

Research Article

Main hazards of Ethyl Alcohol and its additives among workers in Abou-Qurkas sugar factory, El-Minia governorate, Egypt

Esam M. Mekheimer**, Ayman S. El-Khateeb*, Refaat RS*, Refaat TM*, El-Rehany MA*** and Zayet HH****.

* Department of Community and Industrial Medicine, El-Minia Faculty of Medicine,

** Head of medical department, Abu-Qurkas Sugar factory.

*** Dean of Faculty of pharmacy, Draya University.

****Industrial Medicine and Occupational Diseases Department, Cairo Faculty of Medicine.

Abstract

Objective: Prolonged heavy exposure to ethanol can cause significant and permanent damage of many of the human organs. The aim of this study was to detect the possible effects of ethanol and its additives on exposed workers employed in the distillation department of Abou-Qurkas sugar factory. **Patients and methods:** The study included 84 workers who were divided into 2 groups: exposed group included 43 workers in distillation part of the factory, and non-exposed group: 41 workers employed in different sections of the factory other distillation part. The participants were investigated for blood count, liver function tests, renal function tests, random blood glucose, lipid profile, serum ethanol level, and abdominal ultrasonographic findings. **Results:** There were statistically non-significant differences in demographic characteristics, special habits, systemic diseases, vital signs, hematological parameters, liver function, lipid profile, and renal function. There was no statistically significant difference between both groups in regards of levels of serum ethanol (6.90 ± 4.23 mg/dl in exposed, versus 5.64 ± 3.36 mg/dl in non-exposed). The abdominal ultrasonographic findings did not reflect significant hazardous abdominal disease associated with ethanol exposure. **Conclusion:** The harmful health effects of ethanol exposure were absent among workers at our sugar factory due to absence of exposure to excessive limits of ethanol. Application of preventive measures and regular revision of the limits of occupational exposure are recommended to mitigate the risk of chemical hazards.

Keywords: Ethanol, denatured alcohol, sugarcane, occupational disease, hazard

Introduction

In Egypt, about 78% of total sugar production in Egypt is from cane sugar. There are a total of nine factories for sugar production and refining in different governorates of Upper Egypt^[1]. One of these factories is Abu-Qurkas Sugar factory. The distillation part within the factory concerned with ethanol production contains 103 workers all of them are permanent and exposed to ethyl alcohol and its derivatives.

Ethanol have many alternative names like pure alcohol, drinking alcohol, ethyl alcohol and grain alcohol, it is a colorless liquid, volatile, flammable and used as

alcoholic beverages and in modern thermometers, medically it used as a psychoactive material,^[2] Ethanol is the

essential constituent in alcoholic drinks, so most jurisdictions added some agents to the ethanol to make them unfit to drink. These called denatured substances, which are methanol (0.025%), kerosine (0.005%) and bone oil (0.0025%) to obtain denatured alcohol, which is toxic and undrinkable^[3].

Ethanol act on central nervous system as a depressant and has powerful psychoactive effects in sub-lethal doses. Prolonged heavy use of ethanol can cause significant and long standing damage to the brain and

other organs^[4]. Methanol is an additive material and significantly toxic. If consumption with small dose as 10mL, it can cause permanent blindness by destruction of the optic nerve. The usual fatal dose is 100–125 mL^[5].

The aim of this study were to detect the possible effects of ethanol and its additives (methanol, bone oil and kerosine) on exposed workers employed in the distillation department of Abou-Qurkas sugar factory.

Patients and methods

This cross-sectional study conducted at Abou-Qurkas factory for sugar industry (distillation part concerned with alcohol production), El-Minia Governorate, between January and December 2013. The study included 84 workers who employed in different sections of the factory. We followed the research ethics regulation of El-Minia University Committee (MUC). A formal approval was taken from the manager of the factory to facilitate the study, as well as informed consents were taken from workers to participate in the study.

The workers were divided into 2 groups: Exposed group included 43 workers in distillation part of the factory, and non-exposed group: 41 workers employed in different sections of the factory other distillation part. All subjects were interviewed using a specially designed interviewing questionnaire. The interviewing sheet was modified according to previously pilot study. The questionnaire included: Personal history (name, age, residence, etc...), occupational history (history of previous job, duration of the present job, duration of exposure), special habits (smoking, drug abuse), and history of systemic diseases (hypertension, diabetes mellitus).

Clinical examination of all workers included: general examination, ophthalmic examination, and vital signs in regard to pulse (rate/minute, rhythm), blood pressure (pre and after-shift), respiratory rate and temperature.

Investigations were carried out for all workers and included: Liver functions, Kidney functions, complete blood count, blood glucose level, and abdominal ultrasonography.

The Polymer Technology System (PTS) strips for use with Cardio Chek brand analyzer were used to measure blood sugar level, total cholesterol (TC), high density lipoprotein (HDL), low density lipoprotein (LDL) and triglycerides (TG) in whole blood and finger-stick blood.

The statistical analysis was performed using Statistical Package for Social Science (SPSS) for windows version 20. Continuous data were expressed as mean and standard deviation (SD), while categorical data were expressed as number and percent. Student (t) test was used for comparing means of two groups. Paired student (t) test was used for comparing the same group with a variable. ANOVA test used to compare means of more than two groups. Chi-square (χ^2) test was used to compare the qualitative data between two or more groups. A P-value of 0.05 was considered the limit below which the difference of the values would be statistically significant.

Results

There were no statistically significant differences between exposed and non-exposed workers in terms of demographic characteristics (age and residence), special habits, history of systemic diseases (smoking, diabetes mellitus and hypertension), and vital signs [Table 1].

There were no statistically significant differences between both groups in liver function tests (total bilirubin, total protein, serum albumin, ALT, AST, ALP, prothrombin time), renal function tests (serum creatinine, BUN), hematological parameters (Hb, TLC, RBCs, platelets count), random blood sugar, and lipid profile (total cholesterol, TG, HDL, LDL) [Table 2].

There was no statistically significant difference between both groups regarding the levels of serum ethanol (6.90 ± 4.23

mg/dl in exposed, versus 5.64±3.36 mg/dl in non-exposed, $P<0.05$) [Figure 1].

The findings of abdominal ultrasound were normal in 90.7% of exposed group versus 80.5% of non-exposed group, while it were abnormal in 9.3% of exposed group versus 19.5% of non-exposed group ($P<0.05$) [Table 3]. There was no statistically

significant difference in ultrasonography findings between both groups regarding presence of fatty liver (4.7% in exposed versus 4.8% in non-exposed, $P<0.05$), gallbladder disease (2.3% in exposed versus 2.4% in non-exposed, $P<0.05$), and benign prostate hypertrophy (0% in exposed versus 4.8% in non-exposed, $P<0.05$) [Table 3].

Table 1: Demographic characteristics, special habits, systemic diseases and vital signs in the studied groups

Variables	Exposed (n=43)	Non-exposed (n=41)	P-value
Age (years)	41±7.5	42.4±8.9	0.43
Residence: Rural/Urban	26/17	27/14	0.68
Smoking	38 (88.4%)	35 (85.3%)	0.67
Hypertension	3 (7%)	5 (12.1%)	0.42
Diabetes mellitus	3 (7%)	6 (14.6%)	0.26
Heart rate (beat/min.)	73.06±10.10	74.80±9.49	0.41
SBP (mmHg)	117.44±10.48	113.46±10.17	0.08
DBP (mmHg)	75.34±9.34	74.23±9.02	0.58
Respiratory rate (breath/min.)	16.1±2.3	15.2±2	0.06
Temperature (°C)	36.48±0.50	36.53±0.50	0.64

SBP: systolic blood pressure. DBP: diastolic blood pressure

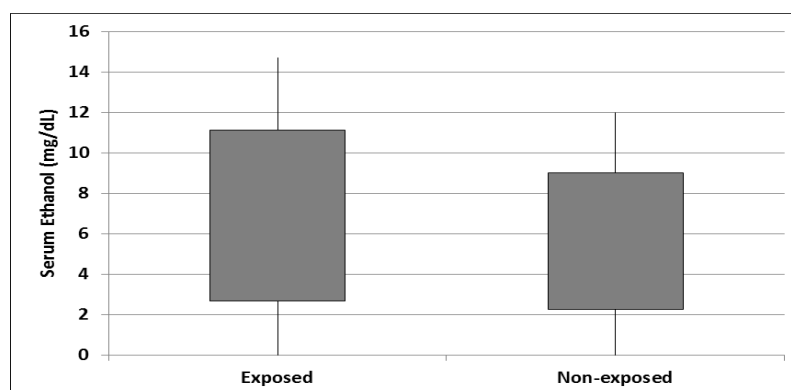
Table 2: Laboratory results in the studied groups

Variables	Exposed (n=43)	Non-exposed (n=41)	P-value
Bilirubin (mg/dl)	0.66±0.31	0.64±0.34	0.77
Total protein (g/dl)	6.90±0.81	6.84±0.88	0.74
Serum albumin (g/dl)	4.20±0.83	3.88±0.81	0.07
ALT (IU/L)	31.67±13.22	36.65±13.03	0.08
AST (IU/L)	27.18±11.86	30.23±12.88	0.26
ALP (IU/L)	79.06±20.17	74.57±16.25	0.26
PT (Seconds)	11.58±1.11	11.38±1.06	0.40
Serum creatinine (mg/dl)	0.84±0.16	0.83±0.15	0.76
Blood urea (mg/dl)	14.16±3.77	15±2.92	0.25
Hb (g/dl)	14.1±0.9	13.7±1	0.06
TLC ($\times 10^3$ cells/mm ³)	6.744±2.237	6.423±2	0.49
RBCs ($\times 10^6$ cells/mm ³)	4.90±0.28	4.94±0.33	0.55
Platelets ($\times 10^3$ cells/mm ³)	294.37±77.74	286.46±72.68	0.63
RBS (mg/dl)	90.86±24.08	90.53±24.73	0.95
Total Cholesterol (mg/dl)	202±26.74	192.6±19	0.06
TG (mg/dl)	102.23±51	100±54.16	0.84
HDL (mg/dl)	56±10	61±14.84	0.07
LDL (mg/dl)	118.65±12.91	120.50±11.79	0.49

AST: Aspartate Aminotransferase. ALT: Alanine Aminotransferase. ALP: Alkaline Phosphatase. PT: Prothrombin Time. Hb: Hemoglobin, TLC: Total leucocytic count. RBCs: Red blood cells. RBS: Random blood sugar. TG: Triglycerides. HDL: High density Lipoprotein, LDL: Low density Lipoprotein.

Table 3: Finding of abdominal ultrasound in the studied groups

Variables	Exposed (n=43)	Non-exposed (n=41)	P-value
Normal Ultrasound	39 (90.7%)	39 (80.5%)	0.23
Abnormal Ultrasound	4 (9.3%)	8 (19.5%)	0.23
Fatty liver	2 (4.7%)	2 (4.8%)	0.87
Gallbladder disease	1 (2.3%)	1 (2.4%)	0.97
Renal stones or abnormality	1 (2.3%)	3 (7.3%)	0.28
Benign prostate hypertrophy	0	2 (4.8%)	0.19

**Fig. 1:** Boxplot of mean±SD and range of the serum ethanol in the studied groups

Discussion

The main finding of the present study is the absence of hazardous effect of chronic excessive ethanol exposure on hemodynamics, hematological parameters, blood sugar level, renal and liver function as well as lipid profile. These findings reflect absence of harmful health effects of ethanol exposure that may be attributed to absence of exposure to repeated hazardous excessive limits of ethanol.

Liver damage from excessive chronic ethanol exposure is diagnosed by abnormal liver function tests in addition to abnormalities on abdominal ultrasound^[6]. Excessive ethanol alcohol exposure can have profound bad effects on the kidneys and their function as acid-base balance, electrolyte, maintaining the body's fluid, leaving alcoholic workers vulnerable to a most of kidney-related diseases^[7]. Exposure to ethanol alcohol has only modest effects on platelet aggregation and hemorheological parameters^[8], and these effects are mainly dose-dependent^[9]. Excessive exposure has been also associated with higher glucose levels, therefore increasing the risk of both diabetes and metabolic syndrome^[10]. Chronic excessive exposure to ethanol alcohol affects fat metabolism and

increases adipose tissue lipolysis which leads to deposition of ectopic fat within the liver and the development of alcoholic fatty liver disease^[11]. Absence of these abnormalities in our study is consistent with limited exposure to hazardous ethanol doses.

Also, we found no statistically significant difference between both groups in regards of levels of serum ethanol (6.90 ± 4.23 mg/dl in exposed, versus 5.64 ± 3.36 mg/dl in non-exposed). These findings indicate non-toxic and non-critical levels of serum ethanol among exposed sugarcane workers. Adherence with occupational safety measures may explain the negative serum levels of ethanol. Thus, the inhalatory and dermal routes of ethanol exposure may deliver very low amounts of ethanol. Moreover, the small sample size may explain the minor statistically non-significant differences.

The abdominal ultrasonographic findings did not reflect significant hazardous abdominal disease associated with ethanol exposure. We found no statistically significant difference between both groups in regard to presence of fatty liver and gallstones that caused by the excessive

accumulation of lipids within the liver cells which encompasses a morphological spectrum consisting of hepatic steatosis (fatty liver) and steatohepatitis that can progress to cirrhosis and hepatocellular carcinoma^[12, 13]. Regular exposure to ethanol reduces the risk to develop gallstones compared to non-exposed population, however infrequent exposure showed no significant association with risk^[14,15].

In conclusion, occupational health concerned with health safety in the workplace and strongly focusing on primary prevention of hazard. The risks of ethanol in workplace results mainly from inhalation of ethanol vapor and/or from skin contact. Excessive exposure to ethanol causes damage of major organs including heart, brain, liver and kidney. This study recommends regular revision of the limits of occupational exposure to mitigate the risk of chemical hazards. Also, successful application of preventive measures is recommended to eliminate any hazardous effect of ethanol during its production in the distillation part of sugar factory.

References

1. World Bank. Industrial pollution prevention: sugar manufacturing. Draft Technical Report, Environment Department, Washington. D.C., 1995, pp. 401-5.
2. McMurry J. Organic Chemistry. 6th ed. Belmont, CA: Brooks/Cole, 2004.
3. UK Stationery Office. The Denatured Alcohol Regulations 2005. Statutory Instrument 2005 No. 1524, 2005.
4. Seitz HK, Stickel F. Molecular mechanisms of alcohol-mediated carcinogenesis. *Nat Rev Cancer*. 2007; 7(8):599-612.
5. Vale A. *Methanol. Medicine*. 2007; 35(12): 633-4.
6. Berg JM, Tymoczko JL, Stryer L. Section 30.5 Ethanol Alters Energy Metabolism in the Liver. In: *Biochemistry*. 5th edition. New York: W H Freeman; 2002.
7. Epstein M. Alcohols's Impact on Kidney Function. *Alcohol Health & Research World*. 1997;21(1):84-93.
8. Fehr M, Galliard-Grigioni KS, Reinhart WH. Influence of acute alcohol exposure on hemorheological parameters and platelet function in vivo and in vitro. Selected Proceedings of the 14th European Conference for Clinical Hemorheology and Microcirculation, Dresden, Germany, 27-30 June, 2007.
9. Zhang QH, Das K, Siddiqui S, Myers AK. Effects of acute, moderate ethanol consumption on human platelet aggregation in platelet-rich plasma and whole blood. *Alcohol ClinExp Res*. 2000;24(4):528-34.
10. Athyros VG, Liberopoulos EN, Mikhailidis DP, Papageorgiou AA, Ganotakis ES, Tziomalos K, Kakafika AI, Karagiannis A, Lambropoulos S, Elisaf M. Association of drinking pattern and alcohol beverage type with the prevalence of metabolic syndrome, diabetes, coronary heart disease, stroke, and peripheral arterial disease in a Mediterranean cohort. *Angiology*. 2007;58:689-97.
11. Steiner JL, Lang CH. Alcohol, adipose tissue and lipid dysregulation. *Biomolecules*. 2017; 7(1).doi: 10.3390/biom7010016.
12. Reddy JK, Rao MS. Lipid metabolism and liver inflammation. II. Fatty liver disease and fatty acid oxidation. *American Journal of Physiology-Gastrointestinal and Liver Physiology* 2006; 290:G852-8.
13. Bedogni G, Nobili V, Tiribelli C. Epidemiology of fatty liver: an update. *World Journal of Gastroenterology* 2014;20:9050-4.
14. La Vecchia C, Decarli A, Ferraroni M, Negri E. Alcohol drinking and prevalence of self-reported gallstone disease in the 1983 Italian National Health Survey. *Epidemiology* 1994;5 (5):533-6.
15. Leitzmann MF, Giovannucci EL, Stampfer MJ. Prospective study of alcohol consumption patterns in relation to symptomatic gallstone disease in men. *Alcohol ClinExp Res*. 1999;23 (5): 835-41.