Abstract

Background

Past studies investigating the association between blood pressure (BP) and cigarette smoking habits among United States adults have provided mixed results. In this study, the association is further investigated by examining how cigarette smoking affects systolic blood pressure (SBP) in U.S. adult smokers and ex-smokers aged 20-68 via The National Health and Nutrition Examination Survey (NHANES) data.

Methods

A complete case analysis from NHANES 2009-2010 and 2011-2012 data (n = 2,006) using a sequence of nested linear regression models was performed to investigate the relationship between current smoking status and SBP. Age, gender, race, BMI, drug usage, physical activity, and estimated days in the past year consuming alcohol were identified as appropriate confounders, as well as an interaction term between alcohol consumption and smoking status.

Results

Our analysis showed an estimated mean increase of 1.97 mmHg in SBP (95% CI: 0.67, 3.27) ($p < 0.05$) in smokers compared to ex-smokers. After introducing an interaction term between alcohol consumption and smoking status, we found a significant interaction between smoking status and alcohol intake ($p = 0.04$). The effect of smoking on SBP was stronger on average among participants who consumed more alcohol in the prior year.

Discussion

The relationship between smoking status and SBP is significantly modified by alcohol consumption. Future studies should investigate the role of EMM on the relationship between smoking status and SBP.

Background

Motivation

Nearly 1 in 2 adults in the U.S. are hypertensive (suffer from abnormally high blood pressure). 21.5% of hypertensive adults are recommended only lifestyle modifications as treatment [1]. Hypertension raises concern as it can lead to heart disease and stroke, which are both among the top five leading causes of death in the United States [2]. Blood pressure (BP) is measured in two measurements: systolic blood pressure (SBP) — the pressure exerted by blood against the walls of arteries when the heart beats, and diastolic blood pressure (DBP) — the pressure exerted by blood against the walls of arteries between heartbeats. Hypertension is formally defined as SBP $\geq$140 mmHg or DBP $\geq$90 mmHg. Cigarette smoking is widely accepted as a leading risk factor for atherosclerosis: a significant buildup of plaque in the arteries [3].

Surprisingly, studies investigating the association between cigarette smoking and SBP have produced paradoxical results [4]. Some studies investigating smoking and SBP have identified significant associations between presence of smoking habit and increased SBP and arterial stiffness [4, 5, 6]. Other studies have identified significant associations between smoking and decreased SBP, which is typically attributed to the lower BMI of smokers [6], and some studies have found an insignificant association between smoking habit and SBP [4]. Therefore, there is still motivation to investigate the association between SBP and cigarette smoking habits.

Literature Review

Current and past research indicates there are many other risk factors for hypertension: age, family history, and more, but dissimilarly, if found as a risk factor, the detrimental effects of cigarette smoking are preventable [7, 8]. An array of confounders that have been observed to influence both smoking status and SBP were identified from a carefully selected combination of

literature that closely reflects our study population. Regarding age, younger adults are more likely to smoke [9], and older adults are more likely to be hypertensive [10]. Regarding gender, men are more likely to smoke [9, 11] and be hypertensive [12, 13]. Research has reported different incidences of smoking [14] and hypertension across different races [10]. BMI has been shown to be negatively associated with smoking [15, 16] and positively associated with hypertension [17, 18]. Heavier alcohol consumers are more likely to smoke [19, 20] and have higher SBP [21]. Marijuana use has been shown to be positively associated with both cigarette smoking [22] and hypertension [23]. Similar associations have been seen for hard drug use [24, 25]. Increased physical activity has been historically associated with lower incidences of smoking [26], as well as preventative of higher SBP [27].

Studies have indicated that alcohol and cigarettes can have a synergistic detrimental effect on BP and cardiovascular issues [28, 29]. These studies originated from Germany [28] and Japan [29], and one study slightly younger than our target population [28], and the other including only men [29], but the presented reasons for the differing effect of smoking status on SBP by alcohol consumption (increased oxidative stress in vascular tissue and endothelial dysfunction [28], sympathetic nervous system stimulation [29]) were not shown to be country-, age-, or sex-dependent. Moreover, the difference in these study groups only strengthens the argument that this interaction is not specific for certain subgroups and is applicable to this study.

Materials and Methods

Study Population

The National Health and Nutrition Examination Survey (NHANES) is a large, annual cross-sectional study of approximately 5,000 U.S. individuals that provides demographic, health, lifestyle, and physical information on U.S. adults and children via interviews and physical

examinations. Data from 2009-2010 and 2011-2012 is used for this analysis. Weights stemming from deliberate oversampling are kept, and the data is treated as a random U.S. population sample.

There were an increased number of missing values for current smoking habit (n = 6,789 missing values), drug usage (n = 4,990 missing values), and alcohol consumed in the last year (n = 4,078 missing values) compared to other variables in our models. This is likely not random, as these topics are more sensitive than others, and this phenomenon introduces bias and affects our ability to easily generalize to the entire population. After removing incomplete samples, the population for this study includes U.S. adult smokers and ex-smokers aged 20-68.

Variables

This analysis is interested in investigating the relationship between current cigarette smoking habit and SBP, as well as the difference in effect of smoking on SBP among varying levels of alcohol consumption, in U.S. adults only.

Predictor of Interest

The predictor of interest is current smoking habit. This value was self-reported by the individual, so the possibility of response bias exists. Smoking was categorized as either “yes”, “no”, or “NA”, where “yes” and “no” correspond to whether a participant currently smokes, given that they have smoked ≥100 cigarettes in their lifetime. “NA” represents either missing data or participants who have smoked <100 cigarettes in their lifetime. For this study, ex-smokers are individuals who are not currently smoking, but have smoked an estimated ≥100 cigarettes in their lifetime. Our study population has 1,083 current smokers and 923 ex-smokers.

Outcomes

The outcome variable is SBP, which is the mean of three to four collected blood pressure readings for each participant, with the first measurement excluded. In cases without enough valid recordings or measurements, the first blood pressure reading was reported as the mean instead.

Covariates

The age of each participant was recorded in years with participants over the age of 80 recorded as 80. Gender was recorded as male or female. Race was categorized as Mexican, Hispanic, White, Black, or other. BMI, measured in kg/m^2. The number of days that the participant consumed alcohol in the past year was self-reported and estimated by the participant. Drug use is determined by a self-reported "yes" or "no" response regarding whether the participant used marijuana at least once a month for a year or has ever tried cocaine, crack cocaine, heroin or methamphetamine. Lastly, physical activity was self-reported by participants, recorded as "yes" or "no" regarding participation in moderate or vigorous intensity physical activities.

Statistical Analyses

Two sample t-tests were used to compare the difference between smokers and ex-smokers for SBP, DBP, estimated prior year's alcohol consumption, BMI, and age; a chi-squared test was used to evaluate this difference for gender (Table 1b). Simple linear regressions (SLR) were fitted between SBP and each explanatory variable to check the unadjusted correlations and associations between SBP and the literature review-selected variables (Table 2). Subsequently, a multiple linear regression (MLR) was fitted between SBP and current smoking status, adjusting for the other confounders (Table 3a). Lastly, our final model is a MLR model with an interaction term between alcohol consumption and current smoking status (Table 3b). R version 4.1.2 was used for the entire analysis.

Model Diagnostics

Two sample t-tests and chi-square tests were used to compare the difference between complete cases and incomplete cases. Discussed more in depth in the discussion, other techniques such as VIF, partial regression plots, and more were used to diagnose the model.

Results

Table 1a. Complete vs. Incomplete Cases

Characteristic	N	Complete, N = 2,006	Incomplete, N = 7,994	p-value
Age	10,000	41 (12)	36 (24)	<0.001
Gender	10,000			<0.001
female		829 (41%)	4,191 (52%)	
male		1,177 (59%)	3,803 (48%)	
BMI	9,634	28 (6)	26 (8)	<0.001
Drugs	5,010	1,207 (60%)	536 (18%)	<0.001
PhysActive	8,326	1,001 (50%)	3,648 (58%)	<0.001
Smoker	3,211	1,083 (54%)	383 (32%)	<0.001
AlcoholYear	5,922	87 (107)	69 (100)	<0.001
BPSysAve	8,551	119 (15)	118 (18)	0.002
BPDiaAve	8,551	72 (12)	66 (15)	<0.001
Race1	10,000			<0.001
Black		200 (10.0%)	997 (12%)	
Hispanic		92 (4.6%)	518 (6.5%)	
Mexican		158 (7.9%)	857 (11%)	
White		1,434 (71%)	4,938 (62%)	
Other		122 (6.1%)	684 (8.6%)	

Table 1b. Ex-Smokers vs. Smokers Profiles

Characteristic	**N**	**0**, N = 923	**1**, N = 1,083	**p-value**
Age	2,006	44 (11)	39 (12)	**<0.001**
Gender	2,006			>0.9
female		381 (41%)	448 (41%)	
male		542 (59%)	635 (59%)	
BMI	2,006	29 (6)	28 (6)	**<0.001**
Drugs	2,006	515 (56%)	692 (64%)	**<0.001**
PhysActive	2,006	544 (59%)	457 (42%)	**<0.001**
AlcoholYear	2,006	95 (112)	80 (103)	**0.002**
BPSysAve	2,006	119 (14)	119 (16)	0.8
BPDiaAve	2,006	73 (12)	71 (12)	**<0.001**
Race1	2,006			**<0.001**
Black		59 (6.4%)	141 (13%)	
Hispanic		39 (4.2%)	53 (4.9%)	
Mexican		78 (8.5%)	80 (7.4%)	
White		712 (77%)	722 (67%)	
Other		35 (3.8%)	87 (8.0%)	

Baseline Characteristics

Using the complete data, we compared the demographic information between smoker and ex-smoker groups. The comparison results are shown in Table 1b. Comparing all the variables of interest, the smokers tend to have lower mean age (39 vs. 44, $p<0.001$), lower mean BMI (28 vs. 29, $p<0.001$), higher percentage of drug usage (64% vs. 56%, $p<0.001$), lower percentage of physical activity (42% vs. 59%, $p<0.001$), fewer days drinking alcohol in the year (80 vs. 95, $p=0.002$), lower average diastolic blood pressure (71 vs. 73, $p<0.001$), and significantly different distribution of races such as more blacks in the group (13% vs. 6.4%, $p<0.001$). The results may suggest that the profiles of smokers and ex-smokers may be different according to the dataset. The average systolic blood pressure between smokers and ex-smokers are almost the same, without adjusting other characteristics (119 vs 119, $p=0.8$).

Table 2. Unadjusted Analysis (SLR) Table

	Estimate	Std. Error	t value	95%CI	Pr(>|t|)
Age_c					
	0.39	0.03	13.9	0.33,0.44	<0.01
BMI_c					
	0.3	0.05	5.59	0.19,0.4	<0.01
AlcoholYear					
	0.02	0	5.95	0.01,0.03	<0.01
Gender					
female	Reference	-	-	-	-
male	7.08	0.68	10.43	5.75,8.41	<0.01
PhysActive					
No	Reference	-	-	-	-
Yes	-3.23	0.68	-4.74	-4.57,-1.89	<0.01
Smoker					
0	Reference	-	-	-	-
1	0.2	0.69	0.29	-1.15,1.55	0.77
Drugs					
No	Reference	-	-	-	-
Yes	2.03	0.7	2.91	0.66,3.4	<0.01
Race1					
Black	Reference	-	-	-	-
Hispanic	-8.02	1.92	-4.18	-11.78,-4.26	<0.01
Mexican	-2.81	1.62	-1.73	-5.98,0.37	0.08
White	-4.15	1.15	-3.62	-6.41,-1.9	<0.01
Other	-10.01	1.75	-5.73	-13.44,-6.58	<0.01

Simple Linear Regression

For the SLR and MLR models, age was centered at 40 and BMI was centered at 27. The unadjusted relationship between SBP and each of the covariates: age, BMI, days consuming alcohol over the past year, gender, physical activity, drugs, and race is significant.

Without adjusting for confounders, the average difference in SBP between smokers and ex-smokers is insignificant ($p = 0.8$). The mean SBP for all individuals was 119.20 mmHg. The mean age for all individuals was 41.28 years. The study population consists of 829 females (41%) and 1,177 males (59%).

Table 3a. Main Effects Model

	Estimate	Std. Error	t value	95%CI	Pr(>|t|)
Intercept					
	115.58	1.33	86.87	112.97,118.19	<0.01
Age_c					
	0.35	0.03	12.65	0.3,0.41	<0.01
BMI_c					
	0.26	0.05	5.18	0.16,0.36	<0.01
Gender					
female	Reference	-	-	-	-
male	6.59	0.64	10.26	5.33,7.85	<0.01
Drugs					
No	Reference	-	-	-	-
Yes	0.59	0.65	0.91	-0.68,1.87	0.36
PhysActive					
No	Reference	-	-	-	-
Yes	-1.36	0.66	-2.06	-2.66,-0.06	0.04
Smoker					
0	Reference	-	-	-	-
1	1.97	0.66	2.97	0.67,3.27	<0.01
Race1					
Black	Reference	-	-	-	-
Hispanic	-5.51	1.78	-3.1	-9,-2.02	<0.01
Mexican	-1.56	1.51	-1.03	-4.53,1.41	0.3
White	-3.04	1.07	-2.84	-5.14,-0.94	<0.01
Other	-8.2	1.62	-5.06	-11.37,-5.02	<0.01
AlcoholYear					
	0.01	0	4.8	0.01,0.02	<0.01

Table 3b. Full Model with Interaction

	Estimate	Std. Error	t value	95%CI	Pr(>|t|)
Intercept					
	116.01	1.35	86.18	113.37,118.65	<0.01
Age_c					
	0.36	0.03	12.7	0.3,0.41	<0.01
BMI_c					
	0.26	0.05	5.21	0.16,0.36	<0.01
Gender					
female	Reference	-	-	-	-
male	6.53	0.64	10.16	5.27,7.79	<0.01
Drugs					
No	Reference	-	-	-	-
Yes	0.61	0.65	0.94	-0.66,1.89	0.35
Race1					
Black	Reference	-	-	-	-
Hispanic	-5.4	1.78	-3.03	-8.89,-1.91	<0.01
Mexican	-1.54	1.51	-1.02	-4.51,1.43	0.31
White	-2.87	1.07	-2.67	-4.98,-0.77	0.01
Other	-8.17	1.62	-5.05	-11.35,-5	<0.01
PhysActive					
No	Reference	-	-	-	-
Yes	-1.33	0.66	-2.02	-2.63,-0.04	0.04
Smoker					
	0.95	0.83	1.14	-0.68,2.57	0.25
AlcoholYear					
	0.01	0	2.04	0,0.02	0.04
Smoker:AlcoholYear					
	0.01	0.01	2.04	0,0.02	0.04

Multiple Linear Regression

After adjusting for confounders, smokers had a significant increase in SBP compared to ex-smokers — an estimated increase of 1.97 mmHg on average (95% CI: 0.67, 3.27) ($p < 0.05$).

The alcohol and smoking interaction introduced to the main effects model is significant ($p = 0.04$). The interaction plot shows that there is an overlap of confidence intervals between smokers and ex-smokers only when the estimated days consuming alcohol in the last year is less than approximately 100 (Fig. 1). Indicated by the steeper slope, as more alcohol is consumed, the difference in SBP between smoking status becomes more obvious. Additionally, an ANOVA test compared the full model to the restricted main effects model to test a null hypothesis that the effect of smoking on SBP is insignificant (Table 4). The result, ($F_{2,1995} = 6.495$, $p = 0.0015$), provides strong evidence to reject the null hypothesis and support the claim that the effect of smoking is significant.

Fig. 1. Interaction Plot for SBP

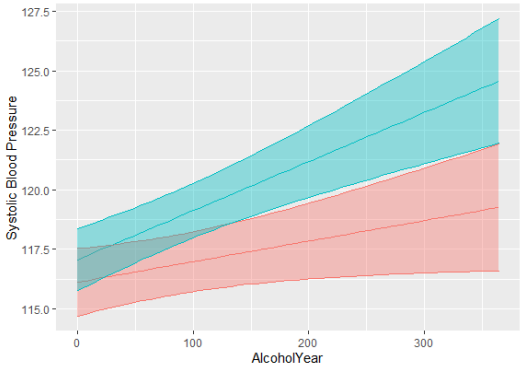


Table 4. ANOVA Table for Restricted Model vs. Full Model

	Res.Df	RSS	Df	Sum of Sq	F	Pr(>F)
Restricted Model	1993	387927.2				
Full Model	1995	390455.6	-2	-2528.388	6.494874	0.0015434

On average, the effect of smoking on SBP was higher among those who consumed more alcohol. For example, we estimated that among participants who did not drink in the past year, smokers tended to have 0.95 mmHg higher SBP on average compared to ex-smokers. Among participants who consumed alcohol 48 days (median value), smokers tended to have 1.52 mmHg higher SBP on average compared to ex-smokers. Among participants who consumed alcohol for 156 days (third quartile), smokers tended to have 2.81 mmHg higher SBP on average compared to ex-smokers. All predictions were adjusted for other covariates.

Comparing the main effects model and the interaction model, adjusted R^2 is investigated. The interaction model has larger adjusted R^2 (0.1742) than the main effect model (0.1729).

Conclusion and Discussion

In summary, the effect of smoking on SBP is significantly modified by alcohol consumption, a result consistent with prior research [28, 29]. In the main effects model, the estimated mean difference in SBP is significantly higher in smokers compared to ex-smokers. This result is consistent with some research papers, but not others [4, 6]. This may result in different population and demographic characteristics between different studies since there are multiple confounders that might contribute to the effect. Moreover, the interaction between alcohol and smoking status might also be a main cause of conflict in literature.

It is crucial to recognize that unweighted NHANES data does not accurately reflect the U.S. population; participants 80+ are recorded as 80, and certain population subgroups, such as racial minorities and low-income Americans, are deliberately oversampled in order to increase health status indicator estimates for those subgroups [30]. Weights are created in NHANES data to accomodate for the aforementioned oversampling; therefore, duplicate samples in the data were maintained. The data was reduced from 10,000 samples to 2,006 samples due to the presence of missing data for the predictor of interest (current smoking habit), outcome (SBP), or confounders (BMI, drug use, physical activity, past year's alcohol consumption). The decision to perform a complete case analysis is supported by other published analyses that perform complete case analyses on data from comparable national health surveys [31, 32, 33]. Other methods for dealing with missing data are used to great success, such as hot deck imputation and reweighting [34]. However, performing complete case analyses introduces no simulated data, and all observations are as accurate as possible. By performing complete case analysis, a significant

difference of characteristics between complete cases and incomplete cases materializes (Table 1a). Consequently, our results and conclusions might lose generalizability to the entire population.

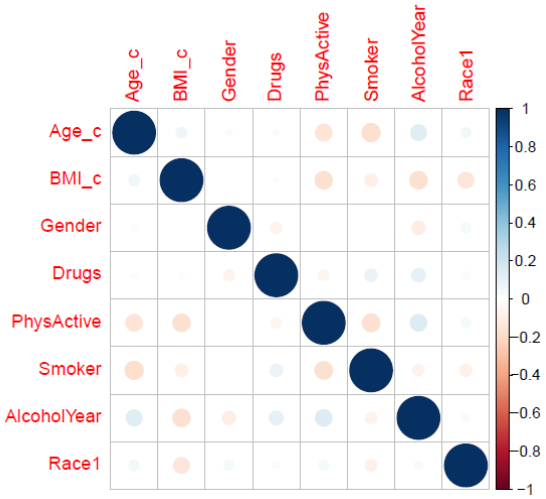
For pre-analysis and model diagnostic after fitting the model, VIF calculations did not raise any multicollinearity concerns (Appendix 1). Since the data was not time series or clustered, we assume the observations are independent. From the partial regression plots (Appendix 2.1), we observe that continuous variables age and BMI each follow a roughly linear relationship with SBP. This supports our decision of not including any nonlinear terms in the model. From the fitted value vs. residual plot (Appendix 2.3), we observe that the points are roughly, randomly scattered around $y = 0$. Therefore, our assumption of equal variances holds. For the Q-Q plot (Appendix 2.2), we could observe that the residuals are slightly right skewed, and this would violate the normality assumption. However, since our sample size is large (n = 2,006), by the central limit theorem, the normality assumption is roughly met.

Outliers were investigated via Cook's distance (Appendix 3) and leverage vs. residual plots (Appendix 4). We observed that observation 1602 could be an outlier, but after investigating the data, it was found that this individual had reportedly consumed alcohol 364 days in the prior year. Since this is possible, and could not be concluded as a typo, observation 1602 was kept in our analysis.

Future work can investigate the relationship between SBP and e-cigarettes, vaping, and more, as the consumption of tobacco and nicotine changes with time. Moreover, data collected with the specific intent of analyzing this relationship will mitigate the level of missing data, reduce bias, and increase accuracy in future work. From this study, cigarette smoking was shown to be a risk factor for hypertension, and this risk factor is exacerbated by alcohol consumption.

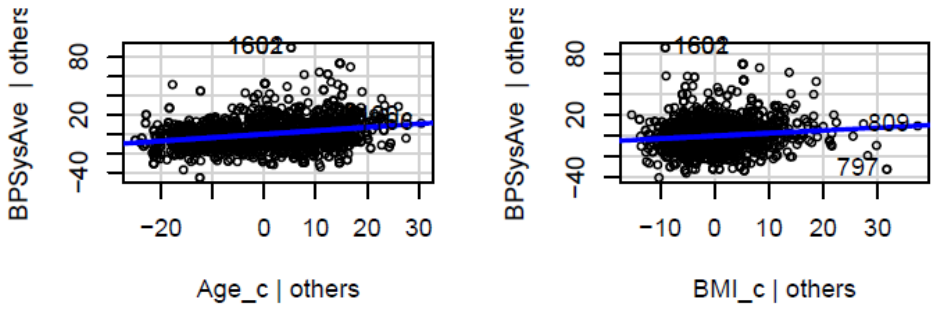
Appendix

Appendix 1. Multicollinearity Diagnostics

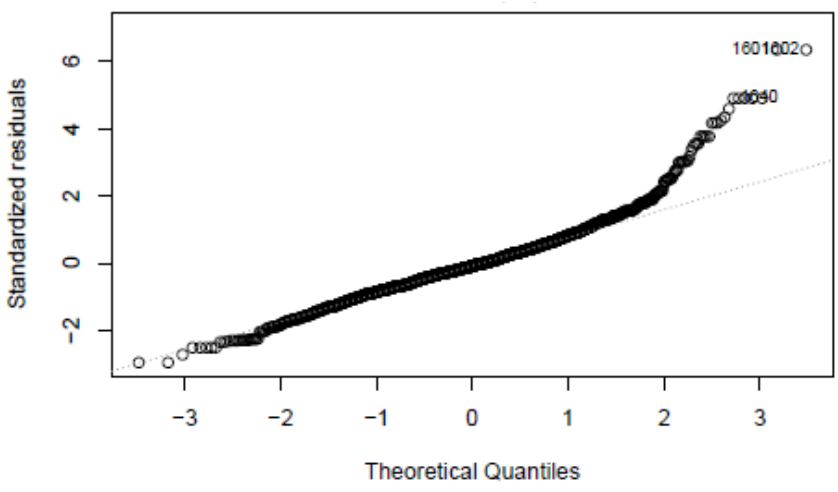


	GVIF	Df	GVIF^(1/(2*Df))
Age_c	1.113896	1	1.055413
BMI_c	1.090924	1	1.044473
Gender	1.032965	1	1.016349
Drugs	1.049163	1	1.024286
Race	1.135374	4	1.015997
PhysActive	1.125995	1	1.061129
Smoker	1.759956	1	1.326633
AlcoholYear	2.129582	1	1.459309
Smoker:AlcoholYear	2.585673	1	1.608003

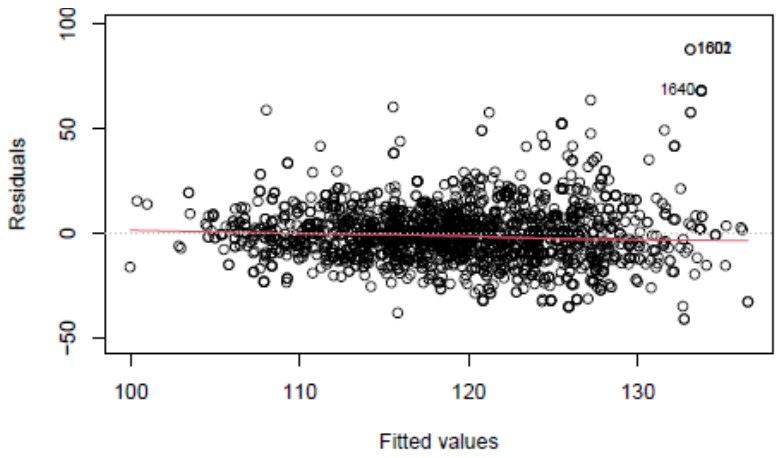
Appendix 2.1. Partial Regression Plots



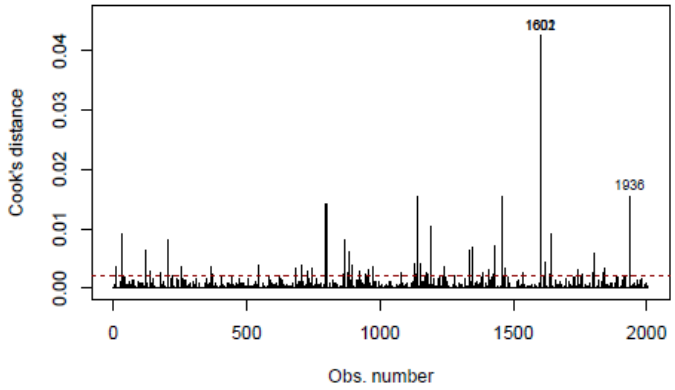
Appendix 2.2. Q-Q Plot



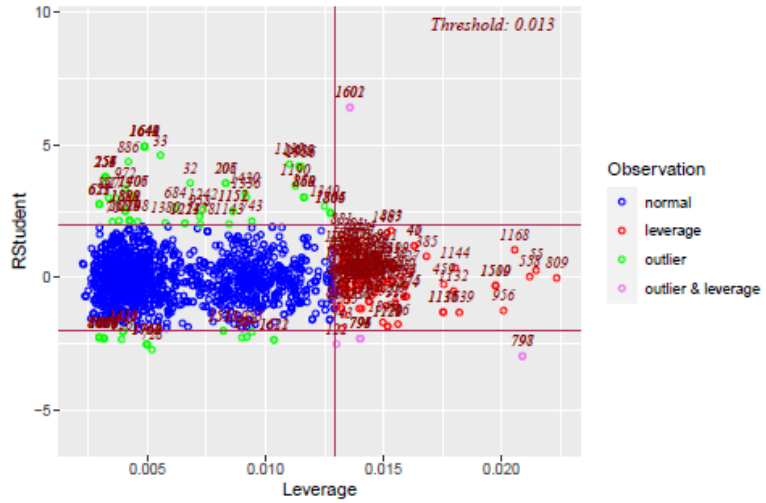
Appendix 2.3. Fitted value vs. Residual plot



Appendix 3. Cook's Distance Plot



Appendix 4. Leverage vs. Externally Studentized Residuals Plot



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Author Contributions

Sweeney, Michael: Conducted literature review. Prepared final transcript.

Tao, Xueting: Main data analysis, conclusion part. Method of model diagnostics

Tiernon, Grace: Conducted literature review to research the background, described the variables, and prepared the final transcript.

Xu, Haisheng: Interpreting a part of results including baseline characteristics, comparisons between smokers and ex-smokers, and unadjusted analysis results.

Xu, Mengqi: Responsible for the methods of statistical analysis in the paper and presentation

Zhang, Han: Main analysis and multiple linear regression part in the paper and presentation.