Effects of potentially modifiable risk factors on the health of adults in the Eastern Province of KSA

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Abstract

Objectives: The purpose of this study was to investigate the association between selected major modifiable risk factors including life style habits, household income and smoking on health.

Methods: This cross-sectional study was conducted during 2015–2016 among 104 healthy men aged 38 ± 8 years. The data were collected using a self-administered questionnaire that enquired about clinical information about blood pressure and body mass index. Venous blood samples were taken to assess the fasting blood glucose (FBG), lipid profile, high density lipoprotein and triglyceride.

Results: Current smoking status and consumed energy drinks were significantly positive risk factors for increased systolic blood pressure and FBG, respectively. Participants with monthly income of more than 10,000 Saudi Riyals showed significantly lower diastolic pressure than those with lower income. However, there was a significant decrease in body weight among those who consumed vegetables.

Conclusion: This study highlights the effect of major modifiable risk factors on health. There is a great need for improving and enhancing a healthy lifestyle behaviour.

Keywords: Blood pressure; Habits; Lipid profile; Sedentary; Smoking

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Introduction

Human health is influenced by many factors, and cultural and social transitions during the process of economic development are considered to influence the pace of the pathogenesis of many diseases emerging in developing countries. The negative associations between socioeconomic factors and health in such societies have been reported in many studies. For example, socio-demographic characteristics, such as income, schooling, race, obesity, physical inactivity, and smoking, are well-known factors associated with an increase in blood pressure.

With "Westernization" and the increasing predominance of sedentary lifestyles, people from developing countries are engaging in unhealthy behaviours that contribute to the development of obesity. For instance, the trend towards increased consumption of artificially sweetened soft drinks, sports drinks, high-energy beverages, and coffee products is observed among adolescents and is linked to the obesity epidemic. The prevalence of obesity is increasing worldwide at an alarming rate in both developing and developed countries, and it has become a serious pandemic health problem. Obesity is a risk factor for many conditions, such as coronary heart disease, metabolic syndrome, type 2 diabetes mellitus, certain cancers, hypertension, and dyslipidaemia, and it is estimated to be the fifth leading cause of mortality worldwide.

In KSA, the findings of various studies on the relationship between obesity and lifestyle have not been encouraging. The rapid socioeconomic and cultural revolution that has occurred in KSA since the discovery of oil is associated with a sharp increase in the rate of obesity. Dietary changes have been implicated in the increasing prevalence of both overweightness and obesity among Saudi adults, adolescents and children in the past few decades. Studies have shown increased consumption of refined foods and animal products at the expense of vegetables and fruits among Saudi Arabians during the past few decades. However, limited studies that quantify these behaviours are available. Most related studies are either surveys or retrospective use of self-reporting surveys and do not investigate the relationship between lifestyle behaviours and health risks such as diabetes mellitus and cardiovascular risk factors such as lipid profile, blood glucose parameters, and blood pressure. Thus, the current study aimed to investigate the association between selected major modifiable risk factors, including lifestyle habits and smoking on indicators of metabolic syndrome including body mass index (BMI); arterial blood pressure; and levels of fasting blood glucose (FBG), high-density lipoprotein (HDL), and triglycerides (TG) of men in Eastern Province, KSA.

Materials and Methods

Participant characteristics

This cross-sectional study was conducted between 2015 and 2016. A total of 104 men who had not been diagnosed with any component disease of metabolic syndrome as defined by the International Diabetes Federation (IDF) and without any immobility-causing disorders were recruited to participate in this study. Participants were selected using convenience sampling. The study details were distributed via the noticeboard at Imam Abdulrahman Bin Faisal University, at some Eastern Province community centres, and via the Whats App to all members of Saudi Diabetes and Endocrinology Association at the Eastern Province of KSA. The participants were from Imam Abdulrahman Bin Faisal University, two schools at Al-Khobar and Saihat, and some Dammam city centres those who expressed an interest to participate and were eligible to participant signed a written informed consent form, according to the Helsinki Declaration. Further, the study protocol was approved by the Internal Review Board of Imam Abdulrahman Bin Faisal University (IRB No. 2014-14-221).

Study procedure

All participants completed a self-administered questionnaire manually. This questionnaire includes items from previously published tools. Through a pilot study of the questionnaire previously conducted among urban and rural students in KSA, it was found to be valid and reliable.

Subjects’ weight was measured using an ordinary scale (portable balance) with indoor clothing on but without shoes. Height was measured to the nearest millimetre with the subjects standing without footwear using a measuring tape that was part of the weighing scale. Subjects were categorized as underweight (BMI, < 18 kg/m²), lean (BMI, 18–25 kg/m²), overweight (BMI, 25–30 kg/m²), or obese (BMI, > 30.0 kg/m²). With an electronic sphygmomanometer (Omron M6 Comfort [HEM-7223-E]; Omron Healthcare Co., Ltd., Kyoto, Japan), the average of three consecutive blood pressure readings recorded with a 5-min interval was obtained using an appropriately sized cuff as each participant sat with their arms supported at heart level. The cuff was wrapped around the upper arm loosely enough to allow two fingers to be easily placed under it. Systolic and diastolic blood pressures (SBP and DBP, respectively) were recorded digitally, and the values appeared on the screen. At a private laboratory, venous blood samples were drawn from an antecubital vein after the participants fasted overnight for a minimum of 10 h. The laboratory analysed the blood samples to assess the following components of the fasting blood profile: HDL, TG, and plasma glucose.

In this study, quantitative methods were used to investigate the health risk factors. The t-test and analysis of variance (ANOVA) were used to analyse differences between lifestyle-related health risk factors. Data were presented as the mean values and standard deviations and analysed using SPSS Version 22. Statistical significance was set at the 5% level. Binary logistic regression was used to determine the influence of independent variables on metabolic syndrome.

Results

The baseline characteristics and demographic data of the study participants are presented in Table 1.

The mean age of the 104 participants was 38 ± 8 years and the mean BMI was 29 ± 5 (overweight category). The mean recorded SBP and DBP were 120 ± 12 and 78 ± 8 mmHg, respectively. As shown in Table 2, the mean TG level was 129 ± 22 mg/dl and HDL level was 42 ± 9 mg/dl.
Leisure time commitments were divided into three subgroups, as shown in Table 3. Most study participants (51%) spent 1–3 h of leisure time on the computer. In terms of dietary habits (Table 4), 38% of participants consumed 2–3 fruits and vegetables per day. Interestingly, most participants (87%) did not consume energy drinks, and only 40% consumed ≤1 of soft drinks per day.

The effect of independent variables (lifestyle factors) on metabolic syndrome components showed four significant associations (Table 5). Participants with high income (>$10,000 SR a month) had significantly lower DBP (76.5 ± 8.7 mm Hg; p = 0.03) than those with medium income (5001–10,000 SR a month; 77.4 ± 7.5 mm Hg) or low income (≤5000 SR a month; 84.1 ± 7.6 mm Hg). SBP differed significantly between individuals who smoked cigarettes for 1–5 years (130.1 ± 13 mm Hg) and non-smokers (118.2 ± 10.7 mm Hg) or individuals who smoked for 6–10 years (114.5 ± 4.6 mm Hg; p = 0.02). Participants who consumed 1 or more vegetables per day had a significantly lower BMI (27.56 ± 4.23 kg/m²) than those who consumed vegetables less frequently (30.65 ± 6.5 kg/m²; p = 0.03). Lastly, participants who consumed energy drinks more than once a week had significantly higher FBG levels (105.1 ± 13.1 mg/dL) than those who did not (96.5 ± 7.6 mg/dL; p = 0.001).

Binary logistic regression was used to determine the odd ratios between lifestyle variables and the occurrence of metabolic syndrome. The variables most strongly associated with the occurrence of this disease were medium to high income and frequent snacking. Participants who consumed snacks 1–3 times a day were 1.46 times more likely to have metabolic syndrome, and those who consumed snacks more than 3 times a day were 3.68 times more likely. Further, participants who earned more than 10,000 SR a month were 1.2 times more likely to have metabolic syndrome.

Discussion

The present study was conducted to assess the role of various lifestyle habits on metabolic risk factors. The findings suggest that there are significant relationships between...
certain lifestyle behaviours and health risk factors. DBP was elevated in the medium-income group, and high income was associated with the occurrence of metabolic syndrome. Cardiovascular disease is now endemic worldwide and no longer limited to economically developed countries. About a third of all deaths in middle-income countries are caused by cardiovascular disease. Socioeconomic status, mainly when measured by income, can vary greatly with time, and there is evidence that blood pressure is sensitive to fluctuations in this variable. Most of the disease burden caused by high blood pressure is borne by low- and middle-income countries. Blood pressure is also considered a physiological consequence of differential exposure to social, physical, and psychological stressors. Despite this, there is limited information linking contextual socioeconomic status and blood pressure in middle- and low-income countries. A better understanding of the pathways linking economic status to blood pressure and metabolic syndrome is essential for designing interventions to reduce hypertension in rapidly developing countries where the prevalence of hypertension is increasing. The direction and magnitude of relationships are not well understood with respect to income as a contributor to hypertension. It could be that in men with very limited economic prospects and education, high income acts as a buffer against psychosocial stress.

Frequent snacking is associated with the occurrence of metabolic syndrome. Economic changes and cultural habits that lead to sedentary lifestyles are usually associated with snack consumption. Socioeconomic factors may be markers of a diverse range of individual- and community-related factors associated with health risks. Many studies have found relationships between socioeconomic indicators and factors such as dietary patterns, physical activity, and availability of healthy foods. Additionally, multiple elements of communities, such as the social environment, social services, resource allocation, and population heterogeneity, interact to shape health indicators through complex, indirect pathways. Meanwhile, in middle-income developing highly westernized countries, similar factors may explain the socioeconomic factor-associated gradient in blood pressure. To curb hypertension, prophylactic and interventional measures are needed, wherein strategies centred on the individual and the community can be combined.

Another important finding of our study was the inverse relationship between total daily vegetable intake and body weight. This finding was in agreement with several other interventional and observational studies, supporting the beneficial role of high dietary fibre intake in preventing weight gain, promoting weight loss, and maintaining a healthy body weight. Findings from different studies support the beneficial role of dietary fibre in body-weight regulation. Pereira et al. proposed physiologic mechanisms by which vegetable consumption helps in weight management. First, fibre-rich foods tend to be more satiating because of their relatively lower energy density and palatability compared to low-fibre foods. Second, dietary fibre, especially soluble fibre, can increase the viscosity of food and slow down digestion, which stimulates the release of gut hormones such as cholecystokinin and glucagon-like peptide 1 and promotes satiety. In addition, dietary fibre could provide a mechanical barrier for the enzymatic digestion of other macronutrients, such as fats and starch, in the small intestine. Moreover, the slower digestion and absorption rate of carbohydrates leads to a reduced postprandial blood glucose response, which over the long term, improves insulin sensitivity and influences fuel partitioning to favour fat oxidation. At present, the benefits of increased vegetable intake in the prevention and treatment of obesity and associated diseases such as diabetes type 2 and cardiovascular diseases are considered to be derived mainly from soluble fibre.

In the present study, we found that consumption of energy drinks was accompanied by a significant increase in FBG levels. Many health problems are reportedly caused by the consumption of energy drinks. Sugar and caffeine may also synergistically increase postprandial hyperglycaemia. It has been reported that energy drinks block insulin-stimulated glucose and reduce glucose uptake. Additionally, the calories provided by energy drinks have many other adverse effects such as calcium deficiency, dental problems, hypertension, and increased BMI. Thus, our results are consistent with those of other studies that reported that consumption of energy drinks could lead to diabetes because of imprecise and incomplete compensation for energy consumed in liquid form.

We found that SBP was significantly higher in moderate smokers (1–5 years) than in those who never smoked, but no such differences were seen in DBP or for long-term smokers. Another study had similar findings. Paradoxically, several studies have reported that blood pressure in smokers is the same as or lower than that in nonsmokers. Stranger still, some studies observed that SBP decreased as the level of cigarette consumption increased. Smoking is the most common cause of avoidable cardiovascular mortality worldwide, and a wealth of research demonstrates the pathophysiological mechanisms by which smoking causes an increase in blood pressure. The mechanism of nicotine-induced hypertension is believed to be via activation of the sympathetic nervous system with release of epinephrine and norepinephrine, which increases myocardial oxygen consumption through a rise in blood pressure, heart rate, and myocardial contractility. Chronic diseases such as combined chronic diseases, diabetes mellitus, cardiovascular disease, and cancer share major risk factors beyond genetics and social inequalities, including smoking and unhealthy diets.

In the present study, leisure time did not differ significantly between the groups and health outcomes. It should be noted that sedentary time, such as daily sitting hours, was not measured in the current study. Previous studies reported no significant association between self-reported sitting time and weight gain. Future studies should include objective measures that can help categorize individuals from the general population on the basis of daily activity and the extent of sedentary behaviour.

The main limitation of this study is its small sample size, although the sample was carefully selected from the main cities of Al-Khobar, Dammam, and Saihat in Eastern Province, KSA. Further large-scale studies are necessary to examine the association between lifestyle habits and metabolic syndrome markers. In addition, sitting time was not recorded and analysed, neither over the entire day nor separately during work and free time.
Conclusion

The significant relationship between the studied risk factors and adverse health outcomes could indicate an emerging health risk. There is a great need to encourage individuals to adopt a healthy lifestyle by tackling risk factors at the societal, community, and individual levels. Further, there is an obvious paucity of research on health risk factors in the Saudi population, and this research gap must be bridged.

Authors’ contribution

MTH and SAA performed the measurements, and AMA was involved in planning and supervised the work, MTH contributed to the interpretation of the results. SAA and AMA processed the experimental data, drafted the manuscript and designed the figures and tables. All authors discussed the results and commented on the manuscript and approved the final draft and responsible for the content of manuscript.

Conflict of interest

The authors have no conflict of interest to declare.

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References

1. Organization WH. Obesity: preventing and managing the global epidemic. World Health Organization; 2000.
2. Abbas F, Brown BW, Lamendola C, McLaughlin T, Reaven GM. Relationship between obesity, insulin resistance, and coronary heart disease risk. J Am Coll Cardiol 2002; 40(5): 937–943.
3. Höflermaan DA, Antunes JLF, Silva DAS, Peres MA. Is income area level associated with blood pressure in adults regardless of individual-level characteristics? A multilevel approach. Health Place 2012, 18(5): 971—977.
4. Gasbarrini A, Piscaglia AC. A natural diet versus modern western diets? A new approach to prevent “Well-Being Syndromes”. Dig Dis Sci 2005; 50(1): 1–6.
5. Tordoff MG, Alleva AM. Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight. Am J Clin Nutr 1990; 51(6): 963–969.
6. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. Am J Clin Nutr 2006; 84(2): 274–288.
7. Yang P, Zhou Y, Chen B, et al. Overweight, obesity and gastric cancer risk: results from a meta-analysis of cohort studies. Eur J Cancer 2009; 45(16): 2867–2873.
8. Freedland SJ, Wen J, Wuerstle M, et al. Obesity is a significant risk factor for prostate cancer at the time of biopsy. Urology 2008; 72(5): 1102–1105.
9. Nguyen NT, Magno CP, Lane KT, Hinojosa MW, Lane JS. Association of hypertension, diabetes, dyslipidemia, and metabolic syndrome with obesity: findings from the National Health and Nutrition Examination Survey, 1999 to 2004. J Am Coll Surg 2008; 207(6): 928–934.
10. James WPT, Jackson-Leach R, Ni Mhurchu C, Kalamar E, Shuyeghi M, Rigby NJ, et al. Overweight and obesity (high body mass index). In: Ezati M, Lopez AD, Rodgers A, Murray CJL, editors. Comparative quantification of health risk: global and regional burden of disease attributable to selected major risk factors. Vol. 1. Geneva: World Health Organization; 2004. pp. 497—596.
11. Alsafi MA, Hakim IA, Harris RB, et al. Prevalence and risk factors of obesity and overweight in adult Saudi population. Nutr Res 2002; 22(11): 1243–1252.
12. Al-Nuaim AA, Bangboye EA, Al-Rubeaan KA, Al-Mazrou Y. Overweight and obesity in Saudi Arabian adult population, role of sociodemographic variables. J Community Health 1997; 22(3): 211–223.
13. Al-Nozha M. Prevention of coronary artery disease-time for action! Ann Saudi Med 1995; 15(4): 309.
14. Alkahtani S, Elikilany A, Alhariri M. Association between sedentary and physical activity patterns and risk factors of metabolic syndrome in Saudi men; a cross-sectional study. BMC Public Health 2015; 15(1): 1234.
15. Organization WH. The world health report 2002: reducing risks, promoting healthy life. World Health Organization; 2002.
16. Matthews KA, Kiefe CI, Lewis CE, Liu K, Sidney S, Yunis C. Socioeconomic trajectories and incident hypertension in a biracial cohort of young adults. Hypertension 2002; 39(3): 772–776.
17. Lawes CM, Vander Hoorn S, Rodgers A. Global burden of blood-pressure-related disease, 2001. Lancet 2008; 371(9623): 1513–1518.
18. Spruijl TM. Chronic psychosocial stress and hypertension. Curr Hypertens Rep 2010; 12(1): 10–16.
19. Reddy KS, Yusuf S. Emerging epidemic of cardiovascular disease in developing countries. Circulation 1998; 97(6): 596–601.
20. Mendez MA, Cooper R, Wilks R, Luke A, Forrester T. Income, education, and blood pressure in adults in Jamaica, a middle-income developing country. Int J Epidemiol 2003; 32(3): 400–408.
21. Florey LS, Galea S, Wilson ML. Macrosocial determinants of population health in the context of globalization. Macrosocial Determinants of Population Health. Springer; 2007. pp. 15–51.
22. Yen IH, Kaplan GA. Poverty area residence and changes in physical activity level: evidence from the Alameda County Study. Am J Public Health 1998; 88(11): 1709–1712.
23. Diez Roux AV, Nieto FJ, Caulfield L, Tyrroler HA, Watson RL, Szkoł M. Neighbourhood differences in diet: the Atherosclerosis Risk in Communities (ARIC) Study. J Epidemiol Community Health 1999; 53(1): 55–63.
24. Diez Roux AV, Mair C. Neighborhoods and health. Am N Y Acad Sci 2010; 1186(1): 125–145.
25. Liu S, Willett WC, Manson JE, Hu FB, Rosner B, Colditz G. Relation between changes in intakes of dietary fiber and grain products and changes in weight and development of obesity among middle-aged women. Am J Clin Nutr 2003; 78(5): 920–927.
26. Birkhedtvedt G, Aaseth J, Flørholmen J, Rytting K. Long-term effect of fibre supplement and reduced energy intake on body weight and blood lipids in overweight subjects. Acta Medica (Hradec Kralove)/Universitas Carolina, Facultas Medica Hradec Kralove 1999; 43(4): 616–622.
27. Newby P, Maras J, Bakun P, Muller D, Ferrucci L, Tucker KL. Intake of whole grains, refined grains, and cereal fiber measured with 7-d diet records and associations with risk factors for chronic disease. Am J Clin Nutr 2007; 86(6): 1745–1753.
28. Pereira MA, Ludwig DS. Dietary fiber and body-weight regulation: observations and mechanisms. Pediatr Clin N Am 2001; 48(4): 969–980.
29. Kolnes A, Ingvaldsen A, Bolling A, et al. Caffeine and theophylline block insulin-stimulated glucose uptake and PKB phosphorylation in rat skeletal muscles. *Acta Physiol 2010*; 200(1): 65–74.

30. Ludwig DS, Peterson KE, Gortmaker SL. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. *Lancet 2001*; 357(9255): 505–508.

31. Seifert SM, Schaechter JL, Hershorin ER, Lipshultz SE. Health effects of energy drinks on children, adolescents, and young adults. *Pediatrics 2011*; 127(3): 511–528.

32. Primatesta P, Falaschetti E, Gupta S, Marmot MG, Poulter NR. Association between smoking and blood pressure evidence from the health survey for England. *Hypertension 2001*; 37(2): 187–193.

33. Seltzer CC. Effect of smoking on blood pressure. *Am Heart J 1974*; 87(5): 558–564.

34. Berglund G, Wilhelmsen L. Factors related to blood pressure in a general population sample of Swedish men. *Acta Medica Scandinavica 1975*; 198(1–6): 291–298.

35. Teo KK, Ounpuu S, Hawken S, et al. Tobacco use and risk of myocardial infarction in 52 countries in the INTERHEART study: a case-control study. *Lancet 2006*; 368(9536): 647–658.

36. Cryer PE, Haymond MW, Santiago JV, Shah SD. Norepinephrine and epinephrine release and adrenergic mediation of smoking-associated hemodynamic and metabolic events. *N Engl J Med 1976*; 295(11): 573–577.

37. Najem B, Houssière A, Pathak A, et al. Acute cardiovascular and sympathetic effects of nicotine replacement therapy. *Hypertension 2006*; 47(6): 1162–1167.

38. Helen Ann Halpin S, Morales-Suarez-Varela MM, Martin-Moreno JM. Chronic disease prevention and the new public health. *Public Health Rev 2010*; 32(1): 120.

39. Ball K, Brown W, Crawford D. Who does not gain weight? Prevalence and predictors of weight maintenance in young women. *Int J Obes 2002*; 26(12): 1570–1578.

40. Blanck HM, McCullough ML, Patel AV, et al. Sedentary behavior, recreational physical activity, and 7-year weight gain among postmenopausal US women. *Obesity 2007*; 15(6): 1578–1588.

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