Letters to the Editor

Re: Article by Knott and Harr, *Canadian Journal on Aging/La Revue canadienne du vieillissement*, Vol. 16 no. 4, 1997

I am writing with some concern about the article cited above. This article explores a number of smoking related changes in the aging brain, which is of much theoretical significance due to the known roles of acetylcholine and nicotine receptors in dementia of the Alzheimer type. Unfortunately, the authors only cite the studies finding a negative correlation of Alzheimer disease with smoking and do not cite some of those finding that dementia overall may increase in smokers. Some of the methodology of the "smoking positive studies" deserve critical commentary, which is not provided in this paper. On page 649, last paragraph, the article states that, "Although smoking nicotine has often been assumed to only impact negatively on parameters associated with aging and health status . . . the influence of smoking on aging-related brain function status per se has received little attention . . .". This sounds like the authors are suggesting that smoking isn't really all that bad. This is in spite of the very well documented major health risks associated with smoking, risks that far outweigh any short-term cognitive benefit.

I wonder too, whether the methodology of this study might involve a major selection problem, as "healthy" old and young people only were enrolled; also, the healthy elderly smoking population is a very special group, given that most of their smoking cohort has long joined the ranks of the non-healthy, and is thus not being sampled.

The authors' conclusions are also not qualified by appropriate information regarding the total health risks of smoking: for example the increase in vascular dementia as opposed to DAT, not to mention the overall increased morbidity in and mortality of smokers The use of certain forms of nicotine itself might indeed have some promise in the symptoms of DAT, but, as a responsible professional, one can and should most certainly not advocate nicotine dispensing by smoking to improve dementia, or other general health problems. In the final analysis, this paper, although very interesting to many researchers, provides further rationalization for confirmed smokers to keep smoking, and ammunition for the multinational tobacco companies in their battle to keep people smoking as well as to encourage others to start. This was perhaps not intended as such by the authors, but is implied in their results nevertheless.

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Author's Reply:

The concerns of Dr. Thorpe over our paper (Knott & Harr, *Canadian Journal on Aging/La Revue canadienne du vieillissement*, 1997, Vol. 16 no. 4, pp. 647–664) are appreciated but they appear to go beyond the intention and scope of the study. Our objective of comparing brain function (EEG) profiles of elderly smokers and non-smokers was based on a number of papers which had reported a negative association between smoking and dementia of the Alzheimer type (DAT) and by the suggestion from these reports that smoking may exert a neuroprotective effect. Dr. Thorpe is of the opinion that we should have included a critique of the methodological problems associated with these reports but as the smoking-DAT association was not the primary aim of our study we did not think a review of the weaknesses and strengths of these numerous reports was appropriate. However, in fairness to the outcome of these reports, it should be pointed out that improved methodological studies published after our paper continue to find a negative association between smoking and DAT although there is some indication that it may be specific to males with a family history of DAT (e.g., Salib & Hiller, 1997).

The use of healthy elderly subjects in our sample appears to be problematic for Dr. Thorpe as it may have introduced a bias in that they would not be representative of the elderly smoking population who, according to Dr. Thorpe, suffer chronic ill effects from smoking. Our decision to include only healthy non-smokers and smokers was based on sound scientific design principles which required the exclusion of confounding factors (e.g., cardiovascular and respiratory illnesses) which could potentially affect brain function status and hence, prevent a clear assessment of the central effects of smoking history per se.

Dr. Thorpe is of the opinion that our paper should have included information on the health risks of smoking lest readers view this study as an advertisement for smoking and its benefits for dementia and other general health problems. Contrary to Dr. Thorpe's contention that this study may be promoting smoke-inhaled nicotine as an alternative treatment for DAT, our paper specifically states in the final discussion paragraph that treatment trials with nicotine, not smoking, are the recommended methodology for exploring the putative cognitive-enhancing effects of this agent in DAT.

Reference

Salib, E., & Hillier, V. (1997). A case-controlled study of smoking and Alzheimer's Disease, *International Journal of Gerontological Psychiatry*, *12*, 295–300.

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