Published in final edited form as:

Alcohol Clin Exp Res. 2013 May ; 37(5): 804–810. doi:10.1111/acer.12041.

LONGITUDINAL ASSOCIATIONS BETWEEN SMOKING CESSATION MEDICATIONS AND ALCOHOL CONSUMPTION AMONG SMOKERS IN THE INTERNATIONAL TOBACCO CONTROL FOUR COUNTRY SURVEY

Sherry A. McKee, Ph.D.¹, Kelly C. Young-Wolff, Ph.D., M.P.H.¹, Emily L.R. Harrison, Ph.D.¹, K. Michael Cummings, Ph.D., M.P.H.², Ron Borland, Ph.D.³, Christopher W. Kahler, Ph.D.⁴, Geoffrey T. Fong, Ph.D.⁵, and Andrew Hyland, Ph.D.²

¹Department of Psychiatry, Yale University School of Medicine, 2 Church Street South, Suite 109, New Haven, CT 06519 USA

²Department of Heath Behavior, Roswell Park Cancer Institute, Elm & Carlton Streets, Buffalo, NY 14263 USA

³Victoria Health Center for Tobacco Control, The Cancer Council Victoria, 1 Rathdowne St, Carlton VIC 3053, Australia

⁴Center for Alcohol and Addiction Studies, Brown University Box G-S121-5, Providence, RI 02912, USA

⁵Department of Psychology, University of Waterloo, Waterloo, 200 University Avenue West, Waterloo, Ontario, Canada N2L 3G1 and Ontario Institute for Cancer Research, Toronto, Ontario, Canada M5G 0A3

Abstract

Background—Available evidence suggests that quitting smoking does not alter alcohol consumption. However, smoking cessation medications may have a direct impact on alcohol consumption independent of any effects on smoking cessation. Using an international longitudinal epidemiological sample of smokers, we evaluated whether smoking cessation medications altered alcohol consumption independent of quitting smoking.

Methods—Longitudinal data were analyzed from the International Tobacco Control Four Country Survey (ITC-4) between 2007 and 2008, a telephone survey of nationally representative samples of smokers from the United Kingdom, Australia, Canada, and the United States (n=4,995). Quantity and frequency of alcohol consumption, use of smoking cessation medications (varenicline, nicotine replacement (NRT), no medications), and smoking behavior were assessed across two yearly waves. Controlling for baseline drinking and changes in smoking status, we evaluated whether smoking cessation medications were associated with reduced alcohol consumption.

Results—Varenicline was associated with a reduced likelihood of any drinking compared to nicotine replacement (O.R. = 0.56; 95% CI = 0.34–0.94), and consuming alcohol once a month or

CORRESPONDING AUTHOR: Sherry A. McKee, Ph.D., Department of Psychiatry, Yale University School of Medicine, 2 Church St. South, Suite 109, New Haven, CT, USA, 06519. Phone: (203) 737-3529, Fax (203) 737-4243, sherry.mckee@yale.edu.

Declarations of interest

SM received an investigator initiated grant from Pfizer in 2009 to study varenicline's interactions with alcohol. KYW, EH, MC, CK, RB, GF and AH have no conflicts to report.

more compared to nicotine replacement (O.R. = 0.43; 95% CI = 0.27–0.69) or no medication (O.R. = 0.63; 95% CI = 0.41–0.99). Nicotine replacement was associated with an increased likelihood of consuming alcohol once a month or more compared to no medication (O.R. = 1.14; 95% CI = 1.03–1.25). Smoking cessation medications were not associated with more frequent drinking (once a week or more) or typical quantity consumed per episode. Medication effects on drinking frequency were independent of smoking cessation.

Conclusions—This epidemiological investigation demonstrated that varenicline was associated with a reduced frequency of alcohol consumption. Continued work should clarify under what conditions nicotine replacement therapies may increase or decrease patterns of alcohol consumption.

Keywords

alcohol; varenicline; nicotine replacement; epidemiological; longitudinal; smoking cessation

INTRODUCTION

Alcohol consumption and tobacco use are highly comorbid (Grant et al., 2004), interact at electrophysiological, pharmacological, genetic, and neurochemical levels (Dani and Harris, 2005; Davis and de Fiebre, 2006; Gonzales and Weiss, 1998; Larsson et al., 2004; Söderpalm et al., 2000), and are associated to such a degree that smoking can be used as a clinical indicator for alcohol use disorders (McKee et al., 2007). The comorbidity between alcohol consumption and smoking is of particular concern given evidence that heavy alcohol consumption is associated with reduced odds of successful smoking cessation in longitudinal community studies (Augustson et al., 2008; Dollar et al., 2009; Kahler et al., 2009), and greater risk for smoking cessation failure in clinical trial and laboratory studies (Humfleet et al., 1999; McKee et al., 2006).

Although heavier alcohol consumption predicts a decreased likelihood of smoking cessation, most studies find that quitting smoking does not alter alcohol use (Gordon and Doyle, 1986; Kahler et al., 2010; Nothwehr et al., 1995). For example, smoking cessation was not associated with changes in alcohol consumption over time in an 18-year longitudinal study of men (Gordon and Doyle, 1986). More recently, a prospective cohort study of smokers in Australia, Canada, the UK and the US found that individuals who achieved sustained smoking cessation were no more likely to reduce their alcohol consumption than individuals who continued to smoke (Kahler et al., 2010). Therefore, it has been suggested that treatments that target smoking cessation are unlikely to lead to meaningful changes in drinking unless they include components that directly impact alcohol consumption (Kahler et al., 2010).

Currently approved smoking cessation medications (nicotine replacement, varenicline, and bupropion) all target the nicotinic acetylcholine receptors (nAChRs) to some extent. Reinforcing effects of alcohol are thought to be mediated, in part, by dopamine release in the nucleus accumbens (NA) and the ventral tegmental area (VTA) (Gonzales and Weiss, 1998; Middaugh et al., 2003). It has been hypothesized that alcohol may produce mesolimbic activation, at least in part by its effects on central nAChRs (Blomqvist et al., 1996; Ericson et al., 1998; Soderpalm et al., 2000). Thus, smoking cessation medications may have a direct impact on alcohol consumption independent of any effects on tobacco cessation.

Findings from studies that have investigated the effects of nicotine on alcohol intake and reactivity are mixed. Results from preclinical studies indicate that acute nicotine administration decreases alcohol self-administration behavior (Katner et al., 1997; Nadel et al., 1998), whereas chronic nicotine administration increases alcohol intake in alcohol-

experienced rats (Blomqvist et al., 1996; Clark et al., 2001; Smith et al., 1999; Soderpalm et al., 2000). Additionally, nicotine has been shown to facilitate the acquisition of alcohol self-administration (Smith et al., 1999) and reinstate alcohol seeking behavior (Lê et al., 2003). Human laboratory studies have found that nicotine replacement reduced the sedating and intoxicating effects of alcohol in light to moderate drinking daily smokers (Perkins et al., 1995), and conversely, increased subjective intoxication and alcohol and tobacco craving in a sample of moderate to heavy-drinking daily-smoking males (Kouri et al., 2004).

Studies of the effect of nicotine on human alcohol self-administration have also produced mixed findings. Acheson and colleagues (2006) examined the effect of transdermal nicotine replacement on responses to an initial priming drink and subsequent alcohol self-administration behavior in light-smoking social drinkers. Nicotine replacement was not associated with heart rate or ratings of desire for an additional drink following the priming drink, but was found to subsequently increase alcohol consumption in men and decrease alcohol consumption in women. McKee et al. (2008) examined the effect of transdermal nicotine replacement on response to a priming drink and subsequent alcohol self-administration in a sample of daily-smoking heavy drinkers. Nicotine replacement attenuated subjective and physiological responses to alcohol and delayed the initiation of drinking (McKee et al., 2008).

Varenicline, a partial nicotinic agonist, is an effective smoking cessation pharmacotherapy (Cahill et al., 2011) and shows some promise as a potential intervention for alcohol consumption. Varenicline is associated with reduced ethanol seeking (Steensland et al., 2007), decreased ethanol self-administration (Kamens et al., 2010; Steensland et al., 2007), and attenuated dopamine response to alcohol and nicotine co-administration (Ericson et al., 2009). In heavy drinking smokers, varenicline has been shown to reduce alcohol craving (McKee et al., 2009), decrease alcohol consumption (Fucito et al., 2011; McKee et al., 2009; Mitchell et al., 2012), and potentiate the aversive effects associated with alcohol consumption (Childs et al., 2012).

Taken together, evidence indicates that both nicotine replacement and varenicline may alter alcohol consumption. Such results highlight the potential importance of examining the impact of smoking cessation medications on alcohol consumption. Available findings were obtained in primarily heavy drinking samples, and it remains to be determined whether smoking cessation medications alter drinking in an epidemiological sample of smokers. We used longitudinal data from the International Tobacco Control Four Country Survey (ITC-4), a telephone survey of nationally representative samples of smokers in the United Kingdom, Australia, Canada, and the United States to: a) examine the effect of tobacco cessation medications on alcohol consumption among smokers who used either varenicline, nicotine replacement, or no smoking cessation medications in the preceding year, and b) evaluate whether the effect of smoking cessation medications on alcohol consumption was independent of any effects on smoking cessation. Based on available data, we hypothesized that varenicline would demonstrate reductions in the frequency and quantity of alcohol consumption independent of any effects on smoking cessation. Findings with respect to the effects of nicotine replacement therapy on alcohol consumption are mixed and therefore we make no predictions about the direction of effect for these tobacco cessation medications on alcohol consumption.

MATERIALS AND METHODS

Data source

The data used in this analysis come from Waves 6 and 7 of the International Tobacco Control Four Country Survey (ITC-4). The ITC-4 Survey is a prospective cohort study

designed to evaluate the psychosocial and behavioral impact of key national-level tobacco control policies enacted in the United Kingdom, Australia, Canada, and the United States. Data were collected through random digit-dialed telephone surveys of adult smokers (aged 18 years and older). Wave 6 was conducted between September 2007 and January 2008, and Wave 7 was conducted between October 2008 and June 2009 (retention rate = 75%). A complete description of survey and data management procedures for the larger study have been published elsewhere (Fong et al., 2006; Thompson et al., 2006).

The sample for the analysis included Wave 6 current smokers (total $n=4,995$; $n=1,342$ Australia, $n=1,281$ Canada, $n=1,191$ United Kingdom, $n=1,181$ United States) who participated in both Waves 6 and 7. The sample was primarily White (88.9%), evenly divided across gender (male, 52.9%) and education level (low education=49.2%), with a mean age of 44.5 (SD=18.7) years. The majority (76.4%) of the sample smoked 20 or fewer cigarettes per day, and consumed alcohol in the past year at the baseline wave (Wave 6=81.4%; see Table 1).

Measures

Drinking behavior—Frequency and quantity of alcohol consumption were assessed at each wave using items recommended by the National Institute on Alcohol Abuse and Alcoholism's (NIAAA) Task Force on Recommended Alcohol Questions (NIAAA, 2003). For drinking frequency, individuals were asked, “*During the last 12 months about how often did you have any kind of drink that contained alcohol?*” For each wave, responses were coded in 3 ordinal categories of drinking frequency, from any drinking in past year (yes/no), drinking once a month or more (yes/no), to drinking once a week or more (yes/no). For drinking quantity, individuals were asked, “*On a typical day when you did drink alcohol, how many alcoholic drinks did you have?*”. The definitions of a standard drink were equated for alcohol content across countries. In Canada and the United States a standard drink is equivalent to 0.6 oz of pure alcohol which is equivalent to approximately 2 alcohol units in the United Kingdom and Australia (e.g., 5 oz wine or 12 oz can of beer (CA & US); 5oz / 150 mL wine or 13 oz can of beer (UK); 150 mL of wine or a 375 mL can or stubby of beer (AU)). For each wave, responses were coded in ordinal categories, from at least 1 drink per episode (yes/no), at least 3 drinks per episode (yes/no), to 5 or more drinks per episode (yes/no).

Medication groups—For the primary independent variable, participants were categorized as to whether they had used smoking cessation medications (varenicline, nicotine replacement, no medication) in the past year. At Wave 7, respondents were asked three questions about their past year use of smoking cessation medications: 1) “*The last time you used medications to quit smoking, which product or combination of products did you use? This includes both NRTs and prescription medications.*”; 2) “*Have you used any other stop-smoking medications in order to quit or stay quit?*”; 3) “*Which product or combination of products did you use to cut down on the amount you smoke, to cope with non-smoking situations, and/or for reasons other than quitting? This includes both NRTs and prescription medications.*” Past analyses of ITC data has shown that a significant proportion of smokers use smoking cessation medications for reasons other than quitting (Hammond et al., 2008). Thus, we elected to include individuals who used smoking cessation medications, regardless of the reason. Respondents were sorted into mutually exclusive groups and dual users were excluded.

The Varenicline group ($n=291$) consisted of respondents who reported using Chantix/Champix in response to at least one of the questions about their use of smoking cessation medications in the past year. The Nicotine Replacement Therapy (NRT) group ($n=751$)

consisted of those respondents who reported using NRT (patch, gum, lozenge, and/or tablets). The No Medication group (n=3,953) consisted of those respondents who reported that they had not used any medications for smoking cessation in the past year. A small number of respondents (n=75) reported using Zyban (bupropion) in the past year. Due to the small sample, this group was excluded from further analysis.

Control variables—In addition to the demographic variables included in Table 1, heaviness of smoking and whether respondents quit smoking were evaluated as potential covariates when examining the effect of medication use on drinking behavior.

Heaviness of Smoking Index (HSI)—Heaviness of Smoking Index, an established measure of smoking dependence (Heatherton et al., 1989) was a composite variable of cigarettes per day (cpd) and time to first cigarette of the day that was calculated for Wave 6. Scores ranged from 0 to 6, with higher scores indicating greater dependence. In this sample we categorized those with a high HSI (scores 3–6) and those with a low HSI (0–2).

Quit smoking—At Wave 7, if respondents reported that they had quit smoking in the past year (*Have you made any attempts to stop smoking since we last talked with you?*) and reported that they don't smoke at all now (*Have you had any cigarettes, even a puff, since you quit smoking?*) or smoke less than monthly (*Do you currently smoke daily, weekly, or monthly?*), there were coded as having quit smoking. Otherwise, they were coded as not quit (i.e., current smokers).

Statistical analysis

Analyses were conducted with Stata version 11 with survey (svy) commands to account for the complex sampling design of the ITC-4 Survey. All analyses were weighted for the cross-sectional design and non-response. Chi-square analyses were conducted to compare demographic variables across smoking cessation medication groups (Varenicline, NRT, No Medication) and between medication status and measures of frequency of alcohol drinking. Logistic regression analyses were conducted to assess bivariate relationships between drinking frequency (any drinking, drinking 1/day month or more, drinking 1/day week or more) and quantity (3+ drinks, 5+ drinks) variables with medication status at Wave 7. For all analyses, covariates included alcohol- and smoking-related variables, and demographic variables that were significantly associated with medication group (Wave 6 drinking frequency and quantity, quit smoking, Heaviness of Smoking Index, gender, and race). Two general models were analyzed. In the first model, the No Medication group was the reference group against which the Varenicline and NRT groups were compared. In the second model, the NRT group was the reference group against which the Varenicline and No Medication groups were compared. We also investigated country-specific effects. Although rates of varenicline use were lower in the UK (7.6%, Canada 28.5%, US 36.4%, Australia 27.5%), the primary findings were similar across countries; hence, aggregated data are reported.

RESULTS

Demographic variables are presented in Table 1. Baseline differences in gender and race were found across the medication groups. The Varenicline group was more likely to be female and the No Medication group was more likely to include Non-Whites. Quitting smoking was greater in the Varenicline group, than in the NRT or No Medication groups. At Wave 6, those reporting drinking more than 1× per week or drinking 3 or more drinks per episode were less likely to have used Varenicline compared to NRT or No Medication.

Table 2 presents results examining the increasing ordinal categories of past year drinking frequency by medication groups. Smokers who used Varenicline compared to NRT were less likely to engage in any drinking and less likely to drink once a month or more. Smokers who used Varenicline compared to No Medication were also less likely to drink once a month or more. However, smokers who received NRT compared to No Medication were more likely to drink once a month or more. There were no significant medication effects demonstrated for drinking once a week or more. There were no significant effects of medication associated with typical drinking quantity.

Although analyses were adjusted for Wave 6 drinking, quitting smoking, and significant demographic variables, we conducted additional analyses to ensure medication effects were not associated with quitting smoking or attempting to quit smoking. When analyses were unadjusted for quitting smoking, there was no discernable change to the findings. For drinking once a month or more, results comparing Varenicline to NRT (O.R. = 0.42; 95% CI 0.26–0.68), Varenicline to No Medication (O.R. = 0.61; 95% CI 0.40–0.93), and NRT to No Medication (O.R. = 1.13; 95% CI 1.06–1.25) were not substantively altered. Further, when quitting smoking was evaluated as an interaction term with medication group in the logistic regression, it was not a significant predictor of alcohol use. We also evaluated whether engaging in a quit attempt was a significant independent variable in the regression models. There were no significant main or interactive effects associated with engaging in a quit attempt.

DISCUSSION

To our knowledge, this is the first investigation to evaluate the effect of varenicline and nicotine replacement on alcohol use in an international longitudinal, epidemiological sample of smokers. Our primary hypothesis that use of varenicline would be associated with reductions in alcohol consumption was supported. Varenicline was associated with a reduced likelihood of any drinking and a reduced likelihood of drinking at least once a month or more when compared to smokers who used nicotine replacement products. Compared to smokers who did not use any medications, smokers who used varenicline reported lower frequency of drinking at least once a month or more. Importantly, the associations between varenicline and lower alcohol consumption were independent of quitting smoking, even though varenicline increased rates of smoking abstinence. That is, the reduction of alcohol use among varenicline users was the same among individuals who had quit smoking and among those who continued to smoke. Other epidemiological investigations have failed to document reductions in alcohol consumption following smoking cessation (Kahler et al., 2010; Nothwehr et al., 1995), and our findings suggest that the observed reductions in alcohol use in the current study were attributable to the use of varenicline and not to smoking cessation.

Varenicline use was not associated with more frequent drinking (once a week or more). However, at the baseline wave, weekly drinkers were less likely to use varenicline which may reflect prescription patterns. It is possible that weekly drinkers were less likely to seek out a prescription medication for smoking cessation. Moreover, doctors may be less likely to prescribe varenicline to these drinkers out of concern of adverse events (Huang et al., 2012). While regulatory bodies advise about the potential neuropsychological events associated with varenicline, large research initiatives have yet to document such adverse outcomes (Gunnell et al., 2009; Kasliwal et al., 2009; Tonstad et al., 2010). In our own work (McKee et al., 2009), we have found that varenicline, combined with alcohol, was well tolerated in a small sample of heavy drinkers.

Our laboratory studies have demonstrated that the effect of varenicline on reducing alcohol consumption was due to an increased likelihood of complete abstinence during the self-administration period, rather than reductions in the number of drinks during an episode of drinking (McKee et al., 2009). This pattern was replicated in our epidemiological sample of current smokers in which varenicline was associated with a reduction in reported frequency of consumption, but if drinking commenced, the reported quantity consumed was not altered. Based on these findings, we speculate that varenicline may reduce drinking in a manner somewhat similar to how it is hypothesized to work for smoking. Varenicline acts as a partial agonist at $\alpha 4\beta 2$ receptors stimulating adequate levels of dopamine release which may prevent alcohol craving and reduce the frequency of consumption (McKee et al., 2009). However, varenicline use was not associated with reduced consumption per episode suggesting that the competitive antagonist effects demonstrated in smoking may not occur for alcohol use. Consistent with this hypothesis, co-administration of alcohol and varenicline did not attenuate extracellular accumbal dopamine levels, when compared to alcohol alone (Ericson et al., 1998).

The effects of nicotine replacement therapies on alcohol consumption have been mixed. Results from the current study indicated that nicotine replacement was associated with greater alcohol consumption, consistent with Acheson et al. (2006) findings for light-smoking social drinking men. However, our results are inconsistent with previous findings that showed that nicotine replacement therapy reduced alcohol consumption among light-smoking social drinking women (Acheson et al., 2006) and among daily-smoking heavy drinkers (McKee et al., 2008). Methodological differences across studies may account for inconsistent results, as the effects of nicotine on alcohol consumption are thought to vary with gender, dose of alcohol, type and dose of nicotine replacement, and degree of experience with alcohol and nicotine (McKee et al., 2008). Taken together, our results further highlight the complex association between nicotine replacement and alcohol consumption and point to the need for additional research that examines how nicotine replacement therapies alter alcohol consumption and further delineates the factors that moderate this association.

An important strength of the study is the use of the large-scale international longitudinal sample to address the study aims, where we obtained consistent findings across countries. Limitations include the loss to follow-up, although the findings from the cohort analysis should be internally valid. The data are self-report and may not accurately reflect some measures, although any bias in alcohol consumption reporting is likely to be uniform across waves. Additionally, our measures of past-year alcohol consumption and heaviness of smoking do not reflect fluctuations in these behaviors over the course of the year. It should also be noted that expectancy effects of smoking cessation medications on self-reported alcohol use were unlikely given that the public is unaware of the possible linkage and the survey questions about cessation medications and alcohol use were separated by many sections. Another important limitation is that the length of medication use, timing of use, and dose were unknown; however, findings regarding medication effects on smoking cessation are consistent with the clinical literature. Thus we would expect use to be around 12 weeks, making it likely that most of the sample would have either not currently be using or to have recently stopped medication use. Clarifying factors pertaining to the timing of alcohol consumption and medication use would likely remove error variance and would serve to strengthen relationships between smoking cessation medications and alcohol use. In particular, knowledge of such factors would allow us to test if the observed effect was primarily associated with concurrent varenicline use, and, if there is a persistence effect, how long it lasts.

In summary, the use of varenicline was associated with a reduced overall frequency, but not overall quantity, of past-year alcohol consumption in an epidemiological sample of smokers, and these effects were independent of actual or attempted smoking cessation. This suggests that individuals in the community who are prescribed varenicline for smoking cessation may also experience concomitant reductions in alcohol consumption, yielding an unexpected benefit to this treatment. Thus, our study has clinical implications for the use of varenicline for smoking cessation among smokers who also drink, an important and substantial group of smokers for whom the relative risk of morbidity and mortality is increased (Blot et al., 1988; Gruzza and Beirut, 2007; Klatsky and Armstrong, 1992; Marrero et al., 2005; Pelucchi et al., 2007; Rosengren et al., 1988; Vallant et al., 1991). Supporting clinical trial and laboratory findings (Childs et al., 2012; Fucito et al., 2011; McKee et al., 2009; Mitchell et al., 2012), these epidemiological data also suggest that varenicline inhibits alcohol consumption among smokers and should be further evaluated as a potential therapeutic to reduce alcohol consumption. Continued work should clarify under what conditions nicotine replacement therapies may increase or decrease patterns of alcohol consumption.

Acknowledgments

Sources of support

This work was supported by NIH grants R01AA015596 (McKee), R01AA017976 (McKee), CTSA-UL1RR024139, T32AA015496. The ITC Four Country Survey is supported by grants from the U.S. National Cancer Institute (R01 CA100362), and (P50 CA111236), the Canadian Institutes of Health Research (79551), the National Health and Medical Research Council of Australia (450110), Cancer Research UK (C312/A6465), and the Ontario Institute for Cancer Research (Senior Investigator Award).

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Table 1

Distribution of Demographic Characteristics and Alcohol Drinking Frequencies, n=4,995 from Waves 6 (2007) and Wave 7 (2008) of the International Tobacco Control Four Country Survey

Variables	All n=4,995	All %	Varenicline ^d (n=291, 5.1%)		NRT ^b (n=751, 15.3%)		No Medication (n=3,953, 79.6%)	
			n	%	n	%	n	%
Gender**								
Male	2,130	52.9	96	39.0	295	47.4	1,739	54.9
Female	2,865	47.1	195	61.0	456	52.6	2,214	45.1
Race/Ethnicity*								
White	4,531	88.9	270	91.8	699	92.2	3,562	88.1
Non-White	458	11.1	20	8.2	51	7.8	387	11.9
Age at Wave 6 (mean, SD)	44.5, 18.7		47.3, 18.2		44.0, 17.8		44.4, 18.9	
Education Level Wave 6								
Low	2,550	49.2	127	41.2	368	48.7	2,055	49.8
Medium or higher	2,432	50.8	164	58.8	381	51.3	1,887	50.2
Income Wave 6								
Moderate or lower	3,084	66.8	190	64.2	449	63.5	2,445	67.6
High	1,568	33.2	87	35.8	245	36.5	1,236	32.4
Cigarettes per Day Wave 6								
1–20 cigarettes per day	3,283	76.4	201	81.3	485	74.6	2,597	76.4
+20 cigarettes per day	1,021	23.6	53	18.7	162	25.4	806	23.6
Heaviness of Smoking Index Wave 6^c								
Low (0–2)	1,742	40.3	98	42.1	272	40.5	1,372	40.1
High (3–6)	2,544	59.7	155	57.9	372	59.5	2,017	59.9
Quit Smoking Wave 7**	537	11.1	78	27.1	133	17.4	326	8.9
Wave 6 Alcohol Drinking Frequency								
Any drinking (yes/no)	3,905	81.4	224	81.1	585	80.7	3,096	81.6
Drink 1 day/month or more (yes/no)	3,013	63.8	164	60.1	451	64.0	2,398	64.0
Drink 1 day/week or more (yes/no)*	2,310	48.9	116	41.1	352	49.5	1,842	49.3

Variables	All n=4,995	All %	Varenicline ^d (n=291, 5.1%)		NRT ^b (n=751, 15.3%)		No Medication (n=3,953, 79.6%)	
			n	%	n	%	n	%
Quantity per episode								
1+ drinks (yes/no)	3,879	81.1	223	81.1	581	80.2	3,075	81.3
3+ drinks (yes/no) *	2,062	47.2	97	36.9	329	49.5	1,636	47.4
5+ drinks (yes/no)	910	22.5	40	18.4	125	21.5	745	23.0
Wave 7 Alcohol Drinking								
Frequency								
Any drinking (yes/no)	3,768	79.7	208	76.1	579	80.9	2,981	79.6
Drink 1 day/month or more (yes/no)	2,808	61.2	139	52.0	443	64.4	2,226	61.1
Drink 1 day/week or more (yes/no)	2,115	45.3	109	43.9	335	47.6	1,671	44.9
Quantity per episode								
1+ drinks (yes/no)	3,741	79.5	206	75.9	574	80.7	2,961	79.5
3+ drinks (yes/no) *	1,961	46.4	96	38.3	296	47.7	1,569	46.6
5+ drinks (yes/no)	883	23.1	45	17.8	123	22.9	715	23.5

n (unweighted) % (weighted);

* $p < .05$;

** $p < .01$;

^a Medication use was assessed at Wave 7 and retrospectively reported on for the past year;

^b NRT = Nicotine Replacement Therapy;

^c Heaviness of Smoking Index is a composite score of cigarettes per day and time to first cigarette. Scores range 0–6, with higher scores indicating heavier smoking

Table 2

Odds Ratios for Drinking Frequencies by Smoking Cessation Medication Groups n=4,995 from Waves 6 (2007) and Wave 7 (2008) of the International Tobacco Control Four Country Survey

Wave 7 Past Year Drinking Frequency	Odds Ratios [^] (95% Confidence Interval)		
	Varenicline ^a vs. NRT (ref)	Varenicline vs. No Medication (ref)	NRT vs. No Medication (ref)
Any drinking (yes/no)	0.56 (0.34–0.94) *	0.74 (0.47–1.15)	1.10 (0.98–1.22)
Drinks 1× month or more (yes/no)	0.43 (0.27–0.69) **	0.63 (0.41–0.99) *	1.14 (1.03–1.25) *
Drinks 1×week or more (yes/no)	0.94 (0.55–1.61)	1.21 (0.74–1.96)	1.09 (0.98–1.21)

[^] Adjusted for the following variables: Wave 6 drinking, Wave 7 quit smoking, gender, race, and Wave 6 Heaviness of Smoking Index;

^a Medication use was assessed at Wave 7 and retrospectively reported on for the past year.

* $p < .05$;

** $p < .01$