

Veracity and Vodka: Can Alcohol Actually Be Good for the Heart?

A statistical analysis by August Dombrow

On November 19, 2009, second-tier news web site Chatahbox rather boldly ran the headline “Good News Guys! Beer is Good for Your Heart,” accompanied by the image of an inebriated Homer Simpson and the results of a recent study. The article demonstrates both the benefits and the hazards of statistics – the ability to quantitatively measure relationships in order to make informed decisions or the ability to lend potentially dangerous claims an air of legitimacy, backed by the appearance of authoritative figures and quotations. In this case, Chatahbox staff writer “Sue” offered the following interpretation:

“According to a Spanish study, daily moderate consumption of alcohol reduces the risk of coronary heart disease in men, by at least 35 percent and downing from three to 11 shots of liquor per day reduced the risk even further, by a whopping 50 percent. Of course excessive drinking can cause other problems, such as liver disease, dementia and stroke, but coronary heart disease would not be a concern.”

Armed with the statistical tools of the past semester, I decided to investigate the matter for myself, testing the claims for veracity and settling the question (at least for myself): can alcohol actually be good for the heart?

The original study, titled “Alcohol intake and the Risk of coronary heart disease in the Spanish EPIC cohort study” and published in the scholarly journal *Heart*, indeed concludes that consuming alcohol in any amount results in a reduced chance of coronary heart disease (CHD). Conducted by researchers from the Basque Public Health Department, the published findings report an inverse association between alcohol consumption and incidents of CHD, a relationship that does not diminish with increased levels of consumption.

Similar studies have been conducted repeatedly over the past several decades, but this was the first to attempt to compensate for the so-called “sick quitters,” those former alcohol consumers who quit due to poor health, medications, or simply old age. Critics of past studies argued that these reformed abstainers resulted in lower rates of CHD among alcohol consumers as their failing health was reflected in the non-drinker data. In addition to the “sick abstainer” phenomenon, the researchers attempted to control for a wide range of potential confounding “lifestyle” variables ranging from age and weight to diet and medications, concluding that no combination of these additional variables altered the association between alcohol and CHD.

The study included 41,438 volunteers, providing information through a series of dietary and lifestyle questionnaires conducted by trained personnel. Based on this information, men were divided into six categories of alcohol consumption: former drinkers, never drinkers, and low-, moderate-, high-, or very high-intake drinkers. Women were divided into five similar groups, with the exception of the very high-intake category. In a series of follow-up interviews, averaging approximately ten years from the start of the study, the researchers identified coronary incidents through a combination of self-reported questionnaires and other relevant records, including hospital databases, myocardial infarction registries, and mortality registries. In this manner, they identified 481 incidents in men and 128 in women, reporting “incident rates” of 300.56/100,000 person-years and 47.93/100,000 person-years.

While many of the statistical methods utilized are beyond the scope of my expertise, I decided to conduct a hypothesis test of my own, focusing on the most controversial aspect of the claim, that is, whether heavy drinkers demonstrated fewer incidents of CHD than lifelong abstainers. To accomplish this I examined the confidence interval for the difference between two proportions, tested hypotheses about proportions, and finally conducted a chi-square test, all using

the data for incidence rates in males (similar statistics for females yielded no statistical significance), standardized by age and smoking status. Curiously, none of my calculations achieved statistical significance using a .05 threshold (the same as in the study), and I could not conclude with the same certainty that a relationship does exist.

I began by calculating the confidence interval for the difference between two proportions, namely the proportion of “never-drinkers” with CHD (population 1) and the proportion of “high-intake (30-90 g / day) drinkers” with CHD (population 2). According to data provided within the published study, 618 never-drinkers reported 25 cases of CHD, or approximately 4%, while only 2.9% (159 / 5502) of high-intake drinkers reported CHD. These figures yielded standard errors of 0.8% for sample proportion 1 and 0.2% for sample proportion 2, resulting in a 95% confidence interval of -0.5% and 2.7%. Consequently, I could not conclude that the true population difference does not include zero, so it remains possible that no difference exists between the two populations.

Next, I conducted a one-sided hypothesis test with the following hypotheses:

Null Hypothesis: There is no difference between the proportion of CHD amongst never-drinkers and high-intake drinkers.

Alternative Hypothesis: Never-drinkers exhibit higher proportions of CHD than high-intake drinkers.

To test this hypothesis, I calculated the sample proportion assuming the null hypothesis is true, resulting in 3%. Using this new figure, I calculated the null standard error (approximately .007237) and a z-score of 1.52. The corresponding p-value, .0655, failed to meet the prescribed alpha value, so I was again unable to validate the findings of the study.

Finally, I attempted a chi-square test for a two-way table, set up as follows:

	CHD Incident? (Observed Counts)		
Group	No	Yes	Total
Never-drinkers	593 (96%)	25 (4%)	618
High-intake	5343 (97.1%)	159 (2.9%)	5502
Total	5936 (97%)	184 (3%)	6120

My null hypothesis proposed that there is no relationship between alcohol consumption and incidents of CHD, while the alternative hypothesis stated that the variables are, in fact, related. The corresponding expected counts were:

	CHD Incident? (Expected Counts)		
Group	No	Yes	Total
Never-drinkers	599.4	18.6	618
High-intake	5936.6	165.4	5502
Total	5936	184	6120

Accordingly, the chi-squared statistic was $(.068 + .008 + 2.2 + .248) 2.524$. Using the .05 cut-off, or 3.83, we can see that it again fails to achieve statistical significance.

So after three failed attempts to verify the study's conclusions, I could only wonder what caused the different outcomes. Where I used the proportion within each category, the researchers translated that data into person-years and then into the number of incidents per 100,000 person-years. Given the reputation of these researchers and the peer-reviewed journal, as well as my own rudimentary grasp of statistical methods, it is plausible that this new figure is the more appropriate measure. But perhaps these additional calculations are responsible for the discrepancies in our outcomes. As mentioned in class, with large enough

data, otherwise negligible differences can achieve statistical significance. Even though my own computations failed to achieve the .05-threshold for statistical significance, they did come very close, and I am not altogether prepared to dismiss the findings of the study.

The health implications of alcohol might be elusive, but global consumption demands a thorough investigation. According to the World Health Organization (WHO), an estimated 2 billion people imbibe, and approximately 1.8 million deaths are attributed to alcohol annually (2). With this degree of consumption, any warnings of negative consequences should be immediately and thoroughly explored; conversely, positive side effects should not be dismissed because of cultural prohibitions.

This is not to say that everyone should start a heavy regimen of alcohol consumption, and the study's authors are very clear when discussing the ramifications of their research. In an article from *The Independent*, lead researcher Larraitz Arriola cautions, "The first thing to say about our research is that alcohol is very harmful. If you drink heavily, you should drink moderately. The more you drink, the worse off you will be." But with headlines reading "Drink half a dozen beers every day and have a healthier heart," it is no wonder that readers become confused – and vulnerable to suggestive articles that seem to validate otherwise reckless habits. While the study is both persuasive and tempting in its presentation, it does not provide a blank check for the dedicated bar patron. In fact, the researchers seem astutely aware of the limitations of their own study, writing:

"Residual or unmeasured confounding variables could be a limitation. Although we adjusted for several potential confounders in our different models, we could never be sure about remaining confounders. Indeed, non-randomised studies are never free of residual confounding." (10)

Ultimately, the study adds to a growing body of research documenting the implications of alcohol consumption on our health, without providing that conclusive piece of evidence for either side of the debate.

Works Cited

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