

Metabolic Syndrome in Alcohol-dependent Men: A Cross-sectional Study

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ABSTRACT


Background: In the context to mental illness metabolic syndrome (MS) has gained significant attention in the last decade. The present research aimed to study the prevalence of MS and its correlates among the alcohol-dependent men at a deaddiction center in Northern India. **Materials and Methods:** A cross-sectional analysis was done for consecutive male subjects who met the diagnosis of alcohol-dependence syndrome currently using alcohol according to the International Clinical Diagnostic criteria- tenth revision mental and behavioral disorder- Clinical description and diagnostic guidelines criteria (ICD-10). The subjects were evaluated for alcohol consumption and the components of MS as per the International Diabetic Federation (IDF) and National Cholesterol Education Program Adult Treatment Panel-III (NCEP ATP-III). **Results:** A total of 200 male subjects were studied: 100 subjects meeting ICD-10 criteria for alcohol dependence currently using alcohol; 50 each of genetically related controls and nongenetically related healthy controls. As per the IDF (with ethnicity specific modifications for waist circumference) and NCEP ATP- III definitions, respectively, MS was found to be less prevalent in alcohol-dependent subjects (27% and 18%) in comparison the healthy controls (30% and 20%). **Conclusion:** Findings of the study suggest that irrespective of the amount the current alcohol intake is associated with a lower prevalence of MS and a favorable effect on serum high density lipoproteins and waist circumference. However, the cross-sectional nature of our study does not allow any definitive causal inference.

Key words: Alcohol dependence, metabolic syndrome, prevalence

INTRODUCTION

Metabolic syndrome (MS) comprises of metabolic risk factors including central obesity, glucose intolerance, hyperinsulinemia, low high density cholesterol (HDL-C), high triglycerides (TG) and hypertension. There is good evidence of it contributing to greater risk for type 2 diabetes mellitus and myocardial infarction or cerebrovascular accident.^[1,2]

Its increasing prevalence in the developing countries is attributed to the increasing affluence of middle class, urbanization, mechanization, changes in diet, and the sedentary habits.^[3] The relation between MS and alcohol use is complex. Alcohol is reported to have a favorable effect on plasma HDL-C levels and insulin sensitivity, a detrimental effect on plasma TG concentration, and may contribute to elevation of blood pressure.^[4-7] However, the relation between alcohol and obesity is reportedly inconsistent.^[8] Research from America and Europe has reported lower prevalence of MS in populations with moderate alcohol consumption; however, alcohol intake quantification was not standardized.^[4-6,9] One study has suggested detrimental effects of alcohol on all the components of MS, except the HDL-C levels.^[10] Another study of adults in Shanghai reported lower prevalence of MS, irrespective of amount of alcohol

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consumption.^[11] Most of the research on correlation between alcohol and MS has included only few heavy alcohol-consuming subjects; only three studies have included alcohol-dependent subjects.^[12-14] The present research aimed to find the prevalence and specified correlates of MS in alcohol-dependent men in comparison to nonalcohol-dependent control groups.

MATERIALS AND METHODS

The study was conducted at the Drug De-addiction and Treatment Centre (DDTC) of the Department of Psychiatry at the multispecialty general hospital of Post Graduate Institute of Medical Education and Research (PGIMER), Chandigarh, India. The study had the ethical clearance from the institute's research committee. The sample comprised of consecutive consenting patients (men aged 30-60 years) who attended the DDTC between 1st July 2009 and 30th June 2010 meeting the diagnostic criteria for alcohol dependence as per the ICD-10 (WHO, 1992).^[15] The subjects were excluded if: They had a comorbid diagnosis of another substance dependence except for tobacco, type 1 diabetes mellitus, autoimmune inflammatory illness; were using medications that effect the components of MS (e.g., steroids); or were nonconsenting. The patients were included after the completion of detoxification with benzodiazepines and prior to starting any pharmacoprophylaxis. A control group of men who were genetically related to the index group but did not meet the ICD-10 criteria for alcohol harmful use and dependence was included in the study as the traits comprising the MS have been shown to be inherited.^[16-18] Another control group of men from the general population, biologically unrelated to the cases and who did not meet the ICD-10 criteria for alcohol harmful use and dependence, was also included in the study.

For the assessment of MS the National Cholesterol Education Program – Third Adult Treatment Panel (NCEP-ATP III)^[19] and International Diabetes Federation (IDF)^[20] criteria were preferred because these represent the consensus definition of MS for both clinical and epidemiological work and also take into cognizance the differential profiles of South Asian subjects. NCEP ATP-III defines MS in men by ≥ 3 of the following measurements: Waist girth ≥ 102 cm; TGs ≥ 1.7 mmol/L, use of fibrates or nicotinic acid; high density lipoprotein (HDL) cholesterol ≤ 1.03 mmol/L, use of fibrates or nicotinic acid; blood pressure $\geq 130/85$ mm of Hg or use of anti-hypertensive medication; fasting glucose ≥ 5.6 mmol/L, use of oral hypoglycemic medication or insulin. In comparison, IDF definition of MS requires central obesity (defined as waist circumference with ethnicity specific values;

which for South-Asian men ≥ 90 cm) and any two of the following factors: TGs ≥ 150 mg/dL (1.7 mmol/L) or specific treatment for this lipid abnormality, serum HDL-cholesterol < 40 mg/dL (1.03 mmol/L) or specific treatment for this lipid abnormality, systolic blood pressure (SBP) ≥ 130 or diastolic blood pressure (DBP) ≥ 85 mm of Hg or treatment of previously diagnosed hypertension, fasting plasma glucose (FPG) ≥ 100 mg/dL (5.6 mmol/L), or previously diagnosed type 2 diabetes. If FPG is above 5.6 mmol/L or 100 mg/dL, oral glucose tolerance test (OGTT) is strongly recommended but is not necessary to define the presence of MS.

The sociodemographic and clinical data were obtained through clinical interviews with the subjects and reliable attendants living with them for at least 2 preceding years. Detailed dietary history was taken by 24-hour recall method i.e., detailed food intake of full single day (day prior to assessment) from early morning to night from the patient and corroborated from the informants. The total calorie, protein and fat intake was calculated as per the tool (Dietetics for You) devised and used by Department of Dietetics, PGIMER. Severity of alcohol dependence in the patients was assessed quantitatively using Alcohol Dependence Scale (ADS) – a reliable and valid research and clinical tool.^[21]

Study subjects were assessed by Self Report Questionnaire (SRQ)^[22,23] and screened for present, past and family history of mental disorder, using a detailed clinical interview. Health Promoting Lifestyle Profile-II (HPLP-II),^[24] used extensively in health promotion research to explore correlates or determinant of health promoting lifestyle, was administered to assess the lifestyle and quantify the health related activities of the subjects. HPLP-II is a 52-item self report instrument in which items are scored from 1 to 4 with 1=never, 2=sometimes, 3=often, and 4=routinely. In our study the subjects were assessed on two subscales/factors, i.e., physical activity and nutritional habit containing 8 and 9 items, respectively, and yielding minimum and maximum scores of 17 and 68, respectively. Waist circumference (in centimetres) was measured using a measuring tape in the horizontal plane midway between the inferior margin of the ribs and the superior border of the iliac crest at the end of a normal expiration. An overnight fasting venous blood sample was drawn under aseptic condition to measure the TGs cholesterol (TG-C), high-density lipoprotein cholesterol (HDL-C), fasting blood sugar (FBS) and the low density lipoprotein cholesterol (LDL-C) on random access auto-analyzer modular-P in the Clinical Biochemistry Laboratory, Department of Biochemistry at our institute. To control for potential confounding factors affecting the study variables (lipid

profile and fasting blood sugar) liver function test and renal function test were done. All anthropometric measurements were done by one of the authors (JA). The body mass index (BMI) was calculated from the weight and height using the formula weight in kg divided by the square of the height in meters (kg/m^2). Blood pressure was measured using standard mercury manometer. Two readings at 5-minute intervals were recorded and if a high blood pressure ($\geq 140/90$) was noted a third reading was taken after 30 min. The lowest of the three readings was taken as the blood pressure. MS was diagnosed if subjects met the criteria according to either the NCEP ATP-III or the IDF definition of MS.

Statistical analysis

The data were analyzed using SPSS version 15.0 for Windows (Chicago, IL). Mean and standard deviation (SD) with 95% confidence interval (CI) were calculated for continuous variables. Frequencies and percentages (%) were calculated for nominal and ordinal variables. Comparison of various continuous variables e.g., socio-demographic, clinical and MS parameters across the three groups were done by using one way Analysis of Variance (ANOVA) and post-hoc analysis. Pearson's Product Moment correlation and Spearman Rank correlation were used to assess the association of sociodemographic and clinical variables and MS. Cohen's κ was used to assess the concordance between the two major diagnostic definitions (IDF and NCEP ATP III) for MS.

RESULTS

A total of 200 males were included in the study. The alcohol dependent ($N=100$), genetic control ($N=50$), and healthy control ($N=50$), groups were similar in age (mean \pm SD: 41.6 ± 7.81 , 42.0 ± 11.94 , and 42.0 ± 7.41 years, respectively). Of the 100 alcohol-dependent subjects, 47% were codependent on tobacco, and 3% were nondependent users of tobacco. While 52% of genetic controls and 32% of healthy controls were substance nonusers, 36% of genetic controls and 42% of healthy controls were nondependent users of alcohol. Across all the study groups commonest alcoholic drinks were whisky (64%, 30% and 66% respectively in patients, genetic and healthy controls) followed by country made liquor/spirit (33%, 10% and 2%, respectively, in patients, genetic and nongenetic controls); the use of beer, wine and other spirits like rum and vodka was minimal.

Majority of study subjects ($n=176$, 88%) were free of any comorbid physical illness. Only 19% of the patients had a past history of treatment or hospitalization for substance related problems. Only 26% of the study

subjects had a family history of physical illness; hypertension was the commonest disorder with a prevalence of 9.5%. A physical illness was present in 15% of patients and 18% of genetic controls; hypertension and diabetes mellitus were the most common comorbidities.

As per IDF, the MS was present in 27%, 22%, and 30% of patients, genetic controls and healthy controls, respectively; there were no significant group differences. As per NCEP ATP-III, the MS was present in 18%, 12% and 20% of patients, genetic controls and healthy controls respectively. The concordance for MS between the two classificatory systems, i.e., NCEP-ATP III and IDF, was low with Cohen's κ being 0.522.

Table 1 shows that the three study groups were significantly different for body mass index, high density lipoproteins, total duration of daily activity, subtotal physical activity score on Health Promoting Lifestyle Profile (HPLP), total and mean HPLP score while being similar for intake of proteins, fats and calories per day and mean height, waist circumference, systolic and diastolic blood pressure, fasting blood sugar levels, low density lipoproteins, serum TGs and subtotal nutritional habits score of HPLP. In the three groups among the subjects who fulfilled criteria for MS as per the IDF the daily duration of activity differed significantly; healthy controls were the most active and genetic controls the least active (mean \pm SD: 52.00 ± 65.70 , 13.88 ± 26.25 , 8.18 ± 9.40 minutes daily respectively; $P=0.008$). The BMI was significantly different across the three groups being highest among the healthy controls and lowest among the patients (mean \pm SD: 28.23 ± 3.39 , 27.44 ± 3.39 , and 25.57 ± 3.71 kg/m^2 respectively; $P=0.045$).

Table 2 compares the clinical profile of patients, and genetic and healthy controls with IDF diagnosed MS. The three groups were similar for current age, age at onset of substance use, waist circumference, FBG, HDL-C, TGs, SBP, DBP, urban/rural location. However, healthy controls had a higher duration of activity (mean \pm SD: 52.0 ± 65.7 vs 13.88 ± 26.25 among patients vs 8.18 ± 19.40 among genetic controls, $P=0.008$), a higher BMI (mean \pm SD: 28.23 ± 2.65 vs 25.57 ± 3.71 among patients vs 27.44 ± 3.39 among genetic controls, $P=0.045$), and less often a sedentary life style (53.33% vs 74.08% among patients vs 81.82% among genetic controls; $P=0.027$).

The clinical and metabolic profile of the subjects with and without MS as per IDF is compared in Table 3. The two groups were similar for duration of alcohol dependence, duration of activity, TG, SBP. Compared to those without MS, the subjects with MS had higher age ($P=0.022$), weight, waist

Table 1: Selected characteristics of the study groups

Variable	Patients Mean (standard deviation) (N=100)	Genetic controls Mean (standard deviation) (N=50)	Healthy controls Mean (standard deviation) (N=50)	ANOVA/F value	P value
Age (years)	41.6 (7.81)	42.0 (11.94)	42.0 (7.41)	0.025	3.767
Daily dietary calorie intake (Kcal)	1895.60 (550.68)	1884.40 (374.28)	1917.20 (333.27)	0.066	0.936
Height (cm)	167.17 (5.87)	167.28 (5.59)	168.80 (5.61)	1.455	0.236
Body mass index (Kg/m ²)	23.00 (3.76)	24.51 (3.69)	25.54 (3.18)	8.879	<0.001 [†]
Waist circumference (cm)	89.54 (10.46)	89.36 (11.01)	89.78 (10.90)	0.019	0.981
Waist circumference ≥90 cm (n;%)	52 (52)	25 (50)	26 (52)		
Systolic blood pressure (mmHg)	122.26 (17.31)	123.32 (16.72)	140.68 (14.41)	1.139	0.322
Systolic blood pressure ≥130 mmHg (n;%)	40 (40)	18 (36)	12 (24)		
Diastolic blood pressure (mmHg)	81.34 (11.24)	80.32 (9.91)	79.38 (6.79)	0.663	0.517
Diastolic blood pressure ≥85 mmHg (n;%)	36 (36)	13 (26)	12 (24)		
Fasting blood glucose (mg/dL)	98.60 (30.24)	90.10 (12.19)	96.00 (22.98)	1.919	0.149
Fasting blood glucose ≥100 mg/dl (n;%)	27 (27)	8 (16)	14 (28)		
High density lipoproteins (mg/dL)	53.15 (16.87)	48.93 (12.61)	44.09 (11.03)	6.548	0.002 [†]
High density lipoproteins <40 mg/dl (n;%)	17 (17)	9 (18)	21 (42)		
Low density lipoproteins (mg/dL)	117.76 (38.74)	122.49 (31.17)	112.05 (33.07)	1.079	0.342
Triglycerides (mg/dL)	158.22 (58.73)	155.73 (54.15)	163.71 (100.76)	0.171	0.843
Triglycerides ≥150 mg/dl (n;%)	51 (51)	20 (40)	22 (44)		
Total duration of daily activity (in min)	16.65 (40.18)	23.40 (36.58)	49.80 (54.87)	9.856	<0.001 [†]
Total HPLP [‡] score	22.06 (4.99)	23.36 (4.60)	24.32 (4.81)	3.857	0.023*
Subtotal physical activity score on HPLP	9.19 (2.67)	9.8 (2.71)	10.4 (2.76)	3.468	0.033*
Subtotal nutritional habits score on HPLP	13.29 (4.00)	13.54 (2.99)	13.92 (2.93)	0.535	0.587
Mean HPLP score (total HPLP score/total items of HPLP)	1.29 (0.29)	1.37 (0.27)	1.43 (0.28)	3.857	0.023*

* $P < 0.05$, [†] $P < 0.01$, [‡]HPLP score – Health-promoting lifestyle profile score

Table 2: MS and its relationship with clinical variables in the study groups

	MS present (as per IDF)			Fischer value/P value
	Patients Mean (standard deviation)	Genetic controls Mean (standard deviation)	Healthy controls Mean (standard deviation)	
Age (years)	43.2 (7.42)	46.18 (11.27)	40.8 (8.67)	1.223/0.303
Age of starting of substance (years)	20.5 (4.49)	18.75 (8.06)	18.5 (1.84)	0.838/0.440
Duration of activity (minutes/day)	13.88 (26.25)	8.18 (19.40)	52.00 (65.70)	5.254/0.008 [†]
Waist circumference (cm)	98.84 (8.09)	100.27 (6.57)	99.26 (7.04)	0.011/0.994
Body mass index (kg/m ²)	25.57 (3.71)	27.44 (3.39)	28.23 (2.65)	3.312/0.045*
Fasting blood glucose (mg/dL)	117.22 (46.83)	98.54 (17.12)	110.80 (33.48)	0.904/0.411
High density lipoproteins (mg/dL)	49.10 (12.34)	45.66 (9.10)	39.56 (10.71)	1.043/0.593
Triglycerides (mg/dL)	203.67 (55.48)	177.46 (68.55)	195.14 (101.76)	1.861/0.394
Systolic blood pressure (mm Hg)	132.00 (14.88)	133.27 (12.47)	128.13 (7.65)	0.632/0.536
Diastolic blood pressure (mm Hg)	86.74 (9.95)	88.54 (9.03)	85.26 (5.53)	0.447/0.642
Locality, n (%)				
Urban	12 (44.45)	7 (63.64)	6 (40)	2.48/0.289
Rural	15 (55.55)	4 (36.36)	9 (60)	6.5/0.0387
Lifestyle, n (%)				
Active	7 (25.92)	2 (18.18)	7 (46.67)	3.125/0.209
Sedentary	20 (74.08)	9 (81.82)	8 (53.33)	7.189/0.027*

* $P < 0.05$, [†] $P < 0.01$, MS – Metabolic syndrome; IDF – International diabetic federation

circumference, BMI, FBG, DBP, SBP > 130 mm Hg, WC, TG > 150 mg/dL ($P < 0.001$), and HDL-C ($P = 0.019$).

Correlation analysis of sociodemographic and clinical variables with the outcome variables for MS showed a significant correlation with weight and body mass index

for all except low high density lipoprotein cholesterol components of MS in the patient group.

DISCUSSION

Prevalence of MS: In the present study the prevalence

Table 3: Clinical and metabolic profile of the subjects with and without MS

Variable	MS as per IDF		P value
	Present (N=53) mean (SD)	Absent (N=147) mean (SD)	
Age (years)	43.1 (8.70)	39.8 (9.04)	0.022*
Duration of dependence (years)	8.5 (7.10)	7.4 (6.46)	0.436
Duration of activity (min)	23.5 (43.71)	27.75 (46.09)	0.559
Weight (kg)	75.85 (10.37)	64.57 (10.54)	<0.001†
Waist circumference (cm)	99.26 (7.39)	86.05 (9.42)	<0.001†
BMI (kg/m ²)	26.71 (3.53)	23.03 (3.33)	<0.001†
FBG (mg/dL)	111.52 (38.83)	90.16 (14.32)	<0.001†
HDL-C (mg/dL)	45.69 (11.83)	51.31 (15.74)	0.019*
TG (mg/dL)	195.81 (73.04)	145.68 (64.58)	0.055
SBP (mm Hg)	131.16 (12.66)	125.67 (85.37)	0.642
DBP (mm Hg)	86.69 (8.65)	78.31 (9.46)	<0.001†
WC ≥90 cm	53 (100)	49 (33.33)	<0.001†
SBP ≥130 mm Hg (%)	38 (71.7)	32 (21.76)	<0.001†
DBP ≥85 mm Hg (%)	35 (66.03)	26 (17.69)	<0.001†
FBG ≥100 (%)	32 (60.37)	17 (11.56)	<0.001†
HDL-C <40 mg/dL (%)	19 (35.85)	28 (19.04)	0.013*
TG ≥150 mg/dL (%)	42 (79.24)	51 (34.69)	<0.001†

* $P < 0.05$, † $P < 0.001$, MS – Metabolic syndrome; BMI – Body mass index; FBG – Fasting blood glucose; HDL-C – Low high density cholesterol; TG – Triglycerides; SBP – Systolic blood pressure; DBP – Diastolic blood pressure; WC – Waist circumference; SD – Standard deviation; IDF – International diabetic federation

of MS was 27%, 22% and 30% for patients, genetic controls and healthy controls respectively by IDF criteria, and 18%, 12% and 20% for patients, genetic controls and healthy controls respectively by NCEP ATP-III criteria. The higher prevalence of MS by IDF criteria can be attributed to the lower cut-off of the waist circumference as compared to the NCEP ATP-III criteria. The reporting of higher prevalence of MS by IDF criteria has been a consistent finding in research from India and other parts of world.^[25-29] However, unlike another study from our region^[25] reporting a high concordance between the IDF and ATP-III criteria we found only a moderate concordance ($k=0.522$) between these two criteria of MS; this might have been because of a lower sample size in our study.

The prevalence of MS in alcohol-dependent subjects in the present study at 27% is within the range of 5-31% reported for alcohol-dependent patients from the western countries.^[12-14] This rate is also in line with the rate of 26.4% reported from our center using the IDF criteria with alcohol and opioid-dependent subjects; however, in that study 17.3% patients were having psychiatric comorbidity and were on psychotropics.^[30] In contrast, the rate of 27% for MS found in the present study is lower than that reported by two community-based cross-sectional studies done in our/similar catchment areas on general population, where prevalence of MS was recorded in 45.3% and

39.5% by modified NCEP ATP-III and IDF criteria respectively in one study,^[25] and in 47% by IDF criteria in the other study.^[26]

Also, a meta-analysis of observational studies from the western countries demonstrated alcohol users to have lower prevalence of MS as compared to the current nondrinkers; however, none of the included studies had sampled alcohol-dependent subjects.^[31]

Alcohol use

In alcohol users, the western research^[10-13] has reported increased rate of MS in those consuming >40-60 g alcohol/day, while rates of MS by IDF were similar in general population and nondependent users. In contrast, in our study the patient group comprised of subjects dependent on alcohol for about 9.7 ± 6.7 years with daily alcohol intake of more than 60 g/day. However, the finding that cannot be ignored is that the rates of MS were 22% in our genetic control group and 30% in healthy control group, clearly showing that no definitive pattern exists between MS and alcohol nonuse or alcohol nondependent use.

Components of MS

The commonest components of MS in the present study were waist circumference >90 cm (52%) and raised TGs (51%); the differences across groups were not significant. The finding of raised TGs among the alcohol-dependent subjects in the present study is consistent with some studies,^[10,11,32] while being inconsistent with others reporting raised TGs to be the commonest metabolic abnormality.^[10,33] However, the relationship of obesity and alcohol consumption has been generally found to be inconsistent.^[8] Some studies show light to moderate drinking as not associated with weight gain while others show significant weight gain in subjects drinking more than 2 drinks per day.^[34-37] Considering all three groups in our study together, all components of MS as per the IDF definition differentiated those with and those without MS ($P < 0.001$). Weight and BMI were correlated with all the components of MS except HDL-C. This implies that the measurement of height, weight and waist circumference should be a part of the initial clinical assessment of all alcohol-abusing patients seeking any medical consultation.

Alcohol, diet and physical activity

Dietary patterns, physical activity and alcohol consumption have been found to be associated with same outcome with respect to the components of MS, cardiovascular disorders and other chronic disorders. Some studies have assessed the pattern of alcohol consumption while others the quantities of daily alcohol intake and diet in relevance to chronic disease outcomes.^[37-39] Decreased physical activity and sedentary lifestyle have been found to be

associated with MS and its components.^[40] In our study we assessed the dietary pattern and physical activity of subjects clinically and on HPLP. We found no difference with respect to the total calorie intake as assessed by recall method as well as on the subtotal nutritional score of HPLP-II in contrast to previous studies where quality of diet and calorie intake was found to be abnormal in subjects consuming higher amounts of alcohol.^[35,36] However, in keeping with an earlier study,^[40] there was significant difference on the physical activity profile of three groups in that subjective reporting of duration of physical activity was more in healthy controls (52 ± 65.70 , 8.18 ± 19.40 and 13.88 ± 26.25 , respectively, in healthy controls, genetic controls and patients; $P < 0.001$) as well as that assessed on the subtotal physical activity score of HPLP-II (10.4 ± 2.76 , 9.8 ± 2.71 , 9.19 ± 2.67 , respectively, in healthy controls, genetic controls and patients; $P < 0.05$).

LIMITATIONS AND CONCLUSIONS

Our study suffered from the following limitations. The sample was not large. All the patients were hospital attendees. The study sample comprised of only men, and the MS related factors affecting gender were not assessed. The data on alcohol consumption was based on self-declaration implying the possibility of under-reporting, and possibly explaining gross variations across the three groups (few subjects reported lifetime nondrinking). The definition of diabetes was based on a single laboratory measurement. Due to lack of sufficient numbers we failed in our intent to assess the effect on MS of the pattern of alcohol use like continuous versus binge drinking. Another limitation was our failure to control the study for the significant confounder of tobacco use. Dietary calculations were based on the recall method and relied heavily on self-reporting by the subjects. Because of these limitations our results cannot be generalized to the community based nonhospital attending alcohol-dependent subjects. However, within these limitations our study indicates that even larger quantity of alcohol consumption is associated with lower prevalence of MS at least in this part of the world. However, this finding must not overshadow the increased morbidity and mortality associated with alcohol dependence. Further research is warranted to confirm these findings with large number of subjects in this category as well as in other regional settings.

REFERENCES

1. Isomaa B, Almgren P, Tuomi T, Forsen B, Lathi K, Nissen M, *et al.* Cardiovascular morbidity and mortality associated with the metabolic syndrome. *Diabetes Care* 2001;24:683-9.
2. Stern MP, Williams K, Gonzalez-Villalpando C, Hunt KJ, Haffner SM. Does the metabolic syndrome improve identification of individuals at risk of type-2 diabetes and/or cardiovascular disease? *Diabetes Care* 2004;27:2676-81.
3. Gupta R, Misra A. Type 2 diabetes in India: Regional disparities. *Br J Diabetes Vasc Dis* 2007;7:12-6.
4. Goude D, Fagerberg B, Hulthe J. Alcohol consumption, the metabolic syndrome and insulin resistance in 58-year-old clinically healthy men (AIR study). *Clin Sci* 2002;102:345-52.
5. Lazarus R, Sparrow D, Weiss ST. Alcohol intake and insulin levels. The normative aging study. *Am J Epidemiol* 1997;145:909-16.
6. Rimm EB, Williams P, Fosher K, Criqui M, Stampfer MJ. Moderate alcohol intake and lower risk of coronary heart disease: Meta-analysis of effects on lipids and haemostatic factors. *Br Med J* 1999;319:1523-8.
7. Marmot MG, Elliott P, Shipley MJ, Dyer AR, Ueshima H, Beavers DG, *et al.* Alcohol and blood pressure: The INTERSALT study. *Br Med J* 1994;308:1263-7.
8. Sakurai Y, Umeda T, Shinchi K, Honjo S, Wakabayashi K, Todoroki I, *et al.* Relation of total and beverage specific alcohol intake to body mass index and waist-to-hip ratio: A study of self-defence officials in Japan. *Eur J Epidemiol* 1997;13:893-8.
9. Rosell M, Faire UD, Hellenius M. Low prevalence of the metabolic syndrome in wine drinkers-is it the alcohol beverage or the lifestyle? *Eur J Clin Nutr* 2003;57:227-34.
10. Yoon YS, Oh SW, Baik HW, Park HS, Kim WY. Alcohol consumption and the metabolic syndrome in Korean adults: The 1998 Korean National Health and Nutrition Examination Survey. *Am J Clin Nutr* 2004;80:217-24.
11. Fan JG, Cai XB, Li XJ, Dai F, Zhu J. Alcohol consumption and metabolic syndrome among Shanghai adults: A randomized multistage stratified cluster sampling investigation. *World J Gastroenterol* 2008;14:2418-24.
12. Kahl KG, Greggersen W, Schweiger U, Cordes J, Correll CU, Ristow J, *et al.* Prevalence of the metabolic syndrome in men and women with alcohol dependence: Results from a cross-sectional study during behavioural treatment in a controlled environment. *Addiction* 2010;105:1921-7.
13. Jarvis CM, Hayman LL, Braun LT, Schwertz DW, Ferrans CE, Piano MR. Cardiovascular risk factors and metabolic syndrome in alcohol and nicotine dependent men and women. *J Cardiovasc Nurs* 2007;22:429-35.
14. Teixeira PJ, Rocha FL. The prevalence of metabolic syndrome among psychiatric inpatients in Brazil. *Rev Bras Psiquiatr* 2007;29:330-6.
15. World Health Organization. Definition, diagnosis and classification of diabetes mellitus and its complications. Report of a WHO consultation. Geneva: World Health Organisation; 1999.
16. Lin HF, Albala BB, Juo SH, Park N, Rundek T, Sacco RL. Heritabilities of the metabolic syndrome and its components in the Northern Manhattan Family Study. *Diabetologia* 2005;48:2006-12.
17. Mitchell BD, Kammerer CM, Blangero J, Mahaney MC, Rainwater DL, Dyke B, *et al.* Genetic and environmental contributions to cardiovascular risk factors in Mexican Americans: The San Antonio Family Heart Study. *Circulation* 1996;94:2159-70.
18. Henkin L, Bergman RN, Bowden DW, Ellsworth DL, Haffner SM, Langefeld CD, *et al.* Genetic epidemiology of insulin resistance and visceral adiposity. The IRAS Family Study design and methods. *Ann Epidemiol* 2003;13:211-7.
19. National Cholesterol Education Program. Executive Summary of the Third Report of the National Cholesterol

- Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA* 2001;285:2486-97.
20. Alberti KG, Zimmet PZ, Shaw J. Metabolic syndrome—a new worldwide definition. A Consensus Statement from the International Diabetes Federation. *Diabet Med* 2006;23:469-80.
 21. Skinner HA, Allen BA. Alcohol dependence syndrome: Measurement and validation. *J Abnorm Psychol* 1982;91:199-209.
 22. World Health Organisation. A User's Guide to the Self Reporting Questionnaire (SRQ); Division of Mental Health World Health Organisation. WHO. Geneva: 1994.
 23. Harding TW, Climent CE, Diop M, Giel R, Ibrahim HH, Murthy RS, *et al.* The WHO collaborative study of strategies for extending mental health care-II: The development of new research methods. *Am J Psychiatry* 1983;140:1474-89.
 24. Walker SN, Sechrist KR, Pender NJ. The health-promoting lifestyle profile: Development and psychometric characteristics. *Nurs Res* 1987;36:76-81.
 25. Mangat C, Goel NK, Walia DK, Aggarwal N, Sharma MK, Kaur J, *et al.* Metabolic syndrome: A challenging health issue in highly urbanized Union Territory of north India. *Diabetol Metab Syndr* 2010;2:19.
 26. Ravikiran M, Bhansali A, Ravikumar P, Bhansali S, Dutta P, Thakur JS, *et al.* Prevalence and risk factors of metabolic syndrome among Asian Indians: A community survey. *Diabetes Res Clin Pract* 2010;89:181-8.
 27. Hu G, Lindstrom J, Jousilahti P, Peltonen M, Sjoberg L, Kaaja R, *et al.* The increasing prevalence of metabolic syndrome among Finnish men and women over a decade. *J Clin Endocrinol Metab* 2008;93:832-6.
 28. Can AS, Bersot TP. Analysis of agreement among definitions of metabolic syndrome in non-diabetic Turkish adults: A methodological study. *BMC Public Health* 2007;7:353.
 29. Hazallah F, Albeti H, Ben Khalifa F. The metabolic syndrome in an Arab population: A first look at the new International Diabetes Federation criteria. *Diabet Med* 2006;23:441-4.
 30. Mattoo SK, Chakraborty K, Basu D, Ghosh A, Kumar V, Kulhara P. Prevalence and correlates of metabolic syndrome in alcohol and opioid dependent inpatients. *Indian J Med Res* 2011;134:341-48.
 31. Alkerwi A, Michel B, Michel V, Jessica B, Lise LM, Adelin A, *et al.* Alcohol consumption and the prevalence of metabolic syndrome: A meta-analysis of observational studies. *Atherosclerosis* 2009;204:624-35.
 32. Baik I, Shin C. Prospective study of alcohol consumption and metabolic syndrome. *Am J Clin Nutr* 2008;87:1455-63.
 33. Kato I, Kiyohara Y, Kubo M, Tanizaki Y, Arima H, Iwamoto H, *et al.* Insulin-mediated effects of alcohol intake on serum lipid levels in a general population: The Hisayama Study. *J Clin Epidemiol* 2003;56:196-204.
 34. Wannamethee SG, Field AE, Colditz GA, Rimm EB. Alcohol intake and 8-year weight gain in women: A prospective study. *Obes Res* 2004;12:1386-96.
 35. Wannamethee SG, Shaper AG. Alcohol, body weight, and weight gain in middle-aged men. *Am J Clin Nutr* 2003;77:1312-7.
 36. Liu S, Serdula MK, Williamson DF, Mokdad AH, Byres T. A prospective study of alcohol intake and change in body weight among US adults. *Am J Epidemiol* 1994;140:912-20.
 37. Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, 2010. vol. D7. Beltsville, MD: Agricultural Research Services, US Department of Agriculture; 2010. p. 6-8.
 38. Breslow RA, Guenther PM, Smothers BA. Alcohol drinking patterns and diet quality: The 1999-2000 national health and nutrition examination survey. *Am J Epidemiol* 2006;163:359-66.
 39. Jacques PF, Tucker KL. Are dietary patterns useful for understanding the role of diet in chronic disease? *Am J Clin Nutr* 2001;73:1-2.
 40. Santos AC, Ebrahim S, Barros H. Alcohol intake, smoking, sleeping hours, physical activity and the metabolic syndrome. *Prev Med* 2007;44:328-34.

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