Prevalence of Smoking in the Elderly

Smoking is one of the major causes of morbidity and mortality in Canada. In fact, it has been called the leading preventable cause of death in North America. This is because smoking is a known risk factor for four of the leading causes of death in the industrialized world—coronary heart disease, cancer, lung disease and stroke—and because it contributes to many other causes of morbidity. While the current prevalence of smoking in Canadians aged 15 years and older declined by 10.3% between 1985 and 1999, the numbers remain high for both men and women (26.8% and 22.9%, respectively, in 1999). In those aged 65 and older, current smoking prevalence decreased by 8.9% over the same time period. However, it is estimated that 11.6% of seniors continue to smoke. The prevalence of smoking is highest in the Atlantic provinces and Quebec, and lowest in Saskatchewan and Ontario.

Impact of Smoking on Health of the Elderly

Mortality

The health-related impact of smoking in the elderly is manifold. The increase in mortality has already been mentioned. A prospective cohort study following over 2,000 elderly European men for a 10-year period set out to determine factors that contribute to death. The major risk factors that were examined included age, smoking, blood pressure, high density lipoprotein (HDL) and non-HDL cholesterol, heart rate and body mass index. The study concluded that cardiovascular risk factors such as age, smoking and heart rate are significant predictors of all-cause mortality. A similar study examined the relationship between smoking and mortality in a Chinese cohort aged 70 years and older and found a significant association between current smoking and combined mortality from three major causes: cancer, cardiovascular disease and respiratory disease.

Cardiovascular Disease

In a review of smoking in the Canadian Journal of Cardiology, Pipe asserted that “the importance of smoking as a principal, preventable risk factor for cardiovascular diseases cannot be overstated”. Smoking increases the incidence of cardiovascular disease (CVD) by 50% and the risk of cardiovascular mortality by 100%. More than 16,000 cardiovascular deaths occur per year in Canada as a result of smoking. In addition, several studies have emphasized the synergism of smoking with other cardiovascular risk factors such as diabetes, hypertension and hypercholesterolemia.

The pathogenetic mechanism by which smoking contributes to CVD is complex. Endothelial dysfunction is thought to be the initial event. Nicotine damages endothelium in animal models, and tobacco smoke increases smooth muscle cell proliferation and adhesion, all of which are factors in the genesis of atherosclerosis. In addition, nicotine increases heart rate, blood pressure, cardiac output, myocardial oxygen demand and vasoconstriction—factors that could contribute to a cardiac event in the context of significant atherosclerosis.

Studies have also revealed that thrombi can form on less severely stenotic vessels in smokers when compared with non-smokers.

Peripheral Vascular Disease

Cigarette smoking accelerates atherosclerosis, leading not only to coronary artery disease (CAD), but also to stroke, aortic aneurysms and peripheral vascular disease (PVD). In fact, smoking is the most significant risk factor for PVD. It is clear that smoking cessation reduces the risk of PVD and its complications. For instance, former smokers’ rates of PVD are greater than never smokers’ rates, but are half that of current smokers’ rates. Complications from PVD in individuals who quit smoking are reduced, and function and overall survival are significantly increased.

Cerebrovascular Disease

It has been estimated that over half of all strokes in the U.S. are attributable to smoking. Smoking increases the risk of stroke by 1.5 to 3.0 times compared to non-smokers, as demonstrated through cohort studies and meta-analyses. Smokers have significantly decreased cerebral blood flow as measured by xenon inhalation, and smoking increases the risk for subarachnoid hemorrhage. However, stroke risk is reduced to baseline by five years after smoking cessation, with a major decrease soon after cessation.

Cancer

Smoking has been shown to be associated with a variety of cancers. There are over 4,000 components of tobacco smoke, and of these, approximately 50 have been demonstrated to be carcinogenic in vivo and in vitro. The molecular basis for the carcinogenic potential of smoking is still being elucidated; however, it is known that metabolic activation of smoke by oxidation in rat lungs creates unstable metabolites that react with DNA to form methylated bases. These methylated bases correspond to point mutations in the k-ras oncogene, an oncogene that is known to be activated in up to 30% of patients with adenocarcinoma of the lung.
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Lung Cancer

Lung cancer has the distinction of being the most frequently diagnosed cancer in the world. It is also the leading cause of death from cancer. Smoking is directly responsible for 83% of lung cancer deaths, and nearly one-third of all cancer mortality in the U.S. This enormous disease burden is a recent development, arising in Western countries in the 1930s and coinciding with patterns of growing tobacco use.

The development of lung cancer from smoking has been largely attributed to polycyclic aromatic hydrocarbons and tobacco-specific nitrosamines, with a small contribution by polonium-210 and volatile aldehydes. A dose-response relationship has been found between the number of cigarettes smoked per day and the increasing risk of death from lung cancer. Finally, there is a risk reduction of lung cancer with smoking cessation. The risk reduction is correlated to the dose, duration, type of cigarette and depth of inhalation. Approximately 10 years after cessation, the risk is about 30-50% of that for patients who continued to smoke during the same period.

Head and Neck Cancer and Oral Cancer

There is a large body of evidence linking smoking, as well as other tobacco use, to carcinomas of the head and neck. In addition, the synergistic effect of tobacco and alcohol on the development of head and neck cancers has been recognized.

Oral cancer is a subset of head and neck cancers. While the origin of oral cancer is multifactorial, most cases can be traced to past and present use of alcohol and tobacco products (cigarettes, cigars, pipes, spit tobacco), or to sun exposure (lip cancer) and exposure to workplace carcinogens. Smokers have a two-fold to 18-fold increase in the risk of developing oral cancer over non-smokers.

Esophageal Cancer

Cigarette smoking is a risk factor for esophageal squamous cell carcinoma. However, there are important gender differences. Women appear to be more susceptible to the carcinogens in tobacco smoke than men because of a complex interaction between the higher level of activation of tobacco procarcinogens by certain cytochrome P-450 enzymes, a slower metabolism of nicotine and a possible role for female sex hormones in tumour development.

However, these relationships are speculative and require further investigation and explanation.

Stomach, Pancreatic and Colon Cancer

Tobacco smoking is related to a modest increased risk of adenocarcinomas of the esophagus and gastric cardia. The risk of distal stomach cancer, however, is associated with the presence of stomach ulcers and with the number of pack-years of smoking.

Smoking also increases the risk of pancreatic cancer in both men and women. The proportion of deaths from pancreatic cancer that are attributable to smoking is estimated at about 30%, and there appears to be a dose-response relationship. This type of cancer has one of the lowest five-year survival rates.

Cigarette smoking has been associated with a two-fold increase in risk of death from colon cancer.

Sources:

National Clearinghouse on Tobacco and Health: A Program of the Canadian Council for Tobacco Control
http://www.ncth.ca/NCTHweb.nsf

Physicians Taking Action Against Smoking— an intervention program to optimize smoking cessation counseling by general practitioners: A program based on a cessation model that identifies factors which influence counseling practices.

Smokers and Ex-smokers Support Group: Details of a comprehensive step-by-step implementation process used to establish a smokers and ex-smokers support group.

Health Canada's Cessation Resources for Professionals
http://www.hc-sc.gc.ca/hessesc/tobacco/prof/cessation_program/other_opt_en.html

This government site provides an Inventory of Canadian Tobacco Cessation Programs and Resources, including an exhaustive list of cessation programs listed by province. Details of various therapies—from acupuncture to nicotine replacement therapy—and their clinical evaluation are listed, in addition to listings of programs available to assist physicians and other health professionals set up a counseling program.

FOR DIRECT LINKS TO THESE RESOURCES, PLEASE VISIT: WWW.GERIATRICSANDAGING.CA
Major Diseases Caused by Smoking

Vascular Diseases

- Cerebrovascular disease
- Cardiovascular disease
- Peripheral vascular disease

Cancers

- Lung
- Esophageal
- Stomach
- Pancreatic
- Colon
- Bladder

Squamous cell carcinomas of the oral cavity

- Hard palate
- Uvula
- Alveolar ridge
- Lateral aspect of the tongue
- Lower lip

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colonic adenomas which are strongly related pathogenetically to colon cancer.9,21 In addition, in a veteran cohort, the risk of death from colorectal cancer was 20% higher in tobacco users when compared with non-users. The association between tobacco exposure and colorectal cancer has demonstrated a dose-response relationship.9,21

Bladder and Cervical Cancer

Bladder cancers are predominantly transitional cell type in origin. A case-control study demonstrated a significant relationship between smoking and the development of invasive cancer of the bladder. The risk was significantly related to duration of smoking and inversely related to both the age at initiation of smoking and the time since smoking cessation.9,22 Aromatic amines, or arylamines, are constituents of tobacco smoke and have been implicated in the carcinogenesis of bladder cancer.9,23 A dose-response relationship has also been found to exist between smoking and cervical cancer.9,24

Chronic Obstructive Pulmonary Disease

It has been well established that smoking, both active and passive, exerts a negative influence on lung function. Cigarette smoke influences all three determinants of FEV1 (forced expiratory volume in one second): peak achieved during early adulthood, duration of the early adult plateau phase, and rate of subsequent decline. On average, heavy male smokers have a 15mL per year larger decline in FEV1 than non-smokers.25 Only 10–15% of cigarette smokers will eventually develop chronic obstructive pulmonary disease (COPD); however, cigarette smoking accounts for about 80–90% of this risk in North America.9 In addition, cigarette smokers have higher COPD mortality than non-smokers, as well as higher rates of other respiratory symptoms, bronchitis and decline of FEV1.

In essence, cigarette smoking accelerates the normal aging process of the lungs, including loss of elastic recoil resulting in expansion of distal airspaces. Functionally, this results in hyper-expansion of the lungs and decreased FEV1. In addition, changes in inflammatory cell infiltrate in the terminal airways contribute to bronchiolitis, which can lead to fibrosis and distortion of the small airways.9 After the first year, smoking cessation can decrease the accelerated rate of decline in FEV1 by half.9,26

Reduction Strategies for Tobacco Use in the Elderly

Numerous studies have illustrated the significant benefits of smoking cessation in the elderly.27-30 As smoking is the leading cause of preventable death in North America, physicians must be vigilant about regularly asking about their patients’ smoking status. They should also provide smoking cessation counseling, including the prescription of nicotine replacement or other cessation aids, and recommend cessation programs as appropriate31 (see Sidebar for resources).

In younger patients, nicotine replacement therapy doubles the long-term smoking cessation success rate compared to placebo, and there is no reason to believe that it would be less efficacious in older smokers.32 Transdermal nicotine is the replacement therapy of choice for older patients because of once-daily administration and tolerability. It is unlikely that concurrent smoking or high doses of nicotine replacement would adversely affect healthy elderly patients. While unstable CAD is a contraindication to nicotine replacement therapy, the risk of CAD exacerbation may be no greater than the risk posed by smoking on its own.32 One study has suggested that use of a nicotine patch for five months in combination with a nicotine nasal spray for one year is a more effective method of quitting smoking than use of a patch alone.33

Another pharmacologic option for smoking cessation is the antidepressant bupropion. A study conducted in U.S. veterans with an average age of 57 years compared 150mg of bupropion sustained release (SR) to 300mg of bupropion SR, and concluded that one month of bupropion SR therapy at 150mg per day may provide the maximum benefit with minimal expense and risk.34 Other important interventions for smoking cessation include counseling and follow-up support.32

Conclusions

The use of tobacco in the elderly population is common and is a cause of significant morbidity and mortality, including cardiovascular disease, peripheral vascular disease, cerebrovascular disease, cancer and chronic obstructive pulmonary disease. However, smoking cessation has benefits even in elderly patients with respect to preventing these diseases or, at the very least, slowing the decline of pulmonary function. Similar pharmacologic and behavioural techniques may be used in the elderly as in their younger counterparts, yielding similar results.32

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References

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