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Research

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Association between Smoking and Anthropometric Characteristics, Biochemical Markers, and Dietary Intake of Pakistani Male Adult Population

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ABSTRACT

Background/Objectives: A community-based study was conducted to compare the nutritional status between smokers and non-smokers in association with dietary, biochemical and socio-economic characteristics.
Methods: A convenient sampling method was used to enroll 100 smokers and 99 non-smokers aged between 46 and 78 years from the urban and semi-urban areas of district Peshawar, Pakistan. Weight, height, waist and hip circumferences of the subjects were taken while body composition was determined by employing a Bodystat Analyzer. A blood sample was taken from each subject for the determination of serum vitamin A and zinc levels. Subjects were interviewed for a 24-hr dietary recall and demographic and socio-economic characteristics. Student's t-test and bivariate analysis were conducted to compare the mean differences and examine the association between different variables of smoker and non-smoker groups.
Results: The results revealed that there was no significant (p>0.05) difference between the mean age, weight, height and body mass index of smokers and non smokers. However, the mean body fat, waist and hip circumference of the smokers were significantly (p<0.05) lower than the non-smokers. Conversely, the mean serum vitamin A (32.30±15.99 µg/dl) of smokers was significantly (p<0.05) higher than non-smokers (26.50±20.44 µg/dl) but the mean serum zinc concentration of smokers (99.76±27.42 µg/dl) was significantly lower than the non-smokers (108.25±32.20 µg/dl).
Conclusions: The study concludes that anthropometric (body mass index), biochemical (vitamin A and zinc status), dietary (energy intake) and socio-economic (income, profession) characteristics failed to establish an association with smoking as most of the indicators of smokers are comparable to non-smokers.

KEYWORDS: Smoking; Anthropometry; Vitamin A; Zinc, Dietary intake; Pakistan.

INTRODUCTION

Smoking has long been implicated as a risk factor for many chronic diseases, including cardiovascular, respiratory and gastrointestinal diseases and a variety of cancers.[1-3] Tobacco smoke contains many oxidants and free radicals that can cause damage to lipids, proteins, DNA, carbohydrates and other bio-molecules.[4,5] It also contains numerous pro-oxidants capable of producing free radicals and enhancing the oxidative stress *in vivo*.[6] Each puff of tobacco contains approximately 1014 oxidant molecules in the tar phase and approximately 1015 in the gas phase including oxygen and nitrogen derived free radicals.[7] These free radicals are consid-

ered to be the major patho-physiological factors responsible for the development of many chronic diseases.[8]

There is a growing body of evidence that oxidants such as reactive oxygen species are involved in the development of cerebrovascular degenerative diseases, hypertension, increased oxidative stress, impaired nitric oxide bioavailability and endothelial dysfunction.[9,10] Production of reactive oxygen species in quantities that overwhelm the endogenous antioxidant defense system is referred to as oxidative stress and involves the oxidation of molecules in ways that impair cellular function.[11] Numerous epidemiological studies have shown that cigarette smoking causes oxidative stress, impaired antioxidant blood levels, increased risk of cancer, cardiovascular diseases, diabetes, pulmonary hypertension, stroke and premature deaths.[12,13] It has also been estimated that tobacco smoking accounts for 33% of cancer related deaths in male and 10% in women.[14] Cigarette smoking alone has been attributed to annually cause about 5 million deaths worldwide; while the number of the deaths is expected to increase to 10 million by the year 2030 with 70% of deaths in low to middle income countries.[15]

In Pakistan, the prevalence of cigarette smoking has been estimated to be 19.4% among the population aged over 14 years causing serious health, economic and social challenges to the society at large.[16] Cigarette smoking, with no minimum age with regards to the legal purchase of tobacco and related products has multiplied the human sufferings by the overwhelming burden of chronic diseases, disabilities and premature deaths. While the increased vulnerability of smokers to chronic diseases and premature deaths have been partly attributed to elevated oxidative stress and reduced blood antioxidant levels; it remains unclear whether lower antioxidant levels are due to decreased dietary intake of antioxidant rich foods or the depletion of circulating antioxidants through chronic smoke exposure.[17]

The damage that can be caused by free radicals could potentially be minimized by the regular intake of dietary nutrients that are an integral part of enzymatic and non-enzymatic antioxidant systems. Important antioxidant enzymes include copper-zinc superoxide dismutase, manganese superoxide dismutase, ceruloplasmin, selenium glutathione peroxidase, glutathione reductase and catalase.[18] Non-enzymatic antioxidants include vitamin E (alpha-tocopherol), vitamin C (ascorbic acid), vitamin A (retinal), pro-vitamin A (carotenoids) and urate.[19]

No study related to nutritional status and dietary intake of smokers and non-smokers was found in Pakistan which is important from a public health perspective to assess their nutritional status, develop appropriate policies and plan of actions to improve their nutrition well being, combat tobacco smoke and mitigate subsequent health hazards. Considering the deleterious effects of smoking on human health, this study was designed to assess and compare the anthropometric and biochemical characteristics, dietary intake of smokers with non-smokers and examine the relationship between anthropometry, biochemical, dietary and demographic and socio-economic characteristics.

MATERIALS AND METHODS

A community-based study was carried out among male adult smokers and non-smokers in the urban and semi-urban areas of district Peshawar, Pakistan. A non-probability convenience sampling procedure was followed to enroll 100 smokers and 100 non-smokers from two different localities. One of the participants from the non-smoking group migrated in the middle of the study so the sample size was reduced to 99. Inclusion criteria for enrollment of smokers: (i). were male adults aged ≥40 years; (ii). had been smoking two or more cigarettes per day for the last five years; and (iii). were free from all types of infectious and chronic diseases. The inclusion criteria for non-smokers were similar to the smokers except that they were non- smokers. Subjects fulfilling the inclusion criteria were informed about the purpose of the study and informed consent was obtained. The study was approved by the Board of Studies, University of Agriculture, Peshawar, Pakistan.

Participants were interviewed for their demographic and socio economic characteristics and their systolic and diastolic blood pressures were measured in the supine position by a sphygmomanometer. Weight and height of the subjects were taken by following the WHO recommended procedures.[20] Body Mass Index (BMI) of the subjects was calculated using the formula (weight (kg)/height (m^2). Waist circumference was measured at the midpoint between the lower rib and the iliac crest; while hip circumference was measured as the maximum circumference around the buttocks and recorded to the nearest 0.1 cm to assess abdominal and central obesity.[21] Waist to hip ratio (WHR) was determined by applying the equation: waist circumference (cm)/hip circumference (cm). The prevalence of abdominal obesity in smokers and non-smokers was assessed by following the recommended waist circumference cut-off value of (>85 cm) and WHR (>0.90) for Asian adult males.[22] The anthropometric data were compared with the corresponding age reference population, i.e. National Centre for Health Statistic data to generate weight-for-age, weight-for-height and height-for-age Z-score of adults.[23] The subjects were categorized as underweight, normal, overweight, and obese according to the WHO cut off values.[24]

Body composition (body fat; lean body mass; water) and body energy requirements were estimated using a Body Stat Analyzer (BSA 1500) (Bodystat LTD, Douglas, Isle of Man). A 24-hr dietary recall was used to interview the subjects for all the foods and beverages that they had consumed during the last 24-hrs. Dietary energy, carbohydrate, protein, fat, vitamin A and zinc intakes of the smokers and non-smokers were calculated using the Food Composition Table of Pakistan.[25]

Blood retinol and zinc levels were determined by taking about 8 ml of blood from subject's antecubital vein follow-

ing an overnight fast. Blood samples were taken by employing a Vacuette blood collection system (Greiner Bio-One, Monroe, NC). The blood samples were immediately transported to the Department of Human Nutrition Laboratory, the University of Agriculture , Peshawar, Pakistan) where they were centrifuged (Hermle Z 200 A, Wehingen, Germany) for the separation of serum at 3000 rpm for 10 minutes, which was then stored at -80 °C until analysis. All the chemical and analytical procedures were carried out in a dim light environment.

Serum vitamin A was determined by following the method of Bieri, et al.[26] on a Perkin Elmer Series 200 HPLC fitted with a UV/V is 200 at 325 nm, and a Hypersil C18 column (250 mm × 4.6 mm ID, 5 µm). The mobile phase was an isocratic mixture of methanol (95%) and water (5%), applied at a flow rate of 1 ml/minute. The standards used were all-trans retinol and retinyl acetate as internal standard (Sigma Aldrich, St. Louis USA). All chemicals and solvents were ultra-purified HPLC grade. Extraction of retinol from the samples was carried out by taking 100 µl serum in a tube to which 100 µl ethanol containing the internal standard was added and the content was mixed in a vortex mixer. The lipid components were extracted by adding 200 µl n-hexane, followed by vortexing for 45 seconds and centrifugation at 3000 rpm for 2 minutes. The solvent layer was transferred and dried under a gentle stream of nitrogen gas at 60 °C. The dried sample was dissolved in 100 µl methanol for injection into the HPLC.

The prevalence of vitamin A deficiency was determined using the WHO recommended cut-off values.[27] Serum zinc was determined by employing an atomic absorption spectrophotometer (Shimadzu, AA 6300, Kyoto, Japan).[28] One ml serum was transferred into a centrifuge tube and 0.4 ml of 24% Trichloroacetic acid (TCA) was added to it and mixed in a vortex for 30 seconds and then 1.0 ml deionized water was added to it. The sample was centrifuged at 3000 rpm for 10 minutes to remove blood proteins, after which the aqueous solution was analyzed on the atomic absorption spectrophotometer. The prevalence of zinc deficiency was assessed by using standard zinc cut-off value of <12 µmol/L.[29]

Data regarding demographic characteristics, socio-economic status, anthropometric measurements, body composition, biochemical assessment and dietary intake were analyzed using SAS (The SAS Institute, Inc., Cary, NC, USA). Simple statistics and bivariate analysis were carried out on the continuous and ordinal data to examine the mean differences in anthropometric measurements, body composition, biochemical and dietary data at 5% level of significance between the smokers and non smokers. Pearson product-moment correlation coefficients were calculated to determine the relationship between different variables.

RESULTS

General characteristics of smokers and non-smokers presented in Table 1 indicate that there was no significant (p>0.05) difference in the mean age between the smokers and non-smokers. The smokers had an average smoking history of ~19 years and smoked an average of 13 cigarettes per day. All the subjects were married: 49% of the smokers lived in a joint family structure; while the remaining 51% lived in a nuclear family structure. No significant difference (p>0.05) was found between smokers and non-smokers with regards to the mean family size; number of children; monthly income; the subject's education; and profession (Table 1). However, a significantly (p<0.05) higher percentage of non-smoker's spouses were uneducated compared to the smoker's spouses. Conversely, there was no significant difference (p>0.05) between the spouse's professions in the two groups but a higher percentage (4%) of smokers' spouses were employed as compared to the spouses (1%) of non-smokers. This suggests that smoking by males with employed spouses is perceived to be a symbol of higher social standing, signifying an improved socio-economic status and enhanced quality of life of the family.

Variable	Smokers (n=100) Mean±SD	Non-smokers (n=99) Mean±SD	*p*-value
Subject Age (Yrs)	45.88±7.29	46.93 ± 8.39	0.35
Smoking Period (Yrs)	19.37±7.80	None	
Cigarettes per day	12.59±7.62	None	
Family Type			
Joint	49%	37%	0.35
Nuclear	51%	62%	
Marital Status			
Married	100%	100%	
Family Size	8.05±3.14	8.32±4.98	0.65
Number of Children	4.07±2.03	4.16±2.44	0.77
Family Income (rupees)/month*	18680.00±13466	21011.11±22948	0.38

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Subject Education			
Nil	19	19	0.05
Middle	18	19	
Matric	24	22	
Intermediate	8	9	
Graduate	26	13	
Postgraduate	5	17	
Subject Profession			
Public Sector	29	31	0.31
Private Sector	36	24	
Business	32	43	
Unemployed	2	2	
Retired	1	0	
Spouse Education			
Nil	42	61	0.00
Middle	20	5	
Matric	14	19	
Intermediate	14	4	
Graduate	6	8	
Postgraduate	4	3	
Spouse Profession			
Housewife	96	98	0.18
Working	4	1	

*1 US $=~100 Pakistani rupees as per (February 2015).

Table 1: General characteristics of smokers and non-smokers.

Anthropometric data as shown in Table 2 indicate that there was no significant ($p>0.05$) difference in the mean weight, height, weight-for-age, height-for-age, weight-for-height Z-scores, body mass index and waist-to-hip ratio between the smokers and non-smokers. However, the mean waist and hip circumferences of smokers were significantly ($p<0.05$) lower than the non-smokers. Data on body composition revealed that smokers had significantly ($p<0.05$) lower mean body fat and lean body mass but there was no significant difference in the mean basal metabolic energy requirements, total energy requirements and systolic and diastolic blood pressure values between the smokers and non-smokers (Table 2).

The mean serum vitamin A and zinc levels revealed that smokers had a significantly ($p<0.05$) higher mean serum vitamin A level but a significantly ($p<0.05$) lower mean serum zinc level than the non-smokers (Table 3). The prevalence of vitamin A deficiency among smokers (21%) was found to be significantly ($p<0.05$) lower than in the non-smokers (41%). However, no significant ($p>0.05$) difference was observed in the prevalence of zinc deficiency between the smokers and non-smokers. The correlation analysis also failed to reveal any significant ($p<0.05$) association between serum vitamin A and zinc levels and the number of cigarettes smoked per day or the duration of smoking.

Dietary nutrients intake results showed that the mean energy and carbohydrate intakes of smokers were significantly ($p<0.05$) higher than the non-smokers (Table 4). Conversely, mean protein and fat intakes of smokers were significantly ($p<0.05$) lower than the non-smokers. Though, the mean dietary vitamin A intake of smokers was slightly higher and dietary zinc intake lower than the non-smokers but the differences were non-significant ($p>0.05$).

Correlation coefficients revealed that there was a significant relationship between weight, height, BMI, waist and hip circumferences, energy, carbohydrates, protein, fat, systolic and diastolic blood pressure but there was a lack of association between the biochemical, anthropometric and dietary intake variables.

DISCUSSION

The hypothesis that smoking adversely affects the nutritional status could not be verified on the basis of the results obtained from anthropometric measurements, serum vitamin A levels and dietary intake of smokers. The lack of significant differences in majority of the physical growth indicators i.e., weight, height, body mass index, weight-for-age, height-for-age and weight-for-height Z-scores between the smokers and non-smokers indicate the insensitivity of anthropometry to detect the adverse effects of smoking. The results are in fair agreement to those of Stolzenberg-Solomon, et al.[30] who reported a non significant difference in the mean weight and height between the

Variable	Smokers (n=100) Mean± SD	Non-smokers (n=99) Mean± SD	*p*-value
Weight (kg)	73.12±13.70	76.17±13.23	0.11
Height (cm)	168.35±6.15	168.84±6.73	0.59
Weight-for-age Z-score	-0.44±1.24	-0.15±1.20	0.10
Height-for-age Z-score	-1.01±0.90	-0.91±0.99	0.43
Weight-for-height Z-score	0.24±1.40	0.53±1.29	0.13
Body mass index	25.77±4.48	26.68±4.17	0.14
Waist (cm)	84.87±11.60	88.61±11.32	0.02
Hip (cm)	94.44±9.49	97.96±8.85	0.00
Waist-to-hip ratio	0.89±0.06	0.90±0.07 0.90± 0.0	0.48
Body fat (%)	23.00±4.76	24.90±4.63	0.00
Lean body mass (%)	76.99±4.76	75.20±4.70	0.00
Basal metabolic energy requirements (Kcal)	1665.32±245.31	1718.70±236.46	0.12
Total energy requirements (Kcal)	2717.32±346.73	2733.30±483.25	0.79
Blood pressure (mm Hg)			
Systolic (mm Hg)	116.70±12.27	116.62±11.54	0.96
Diastolic (mm Hg)	83.20±9.52	82.68±8.15	0.68

Table 2: Anthropometric measurements of smokers and non-smokers.

Variable	Smokers(n=100) Mean±SD	Non-smokers(n=99) Mean±SD	*p*-value
Vitamin A (µg/dl)	32.30±15.99	26.50±20.46	0.03
Zinc (µg/dl)	99.76±27.42	108.25±32.20	0.04
Vitamin A status	**N(%)**	**N(%)**	***p*-value**
Normal	79(79)	58(59)	0.00
Moderate	16(16)	25(25)	
Severe	5(5)	16(16)	
Zinc status			
Normal	76(76)	84(85)	0.12
Deficient	24(24)	15(15)	

Table 3: Vitamin A and zinc status of smokers and non-smokers.

Variable	Smokers (n=100) Mean±SD	Non-smokers (n=99) Mean±SD	*p*-value
Energy (Kcal)	3127.73±389.43	2993.10±448.14	0.02
Carbohydrates (g)	418.96±61.83	374.88±66.14	0.00
Protein (g)	126.96±19.47	141.03±22.43	0.00
Fats (g)	75.89±14.41	94.21±15.65	0.00
Vitamin A (µg)	1407.76±14	1378.80±12	0.06
Zinc (mg)	10.76±6.43	11.59±5.82	0.12

Table 4: Dietary nutrients intake of smokers and non-smokers.

smokers and non-smokers. The results are also consistent with those of Bradley, et al.[31] who also found no significant difference in the mean body mass index between the male smokers and non-smokers. Similarly, Chopra, et al.[32] and AL-Riyami and Afifi[33] also reported no significant difference in the mean BMI between the smokers and non-smokers. Conversely, others[34,35] reported an inverse association between nicotine intake and body weight; while another group of researchers noted a positive association between obesity and number of cigarettes smoked per day.

Interestingly, the markers of overweight and abdominal obesity such as percent body fat, waist circumference and hip circumference of smokers were significantly lower than the non-smokers. The reasons of lower adiposity in smokers may be attributed to their increased energy expenditure, physical activ-

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ity or their increased participation in manual jobs which were not measured in this study. The results are somewhat similar to others[36] who also reported a lower mean waist circumference for smokers. The smokers appear to be advantageous to have lower central obesity which may serve as a barrier against cardiovascular and other chronic diseases. Similarly, no significant difference in the waist-to-hip ratio between the smokers and non-smokers also indicates a non increasing risk of metabolic syndrome for smokers. The results are corroborated by Agarwal[37] and others[33] who reported a non significant difference in the waist-to-hip ratio between the smokers and non smokers.

In general, the Pakistani male adult population is confronted with challenges of poor dietary practices and physical inactivity that have lead to increased prevalence of overweight and obesity. The recent cross sectional studies around the country have revealed that about one thirds of the adult population is overweight and obese with increasing vulnerability to chronic diseases and premature deaths.[38,39] Cereals constitute a staple of Pakistani diet and serve as a major source of carbohydrates, energy, proteins, iron, zinc and other nutrients while animal products, vegetables, fruits and dairy products are less frequently consumed, the imbalanced and less diversified dietary patterns result in micro-nutrient deficiencies which are exacerbated by unhealthy lifestyle practices including smoking and physical inactivity.

No significant difference in the systolic and diastolic blood pressure between the smokers and non-smokers also suggests that smoking alone may not be a causative factor for hypertension rather it may exacerbate the risk by working synergistically with other potential risk factors. Our results are supported by Stolzenberg-Solomon, et al.[30] who reported a non significant difference in the mean systolic and diastolic blood pressures between the smokers and non smokers. But the results are contrary to the generally established fact that smoking causes oxidative stress and is responsible for increasing the risk of coronary artery disease, stroke and cerebrovascular diseases, hypertension and premature deaths.[12,13]

A significantly higher mean serum vitamin A level of smokers than non-smokers also demonstrates that smokers are not at an increasing risk of clinical vitamin A deficiency or for the development of abnormal degenerative changes in eyes such as age-related macular degeneration. Our results on vitamin A levels of smokers are similar to those of Chiu et al.[36] and Liu et al.[40] who reported a higher mean serum vitamin A level for smokers, however, Faure, et al.[41] reported no significant effect of smoking on serum retinol level in male and female French participants of SU.VI.MAX study. These results are somewhat different than those of Hawkins, et al.[42] who reported that smoking doubles the risk of age-related macular degeneration which could be attributed to oxidative stress rather than the vitamin A levels of smokers. The results on vitamin A status of smokers also suggest that either serum vitamin A levels are unaffected by smoking or vitamin A level may not have any potential role in fighting against oxidative stress, suppressing metabolic disorders or degenerative diseases.

Lower mean serum zinc level of smokers but a non-significant difference in the mean dietary intake between the smokers and non-smokers suggest that toxic compounds of tobacco may alter zinc metabolism to the extent of changing the serum zinc level. Decreased serum zinc level of smokers may also be attributed to decreased dietary zinc intake which was lower than the recommended dietary zinc intake of 15 mg/day for adults.[43] The argument of lower dietary zinc intake by smokers was further substantiated by others[44] who reported that smokers had a significantly lower fruits and vegetables consumption as compared to their non-smoking counterparts.

Zinc being an integral component of more than 200 enzymes, catalyzes various oxidation-reduction reactions, its deficiency may affect the metabolism of other nutrients as well as increase the risk of metabolic diseases in smokers by depressing antioxidant enzymes like superoxide dismutase activity.[29] Our results on zinc status of smokers and non-smokers correspond to those of Uz, et al.[45] and Anetor, et al.[46] who also reported significantly lower serum zinc levels in smokers in comparison to non-smokers. The lower serum zinc concentration of smokers compared to non-smokers could also be due to the influence of cadmium, an essential constituent of cigarette smoke that may act as an antagonist to zinc bioavailability.[47,48]

The results on dietary nutrients intake showed that smokers had a significantly higher mean energy and carbohydrate intake than non-smokers. The higher intake of energy and carbohydrates by smokers is somewhat contrary to the generally perceived notion that smoking depresses appetite. It has been reported[49] that nicotine increases energy expenditure and that could lead to increased energy intake while others[34] suggested that nicotine increases serotonin and dopamine levels in the brain that decrease the demand for energy intake and suppress appetite. Our results on energy and carbohydrates intake are in agreement with those of Cade and Margetts[50] who reported a significantly higher intake of energy and carbohydrates in smokers, on the other hand the results are contrary to those of English, et al.[51] who reported that smokers had a lower mean energy and carbohydrates intake than non-smokers.

The lower mean dietary protein and fat intake of smokers could be due to economic or personal reasons or due to decreased appetite caused by nicotine and other toxic compounds of the cigarettes smoke. However, the results do not correspond to those of Faruque, et al.[52] who reported a non significant difference in the dietary protein and fat intake between smokers and non-smokers. Similarly, no significant difference in the mean dietary vitamin A intake between smokers and non-smokers suggest that smoking did not have any significant affect on the dietary vitamin A intake of smokers. No significant difference in

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the mean dietary zinc intake between smokers and non-smokers suggests that dietary habits in terms of zinc intake are almost similar. The results of the study are inconsistent with the generally perceived hypothesis that smoking adversely affects nutritional status, the reasons for inconsistent association between indicators of nutritional status and smoking could be attributed to different degree of indicators' sensitivity owing to their different characteristics. We as authors are satisfied with the results of the study and feel that smoking may have more devastating health implications on the general health than on nutritional status of individuals as indicated by the study's results. A larger similar epidemiological case-control study with an increased number of anthropometric, body composition, biochemical and clinical indicators needs to be conducted to prove or refute the results of the current study.

The study concludes that anthropometric, biochemical, dietary, demographic and socio-economic characteristics are insensitive to the adverse effects of smoking as most of the indicators of smokers are comparable to non-smokers. Due to funding constraints, the sample size of smokers and non-smokers was relatively small that could have limited the statistical power of the study. In addition, all variables of interest such as morbidity and mortality associated with smoking could not be included and that could be one of the limiting factors in this study. Keeping in view the above stated limitations, the results may be used with caution, further studies are needed in the area to augment the study findings.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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