Correspondence

Smoking habits of students in college of Applied Medical Sciences, Saudi Arabia

Sir,

I read with interest the article of Dr Hashim published recently in Saudi Medical Journal.[1] I would like to raise the following points: 1. It is advisable to use a standard definition of the smoking status, to allow comparison of various prevalence estimates. The author-adopted definition of current smoking rather reflects daily smoking. The World Health Organization definition of current smoking is: smoking at the time of survey any tobacco product either daily or occasionally.[2] Besides, the author classifies his participants into current smokers and non-smokers (see methods).[1] In this case, what happens to people in other categories of question 11. For example, those who smoke 3 to 5 times a week do not satisfy the author's definition of smokers yet they can never be considered as non-smokers, where are they put? 2. The use of percentages in tables is misleading. For example in Table 1 the percentages of current smokers between males and females express the share of each gender of the total number of smokers regardless of the difference in the denominator for each sex. As they were reported these percentages tell nothing, and rather confuse the information given in the table. In Table 2, in the gender section, each percentage represents a fraction of the total number in columns + rows. This is again very misleading. A clear cut gender-based reporting of smoking is very important, especially in societies where large gender-based differences in the smoking habits are anticipated.[2] 3. The author states in the results that gender was not associated with smoking attitudes or behaviors. I re-did the cross tabulation between smokers non-smokers and gender male-female (Table 1, last section) and the p value was 0.003. It is apparent that 20% (prevalence of smokers among males) will be significantly different from 9% (prevalence of smokers among females) given the size of the sample in this study. I also re-did the analysis between age groups and smoking status (Table 1, 1st section) and the p value was 0.04 not < 0.000 as the author reported. 4. The author states in the results (abstract) that "the 20-24 year old age group exhibited the highest prevalence of smoking". This result do not appear in the results section of the article, instead the author states that "the 20-24 year old age group exhibited the greatest number of cigarettes smoked per day of smoking $p<0.000$". Anyway, a quick look at Tables 1 and 2 shows that both results are wrong and that the age group ≥ 25 years is the one with highest prevalence of smoking and cigarettes smoked per day. The author failed to use the appropriate sub-group denominator. 5. There is an overlap between the fields (denoting the range of cigarettes smoked) in Table 2, the numbers 10 and 20 are shared between respective ranges. 6. Question 5 of the questionnaire (Appendix 1) asks about the degree of religious belief, yet there is no mention of this variable neither in the results nor in the discussion. 7. The results of questions 10, 13 were not reported. Other categories of smokers identified by question 11 were also not reported, although they are very relevant to this age group, as they reflect various developmental stages of the smoking habit. 8. Question 14 of the questionnaire (Appendix 1) is put rather inaccurately. Smoked seriously can mean different things to different people. Usually the age of first whole cigarette smoked is reported.[2,4] Nevertheless, as in the previous point the results of this question were not reported. Also, I did not understand the meaning of question 19, and I think that the conclusions stated in the discussion cannot be based on the answers to this question. 9. Analysis of factors associated with smoking in this population was carried out for males and females combined. This is inaccurate. We, as well as many others have shown that factors associated with smoking differ according to gender,[3,4] especially in societies where strong taboos against smoking exist. 10. The description of the statistical analysis is incomplete (where did the author use ANOVA, since no differences of more than two means are reported? I assume the author used the Chi square test but it is never mentioned in the statistical methods). The p value was not calculated for numbers in Table 2. The response of non-smokers about the health hazards was not reported in Table 3. Science in the title should be sciences. Innovation in Reference 27[1] should be invasion. Male and female in the results (abstract) should be males and females etc.

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Reply from the Author

I would like to thank Dr Maziak for his thoughtful and important comments on the published article by the title "Smoking Habits of Students in College of Applied Medical Sciences, Saudi Arabia". I understood he raised valid points regarding the

percentages used for the categorical data in Tables 1 and 2 of our published article.[1] The approach to analysis is concentrating on amount of cigarette smoked and accordingly a percentage was computed. In my view as a health educator this is important to health education. So as to target the groups that contribute more to the problem rather than focusing on groups at risk. This is recently known as a population based approach to health education rather than risk approach, which is addressing small target. In addition I agree with Dr Maziak that gender is a significant factor particularly in societies like ours.

Other points he raised with regard to some data included in the questionnaire and not reported in the article. This is because I am planning to submit this data as another communication. The overlap in the field of cigarette consumption in Table 2 is just a typing mistake in the signs ≤ & ≥ which should have read < & >. The ANOVA test was used to make a comparison between 2 means with the current smoker. Many items in the questionnaire were well explained to the study subjects and hence the likelihood of confusion or misunderstanding is minimal.

In conclusion, I appreciate your interest in reading the article and taking the time to raise these fruitful comments.

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Hyperhomocysteinemia: another independent vascular risk factor

Sir,

I read with interest the article by El-Gebali et al on lipid peroxidation and coronary heart disease.[1] They correctly stated that hypertension, diabetes, hyperlipidemia, cigarette smoking and obesity are major cardiovascular risk factors. However, another important vascular risk factor they did not include, and was even not mentioned in their discussion, was hyperhomocysteinemia. It is now widely accepted that markedly elevated plasma levels of homocysteine is an independent determinant for clinical arteriosclerotic outcomes with an effect at least equivalent to that of the established risk factors.[2-8] Both case-control and prospective studies have shown that the risk of stroke, myocardial infarction and mortality increased directly with plasma total homocysteine.[3,4,6-8] Data even suggested a risk increase by 6% to 7% for every umol/L increase in total homocysteine.[8]

The mechanisms by which hyperhomocysteinemia induces arteriosclerosis are only partially understood, but its thrombophilic properties and potential for promotion of low density lipo-protein (LDL) oxidation and endothelial injury have been suggested. Recently, it was shown that enhanced in vivo lipid peroxidation was associated with elevated plasma total homocysteine.[9] El-Gebali et al also failed to provide a convincing explanation for the raised serum uric acid concentrations in their patients with coronary heart disease while it is well known that serum uric acid levels simply reflect the degree of renovascular atherosclerosis.[3,10]

Studies of patients with occlusive vascular disease reveal elevated homocysteine concentrations in 23-47% of patients compared with controls.[11] Genetic polymorphisms for methylenetetrahydrofolate reductase (MTHFR) are believed to modulate the risk of coronary heart disease acting through regulation of homocysteine metabolism, and homozygosity for the common 667C-T mutation in the MTHFR gene is now known to be associated with premature coronary artery disease.[12] Although the prevalence of the genetic polymorphisms of this enzyme varies considerably with race and ethnicity,[13] recent data revealed that also in the Arab population increased plasma homocysteine levels were associated with an

increased risk of coronary heart disease.[14]

In conclusion, it is most likely that the reported finding of increased lipid peroxidation in patients with coronary heart disease was – at least partly – the result of hyperhomocysteinemia. The rationale for including plasma homocysteine measurements in patients with atherosclerotic disease is obvious. Simple, inexpensive, non-toxic therapy with folic acid, vitamin B6, and vitamin B12 reduces plasma homocysteine levels and hence reduces the risk of arteriosclerosis.

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Author declined to reply.

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