**Effect of Smoking on Blood Pressure and Heart Rate**

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Cardiovascular diseases (CVD) are the leading cause of morbidity and preventable deaths (Benowitz & Liakoni, 2021; WHO, 2021a). Nearly 18 million people worldwide die every year from CVD and is attributed to about 32% of all deaths of which 85% is attributed to heart attack and stroke (WHO, 2021a). CVD is an umbrella term for disorders of the heart and blood which include, but not limited to, coronary heart disease, cerebrovascular disease, peripheral arterial disease, and rheumatic heart disease (WHO, 2021a). Hypertension, or high blood pressure (HBP), and tobacco consumption are well known risk factors associated with CVD (WHO, 2021b; WHO, 2022). The WHO (2021b) estimated that 1.28 billion adults worldwide have HBP with less than half of them being unaware. Tobacco consumption is responsible for more than 8 million deaths every year and is attributed to about 12% of all deaths (WHO, 2022). Tobacco consumption is estimated to kill about half of all users (WHO, 2022). Acute effects of smoking include, but not limited to, heart attack, stroke, and sudden cardiac death (Benowitz & Liakoni, 2021). Chronic effects of smoking may include, but not limited to, heart failure, irregular heartbeats, and the buildup of plaque in blood vessels (Benowitz & Liakoni, 2021).

Though the immediate effects of increased heart rate (HR) and blood pressure (BP) after smoking is well documented (Alomari & Al-Sheyab, 2016; Alomari et al., 2014), the long-term effect of smoking on BP is still unclear. While some studies suggested that smoking decreases BP (Alomari et al., 2020; Alomari & Al-Sheyab, 2016; Okubo et al., 2003), others indicated an increase in BP (Al-Safi, 2005; D’Elia et al., 2013; Dochi et al. 2009; Gawlik et al., 2018). What is more, some studies suggested that smoking had no effect on BP (Davarian et al., 2013) while others indicated that smoking increased BP in some but decreased BP in others (Al-Safi, 2005; D’Elia et al., 2013; Pandey et al., 2014).

The conflicting results of the effect of smoking on BP may be due to other confounding factors associated with characteristics of the participants such as age, sex, and body mass index (BMI). A study by Davarian et al. (2013) demonstrated that smoking neither decreased nor increased BP nor did smoking increase the prevalence of HBP at older ages. Instead, the study found that age and BMI are more associated with HBP than other sociodemographic factors. This suggests that the association between smoking and BP is weak or absent. This may be in line with the findings by D’Elia et al. (2013) which showed that ex-smokers had significantly higher diastolic BP (DBP) than current smokers which they attributed to the ex-smokers group having higher BMI than the current smokers group. On the other hand, a study by Alomari & Al-Sheyab (2016) showed that a decrease in systolic BP (SBP), DBP, mean arterial pressure (MAP), pulse pressure, and rate pressure products (RPP) in smokers compared to nonsmokers in adolescent boys after controlling for age, BMI, and obesity. Another study by Alomari et al. (2020) indicated that adolescent boys who consume tobacco via cigarettes, waterpipe, or both had decreased heart rates (HR), DBP, MAP, and RPP than nonsmokers. These effects, however, were not found in adolescent girls. These studies suggest a negative correlation between smoking and BP. However, a study by Pandey et al. (2014) showed different BP levels at different ages. The researchers found that participants who consumed tobacco via cigarettes and chewing tobacco had higher BP at younger ages, but only participants who smoked cigarettes showed lower BP levels at older ages while participants who chewed tobacco remained with high levels of BP. This suggests that the method of tobacco consumption may induce different long-term effects.

Other factors associated with participant behavior in regard to tobacco consumption, such as smoking history and smoking frequency, may also confound the association between smoking and BP. A study by Al-Safi (2005) demonstrated a positive dose-effect correlation on HR in men but not women. This may be in line with the study by Alomari et al. (2020) that showed the effect of smoking associated with adolescent boys but not girls which suggests that smoking may affect males differently than females. The study by D’Elia et al. (2013) also found that never smokers showed lower BP than current smokers, and that ex-smokers, over time, showed similar BP levels and risk for HBP as compared to never smokers. It is commonly believed that infrequent smoking, or social smoking, will not significantly increase one’s risk for CVD or HBP; however, a study by Gawlik et al. (2018) demonstrated that even social smoking elevated BP and total cholesterol levels to that of habitual smokers which they found to be significantly higher than nonsmokers. On the other hand, a study by Okubo et al. (2003) found that current smokers showed lower SBP and DBP than never smokers and ex-smokers. The study also found no significant dose-effect correlation between smoking and BP. Furthermore, in a 5-year follow up study, the researchers found that changes in SBP and DBP of current smokers was lower than that of never smokers and ex-smokers. What is even more interesting, the study also found that participants who quitted smoking within the 5-year follow up showed that their BP levels elevated to that of never smokers. Additionally, participants who began smoking within the 5-year follow up showed reduced BP levels similar or even lower than current smokers. This suggests that the association between duration of smoking history and BP is weak or absent.

Other factors unrelated to tobacco consumption may also confound the association between smoking and BP, such as alcohol consumption, working status, exercising, and family history of HBP. A study by Dochi et al. (2009) found that smokers showed increased risk hypertension and systolic hypertension. The researchers suggested that smoking affects SBP more so than DBP. They also found that the percentage of daily drinkers, nonhabitual exercisers, and shift workers was significantly higher in the smokers group than the nonsmokers group. This suggests that drinking, nonhabitual exercise, and shift scheduling is positively associated with hypertension. While family history of HBP understandably increases one’s risk for HBP, it may be an overlooked factor in many studies. For example, a study by Al-Safi (2005) found that smokers had higher levels of SBP, DBP, and MAP than nonsmokers; however, the same study found that participants with a family history of HBP had significantly higher SBP, DBP, MAP, and HR in both the smokers group and nonsmokers group as compared to participants without a family history of HBP.

Because results of the association between smoking and BP is quite equivocal, additional research is needed to better understand the effects of smoking. It is quite evident and well documented that the consumption of tobacco increases one’s risk for CVD, morbidity, and premature death; however, the mechanisms of how smoking increases one’s risk for CVD is still unclear. Therefore, the purpose of this study is to examine the association between smoking cigarettes and BP by analyzing varying levels of smoking frequency. Specifically, this study predicts that if a person smokes more than 5 cigarettes per day, then their SBP, DBP, and HR will be higher than those who smoke only 1-5 cigarettes per day.

**Methods**

**Participants**

The data used is a subset of the Framingham Heart Study that began in 1948. While the original dataset includes n = 4,434 participants, the current study examined the data of n = 780 participants aged 44-80 years (M = 57.29, SD = 7.27). The individuals were selected based on current smoking status. As such, those in the Framingham dataset who self-reported as nonsmokers were excluded from the analysis.

In this study, 51.28% of the participants were female with an average age of 56.63 years (SD = 6.97). The average age of male participants was 57.98 years (SD = 7.52). For this study, the participants were divided into three groups based on the number of cigarettes smoked each day. Participants who smoked 1-5 cigarettes each day belonged in the light group (n = 93) while those who smoked 6-20 cigarettes each day belonged in the moderate group (n = 471). Those who smoked 21 or more cigarettes each day belonged in the heavy group (n = 216). Please refer to Table 1 for the demographic breakdown of the sample.

**Table 1**

*Age Distribution of Participants by Smoking Status*

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Group | Female | | | Male | | |
|  | N | Mean | SD | N | Mean | SD |
| Light | 57 | 59.00 | 7.50 | 36 | 61.47 | 9.21 |
| Moderate | 265 | 56.59 | 6.86 | 206 | 58.85 | 7.33 |
| Heavy | 78 | 55.03 | 5.53 | 138 | 55.77 | 6.73 |
| Total | 400 | 56.63 | 6.97 | 380 | 57.98 | 7.52 |

*Note.* Light = 1-5 cigarettes per day; Moderate = 6-20 cigarettes per day; Heavy = more than 20 cigarettes per day.

**Materials**

The Framingham Heart Study is a long-term prospective study on the causes of CVD. The sample was gathered from the population of individuals living in Framingham, Massachusetts. The Framingham Heart Study was the first prospective study on CVD and materialized the concept of risk factors for CVD. The original study began in 1948 and included n = 5,209 participants. The participants were examined biennially for blood pressure, medication use, smoking history, cholesterol, BMI, diabetes, and heartrate. Through regular surveillance of hospitals, participant contact, and death certificates, the Framingham Heart Study records the occurrence of Angina Pectoris, Myocardial Infraction, Heart Failure, and Cerebrovascular disease.

**Procedure**

The data used in this study is a subset from the Framingham Heart Study that examined n = 4,434 participants over a 24-year period. As this study is the analysis of previously collected data, typical procedures such as informed consent did not apply. The original study collected data during three examination periods, approximately 6 years apart, from roughly 1956 to 1968. Participants were followed during the course of the study for the occurrence of the following events: Angina Pectoris, Myocardial Infraction, Atherothrombotic Infarction, Cerebral Hemorrhage (Stroke), or death. There was no assignment to conditions during the collection of the data as this was a prospective study to identify the risk of CVD.

**Data Analysis**

To examine the current hypothesis of whether there is a significant difference between varying smoking statuses (light, moderate, heavy) in BP and HR, the data was (1) filtered for current smokers only, and then (2) separated into three groups based on the number of cigarettes smoked each day (light = 1-5 cigarettes, moderate = 6-20 cigarettes, heavy = 21 or more cigarettes). Since this study is comparing three independent groups on quantitative dependent variables, this study will analyze the data conducting three separate ANOVA tests for SBP, DBP, and HR.

To further analyze the data, the participants were grouped by sex to examine if any differences exist. Another set of ANOVA tests for SBP, DBP and HR was conducted for females and males. Then sets of t-tests were conducted within each group based on sex.

**Results**

Results from the between-subjects ANOVA for SBP showed a significant difference based on smoking status, F(2, 777) = 3.08, p < 0.05. Specifically, the SBP of participants in the heavy group (M = 137.55, SD = 21.35) was significantly higher than the SBP of those in the moderate group (M = 133.21, SD = 20.72).

Results from the ANOVA for DBP showed a significant difference based on smoking status only at the 0.1 level, F(2, 777) = 2.47, p < 0.1. Specifically, the DBP of participants in the heavy group (M = 81.78, SD = 10.77) was higher than the DBP of those in the moderate group (M = 79.82, SD = 11.22).

Results from the ANOVA for HR did not show any significant differences, F(2, 77) = 0.10, p > 0.05. Please refer to Table 2 for the descriptive statistics of BP and HR measures. Results from the analysis fail to support the hypothesis.

**Table 2**

*Descriptive Statistics for Blood Pressure and Heartrate Based on Smoking Status*

|  |  |  |  |
| --- | --- | --- | --- |
| Group | Mean SBP | Mean DBP | Mean HR |
| Light | 135.06 (24.33) | 79.69 (11.80) | 78.29 (13.67) |
| Moderate | 133.21 (20.72) | 79.82 (11.22) | 78.76 (12.71) |
| Heavy | 137.55 (21.35) | 81.78 (10.77) | 78.37 (11.85) |

*Note.* Standard deviations are presented in parentheses. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate.

Once the data was further divided by sex, results from the ANOVA for SBP showed a significant difference in females based on smoking status only at the 0.1 level, F(2, 397) = 2.34, p > 0.1. Specifically, results showed that the SBP of female participants in the heavy group (M = 138.49, SD = 24.05) was higher than the SBP of females in the moderate group (M = 132.20, SD = 21.98).

Results from a t-test for HR showed that women in the moderate group had higher HR (M = 79.61, SD = 12.52) than men in the same group (M = 77.66, SD = 12.90) only at the 0.1 level, t(469) = -1.66, p > 0.1. No other significant difference between sex was found. Please refer to Table 3 for the descriptive statistics separated by sex, Figure 1 for a bar graph of BP based on smoking status, and Figure 2 for a bar graph of HR based on smoking status.

**Table 3**

*Descriptive Statistics for Blood Pressure and Heartrate Based on Smoking Status and Sex*

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Group | Female | | | Male | | |
|  | Mean SBP | Mean DBP | Mean HR | Mean SBP | Mean DBP | Mean HR |
| Light | 135.13 (26.37) | 78.95 (10.93) | 78.39 (13.3) | 134.94 (21.05) | 80.88 (13.15) | 78.14 (14.42) |
| Moderate | 132.20 (21.98) | 79.30 (11.16) | 79.61 (12.52) | 134.50 (18.95) | 80.49 (11.30) | 77.66 (12.90) |
| Heavy | 138.49 (24.05) | 81.94 (11.66) | 77.24 (11.66) | 137.02 (19.74) | 81.69 (10.28) | 79.00 (11.95) |

*Note.* Standard deviations are presented in parentheses. SBP = systolic blood pressure; DBP = diastolic blood pressure; HR = heart rate.

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**Discussion**

The effects of tobacco consumption on BP and HR are still unclear. The purpose of this study is to examine the differences in BP and HR between smoking status. Results from the analysis failed to support the hypothesis of light smokers having lower SBP, DBP, and HR than moderate and/or heavy smokers; however, differences were found in moderate and heavy smokers. The results showed that participants who were considered heavy smokers (more than 20 cigarettes each day) had a higher SBP than those who were moderate smokers (6 to 19 cigarettes each day). Those who were heavy smokers also showed higher DBP than those who were moderate smokers; however, the performed ANOVA test resulted with only 90% confidence rather than the standard 95%. This is in line with the studies that found the effects of smoking to increase BP (Al-Safi, 2005; D’Elia et al., 2013; Dochi et al., 2009; Gawlik et al., 2018) and opposed studies that found smoking to decrease BP (Alomari & Al-Sheyab, 2016; Alomari et al., 2020; Okubo et al., 2003). The fact that the higher DBP in heavy smokers was found with less confidence may be in line with the study by Dochi et al. (2009) in which they found that smoking affected the SBP more than DBP.

Surprisingly, no significant difference in BP and HR was found between light (1-5 cigarettes each day) and heavy smokers, and between light and moderate smokers. This may be in line with the study by Gawlik et al. (2018) regarding the effects of social smoking. In their study, they found no significant difference between social smokers and habitual smokers. In this study, though, a significant difference was found between moderate smokers and heavy smokers. Because no significant difference was found between light and heavy smokers, it may be likely that other factors, such as age and BMI, needs to be considered. Indeed, the study by Davarian et al. (2013) suggested that age and BMI were more associated with HBP than other sociodemographic factors, even smoking.

Differences in SBP in was found between moderate and heavy smokers in women but not in men. This supports a finding by Alomari et al. (2020) which showed that adolescent girls who smoked had higher BP levels than adolescent boys who also smoked. This study also found that women who were moderate smokers had higher HR than men of the same smoking status. These results oppose the findings from the study by Al-Safi (2005) in regard to the dose-effect relation on HR being present in men but absent in women. Although, these findings together suggests that tobacco consumption may affect men and women differently.

Results from this study failed to support the hypothesis. SBP and DBP was significantly higher in heavy smokers compared to moderate smokers. When comparing by sex, women who were heavy smokers showed higher SBP than women who were moderate smokers. Also, the HR of moderate smokers was significantly higher in women than in men. No significant differences were found between light and heavy, and light and moderate smokers. These results suggest that smoking does, in fact, increase BP and HR; however, uncontrolled sociodemographic factors, such as age, BMI, and family history of HBP, may have confounded the study. Therefore, additional research on the association between smoking and BP is needed. It would be wise for following studies to add additional control for the factors mentioned above.

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