Published in final edited form as:

Epidemiology. 2015 March ; 26(2): 143–150. doi:10.1097/EDE.0000000000000227.

Risk of Myocardial Infarction Immediately After Alcohol Consumption

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Abstract

Background—Habitual moderate alcohol consumption is associated with a lower risk of acute myocardial infarction (MI) whereas heavy (binge) drinking is associated with higher cardiovascular risk. However, less is known about the immediate effects of alcohol consumption on the risk of acute MI and whether any association differs by beverage type or usual drinking patterns.

Methods—We conducted a case-crossover analysis of 3,869 participants from the Determinants of Myocardial Infarction Onset Study who were interviewed during hospitalization for acute MI in one of 64 medical centers across the United States in 1989–1996. We compared the observed number of times that each participant consumed wine, beer or liquor in the hour preceding MI symptom onset with the expected frequency based on each participant's control information, defined as the number of times the participant consumed alcohol in the past year.

Results—Among 3869 participants, 2119 (55%) reported alcohol consumption in the past year, including 76 within 1 hour before acute MI onset. The incidence rate of acute MI onset was elevated 1.72-fold (95% confidence interval [CI]=1.37–2.16) within 1 hour after alcohol consumption. The association was stronger for liquor than for beer or wine. The higher rate was not apparent for daily drinkers. For the 24 hours after consumption, there was a 14% lower rate

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Conflict of Interest Disclosures: None

(relative risk=0.86 [95% CI=0.79–0.95]) of MI compared with periods with no alcohol consumption.

Conclusions—Alcohol consumption is associated with an acutely higher risk of MI in the subsequent hour among people who do not typically drink alcohol daily.

There is consistent evidence that moderate habitual alcohol consumption is associated with a lower risk of cardiovascular events in subsequent months and years¹ and that heavy episodic (binge) drinking is associated with higher cardiovascular risk. In terms of the short-term consequences, previous studies have reported that alcohol is an acute trigger of sudden cardiac death² and ischemic^{3–7} and hemorrhagic^{8,9} stroke; there are a few discrepant studies on the short-term risk of acute myocardial infarction (MI) in the 12 to 24 hours after intake^{10–12}. However, experimental studies suggest that within 1–2 hours, moderate and high alcohol intake is associated with adverse consequences, including increased blood pressure and heart rate and impaired fibrinolysis.¹³ None of the prior studies on alcohol as a trigger of acute MI has examined this short period of potentially heightened risk. Furthermore, these studies did not examine whether the association differs by beverage type.

Therefore, we evaluated whether consumption of beer, wine or liquor is associated with a higher risk of acute MI in the following hour and the following 24 hours. In addition, we examined whether usual drinking patterns modify the association – much like regular physical activity blunts the acutely higher risk associated with episodes of physical activity.¹⁴

Methods

Study Design

We used the case-crossover design, a design that is appropriate when a transient exposure (alcohol consumption) is associated with an abrupt change in the risk of an acute outcome (nonfatal acute MI).¹⁵ We compared a participant's report of alcohol consumption in the hours prior to MI onset (the case period) with the same participant's report of alcohol consumption in the prior year (the control period). Because control information for each subject is based on his or her own past exposure experience, self-matching eliminates confounding by risk factors that are constant within individuals over the sampling period but that often differ between study subjects.

Study Population

The Determinants of Myocardial Infarction Onset Study was a multicenter cohort study of participants with confirmed acute MI. In the first phase of the study (August 1989–September 1994), 1,937 participants were enrolled in 45 community hospitals and tertiary care medical centers in the United States. In the second phase (October 1994–September 1996), the study was expanded to 64 medical centers across the United States and an additional 1,949 participants were enrolled. Altogether, 3,886 participants (2,627 men and 1,259 women; mean age, 61.4 years) were interviewed a median of 4 days (range=0 to 30) after sustaining an acute MI. For inclusion in the study, the participants were required to meet all the following criteria: English-speaking, creatine kinase level greater than the upper

limit of normal for the clinical laboratory performing the test, positive MB isoenzymes, an identifiable onset of pain or other symptoms typical of infarction, and the ability to complete a structured interview. The institutional review board at each participating center approved the protocol, and each participant provided informed consent.

As previously described,¹⁴ detailed chart reviews and participant interviews were conducted by research personnel trained by personal instruction, a training manual and instructional videocassette, and through ongoing feedback the study coordinator. Approximately one third of the interviews were audiotaped for randomly selected quality control of the coding accuracy. To minimize bias in information ascertainment, the interviewers were not told of the duration of the hypothesized hazard. Data were collected on standard demographic variables and risk factors for coronary artery disease. We identified the time, place and quality of the acute MI pain and other symptoms. If the participants reported that they had experienced any chest pain in the week preceding the acute onset, they were classified as having premonitory symptoms. We asked about the timing of their last exposure to several potential triggers and estimated usual frequency of these factors in the year preceding MI onset. For each type of alcoholic beverage (beer, wine and liquor), we asked open-ended questions about usual frequency of intake over the past year, number of servings consumed at each episode and the last time a drink was consumed, and their usual weekly pattern of total alcohol intake (e.g. every Thursday). In addition, we asked participants about alcohol and food intake during each hour in the 36 hours before symptom onset.

Validity and Reliability of the Questionnaire

In a study of 851 patients evaluated in an emergency department,¹⁶ the Kappa coefficient between self-reported recent alcohol consumption and blood alcohol concentration was 0.75 (95% confidence interval [CI]=0.68–0.81), suggesting that there was good agreement between self-report and blood alcohol concentration. In a subset of 115 participants in the current study whose HDL-C levels were measured during hospitalization, the correlation between self-reported alcohol consumption and HDL-C levels (Pearson $r=0.21$)¹⁷ was similar to the degree found in the Second National Health and Nutrition Examination Survey.¹⁸ In a validation study comparing self-reported alcohol consumption over the prior year to intake from two 1-week dietary records collected ~6 months apart, the Spearman correlation coefficient was 0.90 for women and 0.86 for men.¹⁹ There is also high test-retest reliability for self-reported alcohol consumption. In a study of alcohol consumption and ischemic stroke⁷ that used similar questions about usual and recent alcohol consumption, 25 participants were re-interviewed up to 6 days after their initial interview. There was perfect agreement for reporting alcohol consumption in the hours before stroke onset ($K=1.0$), and the intraclass correlation for usual frequency of alcohol intake was excellent (0.84).

Statistical Analysis

We applied standard methods for stratified data analysis, where the stratifying variable is the individual participant. For the primary analysis, we calculated the Mantel-Haenszel rate ratio²⁰ as a measure of relative risk (RR) to compare the ratio of the observed exposure frequency in the hazard period to the expected frequency based on control information about alcohol consumption in the previous year. In order to calculate the expected frequency of

exposure in the case period, we multiplied the usual annual frequency of alcohol consumption by the hypothesized window of its physiologic effect (1 hour in the primary analysis) to estimate the amount of person-time exposed to alcohol; the unexposed person-time was calculated by subtracting this value from the number of hours in a year. Person-times for all alcohol use were calculated by summing the number of episodes of beer, wine and liquor. The data were analyzed using methods for cohort studies with sparse data in each stratum.²⁰ The Mantel-Haenszel formula is

$$RR_{MH} = \frac{\sum_i \frac{A_{1i}T_{0i}}{T_i}}{\sum_i \frac{A_{0i}T_{1i}}{T_i}}$$

Where A_{1i} and A_{0i} reflect whether the case consumed alcohol during the hazard period, T_{1i} is the number of hours (person-time) exposed to alcohol, T_{0i} is the number of hours (person-time) unexposed to alcohol and T_i is the total person-time for the individual (i.e., 1 year or 8766 hours).

To estimate the length of time from alcohol consumption to MI onset, we calculated RRs for various hypothesized windows of its physiologic effect. We conducted stratified analyses to assess whether the association differed by drink type (beer, wine, liquor), drinking patterns (every day of the week vs. any other level of drinking frequency), sex, age (<65 vs. ≥65 years), smoking status (current vs. never/former), weekly physical activity (<3 vs. ≥3 times per week), prior coronary artery disease defined as history of acute MI or angina pectoris and jointly by beverage type and smoking status. We compared the RRs by means of a Wald test for homogeneity.²⁰ We examined whether the RR was higher among people who usually consumed more drinks per episode by estimating the RR for 1 to 2, 3 and >3 drinks per episode, and we tested for a linear component of trend using a chi-square test for linear trend.²⁰

In order to address potential confounding by time of day and inaccurate recall of usual alcohol consumption, we used information on consumption in each of the 36 hours prior to MI onset to conduct a sensitivity analysis using conditional logistic regression to compare each person's exposure during the hazard period with their exposure at the same time on the day before MI onset. Since alcohol absorption is slower when it is consumed with food, we conducted a sensitivity analysis accounting for the fact that some participants consumed alcohol with meals. Since some individuals may consume or abstain from alcohol in response to premonitory symptoms, we conducted a sensitivity analysis restricted to participants who reported no symptoms in the week prior to MI onset. We also conducted a sensitivity analysis restricted to participants interviewed within 1 week of MI onset. All reported p values are 2-sided. In another sensitivity analysis, we excluded participants reporting other potential triggers (anger, physical activity, lifting ≥50 pounds, sexual activity, caffeine, tobacco and illicit drugs) in the proposed case period.

Results

Among 3,886 participants, we excluded 16 participants with a history of alcohol abuse who subsequently abstained from alcohol consumption and 1 participant who did not have information on regular alcohol consumption, leaving 3,869 participants for these analyses. 2,119 (55%) reported alcohol consumption in the previous year. Table 1 summarizes the characteristics of the study population. Compared with nondrinkers, participants who reported alcohol consumption were more likely to be male and to have ever smoked cigarettes. Among those participants who drank alcohol in the prior year, 367 (17%) reported consumption fewer than one time per month, 452 (21%) reported consumption more than once per month, 786 (37%) reported consumption more than once per week, and 514 (24%) reported consumption at least daily. The median number of drinks per drinking episode was 1 serving. Among those who drank alcohol in the prior year, 552 (26%) reported drinking 3 or more drinks within 1–2 hours at least once in the prior year.

Acute MI Incidence within 1 Hour after Consumption

Of the 2,119 participants who drank alcohol in the past year, 76 (3.6%) reported consumption in the hour before acute MI onset. The rate of MI was 1.72 (95% CI=1.37–2.16) times higher in the hour after consumption compared with periods with no alcohol intake. The RR returned to baseline in the third hour after consumption (Figure 1A).

Among the 76 participants who consumed alcohol in the hour before MI onset, 26 drank beer, 14 drank wine, 32 drank liquor and 4 drank more than one type of alcoholic beverage. The RR for alcohol consumption in the hour before MI onset was stronger for liquor than for beer or wine (test for homogeneity, $P=0.04$; Figure 1B). Patterns were similar in analyses restricted to participants reporting only one drink type in the prior year. Among participants who did not drink alcohol daily, there was a 3.29-fold higher MI rate in the hour after consumption (95%CI 2.46–4.41), but there was no association between alcohol consumption and MI among daily alcohol drinkers (RR=0.95 [95% CI=0.65–1.37]; test for homogeneity, $P<0.001$). We did not have sufficient data to examine the association stratified jointly by beverage type and by drinking patterns. Compared with participants who reported drinking 1–2 servings of alcohol per drinking episode, the RR was higher among people who consumed 2–3 drinks per episode and even higher among participants who reported drinking >3 drinks per episode (test for trend, $P<0.001$). We did not have sufficient data on the last time participants consumed 3 or more drinks within 1–2 hours, so we could not assess binge drinking as a trigger of MI.

Among 926 participants who completed questions on consumption in each of the 36 hours prior to MI onset, 65 reported alcohol intake only in the hour prior to MI onset, 48 reported intake only during the same hour on the previous day, and 25 reported intake during both periods. The rate of MI was 1.35 (95% CI=0.93–1.97) times higher in the hour after alcohol consumption compared with the same time on the previous day. In a sensitivity analysis accounting for meal consumption, the results were not meaningfully altered (1.45 [0.99–2.12]).

Acute MI Incidence within 24 Hours after Consumption

Within 24 hours after consumption, there was a 14% lower rate ($RR=0.86$ [95% $CI=0.79-0.95$]) of acute MI compared with 24 hour periods with no alcohol consumption. This protective association was apparent for beer and wine but not for liquor (test for homogeneity, $P=0.005$; Figure 2). Patterns were similar in analyses restricted to participants reporting only one drink type in the prior year. We combined the first hour with the subsequent 23-hour window to prevent overestimation of a protective effect due to potential error in recalling the exact time of drinking.

Sensitivity Analyses

The RR in the one hour following alcohol consumption was higher among current smokers ($RR=2.35$ [95% $CI=1.68-3.28$]) than among never or former smokers (1.35 [0.98–1.85]; test for homogeneity, $P=0.018$) and it was higher among participants reporting a history of coronary artery disease (2.34 [1.64–3.35]) compared with those reporting no prior MI or angina (1.44 [1.07–1.94]; test for homogeneity, $P=0.04$). Estimates stratified jointly by beverage type and smoking status were highest for current smokers who consumed liquor in the hour prior to MI onset. No other differences by any of the other pre-specified potential modifiers were observed (Table 2).

The results were not meaningfully altered when we excluded 172 participants who reported any premonitory symptoms ($RR=1.78$ [1.40–2.26]) or when we excluded 338 participants who were interviewed more than a week after MI onset (1.82 [1.43–2.31]). The association was slightly lower (1.42 [1.06–1.90]) when we excluded 29 participants who reported that, in addition to alcohol consumption, they also had high levels of anger ($n=1$), physical activity ($n=2$), lifting 50 pounds ($n=1$), caffeine ($n=5$), tobacco ($n=12$), marijuana ($n=1$), cocaine ($n=1$) or a combination of several potential triggers ($n=6$) in the hour prior to MI onset.

Discussion

In this study of 3,869 participants with confirmed acute MI, the rate of MI onset was 72% higher during the hour after drinking alcohol compared with periods with no consumption, and the association was stronger for liquor than for beer or wine. The association was stronger among participants who habitually drank more drinks per episode. The higher risk in the hour following alcohol consumption was not apparent for daily drinkers. The elevated risk in the first hour was followed by reduced risk in the next 23 hours, producing an overall protective association for beer and wine in the 24-hour window before MI. For liquor, an overall association in the 24-window was not observed.

We estimated the proportion of cases whose alcohol consumption was associated with AMI onset in the following hour using the formula for the population attributable fraction.²⁰(p. 296) Based on the prevalence of alcohol consumption in the hour before AMI onset and the RR , approximately 1.5% of the cases in this study were associated with recent alcohol consumption, suggesting that the acute effects of alcohol consumption on AMI risk do not have a large public health impact. However, this estimate may be higher in regions with a

higher prevalence of alcohol consumption. For instance, the adult per capita alcohol consumption is higher in regions such as Russia, the Czech Republic, the Republic of Moldova and Denmark.²¹ Additionally, some regions consume mostly liquor, which may incur greater risk than in regions that consume mostly beer or wine.

Despite consistent evidence on the cardiovascular benefits of habitual alcohol consumption and several studies showing that alcohol may be an acute trigger of sudden cardiac death² and ischemic³⁻⁷ and hemorrhagic^{8,9} stroke, there have only been a few studies on the short-term risk of acute MI following alcohol consumption. One case-crossover study¹⁰ found a higher risk of MI within 12 hours after alcohol consumption, and two case-control studies^{11, 12} found a protective association between moderate alcohol consumption and MI in the following 24 hours. This is the first study to evaluate whether alcohol consumption triggers an MI in the hour after intake and the first to examine whether there are differences in MI risk according to beverage type.

Results from experimental studies suggest that there are both harmful and protective effects of moderate and high alcohol intake within hours immediately following consumption.¹³ On the one hand, alcohol intake is associated with increased blood pressure, which may lead to disruption of vulnerable coronary plaques, increased heart rate,²² interarterial electromechanical delay,²³ platelet activation and impaired fibrinolysis; on the other hand, there are transient improvements in flow-mediated vasodilatation, endothelial function and fibrinolytic factors. Within weeks, there are also improvements in inflammatory markers,²⁴ lipid profile,²⁵ and adipokines and insulin sensitivity.¹³ There is some evidence that the acute effect of alcohol intake varies by beverage type. For example, in a randomized trial of 83 healthy young participants,²⁶ red wine and, to a smaller degree, beer, improved endothelial function within 4 hours, whereas whisky had no effect on vascular endothelium. This may at least partially explain why we found a higher acute risk following liquor consumption than following beer or wine and why we found a lower MI risk within 24 hours of beer or wine but not liquor.

In contrast to the acute harmful effects of alcohol, there is consistent evidence that habitual moderate alcohol consumption is associated with a lower risk of cardiovascular disease¹ and cardiovascular risk factors,¹³ whereas binge drinking is associated with a higher risk of cardiovascular disease²⁷⁻²⁹ and of mortality after MI.^{13, 30, 31} It is possible that the transiently higher risk of MI in the hour after alcohol consumption is outweighed by the health benefits for the following 24 hours, whereas consuming many drinks at once may result in a spike in risk with potential increased long-term harm as well. Consequently, persons who drink large amounts at each episode may primarily experience the acute harmful effect, whereas persons who regularly drink moderate amounts may experience a transiently higher risk that is at least partially counterbalanced by the subsequent cardioprotective consequences. The higher risk in the hour following alcohol consumption was not apparent for daily drinkers. This may be due to the fact that habitual alcohol intake is associated with up-regulation of enzymes that metabolize alcohol, resulting in a lower physiological response to each drink.³²

There are several potential explanations for why we found that the elevated acute MI risk in the hour after alcohol consumption was higher for liquor than for beer or wine and that the lower risk in the 24 hours after consumption was evident for beer and wine but not for liquor. It is possible that, compared with liquor, beer and wine contain more polyphenols and other protective constituents,^{33–35} but it is also possible that the differences are due to the amount of alcohol consumed or to simultaneous food intake rather than the type of alcoholic drink. Because of the high ethanol concentration of liquor, participants who recently drank liquor may have consumed larger amounts of alcohol at each episode than participants who recently drank beer or wine. In the United States, a standard serving of alcohol is defined as 0.6 fluid ounces (~14 grams) of pure alcohol, which is found in 12 ounces of regular beer (~5% alcohol), 5 ounces of wine (~12% alcohol) and 1.5 ounces of distilled spirits (~40% alcohol). These serving sizes may not reflect customary serving sizes; one glass of wine or one can of beer has the same alcohol content as only one shot of whiskey.

Alcohol consumption may have hastened the onset of acute MI for people who would have had an MI in a few hours even in the absence of alcohol intake. Therefore, the susceptible pool would be depleted in the 2–24 hours after the hypothesized hazard of 1 hour after alcohol consumption; this may at least partially explain our finding that alcohol intake is associated with lower MI risk within 24 hours. However, it seems likely that briefly advancing the timing of MI onset would have led to a null association by 24 hours, rather than resulting in estimates indicative of a protective association.

Strengths and limitations

We did not have information on the number of drinks consumed in the hours prior to MI onset, and we had information only on the usual number of drinks per session. Therefore, we cannot directly estimate the dose-response relationship between the amount of alcohol consumed and immediate MI risk. Because some participants may have consumed more than one beverage type during typical drinking episodes, summing across beverage types may have led to an overestimate of the exposed person-time during the preceding year, resulting in an underestimate of the RR. In addition, we did not have data on whether liquor was consumed as a mixed drink. As in other observational studies, information on alcohol consumption was obtained via self-report, and participants may not accurately report consumption. We attempted to minimize recall bias by ensuring participant privacy during the interview and by using a standardized structured interview, and participants were not informed of the duration of the hypothesized hazard period. Additionally, this method mimics clinical care, and assessment of alcohol intake appears reliable⁷ and valid¹⁶ among people seeking medical consultation at a hospital emergency department. Furthermore, our results were similar when we compared each person's exposure during the hazard period to their exposure at the same time on the day before onset of AMI symptoms, rather than their usual frequency of consumption in the prior year. Finally, our results were similar when we excluded participants who were interviewed more than a week after AMI onset.

Because the case-crossover design uses participants as their own controls, there can be no confounding by measured and unmeasured risk factors that are stable over time, but

confounding by factors that change over time within individuals can occur. There is a circadian peak of MI onset in the morning hours, and people may drink more alcohol in the evening and night. However, our results were similar when we compared each person's exposure during the hazard period to their exposure at the same time on the day before onset of MI symptoms. In a sensitivity analysis excluding individuals also exposed to other potential risk factors, our findings were not materially altered. It is possible that some participants abstained from alcohol as a result of premonitory symptoms that are harbingers of the onset of MI,^{11, 36} resulting in an underestimate of recent exposure, but the risk of MI remained elevated when we excluded participants who reported premonitory symptoms.

It is possible that, compared with other MI cases, participants who consumed alcohol are more likely to survive³⁷ and to participate in our study, resulting in an overestimate of the RR and the frequency of AMI associated with alcohol consumption. Alternatively, participants who consume alcohol may be less likely to survive,³⁸ resulting in an underestimate of the RR and the frequency with which this occurs. However, it seems unlikely that MI survival is different for cases attributable to different mechanisms. Moreover, we have previously shown in this study population that neither recent nor habitual alcohol consumption affects MI severity,³⁹ and the association between alcohol consumption and MI is similar for fatal and nonfatal events.⁴⁰ Finally, our results may not be generalizable to people with sociodemographic characteristics different from those of our study participants.

Total alcohol intake and beverage preference cannot fully describe drinking patterns and the complex relationship between alcohol consumption and other lifestyle factors. We did not have enough data to further stratify these subgroups to determine whether other participant characteristics were responsible for the heterogeneity in the observed RRs. Nevertheless, these results suggest that there is a transiently higher risk of MI onset in the hour after alcohol consumption among people who do not drink alcohol every day, and the elevated risk is higher for liquor than beer or wine.

In conclusion, our findings highlight the complex association between alcohol consumption and acute MI onset, including higher MI risk in the hour after intake and lower risk in the following 24 hours. The harmful and protective effects of moderate alcohol consumption are influenced by the amount of alcohol consumed, the time course of consumption (i.e. drinking patterns) and, potentially, by beverage type. Therefore, it is difficult to make simple recommendations about alcohol consumption. In the context of prior research, this study adds to the evidence that, while beer, wine, and, particularly, liquor, may be associated with a transiently higher risk of AMI among people who do not drink alcohol daily, moderate habitual consumption may be protective.

Acknowledgments

Funding/Support: This work was supported by grants HL-120505 and HL-41016 from the National Institutes of Health, United States. Johanna van der Bom was supported by a Stipendium from the Niels Stensen Foundation.

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Figure 1.

Association of alcohol consumption with acute MI in the next hour. The error bars indicate the 95% confidence limits. The dashed line indicates the baseline risk. **A.** Relative risk of acute myocardial infarction after alcohol consumption by time of onset. Each of the hazard periods before MI onset was assessed as independent hazard periods, and each window was compared with exposure during the control period of one year. **B.** Relative risk of acute myocardial infarction in the hour after consumption of beer, wine or liquor.

Figure 2.

Association of the consumption of beer, wine or liquor with acute myocardial infarction in the subsequent 24 hours. The error bars indicate the 95% confidence limits. The dashed line indicates the baseline risk.

Table 1Characteristics^a of the Determinants of Myocardial Infarction Onset Study participants.

	Alcohol Consumption in the Past Year	
	Yes (n=2119)	No (n=1750)
Age (years); mean (SD)	58.9 (12.3)	64.6 (12.8)
Female	493 (23)	764 (4)
White race	1899 (90)	1506 (86)
Married	1477 (70)	1009 (58)
Income (US\$); mean (SD)	41,1312 (17,616)	36,726 (16,184)
Education		
Less than high school	351 (17)	485 (28)
Completed high school	808 (38)	776 (44)
Some college	924 (44)	442 (25)
Body mass index (kg/m ²)	27 (5)	28 (6)
Smoking status		
Never	428 (20)	586 (34)
Former	905 (43)	653 (37)
Current	776 (37)	494 (28)
History of		
Hypertension	858 (41)	832 (48)
Diabetes mellitus	286 (14)	539 (31)
Angina	451 (21)	498 (29)
Myocardial infarction	515 (24)	549 (31)
Coronary artery disease	729 (34)	753 (43)
Regular use of		
ACE inhibitors	227 (11)	286 (16)
Aspirin	889 (42)	652 (37)
Beta blockers	428 (20)	402 (23)
Calcium channel blockers	449 (21)	474 (27)
Digoxin	107 (5)	150 (9)
Index Hospitalization		
Thrombolytic use	954 (45)	590 (34)
Congestive heart failure	239 (11)	277 (16)
Ventricular tachycardia	233 (11)	146 (8)
Peak Creatine Kinase level (U/L)	1621 (1886)	1380.(1726)
Physical Activity (times/week)		
<3	1704 (80)	1545 (88)
3	414 (20)	205 (12)

Missing data on education (n=83), income (n=317), smoking status (n=27)

^aNo. (%) unless otherwise indicated.

Table 2

Risk (RR) of acute myocardial infarction within 1 hour after alcohol consumption, according to patient characteristics, among the Determinants of Myocardial Infarction Onset Study participants.

	No. Exposed in Past Hour	No. Exposed in Past Year	RR (95%CI)	Test of Homogeneity ^a
All Patients	76	2119	1.72 (1.37–2.16)	
Drink Frequency				
Daily	29	488	0.95 (0.65–1.37)	<i>P</i> <0.001
Not daily	47	1631	3.29 (2.46–4.41)	
Drink Type in Past Year				
Beer Only	12	415	1.54 (0.88–2.69)	<i>P</i> =0.10
Wine Only	3	258	0.98 (0.32–2.98)	
Liquor Only	10	276	3.14 (1.66–5.94)	
Usual Drinks Per Episode				
1 – 2	19	979	1.48 (0.95–2.32)	<i>P</i> <0.001 ^b
3	15	376	1.59 (0.94–2.67)	
>3	38	659	1.98 (1.43–2.74)	
Sex				
Men	64	1626	1.68 (1.31–2.15)	<i>P</i> =0.61
Women	12	493	1.97 (1.11–3.53)	
Age, y				
<65	55	1390	1.83 (1.40–2.39)	<i>P</i> =0.41
65	21	729	1.48 (0.95–2.29)	
Smoking Status				
Never/Former	39	1333	1.35 (0.98–1.85)	<i>P</i> =0.02
Current	36	776	2.35 (1.68–3.28)	
Beverage Type and Smoking Status				
Beer				
Never/Former smoker	12	847	1.04 (0.59–1.84)	<i>P</i> =0.26
Current smoker	13	520	1.63 (0.95–2.80)	
Wine				
Never/Former smoker	11	831	1.41 (0.79–2.55)	<i>P</i> =0.65
Current smoker	5	332	1.81 (0.74–4.46)	
Liquor				
Never/Former smoker	17	733	1.75 (1.09–2.81)	<i>P</i> =0.02
Current smoker	19	457	3.96 (2.49–6.31)	
Physical Activity (times/week)				
<3	64	1705	1.86 (1.45–2.39)	<i>P</i> =0.15
3	11	391	1.17 (0.65–2.10)	
History of Coronary Artery Disease				
No	44	1390	1.44 (1.07–1.94)	<i>P</i> =0.04
Yes	32	729	2.34 (1.64–3.35)	

^a *P*-value for the χ^2 test for homogeneity, except where indicated

^b
P-value for linear trend