The Relationship Between Alcohol Consumption and Glycemic Control Among Patients with Diabetes: The Kaiser Permanente Northern California Diabetes Registry

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BACKGROUND: Alcohol consumption is a common behavior. Little is known about the relationship between alcohol consumption and glycemic control among people with diabetes.

OBJECTIVE: To evaluate the association between alcohol consumption and glycemic control.

DESIGN: Survey follow-up study, 1994–1997, among Kaiser Permanente Northern California members.

PATIENTS: 38,564 adult diabetes patients.

MEASUREMENTS: Self-reported alcohol consumption, and hemoglobin A1C (A1C), assessed within 1 year of survey date. Linear regression of A1C by alcohol consumption was performed, adjusted for sociodemographic variables, clinical variables, and diabetes disease severity. Least squares means estimates were derived.

RESULTS: In multivariate-adjusted models, A1C values were 8.88 (lifetime abstainers), 8.79 (former drinkers), 8.90 (<0.1 drink/day), 8.71 (0.1–0.9 drink/day), 8.51 (1–1.9 drinks/day), 8.39 (2–2.9 drinks/day), and 8.47 (≥ 3 drinks/day). Alcohol consumption was linearly ($p < 0.001$) and inversely ($p = 0.001$) associated with A1C among diabetes patients.

CONCLUSIONS: Alcohol consumption is inversely associated with glycemic control among diabetes patients. This supports current clinical guidelines for moderate levels of alcohol consumption among diabetes patients. As glycemic control affects incidence of complications of diabetes, the lower A1C levels associated with moderate alcohol consumption may translate into lower risk for complications.

KEY WORDS: diabetes; alcohol consumption; hemoglobin A1C.

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INTRODUCTION

Diabetes is a significant and growing health problem. The prevalence of diabetes is 7% in the U.S. population, and the

prevalence of type 2 diabetes continues to grow in parallel with the prevalence of obesity.¹ Diabetes is a strong risk factor for cardiovascular disease and end stage renal disease, is the most common cause of blindness among working-age adults, and is responsible for half of non-traumatic lower extremity amputation in the United States.^{1–8} In 2002, per capita medical expenditures for people with diabetes were \$13,243, compared to \$2,560 for persons without diabetes, and total direct medical expenditures exceeded \$91 billion.⁹

Glycemic control is a strong predictor of diabetes complications, particularly microvascular complications. As such, regular testing of hemoglobin A1C (A1C) is a cornerstone of proper diabetes care, and achievement of A1C < 7.0% has been promulgated by the American Diabetes Association (ADA) as a goal for diabetes care.¹⁰ However, the proportion of patients with type 2 diabetes who have achieved this goal appears to be declining.¹¹

Given the tremendous disease burden and financial costs associated with diabetes complications, understanding modifiable predictors of diabetes disease course has great public health significance. Alcohol consumption is a very common, potentially modifiable behavior that may impact diabetes disease course. Half of all people age 12 or older in the United States currently consume alcohol.¹² Alcohol consumption has been shown in several large cohort studies to predict diabetes incidence in a U-shaped distribution.^{13–18} That is, moderate alcohol consumption of 1–3 drinks per day is associated with lower incidence rates of diabetes than either abstinence or heavy consumption. Despite alcohol consumption being a common behavior, there is virtually no data on how alcohol is related to glycemic control in diabetes. Current ADA guidelines about recommended alcohol consumption are based on studies that enrolled mainly non-diabetic persons.¹⁹ The purpose of the current study was to fill this gap in knowledge by investigating the cross-sectional associations between alcohol consumption and glycemic control among a large ($n = 38,564$) representative sample of people with diabetes who receive medical care in a nonprofit, group model, integrated care setting. We hypothesized that moderate alcohol consumption would be associated with lower A1C than either abstinence or heavy drinking.

METHODS

Setting

The Kaiser Permanente Northern California Medical Care Program (KP) is a nonprofit health plan that is one of the largest and oldest integrated, group model health plans in the

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United States. KP currently provides comprehensive medical services through 17 hospitals and 32 outpatient clinics to ~3.2 million health plan members in the 14-county San Francisco Bay and Sacramento metropolitan regions. Approximately 30% of the population in the catchment area are KP members. The sociodemographic characteristics of KP members are representative of the underlying population, except with respect to income, where the very poor and very wealthy are somewhat under-represented relative to the general population.²⁰⁻²² The ethnic composition is quite close to that of the U.S. census enumerated population in the Bay Area, Metropolitan Statistical Area (MSA): 7% African American versus 9% in the underlying MSA population, 10% versus 11% Asian/Pacific Islander, 10% versus 14% Hispanic, and 71% versus 64% white. KP maintains comprehensive electronic administrative and clinical databases that are linked through a unique medical record number assigned to each member.

This study was approved by the Kaiser Foundation Research Institute Institutional Review Board.

Source Population

The *Kaiser Permanente Northern California Diabetes Registry* (the "registry"), for which methods have been described previously,²³⁻³¹ is a well-characterized population that has been maintained continuously since its establishment in 1993. Registry eligibility is based on multiple sources of data including pharmacy (diabetes medication prescriptions), labo-

ratory ($A1C \geq 7\%$), and outpatient, emergency room and hospitalization diagnoses of diabetes. The registry was 99.5% sensitive for diagnosed diabetes, compared with self-report, as of January 2003. All automated clinical information (pharmacy, laboratory, outpatient and inpatient diagnoses and procedures) is downloaded annually to provide a comprehensive, longitudinal follow-up of each registry member.

In an attempt to collect data not captured electronically, including self-care and lifestyle behaviors, diabetes symptoms and type of diabetes, all members of the KP Diabetes Registry were surveyed by mail and computer-assisted telephone interview in 1994-1997. A total of 77,722 persons responded, yielding an 83% response rate. We excluded the 11,726 survey respondents (15% of respondents) who did not respond to the first wave of mailed surveys and were then administered a shortened version of the survey that did not include questions about alcohol consumption. We further excluded 4,485 respondents (5.8%) who did not answer the alcohol questions and 22,947 respondents (29.5%) who did not have an A1C measurement within 1 year of survey completion, leaving 38,564 people (41% of the Diabetes Registry) in the study cohort (Fig. 1).

Measures of Alcohol Consumption and Glycemic Control

Survey respondents were asked about frequency of alcohol intake (days per month, in the past year) and usual consump-

Figure 1. Cohort creation.

tion (number of drinks usually consumed on a day of any intake, during the past year). These questions were derived from a modified AUDIT-C questionnaire.³² From these data, we calculated average daily alcohol consumption. Survey-derived self-reported alcohol consumption is a reliable measure of alcohol intake. Mailed survey and interview methods have been demonstrated in previous studies to yield comparable results.^{33–36}

We used A1C as the measure of glycemic control. The first A1C level obtained within 1 year (and for sub-analysis, 2 years) after each subject's survey date was determined from laboratory databases. All assays were conducted at Kaiser's centralized laboratory.

Assessment of Covariates

Compliance with recommended diabetes self-care behaviors was determined from survey self-report and computerized pharmacy records. Cigarette smoking and use of diet and exercise as treatment for diabetes were self-reported. We employed a validated algorithm³⁷ to determine medication-taking adherence, based on pharmacy refills. We defined poor medication refill adherence as having been without sufficient oral hypoglycemic medications at least 20% of the days of the calendar year of survey response. Compliance with the then-current ADA recommendations for self-monitoring of blood glucose (SMBG) was determined among the subset of patients who were taking diabetes medications. Self-monitoring of blood glucose frequency was based on a validated measure of strip utilization.^{26,27} No recommendations for SMBG frequency had been promulgated for those whose diabetes was not treated with medication. Recommended SMBG frequency was at least 3 times daily among people with type 1 diabetes and at least once daily among those with medication-treated type 2 diabetes. To account for occasionally forgetting to test, we considered average use of ≥ 2.5 test strips/day in type 1 diabetes or ≥ 0.75 test strips/day in medication-treated type 2 diabetes to be in compliance with recommendations. Type of diabetes was determined from self-reported characteristics, using a published algorithm.²⁷

Statistical Analysis

We grouped diabetes patients into 7 categories of alcohol consumption: lifetime abstainers, former drinkers, and consumers of <0.1 , 0.1 – 0.9 , 1.0 – 1.9 , 2.0 – 2.9 , and ≥ 3 drinks/day. We employed a survey follow-up design (also known as lagged cross-sectional design) to assess glycemic control during the 1 year after survey date. We employed general linear models to regress A1C on alcohol consumption. We report the least squares means estimate from each model presented. All continuous variables except age were specified in their continuous form in all models. Age was first specified as a categorical variable (18 – 34 , 35 – 49 , 50 – 64 , ≥ 65) to assess whether the alcohol–A1C relationship differed across the life span. Because of the possibility that insulin resistance might be associated with differential effect of alcohol by type of diabetes^{38,39} or age,⁴⁰ we assessed effect modification by these variables. Our first models included only alcohol consumption, each putative effect modifier (age or diabetes type), and the associated cross-product term (alcohol $\times$ [age or type]). We considered p values <0.10 on cross-product terms to be significant. There was no

significant effect modification of the alcohol–A1C relationship by age ($p=0.17$) or type of diabetes ($p=0.15$). All models were adjusted for gender, self-reported race/ethnicity, and age (as a continuous variable). Additionally, we included as covariates in our models those factors known to be associated with glycemic control and potentially associated with alcohol consumption, including diabetes duration; use of oral diabetes medications, insulin, and dyslipidemia medications; medication adherence; SMBG; use of diet and exercise to control diabetes; and obesity. Models also controlled for smoking status, educational attainment (high school graduate or less, some college, college graduate), peripheral neuropathy, hypertension, and type of diabetes. We performed tests of significance for alcohol consumption as a predictor of A1C. We additionally performed tests for trend to assess linear relationships between alcohol consumption and A1C, as indicated.

To assess whether any observed associations between alcohol consumption and A1C during the 1 year after baseline were because of selection bias; that is, bias introduced because those study participants who obtained an A1C within that 1-year period were different than those who waited longer to obtain an A1C; we performed a lagged analyses among the 10,546 respondents whose first A1C value after survey completion was obtained 1 to 2 years after baseline.

All analyses were performed with SAS version 9 (SAS Institute, Cary, NC, USA).

RESULTS

To evaluate response bias, we compared patients in our study cohort ($n=38,564$) to those who were excluded because they did not answer the alcohol questions or had no A1C measurement during the study period ($n=27,432$). We found no significant differences in gender ($p=0.13$) or medication adherence ($p=0.43$). Compared to diabetes patients excluded, those in the cohort were 0.3 years older ($p<0.001$), more likely to report white race/ethnicity ($p<0.001$, 60.3% of cohort, 52.7% of those excluded), to have attended college ($p<0.001$, 55.8% and 52.9%), and to be nonsmokers ($p<0.001$, 88.9% and 86.8%). Responders had more severe disease, as evidence by prescriptions for insulin ($p<0.001$, 31.6% and 26.6%), oral diabetes medications ($p<0.001$, 58.0% and 51.8%), and dyslipidemia medications ($p<0.001$, 14.2% and 11.6%). We have previously compared survey respondents ($n=77,722$) to non-respondents ($n=15,919$) and found no significant selection bias, particularly when examining associations.^{26,28}

Characteristics of the study cohort are presented in Tables 1 and 2. Twenty-two percent had never consumed alcohol (denoted “abstainers” in Tables 1 and 2 and Fig. 2), and 27.9% were former drinkers (denoted “former” in Tables 1 and 2 and Fig. 2). Just over 50% reported current alcohol consumption. In unadjusted analyses (Fig. 2), we observed a J-shaped distribution of A1C by alcohol consumption, with the best glycemic control (i.e., lowest A1C values) among consumers of 2–2.9 drinks/day and poorer control among consumers of either ≥ 3 drinks/day or <2 drinks/day. The highest A1C was observed among consumers of <0.1 drinks/day, followed by lifetime abstainers.

In multivariate models, the alcohol–A1C relationship persisted. Alcohol consumption was significantly ($p<0.001$) and inversely ($p=0.001$ for linear trend) associated with A1C,

Table 1. Patient Characteristics by Level of Alcohol Consumption among 38,564 Diabetes Patients

	Lifetime abstainer	Former drinker	Current drinker	p value
N	8,286	10,751	19,527	
N (%)	21.5	27.9	50.6	
Demographic and socioeconomic characteristics				
Mean age (SD)	61.1 (13.0)	61.0 (11.8)	58.1 (13.0)	<0.0001
Age category (%)				<0.0001
18–34	3.2	1.7	4.8	
35–49	15.4	15.8	20.8	
50–64	37.8	40.2	40.1	
≥65	43.5	42.3	34.3	
Female (%)	70.3	47.4	37.8	<0.0001
Ethnicity (%)				<0.0001
White	49.0	58.5	66.1	
African American	10.7	13.2	9.5	
Latino	8.5	8.5	8.1	
Asian	22.4	8.4	6.6	
Other	3.0	2.6	2.2	
Multithnic	6.5	8.8	7.4	
Education (%)				<0.0001
High school or less	52.0	51.2	37.1	
Some college	25.9	29.8	33.7	
College graduate	22.1	19.1	29.2	
Clinical characteristics				
A1C, mean (SD)	8.60 (1.95)	8.52 (1.99)	8.47 (1.94)	<0.0001
Type of diabetes (%)				<0.0001
Type 1	3.0	2.6	6.2	
Type 2	88.5	90.5	86.9	
Unsure	8.5	6.8	6.9	
Duration of diabetes (%)				<0.0001
<10 years	59.2	61.4	65.9	
10–19 years	25.3	24.5	21.3	
≥20 years	15.6	14.1	12.7	
Hypertension (%)	70.2	71.7	65.0	<0.0001
Peripheral neuropathy (%)	31.3	35.0	29.1	<0.0001
Diabetes medication use (%)				<0.0001
None	17.1	16.6	21.0	
Insulin	24.7	22.8	22.5	
Oral medications	49.2	50.3	49.2	
Oral medications + insulin	9.1	10.3	7.3	
Uses antihypertensives (%)	60.0	60.5	53.8	<0.0001
Uses antilipemic (%)	14.4	14.4	14.1	NS
Obesity, BMI ≥ 30 (%)	37.8	42.4	39.5	<0.0001
Self-care behaviors				
Smoking (%)				<0.0001
Never	76.4	38.7	41.4	
Former	17.8	48.9	45.9	
Current	5.9	12.4	12.7	
Uses diet to control diabetes (%)	60.2	63.1	60.9	<0.0001
Uses exercise to control diabetes (%)	49.5	51.4	52.2	0.0003
Refill adherence good *	80.9	80.0	81.3	0.03
Follows SMBG recommendations†	24.7	28.5	23.6	<0.0001

NS not significant

*Good refill adherence is defined as having sufficient supply of medications at least 80% of the time

†Analysis limited to subjects prescribed diabetes medications

although there appeared to be a J-shaped tail of the distribution with increased A1C among the heaviest drinkers. Adjusted A1C values were 8.88 (lifetime abstainers), 8.79 (former drinkers), 8.90 (<0.1 drink/day), 8.71 (0.1–0.9 drink/day),

8.51 (1–1.9 drinks/day), 8.39 (2–2.9 drinks/day), and 8.47 (≥3 drinks/day).

In our assessment of selection bias, as described above, the lagged analysis of A1C among the 10,546 patients whose first A1C measurement after baseline occurred between 1 and 2 years after baseline, as with the primary analysis, found that alcohol consumption was significantly and inversely associated with A1C in both unadjusted and multivariate models (data available upon request).

DISCUSSION

Among 38,564 adult patients with type 1 or 2 diabetes, increasing levels of alcohol consumption predicted lower A1C values through a nadir at consumption of 2–2.9 drinks/day. Ours is the largest study to examine the association between alcohol consumption and glycemic control among diabetes patients. Our results extend the findings of Mackenzie et al.⁴¹ who found that alcohol consumption was associated with lower A1C among 1,024 adults with diabetes who participated in Third National Health and Nutrition Examination Survey. Furthermore, a randomized, controlled trial of moderate alcohol consumption among 109 previously abstentious adults with diabetes found that consumption of 13 g of alcohol daily reduced fasting plasma glucose by 9% compared to non-alcohol consuming controls.⁴² Because of our much larger cohort, we were able to test for different patterns of association by age and type of diabetes and to compare A1C among more precisely defined levels of alcohol consumption.

There are biologically plausible explanations for our findings. Among people without diabetes, moderate alcohol consumption may enhance insulin sensitivity^{43–51} and has been shown to decrease A1C.^{52–56} It is plausible that regular, moderate alcohol consumption by people with diabetes similarly enhances insulin sensitivity and improves glycemic control. If this is the case, it might follow that the effect would be greater among type 2 diabetes patients, as they typically have much greater insulin resistance than those with type 1 diabetes; we did not, however, find different patterns of effect by diabetes type in our cohort.

Our study has several limitations. First, as with all human studies of the relationship between alcohol consumption and clinical outcomes, randomization was not possible. However, our rich database allowed us to employ multivariate models that controlled for the affects of a wide array of putative confounding variables. Second, alcohol consumption was assessed at a single point in time. It is possible that lower A1C values among current compared to former drinkers reflect their generally better state of health, as former drinkers include those who stopped drinking because of poor health. Our data do not support this hypothesis, however, as our data allowed us to differentiate former drinkers (among whom we would expect to find those who stopped drinking because of ill health) from lifetime abstainers, and we found that lifetime abstainers had higher A1C values than former drinkers. Third, we rely on self-report of alcohol consumption. If heavy consumers of alcohol underreported their consumption because of their wish not to report socially undesirable behavior, this would have created a conservative bias. Fourth, despite the large study population, our conclusions were limited by the small number of patients reporting higher levels of alcohol

Table 2. Patient Characteristics by Level of Alcohol Consumption among 19,527 Diabetes Patients who Consume Alcohol

	Average daily number of alcohol-containing drinks consumed in the past year					p value
	<0.1	0.1–0.9	1–1.9	2–2.9	≥3	
N	9252	7448	1715	552	560	
N (%)	24.0	19.3	4.5	1.4	1.5	
Demographic and socioeconomic characteristics						
Mean age (SD)	57.7 (13.0)	57.8 (13.4)	60.5 (11.9)	60.4 (11.3)	57.2 (10.9)	<0.0001
Age category (%)						<0.0001
18–34	5.0	5.5	2.6	2.4	2.5	
35–49	21.7	21.1	15.8	15.8	22.7	
50–64	40.6	38.9	39.9	42.2	46.1	
≥65	32.7	34.5	41.7	39.7	28.8	
Female (%)	50.5	30.3	20.2	10.9	6.8	<0.0001
Ethnicity (%)						<0.0001
White	63.7	66.5	74.8	71.9	69.3	
African American	9.6	9.9	7.7	7.8	10.7	
Latino	9.2	7.0	7.2	8.9	7.9	
Asian	7.9	6.1	4.0	3.3	2.5	
Other	2.3	2.3	1.5	1.6	2.1	
Multietnic	7.3	8.2	4.8	6.5	7.5	
Education (%)						<0.0001
High school or less	39.2	34.7	34.3	34.6	45.3	
Some college	35.6	32.3	30.7	34.4	29.7	
College graduate	25.2	32.9	35.0	31.1	25.0	
Clinical characteristics						
A1C, mean (SD)	8.63 (1.93)	8.41 (1.96)	8.09 (1.77)	8.04 (1.77)	8.22 (2.02)	<0.0001
Type of diabetes (%)						<0.0001
Type 1	4.8	7.9	6.8	4.9	4.6	
Type 2	88.7	84	85.2	88.6	89.8	
Unsure	6.5	7.3	8.1	6.5	5.5	
Duration of diabetes (%)						<0.0001
<10 years	66.6	65.8	62.7	61.8	70.8	
10–19 years	21.5	20.9	22.1	24.3	19.9	
≥20 years	11.9	13.3	15.3	14.0	9.3	
Hypertension (%)	66.1	62.5	67.4	68.3	70.0	<0.0001
Peripheral neuropathy (%)	29.9	27.4	26.8	33.6	37.1	<0.0001
Diabetes medication use (%)						<0.0001
None	19.7	21.7	24.0	23.6	22.5	
Insulin	22.5	23.3	22.0	19.4	17.0	
Oral medications	49.5	48.4	48.4	51.1	54.5	
Oral medications + insulin	8.3	6.6	5.6	6.0	6.1	
Uses antihypertensives (%)	54.8	51.8	55.9	55.6	56.6	<0.0001
Uses antilipemic (%)	14.3	14.2	13.6	15.8	11.1	NS
Obesity, BMI ≥ 30 (%)	45.5	35.9	28.1	29.9	32.3	<0.0001
Self-care behaviors						
Smoking (%)						<0.0001
Never	46.3	41.1	28.3	28.3	20.0	
Former	41.9	47.1	56.5	56.5	52.9	
Current	11.8	11.8	15.2	17.4	27.1	
Uses diet to control diabetes (%)	61.8	62.3	58.7	48.2	48.6	<0.0001
Uses exercise to control diabetes (%)	51.8	54.6	50.2	42.8	40.2	<0.0001
Refill adherence good *	81.0	81.7	80.8	81.5	81.1	NS
Follows SMBG recommendations†	25.5	22.7	21.5	20.4	15.9	<0.0001

NS Not significant

*Good refill adherence is defined as having sufficient supply of medications at least 80% of the time

†Analysis limited to subjects prescribed diabetes medications

consumption. Finally, it is possible that response bias affected our results, as our study cohort composed 41% of the Diabetes Registry. As with other survey studies, respondents tended to be older (although by only 0.3 years) and have higher socioeconomic status than nonrespondents.^{57–59} However, as our analyses determined associations, rather than prevalence of conditions, the risk of selection bias affecting study conclusions is minimal.⁶⁰ Furthermore, our finding of similar patterns of association between alcohol consumption and A1C among the cohort of 10,546 patients who had an A1C measured between 1 and 2 years after baseline suggests that our findings are robust. These 10,546, together with the

39,142 patients in our main analysis, comprise 53% of the Diabetes Registry. We have assessed responder bias in our previous research based on survey responders and found no noteworthy selection bias, particularly when assessing statistical associations.^{26,28}

Our study has several strengths. First, we had data to differentiate lifetime abstainers from former drinkers, who include those who stopped drinking because of poor health. We did not observe a clinically meaningful difference in A1C between lifetime abstainers and former drinkers, suggesting that the lower A1C observed among drinkers was because of the effects of alcohol rather than because of the “healthy

Figure 2. Unadjusted and multivariate adjusted hemoglobin A1C levels (% mean and 95%CI) by average daily alcohol consumption in the past year among 38,564 adult diabetes patients. Multivariate model is adjusted for age, gender, race/ethnicity, educational attainment, obesity, hypertension, peripheral neuropathy, duration of diabetes, medication-treated dyslipidemia, type of diabetes, and self-care behaviors (self-monitoring of blood glucose, medication adherence, abstinence from smoking, use of diet and exercise to control diabetes). Number of patients in each group is indicated on X axis.

drinker effect." Second, the KP membership from which the study population was drawn makes up approximately 30% of the Northern California population and is sociodemographically representative of the general population, giving this study public health relevance and generalizability.^{20-22,61} Third, we were able to control for a wide variety of potential confounding variables, including diabetes self-care behaviors.⁶² Fourth, the finding that the inverse relationship between alcohol and A1C persisted during the period 1-2 years after baseline suggests that the relationship is robust.

Our study findings have implications for clinical practice and future research. Half of our study subjects, who are drawn from a population representative of the underlying population, consume alcohol. Both primary care and addiction medicine clinicians are increasingly interested in how substance use and abuse affect the disease course of patients with chronic diseases such as diabetes. Current ADA recommendations call

for limiting alcohol consumption by adult diabetes patients to no more than 1 or 2 drinks daily for women and men, respectively.¹⁹ Our findings, by demonstrating that moderate alcohol consumption is associated with better glycemic control than abstinence, provide data to support this recommendation. Likely, only meta-analysis will provide more definitive conclusions, as few study populations are as large as ours.

As glycemic control affects incidence of diabetes-related complications, particularly microvascular endpoints, increased understanding of the effects of alcohol consumption on glycemic control may improve the attention that providers pay to assessing alcohol consumption and addressing alcohol use disorders among their diabetes patients. Compared to lifetime abstainers, consumers of 2-2.9 drinks/day had lower A1C by nearly 0.5%. This difference is clinically significant. For example, this is similar to the lowering of A1C obtained with initiation of intensive metformin therapy.⁶³ Furthermore, this may translate into benefits in diabetes-related complications, as a 1% reduction in HbA1c is associated with a 21% reduction of the risk of any diabetes-related endpoint and a 37% reduction in the risk of microvascular complications.⁶⁴ The mechanisms by which alcohol might affect glycemic control deserve further study. Additionally, research should be directed at assessment of whether the associations between alcohol and glycemic control translate into differential incidence of diabetes complications among patients with different levels of alcohol consumption.

Table 3. Parameter Estimates (β) for Main Effects of Alcohol Consumption on A1C, among 39,142 Patients with Diabetes

	Model 1	Model 2
Lifetime abstainers	0.38	0.41
Former drinkers	0.31	0.32
<0.1 drinks/day	0.41	0.43
0.1-0.9 drinks/day	0.20	0.24
1-1.9 drinks/day	-0.13	0.04
2-2.9 drinks/day	-0.17	-0.08
≥3 drinks/day	0	0

Model 1 Unadjusted; Model 2 adjusted for age, gender, ethnicity, educational attainment, obesity, hypertension, peripheral neuropathy, duration of diabetes, medication-treated dyslipidemia, and self-care behaviors (self-monitoring of blood glucose, abstinence from smoking, use of diet and exercise to control diabetes)

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Conflict of Interest: None disclosed.

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