REVIEW ARTICLE

Effects of Smoking and Alcoholism Comorbid in Reactive Oxygen Species, Alcohol and Serotonin Levels

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Received: 07th July 2021; Revised: 03rd September, 2021; Accepted: 29th Octorber, 2021; Available Online: 25th December, 2021

ABSTRACT

The alcoholism and smoking habit are the most important health, economy, and forensic problems in populations. These habits are related to free radicals, neurotransmitter and other molecules. The present study was conducted to estimate reactive oxygen species (ROS), serotonin and alcohol in smoker in alcohol use disorder, results showed that there was a high percent of drunks have a smoking habit (78.94%) and low percent did not smoker (21.01%), the levels of ROS, serotonin and alcohol in study group show decrease in all study variables in the smoker group in non-significant differences, The correlation among study variables pointed that in non-smoker group weak positive correlation observed between ROS and serotonin, while in smoker weak inverse correlation in non-sig, ROS was significant inverse correlation with alcohol in non-smoker (p 0.031), and in the smoker group was non-sig weak invers correlation, the serotonin correlated with ROS in weak inverse relation in smoker while in non-smoker there was weak positive relation in non-sig for both groups, also there was weak inverse relation in smoker and weak positive in non-smoker group in non-significant relations. The current finding concluded that the smoking may be affected in serotonin and alcohol level in addition to the effects of alcohol, thus the health awareness should be applied among populations.

Keywords: Alcoholism, Alcohol, Comorbid, ROS, Serotonin levels, Smoking.

International Journal of Pharmaceutical Quality Assurance (2021); DOI: 10.25258/ijpqa.12.4.23

How to cite this article: Jawad MA, Altimari US, Al-Terehi MN, Hussain KN. Effects of Smoking and Alcoholism Comorbid in reactive oxygen species, Alcohol and Serotonin Levels. International Journal of Pharmaceutical Quality Assurance. 2021;12(4):343-345.

Source of support: Nil. **Conflict of interest:** None

INTRODUCTION

The alcoholism and smoking habit have been increased among the Iraqi population in last decades and these resulted to complex health disorders, the comorbidity between smoking and drinking have been increased, 1,2 this b elongs to numerous factors such as social/peer pressures, alcohol and cigarettes availability encourage drinking and smoking. Moreover, the synergistic effects of the drugs when used together with reinforcing properties regard. And the drugs pharmacological impacts, like alteration in metabolism or cross-tolerance that facilitate co-abuse. The genetic factors also involvement to this comorbidity.³ Current study suggested that the alcohol and tobacco smoking interaction with serotonin, ROS, and alcohol level in comorbidity of drunks and smoking habit. The serotonergic system is also involved in both tobacco smoking and alcohol dependence.^{4,5} The Dysfunctional of serotonin and dopamine system activity may represent a vulnerability to alcohol dependence.⁶

The neurotransmitters have been measured in drunks like serotonin and dopamine the chronic consume of alcohol found lower level or higher or unchanged in sometimes⁷⁻¹² in compare with control group.

METHODOLOGY

Current study included 40 drunks individuals have age ranged (22–59 years) and BMI (22–39 Kg/m²) without any health problems and different abuse durations, its classified to smoker and non-smoker groups, blood samples and data were collected according to ethical approval of ministry of health and environment in Iraq and approval of each contributor, serum isolated to detection serotonin by enzyme-linked immunoassay (ELIZA), ROS by colormetric method and alcohol by the forensic medicine department. Data represented as mean \pm stander error of mean, independent t test used for significantly detection at *p value* <0.05, the correlation also detected at the same significant level.

RESULTS

The levels of ROS, serotonin and alcohol are clarified in Table 1. There was high percent of drunks have a smoking habit (78.94%) and low percent didn't smoker (21.01%) (Figure 1).

The levels of ROS, serotonin and alcohol in study group show that the smoking habit decrease serotonin levels in significant differences (p 0.002), and non-significant decrease in ROS and alcohol levels (Table 2).

The correlation among study variables were detected, in non-smoker group weak positive correlation observed between ROS and serotonin, while smoker weak inverse correlation in non-sig, ROS was significant inverse correlation with alcohol in non-smoker (p=0.031), and in the smoker group was non-sig weak inverse correlation, the serotonin correlated with ROS in weak inverse relation in smoker while in non-smoker there was weak positive relation in non-sig for both groups, also there was weak inverse relation in smoker and weak positive in non-smoker group in non-significant relations (Table 3).

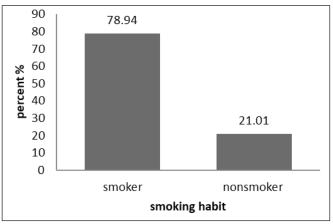


Figure 1: Distribution alcohol use disorder individuals according to the percent of smoking habit

DISCUSSION

The smoking and alcohol are the most important health, economic and forensic problems in the world, investigations pointed to the association between Heavy alcohol drinking and smoking, about 23% of people smoke cigarettes, and more than 50% have alcohol abuse or dependence smoke, however about 30% of smokers are also alcohol dependent. 13-14 The present study found highre percent of smokers than non-smokers, this also found by McKee et al., (2007)¹⁵ who proposed that the smoking habit in daily and occasional are more likely to be hazardous drinkers or to meet criteria for an alcohol abuse diagnosis than a non-smoker. Also the smokers consume twice as much alcohol as nonsmokers and alcohol drinkers used more cigarettes than non-alcohol-dependent smokers per day. 16,17 Serotonin significant decreased in the smoker group in the current study and this agree with Cosgrove et al., (2009)³ who found lower levels of in serotonin transporters in nonsmokers group, and this can be clarified that the smoking suppresses the alcohol-induced elevate in 5-HT transporter availability over the first week of abstinence, evidences refered to several reasons about the differences between smoker and nonsmoker drunks, for instance the genetic variation between them may be contributed in these variations, an investigate suggested that there is a common genetic component that involved in alcohol and nicotine dependence.¹⁸ There was a possible association found between decreased 5-HT transporter availability and the 5-HT genotype in alcoholics¹⁹ in addition to neurochemical variation between drunks who smoker and non-smoker. The tobacco compromised more than 4000 chemical molecules like nicotine, and monoamine oxidase inhibitors that changes the serotonin activity, ^{20,21} low platelet monoamine oxidase inhibitors activity has been detected in both alcoholics and tobacco smokers. 22,23 the ROS found to be didn't affect in the present study, there was slightly difference observed in smoker and nonsmoker groups, this result didn't agree with

Table 1: The ROS, serotonin and alcohol levels in alcohol use disorder

Study variables	ROS ηmol/L	Serotonin mg/mL	Alcohol mg/cm ³
Level	22.90 ± 0.875	121.90 ± 91.43	75.8 ± 41.7

Table 2: The ROS, serotonin and alcohol levels in smoker and nonsmoker group

Study variables	ROS	Serotonin	Alcohol	
Smoker	22.56 ± 1.09	99.21 ± 13.37	79.84 ± 7.95	
Non-smoker	23.76 ± 0.63	202.55 ± 33.75	61.22 ± 7.80	
Sig	0.590	0.002*	0.242	

Independent t test, at p value less than 0.05

Table 3: The correlations among study variables in the smoker and non-smoker group

		ROS		Serotonin	
	Variables	Smoker	Non	Smoker	Non
Serotonin	r	-0.110	0.057		
	P	0.709	0.943		
Alcohol	r	-0.140	-0.969*	-0.216	0.141
	p	0.632	0.031	0.235	0.717

other evidences proved that the smoking induced excessive free radicals production, ²⁴⁻²⁶ this result can be explained that the drunks have a good life style, nutrition and regular exercise that contributed in the reduced ROS and enhance antioxidant activity. The limitation of current study represented by sample size and the sample obtained from drunks during drinking. Nevertheless, can be concluded that the comorbid between alcoholism and smoking have an effect in the serotonin that may be leading to mood disorders and mental health in addition to enhanced alcohol level and this contributed in big health problems.

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