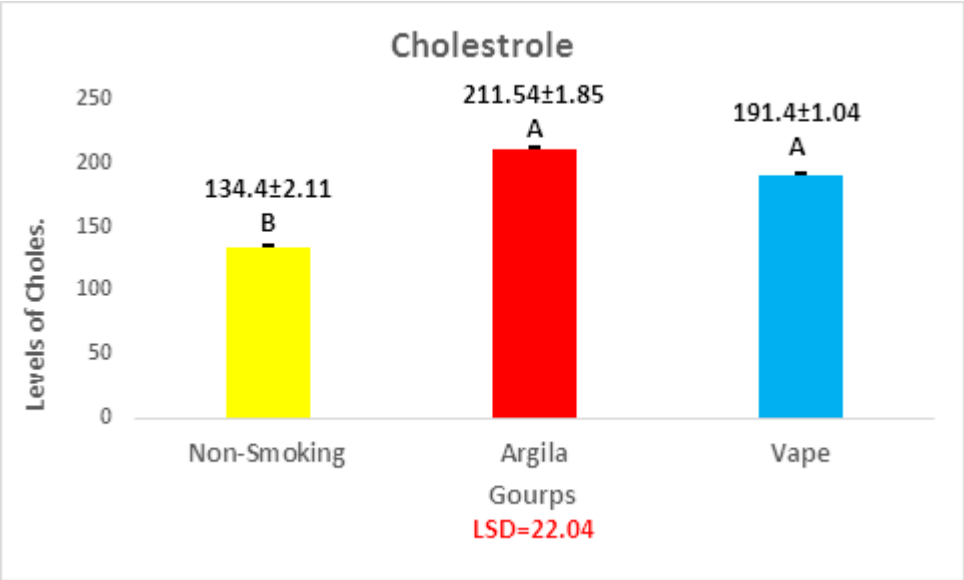

Figure (4): Cholesterol levels between non-Smoking and smoking groups (Argila and Vape).

The results represented as mean ± SE.

Different capital letters denotes a significant differences (P<0.05) between periods.

Similar capital letters denotes non-significant differences (P<0.05) between periods.

Triglyceride

Results shown in figure (5) clarified the Triglyceride increase of the smoking groups (Argila and Vape). It has been found that Triglyceride recorder a significant increase (p<0.05) in smoking groups (Argila and Vape) when compared with Non-Smoking group (152.98±9.11) and (229.1±6.19; 195.48±2.65) respectively.

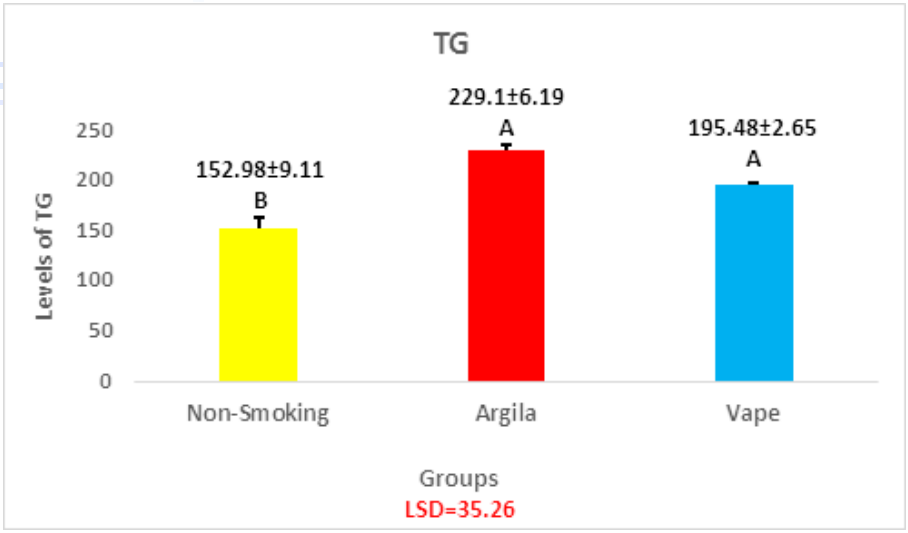

Figure (5): Triglyceride levels between non-Smoking and smoking groups (Argila and Vape).

The results represented as mean ± SE.

Different capital letters denotes a significant differences (P<0.05) between periods.

Similar capital letters denotes non-significant differences (P<0.05) between periods.

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HDL

Results shown in figure (6) clarified the HDL decrease of the smoking groups (Argila and Vape). It has been found that HDL recorder a significant decrease ($p<0.05$) in smoking groups (Argila and Vape) when compared with Non-Smoking group (33.74±0.68) and (22.14±1.39; 25.38±0.45) respectively.

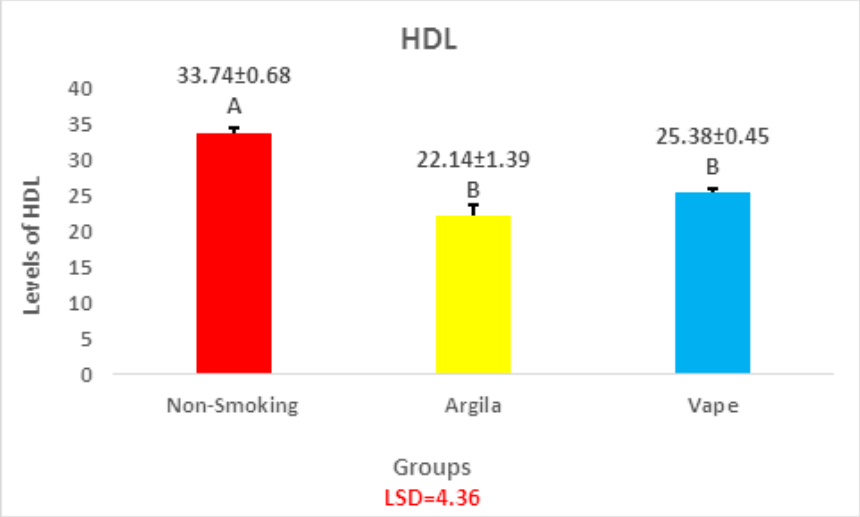

Figure (6): HDL levels between non-Smoking and smoking groups (Argila and Vape).

The results represented as mean ± SE.

Different capital letters denotes a significant differences ($P<0.05$) between periods.

Similar capital letters denotes non-significant differences ($P<0.05$) between periods.

Glucose

The results illustrated in figure (7) showed non-significant differences ($p>0.05$) between all non-Smoking (99.69±0.0042) and smoking (Argila and Vape) groups (99.70±0.0014; 99.71±0.0036) respectively.

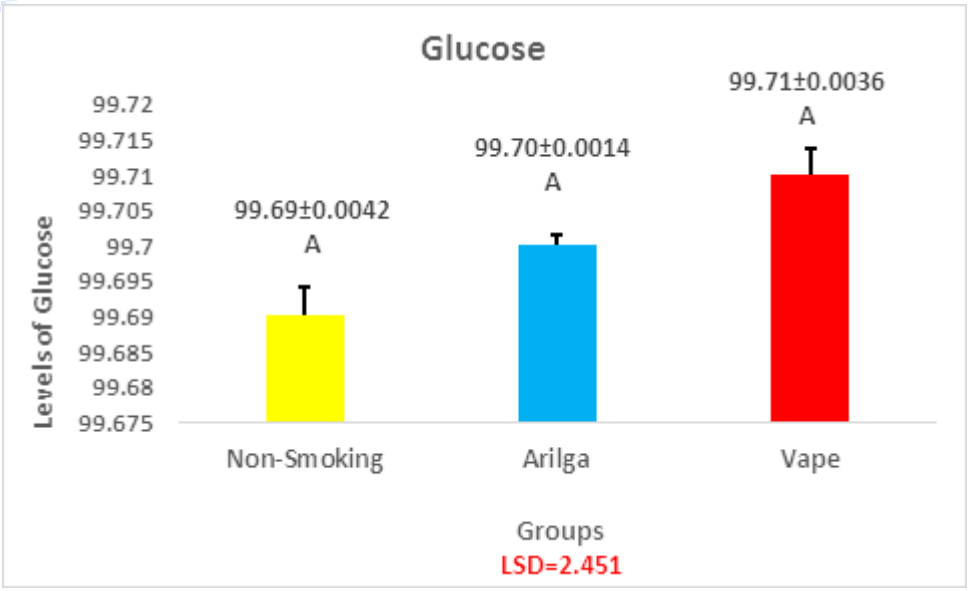

Figure (7): Glucose levels between non-Smoking and smoking groups (Argila and Vape).

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The results represented as mean ± SE.

Different capital letters denotes a significant differences (P<0.05) between periods.

Similar capital letters denotes non-significant differences (P<0.05) between periods.

Discussion

The liver is a key organ with multiple crucial functions. The liver detoxifies the body by removing toxins such as alcohol and narcotics. In addition, the liver is responsible for the metabolisation of nicotine, which has different harmful physiological effects. The aerosols, cartridges, refill liquids, and environmental emissions of e-cigarettes have been shown to include a variety of toxic, carcinogenic, and liver disease-inducing chemical compounds and ultrafine particles (Chen et al., 2021). This study also discovered that WP smokers had higher alkaline phosphatase levels than non-smokers but higher levels of alanine aminotransferase and aspartate aminotransferase. Since oxidative damage to hepatocytes and alterations in liver enzymes are confirmed to occur in the smoking model, Alomari et al. (2020) concluded that stopping smoking can cure liver damage and restore normal levels of liver enzymes in both WP and cigarette smokers. E-vaping increased blood and liver lipids, regardless of whether administered with nicotine. In recent years, diets high in simple carbohydrates and fats that stimulate de novo lipid production have been associated with a dramatic increase in the prevalence of non-alcoholic fatty liver disease (Chen et al., 2021). Another consequence of hepatic insulin resistance is the diversion of glucose from glycogen to de novo lipid synthesis. Nonetheless, this change was inversely related to hepatic lipid levels. It has been demonstrated that exposure to E-vapour causes metabolic changes that cannot be explained by invoking the traditional lipid metabolic regulators. The glycerine base of e-vapour can enter the bloodstream unrestrictedly from the respiratory tract (El. Golli et al., 2016), which may contribute to elevated blood and liver lipids in chow-fed mice exposed to e-vapour. However, evidence suggests that nicotine promotes fat buildup in the liver. According to a human investigation, adding glycerine to a high-fat meal increased plasma-free fatty acid levels, likely via chylomicron formation. While no concrete evidence supports this notion, a toxicology study found that vegetable glycerine preserved its humectant qualities even after being heated (Ababneh et al., 2017). This may occur due to nitrosative stress which is a condition that occurs when the production of highly reactive nitrogen-containing chemicals, such as nitrous oxide, exceed the ability of the human body to neutralize and eliminate them. Nitrosative stress can lead to reactions that alter protein structure thus interfering with normal body functions (Chakraborty *et al*.,2000). The results show that there was no statistically significant difference in serum ALT, AST, ALP and TB between moderate smoker group and control group. From the results of the present study, one can conclude that there is a dose response relationship between the number of cigarettes/day smoked and serum ALT, AST, ALP and total bilirubin levels. From the results, a statistically significant reduction in serum total protein, serum albumin and serum globulin in heavy smoker group were observed. Cigarette smoking is associated with increased oxidative stress. Albumin has antioxidant properties, through binding to copper ions and scavenging HOCl, then the oxidized albumin may be cleared rapidly from the circulation and degraded (Neki,2002). The present study revealed that se-rum protein electrophoretic pattern changed in both heavy and moderate smokers when compared with non-smoker group. To the best of our knowledge and on the basis of active literature searching, other researchers did not investigate the protein fractions by protein-electrophoresis in their studies. From the results, it can be suggested that cigarette smoke contain many potential hepatotoxic substances which affect liver function through its effect on nserum protein electrophoresis fractions, this is due to its effect on their synthesis and metabolism in the liver. There is a great relationship between liver and lipid because all lipid profile parameters are synthesis and metabolized in liver (Jaimes *et al*.,2007). These findings confirm several previous studies that have reported on the association between smoking and liver enzymes (Avti *et al*.,2006; Breitling *et al*.,2009) and extend these findings by examining the role of

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inflammation and the synergistic effects of alcohol drinking. The association between smoking and GGT was to a large extent associated with inflammation, except in the presence of heavy drinking. There was a significant interaction between cigarette smoking and alcohol on GGT. By contrast the association between smoking and ALP was only partially explained by inflammation and was seen irrespective of alcohol intake. Despite the strong positive association between smoking and alcohol intake, which was strongly and positively associated with AST, we observed an inverse relationship between smoking and AST, which persisted even after adjustment for BMI and alcohol intake. The nature of this inverse association is not clear, but has been observed in previous studies (Lee *et al*.,2007). Total cholesterol and triglyceride levels were much greater than normal, whereas high-density lipoprotein (HDL) levels were significantly lower. According to Hallit et al. (2017), the harmful effects of smoking cigarettes and WPs include the promotion of free radical production, which interacts with biological molecules to elevate oxidative stress by increasing lipid peroxidation, the damage to heart muscles and blood vessels, and the possibility of cholesterol reacting with fatty substances on the arterial wall. Chwyeed (2018) examined the effects of shisha smoking on the serum lipid profile in a research of 75 males in the city of Nasirriya and found extremely substantial variations in lipid profile. Cholesterol peroxidation lowers its availability for cellular uptake, accumulating the material in the blood serum. Insulin resistance may potentially play a role in the process by which smoking reduces HDL and increases triglyceride levels. Indicators of insulin resistance syndrome include low HDL and elevated triglycerides. Studies indicate that smokers have a higher degree of insulin resistance (Chwyeed,2018). Compared to non-smokers, cigarette smokers had a higher abdominal adiposity marker due to the unfavourable effects of WPS on TG and HDL levels, as determined by the present study. There may be a connection between WP and e-cigarette stimulation of the hypothalamus-pituitary-adrenal axis, the accompanying elevation in blood cholesterol levels, and the observed significant increase in abdominal fat positively correlated with smoking duration. In addition, WP and e-cigarette release increases lipolysis as well as serum TG and HDL metabolism (Al-Sawalha et al., 2020). Majid et al. (2021) discovered that smoking shisha has the same effect on the lipid profile as smoking cigarettes, stimulating the adrenal sympathetic system, increasing serum free fatty acids levels, and impairing lipoprotein metabolism, which is coronary heart disease risk factors. Majid et al. (2021) reported that HDL levels were considerably lower in cigarette users compared to non-smokers. Contrary to popular perception, the WP likely removes only a tiny fraction of dangerous elements (Rice et al., 2018). We did this study to determine how using a WP and e-cigarette impacts the risk of cardiovascular disease. Although the findings of this study are limited, they suggest that smoking a WP is at least as dangerous as smoking cigarettes. The connection between WP and e-cigarettes and cholesterol and lower HDL is dose-dependent. According to studies referenced in Górna et al. (2020), cigarette smoking decreases HDL by boosting lipase activity. By changing copper metabolism, cigarette smoking may induce cholesterol peroxidation, providing a unifying mechanism for the elevated blood cholesterol levels linked with smoking. Apolipoprotein A-1 is structurally essential for HDL's ability to transport cholesterol back to the liver from the body's peripheral tissues. Because it works as an activator of lecithin-cholesterol acetyltransferase, apolipoprotein A-1 is necessary to reverse transport cholesterol from peripheral tissues to the liver. Greater production of HDL and its receptors reduces the danger associated with using a WP. This significantly decreased HDL and total cholesterol (Al-Numair et al., 2007). The subjects' average smoking duration may have contributed to the normal lipid values seen in their profiles. The prevalence of dyslipidemia in the smoking group indicates that most of these individuals were chronic smokers (defined as more than twenty cigarettes daily). This raises the likelihood of atherosclerosis. Inhaling smoke from shisha and cigarettes amplifies the effects of nicotine and carries the same hazards of alteration and inflammation, which explains why cigarette smoking produces such high levels of oxidative stress (Chen et al., 2021). WP and e-cigarettes have the same effect on glucose tolerance as light tobacco cigarette smoking, so it is not a terrible replacement. Although our studies

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have not established the molecular basis for this improvement in glucose tolerance, a comparison with the groups exposed to nicotine-free e-vapour suggests that the benefit is most likely nicotine-driven. High-sugar diet and nicotine-free e-vaping increased glucose transporters. The increased glucose tolerance of humans exposed to e-cigarettes may be attributable to the downregulation of gluconeogenesis markers. In contrast, the effect of WP and e-cigarettes was stronger in reducing blood sugar levels, consistent with the action of pure nicotine reported elsewhere to stimulate glucose absorption. However, glucose may not always be converted to triglycerides (Al-Sawalha et al., 2020).

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