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Complete List of Authors:	Edjoc, Rojiemiahd; University of Ottawa Heart Institute, Reid, Robert; University of Ottawa Heart Institute, Sharma, Mukul; McMaster University Population Health Research Institute, Neurology Balfour, Louise; University of Ottawa, Psychology Procino, Michael; University of Ottawa,
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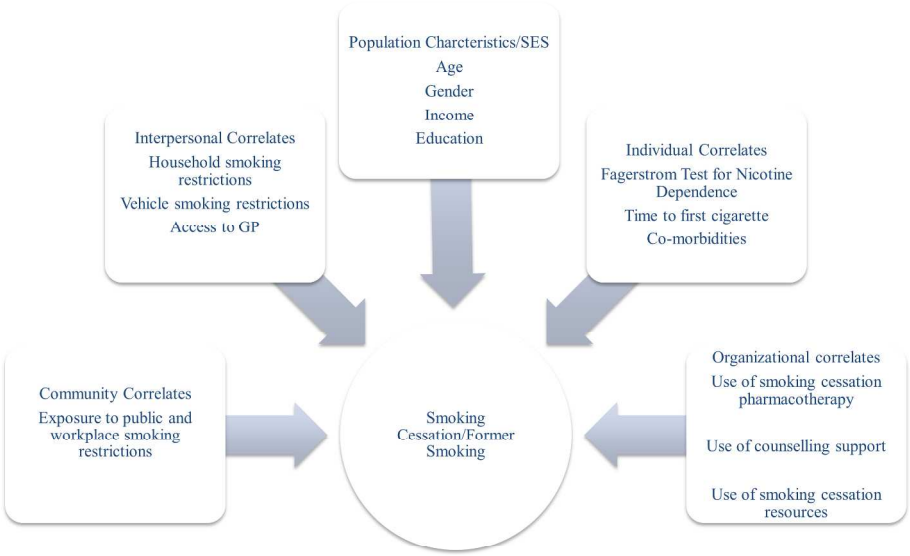
Figure 1: Socio-ecological model for multi-level correlates of former smoking in respondents with cerebrovascular disease



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Correlates of former smoking in patients with cerebrovascular disease: A cross-sectional study

Rojiemiahd K. Edjoc, PhD[1].
Robert D. Reid, PhD., MBA[1]
Mukul Sharma, MD MSc FRCPC[2]
Louise Balfour, PhD[3]
Michael Procino, BSc[3]

[1]Division of Prevention and Rehabilitation, University of Ottawa Heart Institute, Ottawa, Canada
[2]McMaster University, Population Health Research Institute, Hamilton, Ontario, Canada
[3]University of Ottawa, Ottawa, Ontario, Canada

Correspondence to:
R Edjoc PhD
Consulting Epidemiologist
University of Ottawa Heart Institute
40 Ruskin St.
Ottawa, ON
K1Y4W7
Email: redjoc@hotmail.com

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Key words: Smoking, cessation, cerebrovascular disease, stroke, prevention, epidemiology

Word count: 2,491

ABSTRACT

Objective: To identify multi-level correlates of former smoking in patients with cerebrovascular disease.

Design: Secondary data analysis of the Canadian Community Health Survey.

Methods: We used data from the 2007-2008 Canadian Community Health Survey (CCHS). Smoking status (quit smoking completely vs. smoker vs. non-smoker) was described by population characteristics, socio-economic status, individual correlates, interpersonal correlates, community correlates and organizational correlates. The study sample was selected from those respondents of the CCHS that reported they suffered from stroke symptoms. Logistic regression was used to describe the association between quitting smoking and stroke while controlling for multi-level correlates of former smoking. Proportions were weighted to reflect the Canadian population.

Results: There were 383904 respondents who reported to suffer from stroke. From this sample, 211549 respondents (55.1%) reported they were non-smokers and 62960 (16.4%) respondents reported they were smokers. There were 109395 (28.5%) individuals who indicated they were former smokers. At the population characteristics and socio-economic level, female sex (OR 0.4; 95% CI: 0.41 to 0.42) reduced the likelihood of being a former smoker. The age groups 55-69 (OR 1.1; 95% CI: 1.10 to 1.19) and 70-80 (OR 1.6; 95% CI: 1.61 to 1.67) were positively related to former smoking. At the interpersonal level, household (OR 1.1; 95% CI: 1.05 to 1.08) and vehicle (OR 2.9; 95% CI: 2.79 to 2.93) smoking restrictions significantly predicted former smoking. Counselling advice from a physician

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was also a correlate of former smoking (OR 3.7; 95% CI: 3.37 to 4.03). Depression (OR 0.9 CI: 0.89 to 0.91) and alcohol consumption (OR 0.7 95% CI: 0.69 to 0.71) reduced the likelihood of former smoking.

Conclusions: There are multi-level correlates of former smoking in smokers with reported stroke symptoms. Future interventions should be tailored with these correlates in mind to increase the likelihood of cessation.

Article focus

- To elucidate the multi-level correlates of former smoking in smokers who suffer from stroke symptoms.

Key messages

- There are multi-level correlates of former smoking in individuals who reported to suffer from stroke symptoms.
- It is imperative that smoking cessation be incorporated in secondary prevention practice while taking significant co-morbidities such as depression and alcohol consumption into account for this population.

Strengths and limitations of the study

- This study is the first to elucidate correlates of former smoking in this population at a multi-level.
- The size of the study provide adequate power for the statistical analyses
- The cross-sectional nature of the study and self-reported outcomes such as smoking status and the presence of stroke symptoms may result in social desirability bias.

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INTRODUCTION

Smoking is an independent risk factor for incident and recurring stroke.[1, 2, 3]. It has been found that smoking cessation can reduce the relative risk of stroke and transient ischemic attach (TIA) by 50% [4] and stroke related hospitalizations.[5] Despite the supporting evidence regarding the benefits of smoking cessation for smokers with cerebrovascular disease, there is evidence that 89% of these smokers were still smoking 12 months after their event.[6]

Stroke prevention guidelines recommend that healthcare providers strongly advise every smoker who is at high risk for a stroke or TIA to quit, and provide specific assistance with quitting, including counselling and pharmacotherapy.[3, 7]

There are very few published smoking cessation intervention (SCI) studies in stroke and TIA patients. A recent systematic review found a non-significant effect of SCI's on quitting in stroke and TIA patients.[8] The authors found that with the available studies, there was a sub-optimal use of evidence -based approaches to smoking cessation comprised of counselling, pharmacotherapy and follow-up.[8]

More interventions need to be developed by identifying significant correlates of former smoking among these high-risk smokers. The socio-ecological model proposed by Sorensen and associates [9] explicate factors that influence different groups to use or not use tobacco (Figure 1). These factors include population characteristics and socio-economic status (SES), individual, interpersonal, community and organizational factors. Based on this socio-ecological model, the present study elucidated multi-level correlates of former smoking using data from the Canadian Community Health Survey (CCHS). There has not been any study that has systematically explored the impact of multi-level correlates on the cessation attempt and thus is the impetus for the present study.

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METHODS

Data from the 2007-2008 Canadian Community Health Survey were used for the present study. The CCHS is a cross-sectional survey that collects information related to the factors that contribute to health, social and economic determinants of Canadians.[10] The CCHS utilizes a complex sampling strategy with stratification and multiple stages of selection yielding a sample that is representative of 98% of the Canadian population.[10]

Only individuals who reported the effects of stroke were included in the present analysis. Stroke symptoms were defined as those who reported were experiencing stroke symptoms due to a recent stroke or TIA within the last year. No further information was available. From this sample, smoking status (smoking vs. former smoking) was selected as the dependent variable. Important correlates were grouped by population characteristics and SES, individual, interpersonal, community and organizational level. Population characteristics included: sex and age. Socio-economic status included: income and education. Individual level correlates included: co-morbidities such as depression, diabetes mellitus, hypertension, alcohol consumption, and nicotine addiction (as measured by the Fagerström Test of Nicotine Dependence). Interpersonal level correlates included: having household and vehicle smoking restrictions and access to a general practitioner (GP). Community level correlates included: exposure to public and workplace smoking restrictions. Organizational level correlates were defined as the use of smoking cessation resources such as pharmacotherapy (nicotine replacement therapy [NRT] and bupropion) and counselling support provided by a physician or referral to a smoking cessation group. Ideally, varenicline would be included in the list of pharmacotherapy. Unfortunately, at the time of this survey, varenicline was not yet approved for use in Canada and was not collected by the CCHS. Age was re-coded into five categories (ages 12-19; 20-34; 35-54; 55-69 and 70-80+). Due to the

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complex survey design of the CCHS, adjusted weight was calculated for each respondent taking into account national average design effects and the relative sampling weights.

Statistical Analysis

All statistical analyses were performed using Statistical Analysis Software (SAS). Cross-tabulations between reported stroke symptoms (yes or no) and smoking status (smoker, former smoker and non-smoker) were performed while controlling for correlates of interest. A chi-square test of significance was used to determine significant differences between cross-tabulated proportions. Significance was reported at 95% confidence or having a value of $P<0.05$.

A logistic regression model was 'fitted' using the dependent variable of reported former smoking while controlling for each correlate level (population characteristics, socio-economic position, individual, interpersonal, community and organizational level). Significant correlates of former smoking were expressed by odds ratio (OR) point estimates at a 95% confidence level (CI). The method of model building for logistic regression analyses was forward-stepwise selection. The Wald statistic was used for variable selection. Independent variables were identified as significant correlates if the p-value was less than 0.05 ($p<0.05$).

RESULTS

A summary of the characteristics of the study cohort can be found in Tables 1 and 1a. The following table is divided by smoking status (i.e. Non-smoker, Smoker and Former smoker). The overall weighted sample was 383904 individuals who reported to suffer from

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stroke symptoms. From this sample 211549 individuals (55.1%) reported to be non-smokers and 62960 (16.4%) individuals reported to be smokers. There were 109395 (28.5%) individuals who reported to be former smokers.

There were more males who reported they had quit smoking than females (62.9% vs. 37.1%; p<0.0001). In the smoking cohort, more males were continued smokers than females (56.4% vs. 43.6%%; p<0.0001). There were more males than females (50.7% vs. 49.3%; p<0.0001) in the non-smoking cohort. Individuals who quit smoking as well as non-smoking individuals were older than smoking individuals. In general, all cohorts had post-secondary education and were earning an annual income of $ 20 000 - 39 000.

Individuals who quit smoking reported to have higher proportions of household (74% vs. 35.5%%; p<0.0001) and vehicle smoking restrictions (93.7% vs. 0.0%%; p<0.0001) compared to smoking individuals. Exposure to public smoking restrictions (93.3 % vs. 0.6%; p<0.0001) was higher in smokers compared to quitters.

Respondents who were former smokers compared to current smokers reported they used NRT (0.1% vs. 0.0%; p<0.0001) more frequently as well as bupropion (0.4% vs. 0.0%; p<0.0001). Smokers reported more physician counselling (4.5% vs. 0.5%; p<0.0001) than quitters. Smoking individuals reported higher proportions of alcohol consumption (>2 drinks per day; 49.1% vs. 7.9%; p=0.03), more depression (41.7% vs. 35.6%; p<0.0001) and diabetes (20.7% vs. 8.9%; p=0.21) than respondents who were former smokers. Similar trends were found when we compared smoking individuals and non–smokers for alcohol consumption (> 2 drinks per day; 49.1% vs.39.1%; p<0.0001), depression (41.7% vs. 32.8%; p<0.0001) and diabetes (20.7% vs. 8.6%; p=0.21) although not significant.

Logistic regression odds ratios and 95% CIs of significant correlates of former smoking can be found in Tables 2 and 2a. At the population characteristic and socio-economic level, female sex (OR 0.4; 95% CI: 0.41 to 0.42) reduced the likelihood of quitting. Age

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55-69 (OR 1.1; 95% CI: 1.10 to 1.19) and age 70-80 (OR 1.6; 95% CI: 1.61 to 1.67) were significant correlates of former smoking. At the individual level, co-morbidities such as alcohol consumption (OR 0.7; 95% CI: 0.69 to 0.71) and depression (OR 0.9; 95% CI: 0.88 to 0.91) reduced the likelihood of former smoking. At the interpersonal level, household (OR 1.1; 95% CI: 1.05 to 1.08) and vehicle (OR 2.9; 95% CI: 2.79 to 2.93) smoking restrictions significantly predicted former smoking. However at the community level, exposure to workplace and public place smoking restrictions did not significantly predict former smoking. The use of pharmacotherapy such as bupropion significantly predicted former smoking (OR 15.4; 95% of CI: 13.9 to 17.0) while the use of NRT did not. Counselling advice from a physician was also a correlate of former smoking (OR 3.7; 95% CI: 3.37 to 4.03).

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Table 1: Study Cohort Characteristics

Covariates	*Non-Smoker (%)* (n=211 549)	*Smoker (%)* (n=62 960)	*Former Smoker (%)* (n=109 395)	*P-values*
Males	107 191 (50.7)	35 521 (56.4)	68 798 (62.9)	<0.0001
Females	104 358 (49.3)	27439 (43.6)	40 597 (37.1)	<0.0001
Age				
12-19	1975 (0.9)	314 (0.5)	0.0	<0.001
20-34	2259 (1.1)	2432 (3.9)	1649 (1.5)	<0.001
35-54	11 070 (5.2)	12 830 (20.4)	3375 (3.1)	<0.001
55-69	44 277 (20.9)	24 079 (38.2)	26 660 (24.4)	<0.001
70-80+	89 613 (42.4)	19 515 (31.0)	50 456 (46.1)	<0.001
Education				
< secondary	40 722 (19.3)	11 913 (18.9)	19 917 (18.2)	<0.001
Secondary	26 478 (12.5)	7244 (11.5)	14 902 (13.6)	<0.001
Some post secondary	10 367 (4.9)	2390 (3.8)	5583 (5.1)	<0.001
Income				<0.001
None or < 20 000	33 607 (15.9)	13 029 (20.7)	15 686 (14.3)	
20 000 -39 000	56 858 (26.9)	15 931 (25.3)	32 083 (29.3)	
40 000 – 59 000	34 765 (16.4)	11 993 (19.1)	18 891 (17.3)	
60 000 – 79 000	14 422 (6.8)	3928 (6.24)	7712 (7.1)	
80 000 +	26 714 (12.6)	6617 (10.5)	14 739 (13.5)	
Fagerstrom Nicotine Dependency				<0.0001
Very Low	0.0	562 (0.89)	0.0	
Low	0.0	482 (0.8)	0.0	
Medium	0.0	137 (0.2)	0.0	
High	0.0	1288 (2.0)	0.0	
Very High	0.0	326 (0.5)	0.0	

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Table 1a: Study Cohort Characteristics

Covariates	*Non-Smoker (%)* (n=211 549)	*Smoker (%)* (n=62 960)	*Former Smoker (%)* (n=109 395)	*P-values*
Smoking restrictions				
Household	163 099 (77.1)	22 364 (35.5)	80 983 (74.0)	<0.0001
Workplace	23 845 (11.3)	18 740 (29.8)	11 470 (10.5)	0.004
Vehicle	210 504 (99.5)	0.0	102 527 (93.7)	<0.0001
Public	209 436 (99.0)	0.0	102 032 (93.3)	<0.0001
Have access to GP	202 460 (95.8)	55 563 (86.7)	103 830 (94.9)	<0.0001
Smoking cessation aids				
Nicotine replacement therapy (NRT) gum	0.0	0.0	0.0	-
NRT patch	0.0	0.0	65.9 (0.1)	<0.0001
Zyban/Buprion	0.0	0.0	486 (0.4)	<0.0001
MD counseling	0.0	2853 (4.5)	511 (0.5)	<0.0001
One-to-One referral	0.0	326 (0.5)	0.0	<0.0001
Referral to smoking cessation group	0.0	0.0	0.0	-
Alcohol drinking (> 2 drinks/day)	82 752 (39.1)	30 923 (49.1)	4984 (7.9)	0.03
Depression	69 425 (32.8)	26 239 (41.7)	38 989 (35.6)	<0.0001
Diabetes	18 256 (8.6)	13 029 (20.7)	9823 (8.9)	0.21
Hypertension	50 217 (23.7)	4984 (7.9)	22 263 (20.4)	0.02

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Table 2: Correlates of smoking cessation of study cohort

Correlates of smoking cessation	Odds Ratio (OR)	Lower 95% CI	Upper 95% CI
Population characteristics			
Female Sex	0.4	0.41	0.42
Age 12-19	-	-	-
Age 20-34	0.02	0.02	0.03
Age 35- 54	0.03	0.025	0.027
Age 55- 69	1.1	1.10	1.19
Age 70- 80	1.6	1.61	1.67
Socio-economic status			
Less than secondary	0.9	0.88	0.93
Secondary education	1.3	1.22	1.29
Some post-secondary education	1.2	1.23	1.24
Post-secondary	1.1	1.10	1.15
No income or less 20 000	0.7	0.63	0.73
$20 000- 39 000	1.3	1.24	1.29
$40 000- 59 000	1.1	1.06	1.11
$60 000- 79 000	1.5	1.45	1.51
$80 000+	0.7	0.66	0.70
Individual level			
Alcohol Consumption (> 2 drinks/day)	0.7	0.69	0.71
Depression	0.9	0.88	0.91
Hypertension	1.2	1.18	1.24
Diabetes	1.4	1.34	1.38
Fagerstrom Nicotine Dependency	-	-	-

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Table 2a: Correlates of smoking cessation in study cohort contd.

Correlates of smoking cessation	Odds Ratio (OR)	Lower 95% CI	Upper 95% CI
Interpersonal level			
Household smoking restrictions	1.1	1.05	1.08
Vehicle smoking restrictions	2.8	2.72	2.86
Access to GP	1.3	1.24	1.31
Community level			
Public smoking restrictions	-	-	-
Workplace smoking restrictions	-	-	-
Organizational level		-	-
Nicotine replacement therapy (NRT) gum	-	-	-
NRT patch	-	-	-
Zyban/Bupropion	15.4	13.9	17.0
MD counselling	3.7	3.37	4.03

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DISCUSSION

The aim of this study was to identify the correlates of former smoking in smokers with reported stroke symptoms at multi-levels. Income and older age were predictive of former smoking while education at all levels predicted former smoking in this cohort. These results are in line with previous investigations of gender effects,[11, 12, 13] older age [11, 12] and level of income [11, 12] vis a vis smoking and cessation. Koning and associates found that each additional year of education reduced the risk of continued smoking.[14] Their data suggested that people with higher education may be able to better understand the consequences of long-term smoking and may have more resources available for them to quit smoking.[14]

Our findings suggest that co-morbid conditions at the individual level such as alcohol consumption and depression significantly decreased the likelihood of former smoking. These findings are supported by evidence suggesting that cerebrovascular patients experience higher rates of co-morbidity particularly depression.[15, 16, 17] Compared to cardiac patients, patients with a recent stroke suffered a three to five-fold increased risk of depressive disorders [15] as well as higher proportions of alcohol consumption and hypertension.[18]

These findings may have clinical implications particularly for this population, as co-morbid conditions such as depression and increased alcohol consumption are significantly more common in patients who smoke. Considering their association with increased smoking behaviour, co-morbidities may be hindering the success of quitting smoking. The hindering effect of co-morbidity on former smoking is especially problematic as smoking increases blood coagulability, platelet aggregation, thrombus formation and endothelial

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damage,[19] thus increasing the chance of a stroke two-fold [20] and of stroke recurrence.[20, 21] Quitting smoking has been shown to reduce the risk of stroke to that of a non-smoker after 5 years [22] and reduce the risk hazard ratio of stroke recurrence from 1.71 to 1.39 ($p < 0.05$).[23] It is imperative that smoking cessation be incorporated in secondary prevention practice while taking these significant co-morbidities into account. Depression and excessive alcohol consumption might impede cessation in people with cerebrovascular disease. However due to the limitation of cross-sectional studies, we do not know if these co-morbidities existed before or after the reported stroke. Further study regarding the effects of these co-morbidities on cessation using other study designs might be warranted.

Population based interventions such as household, workplace, vehicle and public smoking restrictions have all been found to predict smoking abstinence.[24, 25, 26] They have also been found to reduce cigarette consumption, and initiation and increase smoking cessation rates.[24, 25, 26] These authors suggest that population based interventions are anti-tobacco socialization tools that may promote the internalisation of behavioural norms against the initiation or continuation of smoking. Our results are partially in line with this evidence. We found that household and vehicle smoking restrictions predicted smoking cessation but not so with workplace or public smoking restrictions. It is not known why workplace and public smoking restrictions did not predict smoking cessation especially since their implementation under the Smoke Free Ontario Act [27, 28, 29] in Ontario and similar legislations across Canada. Since their implementation, smoking prevalence in Canada has been dramatically decreased. Perhaps the insignificant effect of public and workplace smoking restrictions may be explained in the decrease of funding in the SFOA in 2007-2008 of 60 million, down 2.5 million from the year before of 62.5 million in 2006-2007.[30, 31] Similar reductions in tobacco control funding can be observed in other provinces.[28, 29] There is a documented association between population interventions effectiveness and sustained funding.[32]

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A similar situation was observed with the California Model in the state of California. The California Model is similar to the SFOA and is a population intervention that used workplace and public place smoking restrictions to de-normalize tobacco use.[32] Pierce and associates found that the initial effect of the California Model to decrease smoking prevalence in the state dissipated as their funding was reduced.[32]

In light of this conundrum, there is evidence that suggests that household and vehicle-smoking restrictions are more effective because they are less regulated.[33, 34] These authors suggest that smoking restrictions such as at home or in a vehicle are effective because those who implement them do so by choice and not through forced legislation [32, 33, 34] thereby increasing the odds of smoking cessation.

At the organizational level, we found that the use of pharmacotherapy such as bupropion and physician counselling increased the odds of former smoking but NRT use did not. According to Fiore and associates, pharmacotherapy along with counselling and follow-up increases the odds of smoking cessation.[35] NRT and bupropion have each been found to be more efficacious than placebo for increasing the odds of smoking cessation.[36]

The lack of effect of NRT may be indicative of the well-documented practice gap in health care in regards to smoking cessation. Young and Ward found that only 32% of physicians provided written materials for their patients and only 28% of physicians set a “quit date” with their patients.[37] Likewise Shaohua and colleagues found that many family physicians feel lack of time was their biggest barrier in terms of implementing smoking cessation practices.[38] Their study found that less than half were willing or able to assist their patients to quit with the use of counselling, pharmacotherapy or arrange a follow-up visit to reinforce the benefits of smoking

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cessation.[38] This is consistent with the stroke population as documented by Mouradian and associates.[6] Perhaps another explanation may be the lack of information regarding the effectiveness of smoking cessation medications and similar interventions in stroke and TIA patients. Furthermore, physicians may be reluctant to prescribe NRT's due to their availability over the counter. Further research is required to determine if the latter explanations are supported by evidence.

Cross-sectional surveys such as CCHS are useful for initial exploratory studies. They are far reaching and reflect "a snapshot" of the population. However there are limitations to our study and they will be explored here. Since both exposure and outcome were measured at the same time, one cannot be certain which is the exposure or the outcome. In other words, the rules for contributory cause cannot be fulfilled. Another limitation is the mode of collection of the data. Social desirability and recall bias for example could play an important role and a source of biases within this study.[39] For example, since smoking status, the presence of stroke symptoms and co-morbidities such as depression were self-reported, special care should be taken when interpreting our results. An example of social desirability effect would be respondents not accurately reporting their smoking status. Since smoking would be an undesirable image for some depending on age, gender or socio-economic status, data obtained might not be representative of the real picture found in the population. Ideally all smoking related measures should be validated bio-chemically with breath samples measuring carbon monoxide levels or coitinine levels measuring the amount of nicotine in the blood. Furthermore, without an expert assessment from a health care professional of stroke symptoms or depression would also limit the generalizability of the results.

CONCLUSION

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We found significant correlates of former smoking at multiple levels in smokers with reported stroke symptoms. Age and education level were significant correlates of smoking cessation at the population and socio-economic level. At the individual level, depression and alcohol consumption reduced the likelihood of cessation while at the interpersonal level, household and vehicle smoking restrictions and access to a GP were found to be significant correlates of former smoking. Public and workplace smoking restrictions were not correlates of former smoking at the community level. Finally, at the organization level the use of bupropion along with physician counselling predicted former smoking.

a. Contributorship Statement

Drs. RE, RD, and MS made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data. Dr. LB and Mr. MP assisted in revising the manuscript critically for important intellectual content. All authors provided approval of the version to be published.

b. Competing Interests

None declared

c. Funding

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Correlates of former smoking in patients with cerebrovascular disease: A cross-sectional study

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Correlates of former smoking in patients with cerebrovascular disease: A cross-sectional study

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Rojiemiahd K. Edjoc, PhD[1].
Robert D. Reid, PhD., MBA[1]
Mukul Sharma, MD MSc FRCPC[2]
Louise Balfour, PhD[3]
Michael Procino, BSc[3]

[1]Division of Prevention and Rehabilitation, University of Ottawa Heart Institute, Ottawa, Canada
[2]McMaster University, Population Health Research Institute, Hamilton, Ontario, Canada
[3]University of Ottawa, Ottawa, Ontario, Canada

Correspondence to:
R Edjoc PhD
Consulting Epidemiologist
University of Ottawa Heart Institute
40 Ruskin St.
Ottawa, ON
K1Y4W7
Email: redjoc@hotmail.com

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ABSTRACT

Objective: To identify multi-level correlates of former smoking in patients with cerebrovascular disease.

Design: Secondary data analysis of the Canadian Community Health Survey.

Methods: We used data from the 2007-2008 Canadian Community Health Survey (CCHS). Smoking status (former smoking vs. smoker) was described by multi-level correlates of former smoking. A multi-level approach for variable selection for this study was used to understand how multiple levels in society can have an impact on former smoking. The study sample was selected from those respondents of the CCHS that reported they suffered from stroke symptoms. Logistic regression was used to predict former smoking in patients with cerebrovascular disease while controlling for multi-level confounders. Proportions were weighted to reflect the Canadian population.

Results: There were 172 355 respondents who reported to suffer from stroke. From this sample, 36.5% were smokers and 63.5% were former smokers. Age groups 55-69 and 70-80 and higher education (secondary education +) were positively related to former smoking. Household and vehicle smoking restrictions significantly predicted former smoking. Counselling advice from a physician and having access to a general practitioner were correlates of former smoking. Finally, the use of buproprion was positively related to former smoking.

*Conclusions***:** There are multi-level correlates of former smoking in smokers with reported stroke symptoms. These correlates include older age groups, higher education, household and vehicle smoking restrictions, pharmacotherapy use (bupropion), access to a general practitioner and counselling advice from a physician.

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Article focus

- To elucidate the multi-level correlates of former smoking in smokers who suffer from stroke symptoms.

Key messages

- There are multi-level correlates of former smoking in individuals who reported to suffer from stroke symptoms.
- It is imperative that smoking cessation be incorporated in secondary prevention practice while taking significant co-morbidities such as depression and alcohol consumption into account for this population.

Strengths and limitations of the study

- This study is the first to elucidate correlates of former smoking in this population at a multi-level.
- The size of the study provide adequate power for the statistical analyses
- The cross-sectional nature of the study and self-reported outcomes such as smoking status and the presence of stroke symptoms may result in social desirability bias.

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INTRODUCTION

Smoking is an independent risk factor for incident and recurring stroke.[1, 2, 3]. It has been found that smoking cessation can reduce the relative risk of stroke and transient ischemic attach (TIA) by 50% [4] and stroke related hospitalizations.[5] Despite the supporting evidence regarding the benefits of smoking cessation for smokers with cerebrovascular disease, there is evidence that 89% of these smokers were still smoking 12 months after their event.[6]

Stroke prevention guidelines recommend that healthcare providers strongly advise every smoker who is at high risk for a stroke or TIA to quit, and provide specific assistance with quitting, including counselling and pharmacotherapy. [3, 7]

There are very few published smoking cessation intervention (SCI) studies in stroke and TIA patients. A recent systematic review found a non-significant effect of SCI's on quitting in stroke and TIA patients.[8] The authors found that with the available studies, there was a sub-optimal use of evidence -based approaches to smoking cessation comprised of counselling, pharmacotherapy and follow-up.[8]

More interventions need to be developed by identifying significant correlates of former smoking among these high-risk smokers. The socio-ecological model proposed by Sorensen and associates [9] explicate factors that influence different groups to use or not use tobacco (Figure 1). These factors include population characteristics and socio-economic status (SES), individual, interpersonal, community and organizational factors. Based on this socio-ecological model, the present study elucidated multi-level correlates of former smoking using data from the Canadian Community Health Survey (CCHS). There has not been any study that has systematically explored the impact of multi-level correlates on the cessation attempt and thus is the impetus for the present study.

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METHODS

Data from the 2007-2008 Canadian Community Health Survey were used for the present study. The CCHS is a cross-sectional survey that collects information related to the factors that contribute to health, social and economic determinants of Canadians. [10] The CCHS utilizes a complex sampling strategy with stratification and multiple stages of selection yielding a sample that is representative of 98% of the Canadian population. [10]

Only individuals who reported the effects of stroke were included in the present analysis. Stroke symptoms were defined as those who reported were experiencing stroke symptoms due to a recent stroke or TIA within the last year. No further information was available. From this sample, smoking status (smoking vs. former smoking) was selected as the dependent variable. Important correlates were grouped by population characteristics and SES, individual, interpersonal, community and organizational level. Population characteristics included: sex and age. Age was re-coded into four categories (ages 12-34; 35-54; 55-69 and 70-80+). Socio-economic status included: income and education. Individual level correlates included: co-morbidities such as depression, diabetes mellitus, hypertension and alcohol consumption. Interpersonal level correlates included: having household and vehicle smoking restrictions and access to a general practitioner (GP). Community level correlates included: exposure to public and workplace smoking restrictions. Organizational level correlates were defined as the use of smoking cessation resources such as pharmacotherapy (nicotine replacement therapy [NRT] and bupropion) and counselling support provided by a physician or referral to a smoking cessation group. Ideally, varenicline would be included in the list of pharmacotherapy. Unfortunately, at the time of this survey, varenicline was not yet approved for use in Canada and

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was not collected by the CCHS. Due to the complex survey design of the CCHS, adjusted weight was calculated for each respondent taking into account national average design effects and the relative sampling weights.

Statistical Analysis

All statistical analyses were performed using Statistical Analysis Software (SAS) using SURVEYFREQ and SURVEYLOGISTIC procedures.

Descriptive analyses were performed to describe smoking status (smoker and former smoker) and correlates of interests in respondents who reported to have stroke symptoms. A chi-square test of significance was used to determine significant differences between cross-tabulated proportions. Significance was reported at 95% confidence or having a value of $P<0.05$.

Logistic regression was used to predict the dependent variable of reported former smoking while controlling for each identified correlate. Significant correlates of former smoking were expressed by odds ratio (OR) point estimates at a 95% confidence level (CI). The method of model building for logistic regression analyses was forward-stepwise selection. The Wald statistic was used for variable selection. Independent variables were identified as significant correlates if the p-value was less than 0.05 ($p<0.05$).

RESULTS

The overall weighted sample was 172 355 individuals who reported to suffer from stroke symptoms. From this sample 36.5% reported to be smokers and 63.5% reported to be former smokers.

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There were more males who reported they were former smokers than females (p<0.0001). In the smoking cohort, more males were smokers than females (p<0.0001). Individuals who were former smokers were older than smoking individuals. In general, this cohort had less than secondary education and were earning an annual income of $ 20 000 - 39 000.

Individuals who were former smokers reported to have higher proportions of household (p<0.0001) and vehicle smoking restrictions (p<0.0001) compared to smoking individuals. Exposure to public smoking restrictions (p<0.0001) was higher in smokers compared to former smokers.

Respondents who were former smokers compared to current smokers reported they used NRT (p<0.0001) more frequently as well as bupropion (p<0.0001). Smokers reported more physician counselling (p<0.0001) than former smokers. Smoking individuals reported higher proportions of alcohol consumption (>2 drinks per day; p=0.03), depression (p<0.0001) and diabetes (p=0.21) than former smokers.

Female sex and co-morbidities such as alcohol consumption and depression reduced the likelihood of former smoking. Age groups 55-69 and 70-80 years old were significant correlates of former smoking. Household and vehicle smoking restrictions significantly predicted former smoking while exposure to workplace and public place smoking restrictions did not. The use of pharmacotherapy such as bupropion significantly predicted former smoking while the use of NRT did not. Counselling advice from a physician and having a GP were correlates of former smoking.

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Table 1: Study Cohort Characteristics

Covariates	*Smoker (%)* (n=62 960)	*Former Smoker (%)* (n=109 395)	*P-values*
Males	56.4	62.9	<0.0001
Females	43.6	37.1	<0.0001
Age			<0.001
12-34	4.2	1.5	
35-54	20.4	3.1	
55-69	38.2	24.4	
70-80+	31	46.1	
Education			<0.001
< secondary	18.9	18.2	
Secondary	11.5	13.6	
Some post-secondary	3.8	5.1	
Income			<0.001
None or < 20 000	20.7	14.3	
20 000 -39 000	25.3	29.3	
40 000 – 59 000	19.1	17.3	
60 000 – 79 000	6.2	7.11	
80 000 +	10.5	13.5	

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Table 1a: Study Cohort Characteristics

Covariates	*Smoker (%)* (n=62 960)	*Former Smoker (%)* (n=109 395)	*P-values*
Smoking restrictions			
Household	35.5	74.0	<0.0001
Workplace	29.8	10.5	0.004
Vehicle	0.0	93.7	<0.0001
Public	0.0	93.3	<0.0001
Have access to GP	86.7	94.9	<0.0001
Smoking cessation aids	0.0	0.0	-
Nicotine replacement therapy (NRT) gum	0.0	0.1	<0.0001
NRT patch	0.0	0.4	<0.0001
Zyban/Bupropion	4.5	0.5	<0.0001
MD counselling	0.5	0.0	<0.0001
One-to-One referral	0.0	0.0	-
Referral to smoking cessation group			
Alcohol drinking (> 2 drinks/day)	49.1	7.9	0.03
Depression	41.7	35.6	<0.0001
Diabetes	20.7	8.9	0.21
Hypertension	7.9	20.4	0.02

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Table 2: Correlates of smoking cessation of study cohort

	Odds Ratio (OR)	Lower 95% CI	Upper 95% CI
Population characteristics			
Female Sex	0.41	0.41	0.42
Age 12-34	0.01	0.001	0.01
Age 35- 54	0.03	0.02	0.03
Age 55- 69	1.20	1.1	1.19
Age 70- 80	1.50	1.61	1.67
Socio-economic status			
Less than secondary	0.90	0.88	0.93
Secondary education	1.25	1.22	1.29
Some post-secondary education	1.24	1.23	1.24
Post-secondary	1.12	1.1	1.15
No income or less 20 000	1.48	1.45	1.52
$20 000- 39 000	1.30	1.24	1.29
$40 000- 59 000	1.10	1.06	1.11
$60 000- 79 000	0.65	0.63	0.67
$80 000+	0.7	0.67	0.7
Individual level			
Alcohol Consumption (> 2 drinks/day)	0.70	0.70	0.71
Depression	0.90	0.88	0.91
Hypertension	1.21	1.18	1.24
Diabetes	1.4	1.34	1.38
Interpersonal level			
Household smoking restrictions	1.10	1.05	1.08
Vehicle smoking restrictions	2.98	2.91	3.06
Access to a GP	1.30	1.23	1.3
Organizational level			
Zyban/Bupropion	15.52	14.03	17.16
MD counselling	1.52	1.39	1.67

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DISCUSSION

The aim of this study was to identify the correlates of former smoking in smokers with reported stroke symptoms at multi-levels. Income and older age were predictive of former smoking while higher education predicted former smoking in this cohort. These results are in line with previous investigations of gender effects, [11, 12, 13] older age [11, 12] and level of income [11, 12, 14] vis a vis smoking and cessation. Our results indicate there are fewer women who are former smokers than men. Reynaso and colleagues outline [13] that there may be several working hypotheses that may explain this result. First, women may respond poorly to nicotine replacement therapy. Second, women are more vulnerable to depression and anxiety symptomatology following cessation. Third, there may be concerns regarding post-cessation weight gain and body-shape concerns for women compared to men. Fourth, women's menstrual cycle effects enhance nicotine withdrawal symptoms following cessation. Fifth, women do not receive or do not respond to the beneficial effects of social support during cessation. [13] They suggest that a varied approach to smoking cessation be taken in light of these hypotheses. For example it is imperative to consider an approach with lengthier treatments for women following NRT termination. [13] Health professionals should also consider adjunct programs during the cessation attempt for women who have concerns about weight gain. [13]

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There should be concurrent treatment programs for women who have a history of anxiety/depression. [13] Finally, the timing of smoking cessation interventions early in the follicular phase of the menstrual cycle should be considered. [13]

In regards to income, there seems to be an inverse effect of those who are at the lower levels of SES and former smoking. A recent review by Hiscock and associates (2011) suggested that the higher smoking prevalence in lower SES groups and lower smoking cessation rates might be a result of the clustering of disadvantages. [14] These disadvantages include: a reduced social support for quitting, low motivation to quit, increased addiction to tobacco, increased likelihood of not completing courses of pharmacotherapy or behavioural support sessions, psychological differences such as lack of self-efficacy, and susceptibility tobacco industry marketing. [14] As a result, quit attempts in this population are significantly less likely to be successful. [14]

Koning and associates found that each additional year of education reduced the risk of continued smoking.[15] Their data suggested that people with higher education may be able to better understand the consequences of long-term smoking and may have more resources available for them to quit smoking.[15]

Our findings suggest that co-morbid conditions such as alcohol consumption and depression significantly decreased the likelihood of former smoking. These findings are supported by evidence suggesting that cerebrovascular patients experience higher rates of co-morbidity particularly depression. [16, 17, 18] Compared to cardiac patients, patients with a recent stroke suffered a three to five-fold increased risk of depressive disorders [16] as well as higher proportions of alcohol consumption and hypertension. [19]

These findings may have clinical implications particularly for this population, as co-morbid conditions such as depression and increased alcohol consumption are significantly more common in patients who smoke. Considering their association with increased

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smoking behaviour, co-morbidities may be hindering the success of quitting smoking. The hindering effect of co-morbidity on former smoking is especially problematic as smoking increases blood coagulability, platelet aggregation, thrombus formation and endothelial damage,[20] thus increasing the chance of a stroke two-fold [21] and of stroke recurrence.[21, 22] Quitting smoking has been shown to reduce the risk of stroke to that of a non-smoker after 5 years [23] and reduce the risk hazard ratio of stroke recurrence from 1.71 to 1.39 ($p < 0.05$).[24] It is imperative that smoking cessation be incorporated in secondary prevention practice while taking these significant co-morbidities into account. Depression and excessive alcohol consumption might impede cessation in people with cerebrovascular disease. However due to the limitation of cross-sectional studies, we do not know if these co-morbidities existed before or after the reported stroke. Further study regarding the effects of these co-morbidities on cessation using other study designs might be warranted.

Population based interventions such as household, workplace, vehicle and public smoking restrictions have all been found to predict smoking abstinence.[25, 26, 27] They have also been found to reduce cigarette consumption, and initiation and increase smoking cessation rates.[25, 26, 27] These authors suggest that population based interventions are anti-tobacco socialization tools that may promote the internalisation of behavioural norms against the initiation or continuation of smoking. Our results are partially in line with this evidence. We found that household and vehicle smoking restrictions predicted smoking cessation but not so with workplace or public smoking restrictions. It is not known why workplace and public smoking restrictions did not predict smoking cessation especially since their implementation under the Smoke Free Ontario Act [28, 29, 30] in Ontario and similar legislations across Canada. Since their implementation, smoking prevalence in Canada has been dramatically decreased. Perhaps the insignificant effect of public and workplace smoking restrictions may be explained in the decrease of funding in the SFOA in 2007-2008 of 60 million, down 2.5 million

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from the year before of 62.5 million in 2006-2007. [, 31, 32] Similar reductions in tobacco control funding can be observed in other provinces. [29, 30] There is a documented association between population interventions effectiveness and sustained funding.[33]

A similar situation was observed with the California Model in the state of California. The California Model is similar to the SFOA and is a population intervention that used workplace and public place smoking restrictions to de-normalize tobacco use.[33] Pierce and associates found that the initial effect of the California Model to decrease smoking prevalence in the state dissipated as their funding was reduced.[33]

In light of this conundrum, there is evidence that suggests that household and vehicle-smoking restrictions are more effective because they are less regulated. [, 34, 35] These authors suggest that smoking restrictions such as at home or in a vehicle are effective because those who implement them do so by choice and not through forced legislation [33, 34, 35] thereby increasing the odds of smoking cessation.

We found that the use of pharmacotherapy such as bupropion and physician counselling increased the odds of former smoking but NRT use did not. According to Fiore and associates, pharmacotherapy along with counselling and follow-up increases the odds of smoking cessation. [36] NRT and bupropion have each been found to be more efficacious than placebo for increasing the odds of smoking cessation.[37]

The lack of effect of NRT may be indicative of the well-documented practice gap in health care in regards to smoking cessation. Young and Ward found that only 32% of physicians provided written materials for their patients and only 28% of physicians set a “quit date” with their patients.[38] Likewise Shaohua and colleagues found that many family physicians feel lack of time was their biggest

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barrier in terms of implementing smoking cessation practices.[39] Their study found that less than half were willing or able to assist their patients to quit with the use of counselling, pharmacotherapy or arrange a follow-up visit to reinforce the benefits of smoking cessation.[38] This is consistent with the stroke population as documented by Mouradian and associates.[6] Perhaps another explanation may be the lack of information regarding the effectiveness of smoking cessation medications and similar interventions in stroke and TIA patients. Furthermore, physicians may be reluctant to prescribe NRT's due to their availability over the counter. Further research is required to determine if the latter explanations are supported by evidence.

Cross-sectional surveys such as CCHS are useful for initial exploratory studies. They are far reaching and reflect "a snapshot" of the population. However there are limitations to our study and they will be explored here. Since both exposure and outcome were measured at the same time, one cannot be certain which is the exposure or the outcome. In other words, the rules for contributory cause cannot be fulfilled. For example, it is possible that respondents stopped smoking years before their stroke diagnosis. Owing to the nature of the cross-sectional design of the CCHS, there is no way to ascertain which of the two (former smoking vs. stroke diagnosis) came first. Unfortunately there is no available variable that quantifies the time-point of cessation in relation to respondents' stroke diagnosis. Another limitation would be the results that found sex and age as significant correlates of former smoking. Unfortunately, these are unmodifiable correlates. Future interventions should take into account modifiable correlates such as the implementation of household and vehicle smoking restrictions and the availability of pharmacotherapy and counselling support. Another limitation is the mode of collection of the data. Social desirability and recall bias for example could play an important role and a source of biases within this study.[40] For example, since smoking status, the presence of stroke symptoms and co-morbidities such as depression were self-reported,

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special care should be taken when interpreting our results. An example of social desirability effect would be respondents not accurately reporting their smoking status. Since smoking would be an undesirable image for some depending on age, gender or socio-economic status, data obtained might not be representative of the real picture found in the population. Ideally all smoking related measures should be validated bio-chemically with breath samples measuring carbon monoxide levels or coitinine levels measuring the amount of nicotine in the blood. Furthermore, without an expert assessment from a health care professional of stroke symptoms or depression would also limit the generalizability of the results.

CONCLUSION

We found significant correlates of former smoking at multiple levels in smokers with reported stroke symptoms. Age and education level were significant correlates of former smoking as well as household and vehicle smoking restrictions and access to a GP. Finally, the use of bupropion along with physician counselling predicted former smoking.

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a. Contributorship Statement

Drs. RE, RD, and MS made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data. Dr. LB and Mr. MP assisted in revising the manuscript critically for important intellectual content. All authors provided approval of the version to be published.

b. Competing Interests

None declared

c. Funding

None

d. Data sharing

No data available

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Correlates of former smoking in patients with cerebrovascular disease: A cross-sectional study

Rojiemiahd K. Edjoc, PhD[1].
Robert D. Reid, PhD., MBA[1]
Mukul Sharma, MD MSc FRCPC[2]
Louise Balfour, PhD[3]
Michael Procino, BSc[3]

[1]Division of Prevention and Rehabilitation, University of Ottawa Heart Institute, Ottawa, Canada
[2]McMaster University, Population Health Research Institute, Hamilton, Ontario, Canada
[3]University of Ottawa, Ottawa, Ontario, Canada

Correspondence to:
R Edjoc PhD
Consulting Epidemiologist
University of Ottawa Heart Institute
40 Ruskin St.
Ottawa, ON
K1Y4W7
Email: redjoc@hotmail.com

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Key words: Smoking, cessation, cerebrovascular disease, stroke, prevention, epidemiology

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ABSTRACT

Objective: To identify multi-level correlates of former smoking in patients with cerebrovascular disease.

Design: Secondary data analysis of the Canadian Community Health Survey.

Methods: We used data from the 2007-2008 Canadian Community Health Survey (CCHS). Smoking status (~~quit smoking completely~~former smoking vs. smoker~~ vs. non-smoker~~) was described by multi-level correlates of former smoking. ~~was described by population characteristics, socio-economic status, individual correlates, interpersonal correlates, community correlates and organizational correlates.~~ A multi-level approach for variable selection for this study was used to understand how multiple levels in society can have an impact on former smoking. ~~We define multi-level as~~ The study sample was selected from those respondents of the CCHS that reported they suffered from stroke symptoms. Logistic regression was used to predict former smoking in patients with cerebrovascular disease ~~describe the association between quitting smoking and stroke~~ while controlling for multi-level confounders~~ correlates of former smoking~~. Proportions were weighted to reflect the Canadian population.

Results: There were ~~383904~~ 172 355 respondents who reported to suffer from stroke. From this sample, ~~211549 respondents (55.1%) reported they were non-smokers and 62960 (~~36.5%~~16.4%) respondents reported they~~ were smokers~~. There were~~ and ~~109395 (28.5%)~~ 63.5% ~~individuals who indicated they~~ were former smokers. ~~At the population characteristics and socio-economic level, female sex (OR 0.4; 95% CI: 0.41 to 0.42) reduced the likelihood of being a former smoker. The a~~Age groups 55-69 ~~(OR 1.1; 95% CI: 1.10 to 1.19)~~ and

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70-80 ~~(OR 1.6; 95% CI: 1.61 to 1.67)~~ and higher education (secondary education +) were positively related to former smoking. ~~At the interpersonal level,~~ H~~h~~ousehold ~~(OR 1.1; 95% CI: 1.05 to 1.08)~~ and vehicle ~~(OR 2.9; 95% CI: 2.79 to 2.93)~~ smoking restrictions significantly predicted former smoking. Counselling advice from a physician and having access to a general practitioner ~~was also a correlate of~~were correlates of former smoking ~~(OR 3.7; 95% CI: 3.37 to 4.03)~~. Finally, the use of buproprion was positively related to former smoking. ~~Depression (OR 0.9 CI: 0.89 to 0.91) and alcohol consumption (OR 0.7 95% CI: 0.69 to 0.71) reduced the likelihood of former smoking.~~

Conclusions: There are multi-level correlates of former smoking in smokers with reported stroke symptoms. These correlates include older ~~certain~~ age groups, higher education, ~~-~~household and vehicle smoking restrictions, pharmacotherapy use (bupropion), ~~-~~access to a general practitioner and ~~s and~~ counselling advice from a physician. ~~Future interventions should be tailored with these correlates in mind to increase the likelihood of cessation~~.

Article focus

- To elucidate the multi-level correlates of former smoking in smokers who suffer from stroke symptoms.

Key messages

- There are multi-level correlates of former smoking in individuals who reported to suffer from stroke symptoms.
- It is imperative that smoking cessation be incorporated in secondary prevention practice while taking significant co-morbidities such as depression and alcohol consumption into account for this population.

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Strengths and limitations of the study

- This study is the first to elucidate correlates of former smoking in this population at a multi-level.
- The size of the study provide adequate power for the statistical analyses
- The cross-sectional nature of the study and self-reported outcomes such as smoking status and the presence of stroke symptoms may result in social desirability bias.

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INTRODUCTION

Smoking is an independent risk factor for incident and recurring stroke.[1, 2, 3]. It has been found that smoking cessation can reduce the relative risk of stroke and transient ischemic attach (TIA) by 50% [4] and stroke related hospitalizations.[5] Despite the supporting evidence regarding the benefits of smoking cessation for smokers with cerebrovascular disease, there is evidence that 89% of these smokers were still smoking 12 months after their event.[6]

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Stroke prevention guidelines recommend that healthcare providers strongly advise every smoker who is at high risk for a stroke or TIA to quit, and provide specific assistance with quitting, including counselling and pharmacotherapy. [3, 7]

There are very few published smoking cessation intervention (SCI) studies in stroke and TIA patients. A recent systematic review found a non-significant effect of SCI's on quitting in stroke and TIA patients.[8] The authors found that with the available studies, there was a sub-optimal use of evidence -based approaches to smoking cessation comprised of counselling, pharmacotherapy and follow-up.[8]

More interventions need to be developed by identifying significant correlates of former smoking among these high-risk smokers. The socio-ecological model proposed by Sorensen and associates [9] explicate factors that influence different groups to use or not use tobacco ~~(Figure 1)~~. These factors include population characteristics and socio-economic status (SES), individual, interpersonal, community and organizational factors. Based on this socio-ecological model, the present study elucidated multi-level correlates of former smoking using data from the Canadian Community Health Survey (CCHS). There has not been any study that has systematically explored the impact of multi-level correlates on the cessation attempt and thus is the impetus for the present study.

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METHODS

Data from the 2007-2008 Canadian Community Health Survey were used for the present study. The CCHS is a cross-sectional survey that collects information related to the factors that contribute to health, social and economic determinants of Canadians. [10] The CCHS utilizes a complex sampling strategy with stratification and multiple stages of selection yielding a sample that is representative of 98% of the Canadian population. [10]

Only individuals who reported the effects of stroke were included in the present analysis. Stroke symptoms were defined as those who reported were experiencing stroke symptoms due to a recent stroke or TIA within the last year. No further information was available. From this sample, smoking status (smoking vs. former smoking) was selected as the dependent variable. Important correlates were grouped by population characteristics and SES, individual, interpersonal, community and organizational level. Population characteristics included: sex and age. Age was re-coded into four categories (ages 12-34; 35-54; 55-69 and 70-80+). Socio-economic status included: income and education. Individual level correlates included: co-morbidities such as depression, diabetes mellitus, hypertension -and- alcohol consumption~~, and nicotine addiction (as measured by the Fagerström Test of Nicotine Dependence)~~. Interpersonal level correlates included: having household and vehicle smoking restrictions and access to a general practitioner (GP). Community level correlates included: exposure to public and workplace smoking restrictions. Organizational level correlates were defined as the use of smoking cessation resources such as pharmacotherapy (nicotine replacement therapy [NRT] and bupropion) and counselling support provided by a physician or referral to a smoking cessation group. Ideally, varenicline would be included in the list of pharmacotherapy. Unfortunately, at the time of this survey, varenicline was not yet approved for use in Canada and was not collected by the CCHS. ~~Age~~

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~~was re-coded into five categories (ages 12-19; 20-34; 35-54; 55-69 and 70-80+).~~ Due to the complex survey design of the CCHS, adjusted weight was calculated for each respondent taking into account national average design effects and the relative sampling weights.

Statistical Analysis

All statistical analyses were performed using Statistical Analysis Software (SAS) using ~~).~~ SURVEYFREQ ~~-~~and SURVEYLOGISTIC procedures.

~~Cross-tabulations between reported stroke symptoms (yes or no) and smoking status (smoker, former smoker and non-smoker) were performed while controlling for correlates of interest.~~ Descriptive analyses were performed to describe smoking status (smoker and former smoker) and correlates of interests in respondents who reported to have stroke symptoms. A chi-square test of significance was used to determine significant differences between cross-tabulated proportions. Significance was reported at 95% confidence or having a value of P<0.05.

LA ~~l~~ogistic regression ~~model was 'fitted'~~ was used to predict ~~using~~ the dependent variable of reported former smoking while controlling for each ~~correlate level (population characteristics, socio-economic position, individual, interpersonal, community and organizational level)~~identified correlate. Significant correlates of former smoking were expressed by odds ratio (OR) point estimates at a 95% confidence level (CI). The method of model building for logistic regression analyses was forward-stepwise selection. The Wald statistic was used for variable selection. Independent variables were identified as significant correlates if the p-value was less than 0.05 (p<0.05).

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RESULTS

~~A summary of the characteristics of the study cohort can be found in Tables 1 and 1a. The following table is divided by smoking status (i.e. Non-smoker, Smoker and Former smoker).~~ The overall weighted sample was ~~383904~~172 355 individuals who reported to suffer from stroke symptoms. From this sample ~~211549 individuals (55.1%) reported to be non-smokers and 62960 (16.4~~37.036.5%~~)~~ ~~individuals~~ reported to be smokers and ~~. There were 109395 (28.5~~63.~~05~~%~~)~~ ~~individuals who~~ reported to be former smokers.

There were more males who reported they were former smokers ~~had quit smoking~~ than females ~~(62.9% vs. 37.1%; (~~p<0.0001). In the smoking cohort, more males were ~~continued~~ smokers than females (~~56.4% vs. 43.6%%;~~ p<0.0001). ~~There were more males than females (50.7% vs. 49.3%; p<0.0001) in the non-smoking cohort.~~ Individuals who were former smokers ~~quit smoking as well as non-smoking individuals~~ were older than smoking individuals. In general, ~~all cohorts had post-secondary education~~ this cohort had less than secondary education and were earning an annual income of $ 20 000 - 39 000.

Individuals who ~~quit smoking~~were former smokers reported to have higher proportions of household ~~(74% vs. 35.5%%;~~ (p<0.0001) and vehicle smoking restrictions (~~93.7% vs. 0.0%%;~~ p<0.0001) compared to smoking individuals. Exposure to public smoking restrictions (~~93.3 % vs. 0.6%;~~ p<0.0001) was higher in smokers compared to ~~quitters~~former smokers.

Respondents who were former smokers compared to current smokers reported they used NRT (~~0.1% vs. 0.0%;~~ p<0.0001) more frequently as well as bupropion (~~0.4% vs. 0.0%;~~ p<0.0001). Smokers reported more physician counselling (~~4.5% vs. 0.5%;~~ p<0.0001) than ~~quitters~~former smokers. Smoking individuals reported higher proportions of alcohol consumption (>2 drinks per day~~; 49.1% vs. 7.9%~~; p=0.03), ~~more~~ depression (~~41.7% vs. 35.6%;~~ p<0.0001) and diabetes (~~20.7% vs. 8.9%;~~ p=0.21) than ~~respondents who were~~ former

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smokers. ~~Similar trends were found when we compared smoking individuals and non-smokers for alcohol consumption (> 2 drinks per day; 49.1% vs.39.1%; p<0.0001), depression (41.7% vs. 32.8%; p<0.0001) and diabetes (20.7% vs. 8.6%; p=0.21) although not significant.~~

~~Logistic regression odds ratios and 95% CIs of significant correlates of former smoking can be found in Tables 2 and 2a. At the population characteristic and socio-economic level,~~ Ffemale sex and co-morbidities such as alcohol consumption and depression ~~(OR 0.4; 95% CI: 0.41 to 0.42)~~ reduced the likelihood of former smokingquitting. Age groups 55-69 ~~(OR 1.1; 95% CI: 1.10 to 1.19)~~ and ~~age~~ 70-80 years old ~~(OR 1.6; 95% CI: 1.61 to 1.67)~~ were significant correlates of former smoking. ~~At the individual level, Cco-morbidities such as alcohol consumption (OR 0.7; 95% CI: 0.69 to 0.71) and depression (OR 0.9; 95% CI: 0.88 to 0.91) reduced the likelihood of former smoking. At the interpersonal level, household~~Household ~~(OR 1.1; 95% CI: 1.05 to 1.08)~~ and vehicle ~~(OR 2.9; 95% CI: 2.79 to 2.93)~~ smoking restrictions significantly predicted former smoking whileg. ~~However at the community level,~~ exposure to workplace and public place smoking restrictions did not. ~~significantly predict former smoking.~~ The use of pharmacotherapy such as bupropion significantly predicted former smoking ~~(OR 15.4; 95% of CI: 13.9 to 17.0)~~ while the use of NRT did not. Counselling advice from a physician and having a GP were~~was also a~~ -correlates of former smoking ~~(OR 3.7; 95% CI: 3.37 to 4.03)~~.

Table 1: Study Cohort Characteristics

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Covariates	*Smoker (%)* (n=62 960)	*Former Smoker (%)* (n=109 395)	*P-values*
Males	56.4	62.9	<0.0001
Females	43.6	37.1	<0.0001
Age			<0.001
12-34	4.2	1.5	
35-54	20.4	3.1	
55-69	38.2	24.4	
70-80+	31	46.1	
Education			<0.001
< secondary	18.9	18.2	
Secondary	11.5	13.6	
Some post-secondary	3.8	5.1	
Income			<0.001
None or < 20 000	20.7	14.3	
20 000 -39 000	25.3	29.3	
40 000 – 59 000	19.1	17.3	
60 000 – 79 000	6.2	7.11	
80 000 +	10.5	13.5	

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Table 1a: Study Cohort Characteristics

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Covariates	Smoker (%) (n=62 960)	Former Smoker (%) (n=109 395)	P-values
Smoking restrictions			
Household	35.5	74.0	<0.0001
Workplace	92.8	80.5	0.004
Vehicle	0.0	93.7	<0.0001
Public	0.0	93.3	<0.0001
Have access to GP	86.7	94.9	<0.0001
Smoking cessation aids	0.0	0.0	-
Nicotine replacement therapy (NRT) gum	0.0	0.1	<0.0001
NRT patch	0.0	0.4	<0.0001
Zyban/Bupropion	4.5	0.5	<0.0001
MD counselling	0.5	0.0	<0.0001
One-to-One referral	0.0	0.0	-
Referral to smoking cessation group			
Alcohol drinking (> 2 drinks/day)	49.1	7.9	0.03
Depression	41.7	35.6	<0.0001
Diabetes	20.7	8.9	0.21
Hypertension	7.9	20.4	0.02

~~Table 1: Study Cohort Characteristics~~

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Covariates	Non Smoker (%) (n=211 549)	Smoker (%) (n=62 960)	Former Smoker (%) (n=109 395)	P values
Males	107 191 (50.7)	35 521 (56.4)	68 798 (62.9)	<0.0001
Females	104 358 (49.3)	27439 (43.6)	37.140 597 (37.1)	<0.0001
Age				
12-19	1975 (0.9)	314 (0.5)	0.0	<0.001
1220-34	2259 (1.1)	27452432 (3.94.2)	1649 (1.5)1.5	<0.001
35-54	11 070 (5.2)	28.412 830 (20.4)	3.13.13375 (3.1)	<0.001
55-69	44 277 (20.9)	38.224 079 (38.2)	24.426 660 (24.4)	<0.001
70-80+	89 613 (42.4)	31.019 515 (31.0)	46.150 456 (46.1)	<0.001
Education				
< secondary	40 722 (19.3)	18.911 913 (18.9)	18.219 917 (18.2)	<0.001
Secondary	26 478 (12.5)	11.57244 (11.5)	13.614 902 (13.6)	<0.001
Some post secondary	10 367 (4.9)	3.82390 (3.8)	5.15583 (5.1)	<0.001
Income				<0.001
None or < 20 000	33 607 (15.9)	20.713 029 (20.7)	14.315 686 (14.3)	
20 000 - 39 000	56 858 (26.9)	25.315 931 (25.3)	29.332 083 (29.3)	
40 000 - 59 000	34 765 (16.4)	19.111 993 (19.1)	17.318 891 (17.3)	
60 000 - 79 000	14 422 (6.8)	6.23928 (6.24)	7.17712 (7.1)	
80 000 +	26 714 (12.6)	10.56617 (10.5)	13.514 739 (13.5)	
Fagerstrom Nicotine Dependency				<0.0001
Very Low				
Low	0.0	562 (0.89)	0.0	
Medium	0.0	482 (0.8)	0.0	
High	0.0	137 (0.2)	0.0	
Very High	0.0	1288 (2.0)	0.0	
	0.0	326 (0.5)	0.0	

Table 1a: Study Cohort Characteristics

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Smoking restrictions				
Household	163 099 (77.1)	35.522 364 (35.5)	74.080 983 (74.0)	<0.0001
Workplace	23 845 (11.3)	10.511 470 (10.5)	0.004	
Vehicle	210 504 (99.5)	29.818 740 (29.8)	93.7102 527 (93.7)	<0.0001
Public	209 436 (99.0)	0.00.0	93.3102 032 (93.3)	<0.0001
Have access to GP	202 460 (95.8)	0.00.0	94.9103 830 (94.9)	<0.0001
Smoking cessation aids				
Nicotine replacement therapy (NRT)	0.0	86.755 563 (86.7)	0.00.0	-
gum	0.0		0.165.9 (0.1)	<0.0001
NRT patch	0.0		0.4486 (0.4)	<0.0001
Zyban/Buprion	0.0	0.00.0	0.5511 (0.5)	<0.0001
MD counseling	0.0	0.00.0	0.00.0	<0.0001
One-to-One referral	0.0	0.00.0	0.00.0	-
Referral to smoking cessation group	82 752 (39.1)	2853 (4.5)4.5 326 (0.5)0.5	4984 (7.9)7.9	0.03
Alcohol drinking (> 2 drinks/day)	69 425 (32.8)	0.00.0	38 989 (35.6)35.6	<0.0001
Depression	18 256 (8.6)		9823 (8.9)8.9	0.21
Diabetes	50 217 (23.7)	49.130 923 (49.1)	22 263 (20.4)20.4	0.02
Hypertension		41.726 239 (41.7)		
		20.713 029 (20.7)		
		7.94984 (7.9)		

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~~Table 2: Correlates of smoking cessation of study cohort~~

Table 2: Correlates of smoking cessation of study cohort

	Odds Ratio (OR)	Lower 95% CI	Upper 95% CI
Population characteristics			
Female Sex	0.41	0.41	0.42
Age 12-34	0.01	0.001	0.01
Age 35- 54	0.03	0.02	0.03
Age 55- 69	1.20	1.1	1.19
Age 70- 80	1.50	1.61	1.67
Socio-economic status			
Less than secondary	0.90	0.88	0.93
Secondary education	1.25	1.22	1.29
Some post-secondary education	1.24	1.23	1.24
Post-secondary	1.12	1.1	1.15
No income or less 20 000	1.48	1.45	1.52
$20 000- 39 000	1.30	1.24	1.29
$40 000- 59 000	1.10	1.06	1.11
$60 000- 79 000	0.65	0.63	0.67
$80 000+	0.7	0.67	0.7
Individual level			
Alcohol Consumption (> 2 drinks/day)	0.70	0.70	0.71
Depression	0.90	0.88	0.91
Hypertension	1.21	1.18	1.24
Diabetes	1.4	1.34	1.38
Interpersonal level			

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Household smoking restrictions	1.10	1.05	1.08
Vehicle smoking restrictions	2.98	2.91	3.06
Access to a GP	1.30	1.23	1.3
Organizational level			
Zyban/Bupropion	15.52	14.03	17.16
MD counselling	1.52	1.39	1.67

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Table 2: Correlates of smoking cessation of study cohort

Correlates of smoking cessation	Odds Ratio (OR)	Lower 95% CI	Upper 95% CI
Population characteristics			
Female Sex	0.41	0.41	0.42
Age 12-19	-	-	-
Age 1220-34	0.012	0.0012	0.013
Age 35-54	0.03	0.025	0.027
Age 55-69	1.21	1.10	1.19
Age 70-80	1.56	1.61	1.67
Socio-economic status			
Less than secondary	0.9	0.88	0.93
Secondary education	1.253	1.22	1.29
Some post-secondary education	1.24	1.23	1.24
Post-secondary	1.12	1.10	1.15
No income or less 20 000	1.480.7	1.450.63	1.520.73
$20 000- 39 000	1.30	1.24	1.29
$40 000- 59 000	1.10	1.06	1.11
$60 000- 79 000	0.651.5	0.631.45	0.671.51
$80 000+	0.70	0.676	0.70
Individual level			
Alcohol Consumption (> 2 drinks/day)	0.7	0.7069	0.71
Depression	0.9	0.88	0.91
Hypertension	1.21	1.18	1.24
Diabetes	1.40	1.34	1.38
Fagerstrom Nicotine Dependency	-	-	-

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Table 2a: Correlates of smoking cessation in study cohort contd.

Correlates of smoking cessation	Odds Ratio (OR)	Lower 95% CI	Upper 95% CI
Interpersonal level			
Household smoking restrictions	1.10	1.05	1.08
Vehicle smoking restrictions	2.988	2.9172	3.062.86
Access to GP	1.30	1.234	1.301
Community level			
Public smoking restrictions	-	-	-
Workplace smoking restrictions	-	-	-
Organizational level		-	-
Nicotine replacement therapy (NRT) gum	-	-	-
NRT patch	-	-	-
Zyban/Bupropion	15.524	13.914.03	17.017.16
MD counselling	3.71.52	3.371.39	4.031.67

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DISCUSSION

The aim of this study was to identify the correlates of former smoking in smokers with reported stroke symptoms at multi-levels. Income and older age were predictive of former smoking while higher education ~~at all levels~~ predicted former smoking in this cohort. These results are in line with previous investigations of gender effects, [11, 12, 13] older age [11, 12] and level of income [11, 12, 14] vis a vis smoking and cessation. Our results indicate there are ~~less~~fewer women who are former smokers than men. Reynaso and colleagues outline [13] that there may be several working hypotheses that may explain this result. First, women may respond poorly to nicotine replacement therapy. Second, women are more vulnerable to depression and anxiety symptomatology following cessation. Third, there may be concerns regarding post-cessation weight gain and body-shape concerns for women compared to men. Fourth, women's menstrual cycle effects enhance nicotine withdrawal symptoms following cessation. Fifth, women do not receive or do not respond to the beneficial effects of social support during cessation. [13] They suggest that a varied approach to smoking cessation be taken in light of these hypotheses ~~gender differences between men and women~~. For example it is imperative to consider an approach with lengthier treatments for women following NRT termination. [13] Health professionals should also consider adjunct programs during the cessation attempt for women who have concerns about weight gain. [13] There should be concurrent treatment programs for women who have a history of anxiety/depression. [13] Finally, the timing of smoking cessation interventions early in the follicular phase of the menstrual cycle should be considered. [13]~~.~~

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In regards to income, there seems to be an inverse effect of those who are at the lower levels of SES and former smoking. A recent review by Hiscock and associates (2011) suggested that the higher smoking prevalence in lower SES groups and lower smoking cessation rates might be a result of the clustering of disadvantages. [14] ~~(Hiscock, Bauld, Amos, Fidler, Munafo 2011).~~ These disadvantages include: a reduced social support for quitting, low motivation to quit, increased addiction to tobacco, increased likelihood of not completing courses of pharmacotherapy or behavioural support sessions, psychological differences such as lack of self-efficacy, and susceptibility tobacco industry marketing. [14] As a result, quit attempts in this population are significantly less likely to be successful. [14]

Koning and associates found that each additional year of education reduced the risk of continued smoking.[145] Their data suggested that people with higher education may be able to better understand the consequences of long-term smoking and may have more resources available for them to quit smoking.[145]

Our findings suggest that co-morbid conditions ~~at the individual level~~ such as alcohol consumption and depression significantly decreased the likelihood of former smoking. These findings are supported by evidence suggesting that cerebrovascular patients experience higher rates of co-morbidity particularly depression. [~~15,~~ 16, 17, 18] Compared to cardiac patients, patients with a recent

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stroke suffered a three to five-fold increased risk of depressive disorders [15~~5~~6] as well as higher proportions of alcohol consumption and hypertension. [18~~8~~9]

These findings may have clinical implications particularly for this population, as co-morbid conditions such as depression and increased alcohol consumption are significantly more common in patients who smoke. Considering their association with increased smoking behaviour, co-morbidities may be hindering the success of quitting smoking. The hindering effect of co-morbidity on former smoking is especially problematic as smoking increases blood coagulability, platelet aggregation, thrombus formation and endothelial damage,[~~19~~20] thus increasing the chance of a stroke two-fold [21~~0~~] and of stroke recurrence.[21~~0~~, 22~~1~~] Quitting smoking has been shown to reduce the risk of stroke to that of a non-smoker after 5 years [23~~2~~] and reduce the risk hazard ratio of stroke recurrence from 1.71 to 1.39 ($p < 0.05$).[23~~4~~] It is imperative that smoking cessation be incorporated in secondary prevention practice while taking these significant co-morbidities into account. Depression and excessive alcohol consumption might impede cessation in people with cerebrovascular disease. However due to the limitation of cross-sectional studies, we do not know if these co-morbidities existed before or after the reported stroke. Further study regarding the effects of these co-morbidities on cessation using other study designs might be warranted.

Population based interventions such as household, workplace, vehicle and public smoking restrictions have all been found to predict smoking abstinence.[~~24,~~ 25, 26, 27] They have also been found to reduce cigarette consumption, and initiation and increase smoking cessation rates.[25, 26, 27~~24, 25, 26~~] These authors suggest that population based interventions are anti-tobacco socialization tools that may promote the internalisation of behavioural norms against the initiation or continuation of smoking. Our results are partially

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in line with this evidence. We found that household and vehicle smoking restrictions predicted smoking cessation but not so with workplace or public smoking restrictions. It is not known why workplace and public smoking restrictions did not predict smoking cessation especially since their implementation under the Smoke Free Ontario Act [~~27,~~ 28, 29, 30] in Ontario and similar legislations across Canada. Since their implementation, smoking prevalence in Canada has been dramatically decreased. Perhaps the insignificant effect of public and workplace smoking restrictions may be explained in the decrease of funding in the SFOA in 2007-2008 of 60 million, down 2.5 million from the year before of 62.5 million in 2006-2007. [~~30~~, 31, 32] Similar reductions in tobacco control funding can be observed in other provinces. [~~28,~~ 29, 30] There is a documented association between population interventions effectiveness and sustained funding.[3~~2~~3]

A similar situation was observed with the California Model in the state of California. The California Model is similar to the SFOA and is a population intervention that used workplace and public place smoking restrictions to de-normalize tobacco use.[3~~2~~3] Pierce and associates found that the initial effect of the California Model to decrease smoking prevalence in the state dissipated as their funding was reduced.[3~~2~~3]

In light of this conundrum, there is evidence that suggests that household and vehicle-smoking restrictions are more effective because they are less regulated. [~~33~~, 34, 35] These authors suggest that smoking restrictions such as at home or in a vehicle are effective because those who implement them do so by choice and not through forced legislation [~~32,~~ 33, 34, 35] thereby increasing the odds of smoking cessation.~~.~~

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~~At the organizational level,~~ W~~w~~e found that the use of pharmacotherapy such as bupropion and physician counselling increased the odds of former smoking but NRT use did not. According to Fiore and associates, pharmacotherapy along with counselling and follow-up increases the odds of smoking cessation. [3~~5~~6] NRT and bupropion have each been found to be more efficacious than placebo for increasing the odds of smoking cessation.[3~~6~~7]

The lack of effect of NRT may be indicative of the well-documented practice gap in health care in regards to smoking cessation. Young and Ward found that only 32% of physicians provided written materials for their patients and only 28% of physicians set a "quit date" with their patients.[3~~7~~8] Likewise Shaohua and colleagues found that many family physicians feel lack of time was their biggest barrier in terms of implementing smoking cessation practices.[3~~8~~9] Their study found that less than half were willing or able to assist their patients to quit with the use of counselling, pharmacotherapy or arrange a follow-up visit to reinforce the benefits of smoking cessation.[38] This is consistent with the stroke population as documented by Mouradian and associates.[6] Perhaps another explanation may be the lack of information regarding the effectiveness of smoking cessation medications and similar interventions in stroke and TIA patients. Furthermore, physicians may be reluctant to prescribe NRT's due to their availability over the counter. Further research is required to determine if the latter explanations are supported by evidence.

Cross-sectional surveys such as CCHS are useful for initial exploratory studies. They are far reaching and reflect "a snapshot" of the population. However there are limitations to our study and they will be explored here. Since both exposure and outcome were measured at the same time, one cannot be certain which is the exposure or the outcome. In other words, the rules for contributory cause cannot be fulfilled. For example, it is possible that respondents stopped smoking years before their stroke diagnosis. Owing to the nature

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of the cross-sectional design of the CCHS, there is no way to ascertain which of the two (former smoking vs. stroke diagnosis) came first. Unfortunately there is no available variable that quantifies the time-point of cessation in relation to respondents' stroke diagnosis. Another limitation would be the results that found sex and age as significant correlates of former smoking. Unfortunately, these are unmodifiable correlates. Future interventions should take into account modifiable correlates such as the implementation of household and vehicle smoking restrictions and the availability of pharmacotherapy and counselling support. ~~that these~~ Another limitation is the mode of collection of the data. Social desirability and recall bias for example could play an important role and a source of biases within this study.[~~39~~40] For example, since smoking status, the presence of stroke symptoms and co-morbidities such as depression were self-reported, special care should be taken when interpreting our results. An example of social desirability effect would be respondents not accurately reporting their smoking status. Since smoking would be an undesirable image for some depending on age, gender or socio-economic status, data obtained might not be representative of the real picture found in the population. Ideally all smoking related measures should be validated bio-chemically with breath samples measuring carbon monoxide levels or coitinine levels measuring the amount of nicotine in the blood. Furthermore, without an expert assessment from a health care professional of stroke symptoms or depression would also limit the generalizability of the results.

CONCLUSION

We found significant correlates of former smoking at multiple levels in smokers with reported stroke symptoms. Age and education level were significant correlates of former smoking as well as ~~of former smokingsmoking cessation, at the population and~~

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socio-economic level. At the individual level, Ddepression and alcohol consumption reduced the likelihood of former smoking cessation while at the interpersonal level, hHhousehold and vehicle smoking restrictions and access to a GPGP were found to be significant correlates of former smoking. Public and workplace smoking restrictions were not correlates of former smoking, at the community level.

Finally, at the organization level the use of bupropion along with physician counselling predicted former smoking.

a. Contributorship Statement

Drs. RE, RD, and MS made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data. Dr. LB and Mr. MP assisted in revising the manuscript critically for important intellectual content. All authors provided approval of the version to be published.

b. Competing Interests

None declared

c. Funding

None

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d. Data sharing

No data available

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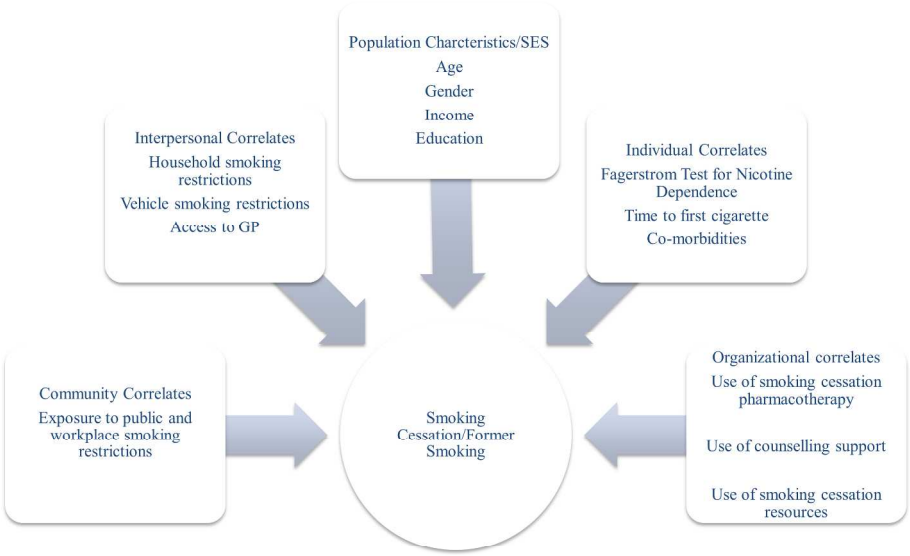
Figure 1: Socio-ecological model for multi-level correlates of former smoking in respondents with cerebrovascular disease



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