Does your heart race for a Fitbit hangover?

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Report: Final report W241 spring 2018

1. Introduction

Fitbits are popular items of wearable technology, particularly for health-conscious individuals and those trying to become healthier. Fitbit collects many different data points, including steps walked, calories burned as well as heart-rate measurements. Several users (MarathonChris, 2016[1]), including ourselves, have observed that one's resting heart rate (RHR) measurement is elevated the day after a few alcoholic drinks. While it is well known that the consumption of alcohol increases an individual's heart rate in the short term (for a few hours during and after), it is not known that there is an increase in the resting heart rate the next day. In the Fitbit community, there have been some suggestions as to the cause. In this report we examine if this observation is accurate via experimentation.

Why would the answer to this question be of interest? As with any questions about health, more knowledge is better. If the answer were that Fitbit's algorithm for calculating this is at fault, at least the 23 million active Fitbit users can better understand how it works. On the other hand, suppose this effect is real and measurable in some or all cases. Then further experiments would be required to determine the actual cause, whether it be dehydration, lack of sleep or an indicator of heart problems in certain individuals.

Take for example an individual with heart disease: knowing that the effects of drinking alcohol on their heart are more prolonged than presupposed may help to deter them from having a few drinks, particularly if they are expecting exertion or stress the next day. Another example is a healthy competitive athlete who likes to relax with a few drinks once a week; will

[1] "Solved: Heart rate day after drinking alcohol - Fitbit Community." 10 Feb. 2016, https://community.fitbit.com/t5/Live-Mindfully/Heart-rate-day-after-drinking-alcohol/td-p/1176594. Accessed 24 Apr. 2018.

their increased heart rate the next day benefit their recovery or reduce the effectiveness of their training?

These and other questions first rely on getting to the answer of the question: does alcohol consumption increase the resting heart rate the next day?

This is the subject of this report and will be covered in the following sections. First, we will introduce the existing literature, followed by the details of our pilot study. After, that we will lay out the design of our experiment and present the results. Finally, we will round up with the conclusions.

2. Literature

Usually searching the internet yields a myriad of results that have to be carefully sorted and analyzed for truth (or at least an educated guess at the likely truth). In this case, however, the search for reasons why the RHR is elevated the day after drinking reveals no documents other than some blogs, and questions on the Fitbit site from MarathonChris[2]. The search shows plenty of information and understanding of the immediate and long-term effects of alcohol, but nothing other than rare blog posts about increased heart rate the next day.

3. Pilot study

Our pilot study attempted to ascertain if the effect is something we could measure using a Fitbit and was conducted using data on a single individual from December 31st to March 5. This study had been done in a less than rigorous manner, using no randomization; however, the data was suitable to evaluate if the experiment was worth conducting, i.e., if an effect could be measured.

The pilot used a within subject design and a difference in differences (DiD) on the heart rate pre and post-treatment, where the treatment was a binary variable indicating when the subject drank alcohol the previous day. It did not use any randomization of treatments and as discussed below had some issues that needed to be accounted for. Table 1 presents the regression results from the pilot.

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[2] "Solved: Heart rate day after drinking alcohol - Fitbit Community." 10 Feb. 2016, https://community.fitbit.com/t5/Live-Mindfully/Heart-rate-day-after-drinking-alcohol/td-p/1176594. Accessed 24 Apr. 2018.

	Dependent variable:			
	Change.HR		Change.HR.Previous.Control	
	(1)	(2)	(3)	(4)
Treatment.Yesterday	1.945***	2.043***	1.719***	1.700***
	(0.404)	(0.468)	(0.398)	(0.456)
Constant	-0.491***	-0.543**	-0.264	-0.200
	(0.190)	(0.254)	(0.177)	(0.231)
Observations	64	45	64	45
R2	0.239	0.271	0.217	0.233
Adjusted R2	0.226	0.254	0.204	0.215
Residual Std. Error	1.332 (df = 62)	1.424 (df = 43)	1.251 (df = 62)	1.313 (df = 43)
Note:				*p<0.1; **p<0.05; ***p<0.01

Table 1: Pilot Regressions

Our initial calculations used a DiD of the RHR on subsequent days, and the results are shown in regression (1) in table 1. We noticed that the intercept (constant) had a highly statistically significant negative result; implying that when the subject did not drink his RHR was continually decreasing. A consistent daily negative change in RHR is impossible, and we realized that this method of taking the DiD included the recovery from treatment as the RHR adjusted back to the original level. Once we realized this, we altered the DiD to be the difference between the current days RHR and the previous control day. The results of this are seen in regression (3) in table 1. In this regression, we no longer see a statistically significant intercept as expected (it is not distinguishable from 0).

There was a second stumbling block in the pilot which we investigated in regressions (2) and (4). The dates of the pilot were after the end of the fall MIDS term and a seasonal vacation, which was the most sedentary time of year for the subject, and the data showed a decline in the subjects RHR as his fitness levels returned to normal. Figure 1 shows this decline.

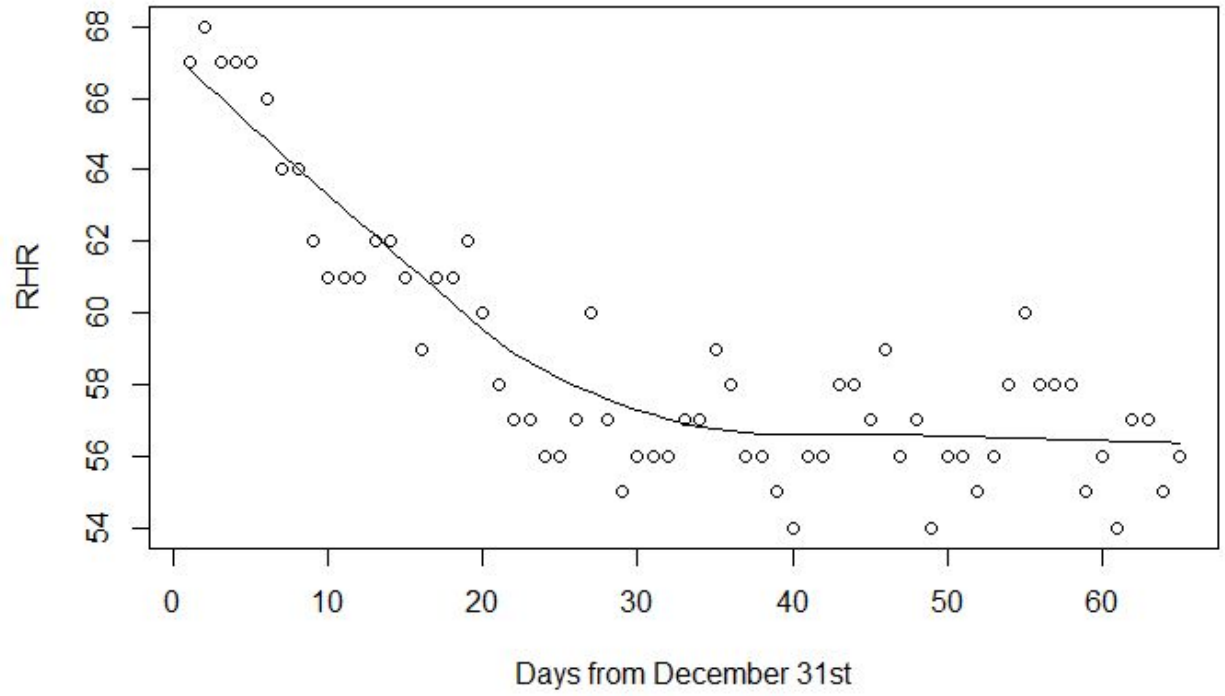


Figure 1: Pilot subjects RHR during the study

Once we had observed the change in figure 1, we took it into account in regressions (2) and (4) by using a smaller set of the data. The results changed slightly, with a decline in the treatment effect, however, it is still highly statistically significant.

Also since the sample size was small (<100) we also performed randomization inference on the data. The results were similar with an ATE of 1.71 and a P-value of 8×10^{-5}, indicating that the observed ATE was unlikely to have occurred by chance (again, highly statistically significant).

3.1 Calculating the sample size

To simplify the calculation of the required sample size we used an algorithm on the website www.sample-size.net. From our pilot, we calculated an effect size of 1.7, with a standard deviation of 1.4. Given that we wanted a high statistical power we chose the probability of type 1 and type 2 errors as 1 in 100 (0.01), we also expected to have about a 1:3 treatment to control measurements. Using these values, we estimated a sample size of 82 with

25 treatments. While this may have been sufficient for the power we needed in this project we erred on the side of caution and gathered over twice this recommendation in our experiment.

3.2 Hypotheses

With the results of the pilot in hand, we set about to design our experiment in a way to prove or disprove the effect we had observed and to also determine if the effect is real or just something specific to Fitbit. To facilitate this, we made a list of the possible effects and what we would need to observe should to prove those results be true. These are presented in table 2. Note, we also had people measure their heart rate manually; this will be discussed further in the design section.

Possible effect	**Results that would be observed**
There is no change in RHR measured by Fitbit or manually	Neither Fitbit nor manual methods will record a statistically significant result
The RHR changes are specific to Fitbit	The Fitbit measured RHR will have a statistically significant outcome but manual will not
The RHR changes are real and observed by both Fitbit and manual measurements	Both manual and Fitbit measurements will show a statistically significant result

Table 2: Possible Effects

The second of these hypotheses is the one we suspected to be most likely. Given that we had measured the effect using a Fitbit in the pilot, but were unable to find literature detailing the cause, it seemed likely that the effect was not real and that it was only going to be found using a Fitbit.

The experiment we designed was to determine which of these hypotheses were true.

4. Design

4.1 Our Design

We design the experiment as follows, with a series of additional caveats we address afterward. We took a subject pool of 15 individuals and randomly assigned them specific days to give themselves a self-determined amount of alcohol. These subjects were typically measured over a two week period where they measured their manual and Fitbit resting heart, cardiovascular exercise, and amount drank every day. We then performed a difference in difference analysis where we used the day before treatment as the control and measured it against the day following treatment to measure the difference in resting heart rate between the days. We performed this analysis in two ways, once using the actual amount of alcohol consumed, and once using a dummy variable to indicate whether that day was a "treatment day" and ignoring the size of the treatment used.

We also tried to avoid interference by giving a recovery of 1 day between treatments so that the RHR could return to normal

The use of a placebo design as it was impractical for several reasons. Firstly individuals were purchasing and administering their treatments, and secondly, there is a physiological effect of alcohol which is easily recognizable and difficult if not impossible to fake.

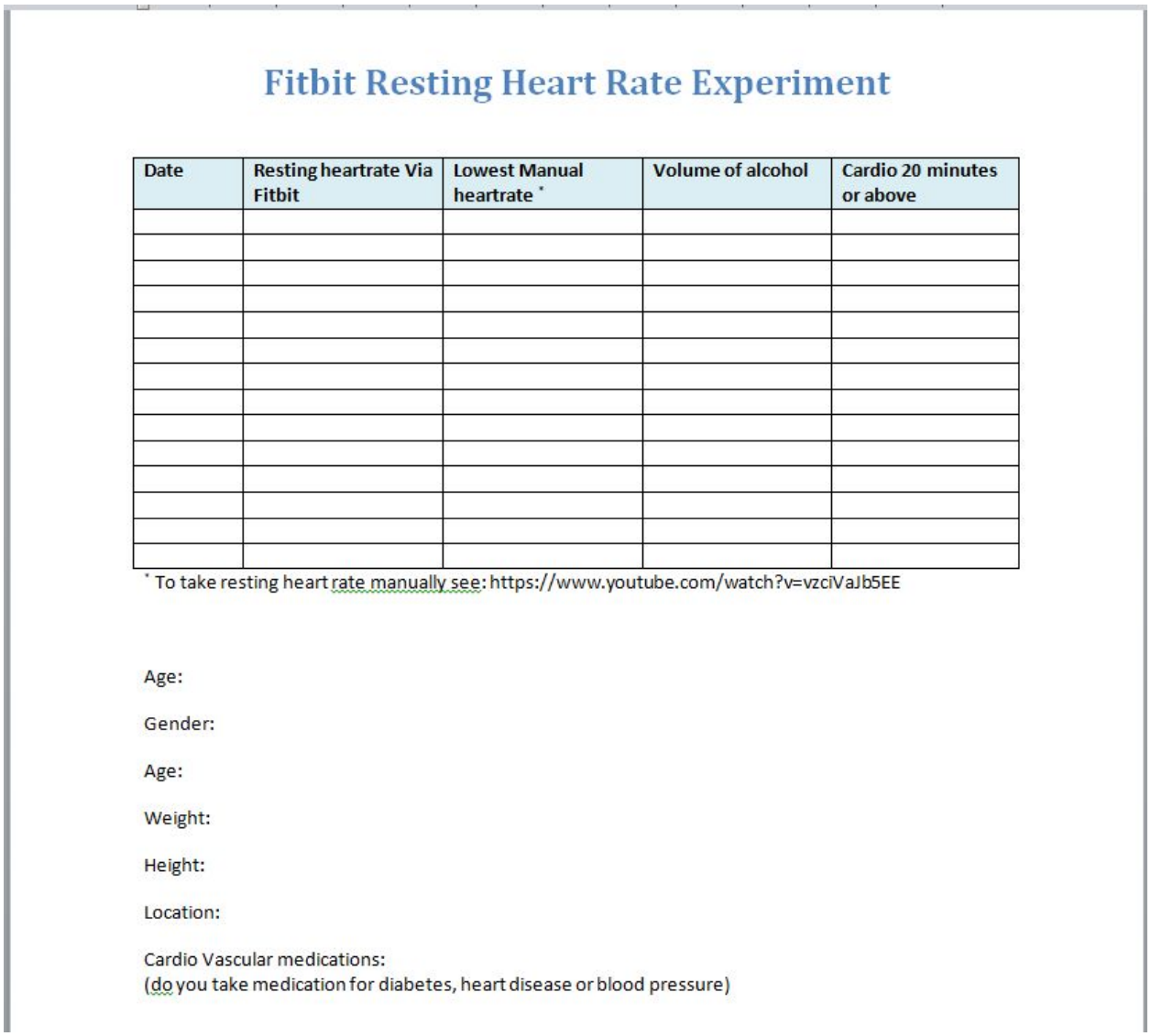
4.2 Data Collected

As for the data we collected, each day our subjects would record the current day of the week, their resting heart rate, and indicated whether they had done at least 20+ minutes of cardiovascular exercise.

The resting heart rate was collected in two ways. Every subject was instructed to, upon waking up, count their heartbeats for two minutes then divide by two. For those who also had a Fitbit, they would also record the resting heart rate given for that day by the Fitbit.

We also collected the following covariates at the end of the study period and assumed they were constant throughout: age, gender, height, weight, and indicated whether they were taking any cardiovascular medication (did not ask what medication specifically).

The data were recorded in a form that we sent to each individual, see figure 2.



Fitbit Resting Heart Rate Experiment

Date	Resting heartrate Via Fitbit	Lowest Manual heartrate *	Volume of alcohol	Cardio 20 minutes or above

* To take resting heart rate manually see: https://www.youtube.com/watch?v=vzciVaJb5EE

Age:

Gender:

Age:

Weight:

Height:

Location:

Cardio Vascular medications:
(do you take medication for diabetes, heart disease or blood pressure)

Figure 2: Form submitted by each subject

4.3 Ideal Design

In an ideal experiment, for a given subject pool, we would randomly assign an amount of alcohol, a day of the week and a time for the treatment to be consumed. We would also record the additional covariate of race, however, given the size and diversity of our subject pool any effects due to race would be impossible to measure. Additionally, we would provide all subjects with a Fitbit and would take multiple manual resting heart rate readings throughout the day to

get more accurate, less variable readings. We would also like to run the experiment on multiple two week periods throughout the year to avoid any possible seasonal effects.

4.4 Caveats

The experiment is built around the treatment effects of alcohol. That being said, in many cases it is difficult to randomize everywhere you might want. For example, we could not randomize the number of drinks that people had for a given treatment. Because of this, we chose to randomize by day of the week to rule out any anticipation effects of the weekend. Once again, it was difficult to fully randomize the day of the week that people chose to self-treat with alcohol. Where possible, we did full randomization of when people drank during the week (4 individuals). For everyone else (who were volunteers in the experiment) we could not fully randomize their drinking schedule. We requested that they did not drink on the same day of the week during the experiment. We believe this to be enough to justify the day of the week being ruled out of any effects measured. For every subject in the pool their week 1 and week 2 treatment schedule was different.

It should also be noted that two subjects had data removed from the study as they drank subsequent days and therefore caused inference effects. A third individual admitted that he only recorded his “heavy drinking days” and did not record the days when he had only 2 or 3 drinks. His readings had to be completely removed as we it seemed likely that all the readings were impacted by interference.

4.5 Assumptions

We make some assumptions in our analysis, and the following list provides more details on these:

1. All participants recorded their resting heart rates correctly and these reflect accurate results. This assumption may be shaky as it is observed that those taking manual heart rates see significantly higher variations from day to day measurements. However, it could be the case that the Fitbit isn’t measuring accurately but averaging out these day to day variations.
2. All participants accurately recorded the amount of alcohol they consumed and that ML conversions we use in the analysis reflect the actual amount of alcohol in those drinks.

Once again, this assumption could be on shaky ground, as significantly larger portions of alcohol consumed would likely have a detrimental effect on one's ability to accurately recall the amount consumed.

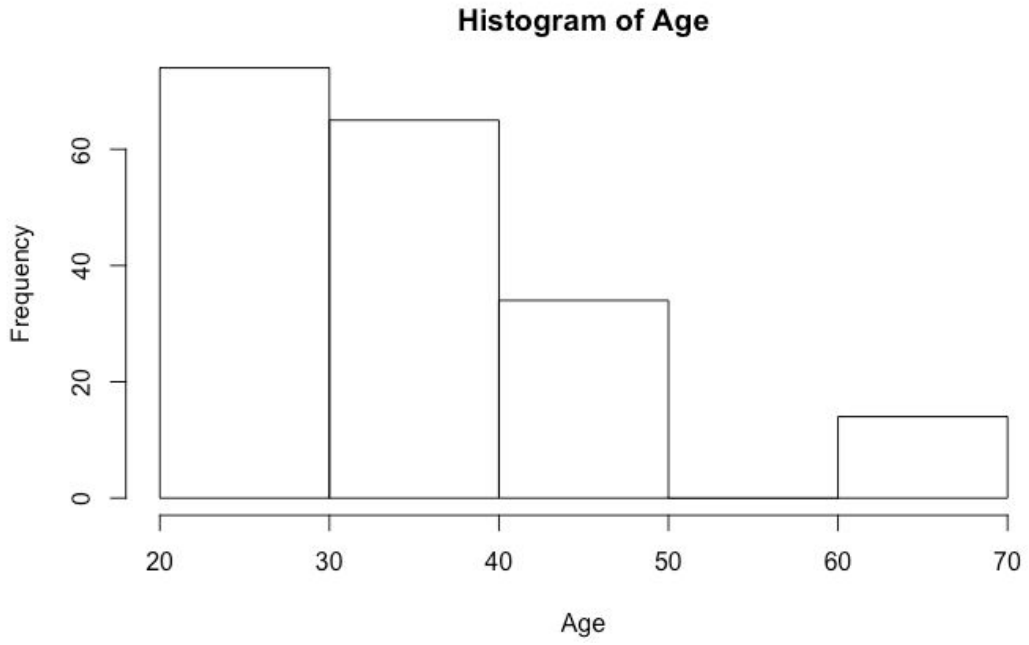
3. Subjects height and weight were accurately provided.
4. There is a linear relationship between resting heart rate and alcohol consumption. We believe this assumption to be accurate for standard amounts of alcohol consumption such as those the subjects were treated with.
5. No anticipation effects on the results
6. The interference effect of the previous treatment needs one day to dissipate
7. Even though we had 190 measurements with 59 treatments our number of subjects used was only 12. During our evaluation, we assume this is a sufficient number for our covariate analysis, though we would prefer to have had a much larger sample size.

5. Results

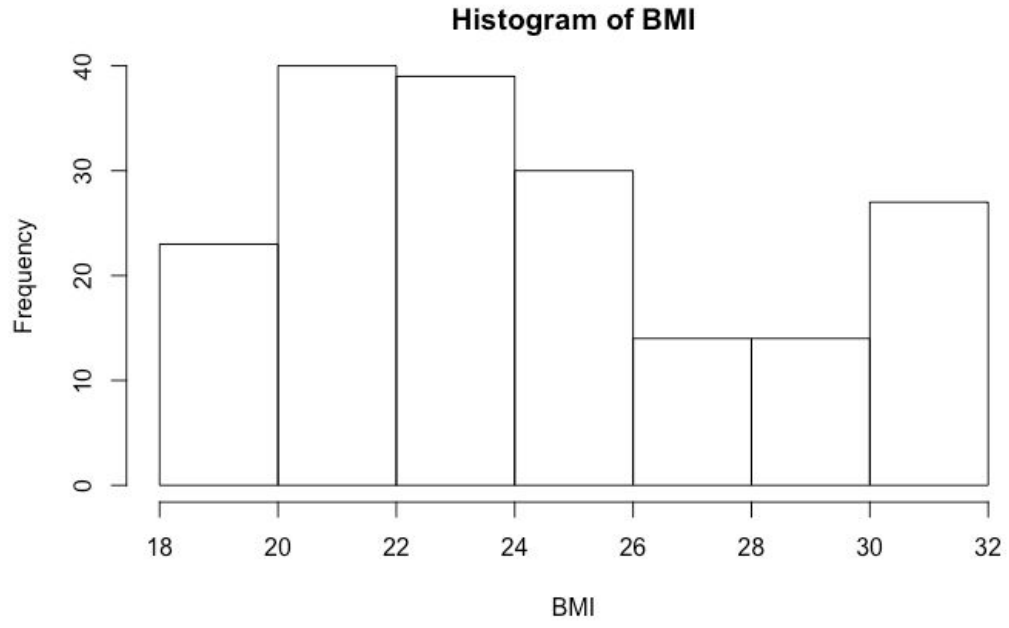
5.1 Covariate Analysis

In accordance with general data analysis, it is important to highlight the overall distribution of key covariates that could factor a role in affecting study outcomes. In this regard, the below dialogue draws focus towards subjects gender, age, and BMI as such covariates are key for this consideration.

Overall the gender split amongst participants within the study was relatively close. After the exclusion of the three subjects with interference, the remaining study group consisted of 7 males and 5 females. In reviewing age across all participants, there is a slight positive skew in the group though nothing out of the ordinary. Overall the group average was approximately 37 years of age. For comparison, the average age in the United States is 37.9 years, and the age across the nation has natural positive skewing (2016 US Census Bureau).



We see that the BMI measures follow a mostly normal distribution with a slight negative skew on the tail end. The overall group average BMI is approximately 25. For comparison, the average BMI in the United States is 26.6 for an adult male and 26.5 for an adult female (CDC).



Given there is no major skewing amongst these three covariates and that such covariates match closely with a common national population there appears to be no cause for concern of major factors at play that might interfere with study results from these covariates. As a final check, we chose to run a joint F-test to understand if the differences across such covariates and height jointly equal zero. The resulting p-value indicates significance and that there is likely correlation between BMI and another covariate. We note that if later analysis reveals such covariates are significant in predicting heart rate change, then it will be worth revisiting this finding.

```
Coefficients:
                                                Estimate Std. Error t value Pr(>|t|)
(Intercept)                                    -3.639196  11.192285  -0.325  0.74566
Female                                          0.110213   0.884155   0.125  0.90102
age                                             0.023952   0.043265   0.554  0.58092
BMI                                            -0.067891   0.122896  -0.552  0.58173
height.inches                                   0.061355   0.177677   0.345  0.73049
Volume.Pure.Alcohol.in.previous.day             0.249915   0.356411   0.701  0.48460
Female:Volume.Pure.Alcohol.in.previous.day     -0.022330   0.031456  -0.710  0.47921
age:Volume.Pure.Alcohol.in.previous.day        -0.001462   0.001214  -1.204  0.23115
BMI:Volume.Pure.Alcohol.in.previous.day         0.009907   0.003758   2.636  0.00955 **
height.inches:Volume.Pure.Alcohol.in.previous.day -0.005871   0.005665  -1.036  0.30223
---
Signif. codes:  0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

Residual standard error: 2.092 on 115 degrees of freedom
  (62 observations deleted due to missingness)
Multiple R-squared:  0.1979,    Adjusted R-squared:  0.1352
F-statistic: 3.153 on 9 and 115 DF,  p-value: 0.001954
```

5.2 First stage analysis

In our initial analysis, we looked at how the volume of alcohol the previous day changed the resting heart rate on its own, then individually with a number of the covariates we collected and finally on many of the covariates. We avoided having height, weight and BMI in a single regression as these are collinear and so would have created issues in the regression. Table 3 shows these results:

	Dependent variable:								
	Change.in.Fitbit.HR.from.previous.control.day								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Volume.Pure.Alcohol.in.previous.day	0.024**	0.023**	0.023**	0.024**	0.024**	0.023**	0.024**	0.024**	0.024**
	(0.008)	(0.009)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.009)
Female		-0.034						0.302	-1.828
		(0.358)						(0.415)	(2.863)
Weight.LBS			0.008					0.012	0.020
			(0.006)					(0.009)	(0.019)
age				0.019				0.001	0.281
				(0.021)				(0.026)	(0.410)
BMI					0.102				
					(0.054)				
exercise						0.322			1.065
						(0.383)			(1.009)
CV.Meds									-1.442
									(1.758)
height.inches									-0.632
									(0.731)
PSTPlus							0.002		-0.300
							(0.060)		(0.507)
Constant	-0.210	-0.193	-1.435	-0.878	-2.528*	-0.343	-0.222	-2.149	32.161
	(0.215)	(0.309)	(0.863)	(0.784)	(1.232)	(0.274)	(0.545)	(1.167)	(40.515)
Observations	125	125	125	125	125	125	125	125	125
R2	0.136	0.136	0.143	0.140	0.147	0.141	0.136	0.146	0.188
Adjusted R2	0.129	0.122	0.129	0.126	0.133	0.127	0.122	0.118	0.131
Residual Std. Error	2.099 (df = 123)	2.108 (df = 122)	2.099 (df = 122)	2.104 (df = 122)	2.095 (df = 122)	2.102 (df = 122)	2.108 (df = 122)	2.113 (df = 120)	2.097 (df = 116)
Note:									*p<0.05; **p<0.01; ***p<0.001

Table 3: Fitbit preliminary regressions on change in resting heart rate

From our analysis in table 3, regression (1) is the simple regression of the treatment on the outcome variable and shows a significant effect of 0.024 change in RHR for each ML of pure alcohol. The constant in this regression is, as expected, not significant indicating that the daily change in RHR is not distinguishable from zero.

Following that, regressions (2) to (7) are regressions using a single covariate to see if there was any significant effect from any of these. We observe in the table that these results did not show any significance, nor did their inclusion alter the standard error of the treatment effect. We did see one strange result, in regression (5) using the BMI covariate we saw a significant constant. This may be by chance, but we decided to check for an HTE with this and will discuss it in a later section.

The second to last regression in table 2 includes the covariates we thought most likely to have an impact, however again there was no significant values and no change to the standard error of the treatment. Then finally regression (9) includes all the covariates and still has no change to the standard error of the treatment. We put this final regression through an F-test to see if the covariates had joint significance, however when we tested to see if all the covariates were equal to zero we got an f-statistic of 0.5852 which means we cannot reject the hypothesis that they are all zero.

We also did the same set of regressions with the manually measured resting heart rate, shown in table 4 (below). The results were very similar with a slightly higher treatment effect of 0.034 change in RHR for each ML of pure alcohol consumed the previous day. Again the addition of covariates did not reveal any significant ones and did not impact the standard error of the treatment effect.

	Dependent variable:								
	Change.in.manual.HR.from.previous.control.day								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Volume.Pure.Alcohol.in.previous.day	0.034**	0.034**	0.033**	0.033**	0.033**	0.034**	0.034**	0.033**	0.037***
	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.011)	(0.012)	(0.011)
Female		0.049						0.0004	-1.158
		(0.506)						(0.739)	(0.903)
Weight.LBS			-0.006					-0.005	0.011
			(0.008)					(0.012)	(0.017)
age				-0.012				-0.008	0.0004
				(0.022)				(0.033)	(0.042)
BMI					-0.028				
					(0.063)				
exercise						0.855			1.747*
						(0.511)			(0.789)
CV.Meds									-2.511
									(1.519)
height.inches									-0.239
									(0.261)
PSTPlus							-0.025		0.152
							(0.069)		(0.118)
Constant	-0.101	-0.125	0.797	0.362	0.591	-0.573	0.065	1.000	12.947
	(0.278)	(0.387)	(1.331)	(0.932)	(1.584)	(0.410)	(0.534)	(1.536)	(16.769)
Observations	149	149	149	149	149	149	149	149	149
R2	0.112	0.112	0.115	0.113	0.113	0.129	0.113	0.115	0.175
Adjusted R2	0.106	0.100	0.103	0.101	0.101	0.117	0.101	0.091	0.127
Residual Std. Error	3.086 (df = 147)	3.097 (df = 146)	3.093 (df = 146)	3.095 (df = 146)	3.095 (df = 146)	3.067 (df = 146)	3.095 (df = 146)	3.113 (df = 144)	3.049 (df = 140)
Note:									*p<0.05; **p<0.01; ***p<0.001

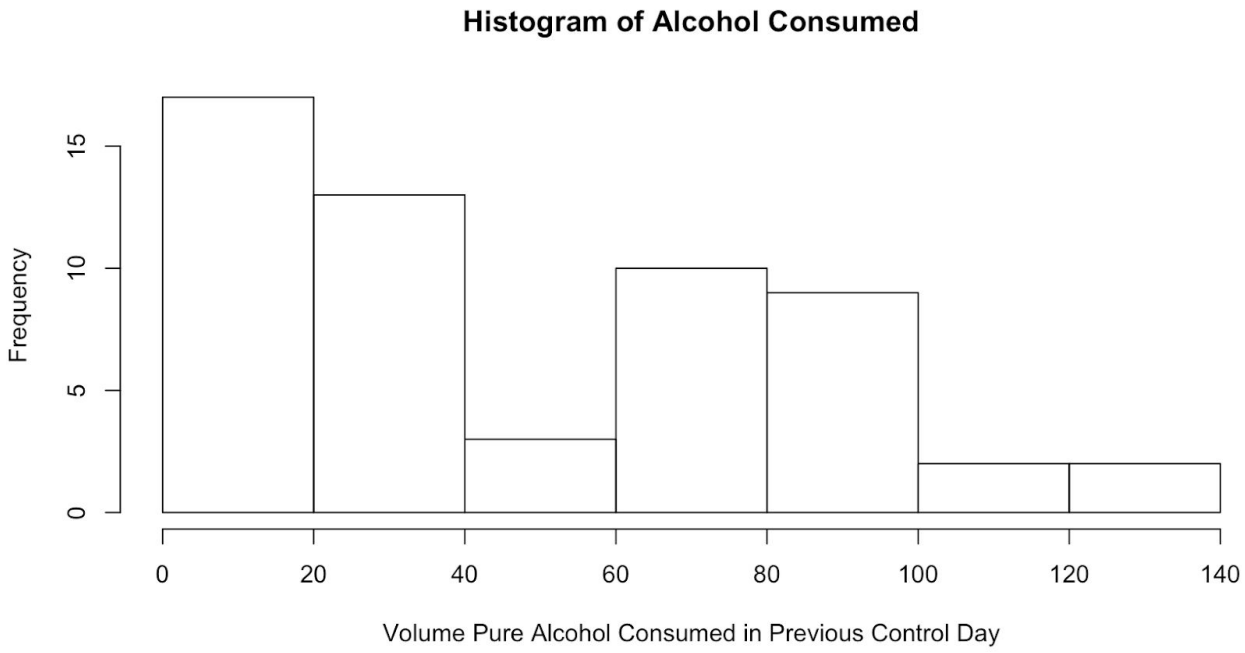
Table 4: Manual preliminary regressions on change in resting heart rate

One curiosity is the statistically significant result of exercise in regression (9). We put this down to chance, and if we use a Bonferroni correction, this result is no longer significant.

5.3 Binary Treatment

```
             Estimate Std. Error t value Pr(>|t|)
(Intercept) -0.098765   0.227490 -0.4342   0.6649
Alcohol1     1.098765   0.439829  2.4982   0.0138 *
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1
```

In analyzing the effects of a binary treatment variable (i.e. Consumed Alcohol, Not Consumed Alcohol), we find that there is still a significant effect on Fitbit measurements (1.0988 at p = 0.0138), despite our treatments being skewed right (below). The effect is consistent across Fitbit and Manual measurements, albeit the Manual measurement showed a higher coefficient, similar to our continuous regression.



5.4 Fixed Effects for Individuals

Here we measure the effects on each person individually (for those with Fitbits). We also we ran the same analysis on those with manual heart rates and found similar results. We see that taking into account individuals results, we still saw a significant effect of alcohol on resting heart rate.

```
                                Estimate Std. Error t value Pr(>|t|)
(Intercept)                    0.0521439  0.4495928  0.1160 0.907869
PersonA2                      -0.0641457  0.5387242 -0.1191 0.905426
PersonCL                      -0.1248155  0.6003252 -0.2079 0.835661
PersonCY                      -0.3659395  0.6205740 -0.5897 0.556553
PersonHoang                   -0.3585093  1.7387372 -0.2062 0.837004
PersonKyle                    -0.6463520  0.4690929 -1.3779 0.170894
PersonPaul                     0.1912314  0.5317432  0.3596 0.719777
PersonWL                      -0.0153492  0.6441160 -0.0238 0.981029
Volume.Pure.Alcohol.in.previous.day  0.0166106  0.0047877  3.4694 0.000733 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1
```

5.5 Heterogeneous Treatment Effects

Heterogeneous treatment effects show how the treatment effect changes with a covariate. We investigated a number of these we thought likely; age, gender, weight, BMI. Age and gender did not reveal any significant HTEs, and interestingly while weight also did not have an HTE, BMI did. The results are in table 5.

	Estimate	Std. Error	t value	Pr(>|t|)	
(Intercept)	-0.2959972	1.5579706	-0.1900	0.849636	
Volume.Pure.Alcohol.in.previous.day	-0.0679437	0.0326623	-2.0802	0.039619	*
BMI	0.0035845	0.0694399	0.0516	0.958916	
Volume.Pure.Alcohol.in.previous.day:BMI	0.0040648	0.0015421	2.6358	0.009494	**

Signif. codes: 0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Table 5: HTE of BMI on the treatment effect

This effect is exciting as it indicates that the BMI has a highly significant impact on the treatment effect, while not related to weight or height individually. This means that for overweight individuals the effect of drinking on their RHR is higher.

6. Conclusion

Our study found that there was a statistically significant increase in Fitbit monitored and manually measured resting heart rate (RHR) within our subjects after the day of alcohol consumption (treatment), as compared to the day before alcohol consumption (control). Among Fitbit monitored RHRs, we found the increase to be **0.024** beats per mL consumed during treatment, and among manually measured RHRs we found the increase to be **0.034** beats per mL consumed during treatment. From a practical sense, this is an increase of between 1-1.5 beats per 2 standard drinks (20 mL pure alcohol). When we analyzed the data using a binary treatment (consumed alcohol vs. not consumed alcohol), we found similar increases in RHR.

In a more in-depth analysis, we included indicator (fixed) variables for each person, which resulted in similar coefficients and significance.

We initially hypothesized that the Fitbit monitored RHR would show an increase, but that it was isolated to Fitbit's algorithm. From the experiment, our initial hypothesis was accurate in that we observed an increase in RHR, however, our second hypothesis was inaccurate as we observed the same effect (even to a greater extent) on manually measured RHR.

6.1 Risks

Although the experiment showed clear effects, there are a few potential risks that should be highlighted.

The first is the potential for measurement error when taking RHRs. Because of the partially manual component of the RHR measurement process, subjects may have mismeasured, either through negligence or lack of knowledge. Measuring heart rate is especially vulnerable to this as subjects may be under slight duress when measuring their heart rate for the first time, which in turns affects the heart rate itself. We assume, though, that these artificial increases in heart rate would only affect the first measurements, which were mostly controls, and thus at worst would decrease (not increase) our measured effect.

Additionally, we assume that all subjects fully report every treatment. In our review of the data, we discovered two subjects who violated the experimental design by having treatments on consecutive days, which forced us to remove their results. Although we believe this has corrected the issue, there is always a possibility of others who have not fully reported.

Finally, since the treatment for this experiment potentially affects memory, the reported alcohol consumption, especially at a higher dosage, may be inaccurate. This affects the variance in total alcohol consumed during treatment and thus increases the standard error in our estimated treatment effect. Based on our discussions with subjects, we've determined that instances of extreme dosage (where this situation may occur) were rare and that for the most part subjects had strong confidence in their treatment dosage. Additionally, our analysis of the treatment as a binary variable (as opposed to continuous) helps mitigate this concern.

6.2 Mediation

Considering the many different side effects of alcohol consumption, it is difficult to say for certain that alcohol itself leads to an increase in RHR. Other factors, such as lack of sleep, additional substances consumed, dehydration, etc. which may be correlated with alcohol consumption may all lead to changes in RHR that are embedded within the treatment effect found in our experiment. To isolate these causes, we would need to set up additional experiments that test these specific factors, separate from alcohol. For example, we could run a future experiment that introduces decreased sleep as a treatment, and measure the effects on RHR for subjects. Alternatively, we could run a similar experiment for reducing water consumption (dehydration) to test a similar hypothesis.

6.3 Generalization

Our experiment found a significant effect among the subjects acquired, but this may not generalize completely, as our subject selection method was not random in regards to the general population. As mentioned in the Design, our subjects were selected among the study's team, their friends and family. Also, all subjects owned or had access to Fitibits, and associated with individuals completing a study for a Masters program. This could potentially be a very niche group of subjects, and thus it would be difficult for us to generalize the effects across the entire population.

6.4 Final Note

From the study, we saw a statistically significant increase in resting heart rate within our subjects the day after consuming alcohol, as compared to the day before. There has been much literature on heart rate changes during alcohol consumption, yet there is little information on the effects of alcohol in the near term after consumption. Given the significant results we've uncovered, especially considering the heterogeneous effects with BMI, we believe the results are promising and the potential impact is significant enough to merit additional research in this area.