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The acute effect of smoking a single cigarette on vascular status, SpO_2, and stress level

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Statistical Analysis C
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Background: Tobacco use is a major cause of cardiovascular and respiratory disease and cancer. It is controversial whether smoking helps relieve stress. This study investigated the acute effect of smoking a single cigarette on vascular status, SpO_2, and stress level.

Material/Methods: 38 non-smokers and 29 smokers were selected as subjects to measure BVOP (Blood Vessel Output Power), BVT (Blood Vessel Tension), RBV (Remained Blood Volume), BVAL (Blood Vessel Aging Level), SpO_2, resistance to stress, and stress score. Pre- and post-experiment changes were recorded during a 20-min interval, with smokers being recorded 5 min after smoking for the post-experiment.

Results: The smoker group showed a significant decrease in vascular status (BVOP, RBV, BVAL) compared with the non-smoker group. SpO_2 showed no difference between the smoker group and the non-smoker group. Smoking showed no effect on stress reduction compared to non-smoking.

Conclusions: The results of the study indicate that smoking causes a negative effect on vascular status and is not beneficial to stress reduction. Therefore, quitting smoking is encouraged for better physical and mental health.

MeSH Keywords: Smoking – adverse effects • Blood Vessels • Stress, Physiological

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Background

About 5 million people die each year from smoking, meaning that 1 of every 10 people are killed by tobacco use. Tobacco use is a major cause of circulatory diseases and cancer, such as heart attack, stroke, lung cancer, laryngeal cancer, oral cancer, and pancreatic cancer. Moreover, smoking during pregnancy heightens the risk of low birth-weight delivery, infant liver disease, and respiratory diseases such as chronic obstructive pulmonary disease. Hence, smoking is the most serious risk to the health of people in Organization for Economic Cooperation and Development (OECD) countries [1]. The current smoking rate (age over 19 years old, standardized) in South Korea is reported to be 47.3% for males and 6.8% for females in 2011 [2]. The male smoking rate is much higher than average (25.94%) among OECD countries, and is ranked 4th of the 41 OECD countries [1].

Approximately 4000 harmful compounds are found in tobacco. Nicotine, tar, and carbon monoxide (CO) are harmful components in smoke. In 1988, Surgeon General of the United States reported that tobacco is addictive due to nicotine, which is as addictive as heroin or cocaine [3]. Nicotine is an alkaloid found in the leaves of tobacco plants. Once it enters the body, nicotine stimulates catecholamine hormones to facilitate the secretion of adrenocortical hormones, leading to vascular contraction [4]. It increases the concentration of epinephrine and norepinephrine in blood by stimulating the sympathetic nervous system and adrenal medulla, increasing heart rate, blood pressure, and cardiac output. Consequently, it may cause cardiovascular diseases such as coronary artery diseases [5–8]. Tar is composed of carcinogens and reduces the lung functions by being very irritating to the lung tissues, paralyzing lung cilia, reducing its cleaning function, making it dark brown, stimulating mucus secretion of airways, and inducing bronchoconstriction to increase airway resistance [9].

Cigarette contains various kinds of gases. The most harmful is carbon monoxide (CO). CO reduces SpO_2 by reducing the ability of hemoglobin to deliver oxygen because CO has high affinity with red corpuscles (250 times more than with oxygen) after reaching the lungs [10]. Normally, SpO_2 should be maintained at more than 95%. If it becomes lower, it might cause the changes in cognitive functions such as attention, memory, and decision-making ability [11].

A study by Kim examining adult eating and health-related habits reports that 76% of male adults used drinking and smoking to relieve stresses [13]. Research investigating workers' drinking, smoking, and stress showed that the major reason for smoking was habit, followed by relieving stress and interpersonal relationships [14]. This indicates that the rate of smoking for relieving stress is high in adults. In addition, smokers felt that smoking helped relieve and manage their stress in everyday life [14]. Conversely, Parrott [15,16] proposed the nicotine-resource model, which suggests that nicotine might help relieve stress and cause relaxation during smoking. Once people stop smoking, however, there is no nicotine effect and the stress level increases. Thus, the stress level of smokers is higher than that of non-smokers. Lee [17] reported that individuals in a mid-level stress group and a high-level stress group were 1.6 times and 2.3 times, respectively, more likely to smoke than those in a low-level stress group. In Park's study [13], the smoking rate in a high stress group was higher than that of a low stress group. These results show that stress level might affect the smoking rate. However, there has been no empirical evidence that people with high levels of stress are more likely to smoke or that people who smoke are more likely to have high levels of stress. Additionally, the mechanism by which smoking relieves stress has not been clearly explained.

A number of cross-sectional and longitudinal studies have investigated questions of the current status of smoking, factors in smoking, associations between smoking and diseases, and relationship between smoking and stress [11]. As the results of these studies have been reported, smokers are aware of harmful effect of smoking. Nevertheless, it is reported that they smoke because of the habit and the belief that smoking helps relieve stress [13]. This might be because few studies have verified the influence of smoking on human body directly through measuring physiological and biochemical variables. Therefore, to develop smoking prevention and cessation programs and prepare for the basic resources to verify its effectiveness in the future, it is required to conduct research to directly measure the change of physiological and biochemical variables and to change the beliefs that smoking helps relieve stress. The aim of this study was to investigate the effect of smoking a single cigarette on blood vessel conditions and physiological changes of stress level and provide empirical evidence for changing the misconceptions of smoking.

Material and Methods

Subjects

The sampling frame for subjects in this study included residents of Eumseong-gun in Chungcheongbuk-do who had received health services from community health care clinics. Of this population, participating subjects were those who understood the research purposes and agreed to participate in this research. Both smokers (consistently smoked for the last year or more) and never-smokers were recruited. The specific criteria for the selection of subjects were no cognitive and communicative problems and no peripheral circulatory disorders. Subjects were asked to not drink alcohol within 24 hours before the study. The general

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Table 1. Mean of general characteristics of the 2 groups.

	Non-smoking group (n=37)		Smoking group (n=29)		χ^2 or t	*p*
Male (%)	17	(45.9)	20	(69.0)	3.50	.061
Female (%)	20	(54.1)	9	(31.0)		
Age (SD)	69.59	(7.71)	69.55	(8.65)	0.02	.983
Height (SD)	151.70	(7.41)	155.93	(9.53)	1.42	.166
Weight (SD)	56.01	(9.63)	55.04	(8.94)	0.26	.778
Hypertension (%)	17	(45.9)	12	(41.4)	0.06	.804
Diabetes mellitus (%)	4	(10.1)	8	(27.6)	3.08	.079

characteristics of the subjects are shown in Table 1. There was no significant difference in age, height, weight, body mass index (BMI), hypertension, or diabetes mellitus between non-smokers and smokers. All protocols and procedures were approved by the Institutional Review Board of Sahmyook University and all subjects signed a statement of informed consent.

Methods

Blood vessel condition

In this study, blood vessel condition was measured by non-invasive APG (SA6000, Medicore, KOREA). APG (Accelerated Photoplethysmograph) automatically analyzes the condition of blood circulation through double-differentiation of the APG record on the fingertip. This method is used for early diagnosis of cardiovascular diseases such as atherosclerosis and peripheral circulatory disorders. This study analyzed blood vessel condition by using Blood Vessel Output Power (BVOP), Blood Vessel Tension (BVT), Remaining Blood Volume (RBV), and Blood Vessel Aging Level (BVAL) [19]. A higher absolute value of BVOP indicates that a vessel is in better condition. The measured result of the test is better when negative (–) value (absolute) increases. BVT, an index indicating the contraction and relaxation of s blood vessel, has a positive (+) value in normal waves. As blood vessels age, BVT decreases and its measured result indicates a negative (–) value. A lower value of RBV indicates that a vessel is in better condition. The measured test result is better when negative (–) value (absolute) decreases. As blood vessels age, BVOP becomes weak, but RBV increases. BVAL is divided into 7 phases using representative waves that are shown most frequently in level analysis. The progression from phase 1 to phase 7 indicates that the blood vessel aging is severe. To examine the blood vessel condition by using APG, subjects were first asked to relax by lying down for about 5 minutes. The vessel condition was then measured for 5 minutes by attaching the sensor to the index finger.

SpO_2

SpO_2 refers to the percentage (%) of the hemoglobin binding with oxygen (%) [20]. SpO_2 was measured by using non-invasive pulse oximetry (9500, Nonin medical, USA). The examinees were first asked to relax for 5~10 min before the test. SpO_2 was then measured for about 1 min by attaching the sensor to the middle finger.

Stress

Stress can be measured by Heart Rate Variability (HRV) through the analysis of heart electrical activity condition, caused by the influence of sympathetic nervous system and parasympathetic nervous system on sinus node [21]. HRV means minute variance between cardiac cycle and the next following cardiac cycle [22]. This study measured stress by stress resistance and stress scores by analyzing electrocardiogram signals by using a non-invasive complex device, HRV tester (SA6000, Medicore, KOREA). This study analyzed HRV by R-wave signals after recording the R-R distance of normal sinus rhythm for 5 min. Based on this result, stress resistance and stress scores were calculated by a formula [19]. During the measurement process, the examinees rested for 5 min lying down. The positive (+) lead of the stress tester was attached to the right wrist, the negative (–) to the left wrist, and grounding to the left ankle.

Procedures

The data was collected over a 3-week period from August 1 to 22 in 2013. For the preliminary study, of the total residents who agreed to participate in the study, the people who did not smoke were assigned to the control group; the people who

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Table 2. Effect of smoking on vascular status, SpO_2 and stress.

Variables	Non-smoking group (n=37)		p	Smoking group (n=29)		p	Pre-test – Post-test		p
	Pre test M ±SD	Post test M ±SD		Pre test M ±SD	Post test M ±SD		Non-smoking M ±SD	Smoking M ±SD	
BVOP	–70.00±11.10	–71.79±18.63	.481	–69.43±13.04	–61.27±16.02	.001	1.79±15.30	–8.16±12.11	.004
BVT	–17.56±13.41	–15.16±7.90	.273	–23.17±12.68	–24.15±13.13	.710	–2.39±13.07	0.98±13.98	.317
RBV	–39.45±13.62	–36.35±12.41	.074	–38.77±15.76	–45.76±12.77	.012	–3.10±10.24	6.99±13.99	.001
BVAL	3.49±1.02	3.32±1.08	.394	3.79±1.32	4.72±1.79	.001	0.16±1.14	–0.93±1.41	.001
SpO_2	96.93±1.41	97.20±0.93	.223	96.75±1.29	96.95±1.00	.297	–0.22±1.06	–0.14±.69	.732
Resistance of stress	107.30±49.70	109.46±50.88	.672	126.55±53.74	130.00±53.18	.326	–2.16±30.83	–3.45±18.57	.844
Stress score	74.32±17.09	73.24±10.02	.720	72.76±20.51	73.45±20.05	.326	1.08±18.22	–0.69±3.71	.609

BVOP – blood vessel output power; BVT – blood vessel tensionl; RBV – remained blood volume; BVAL – blood vessel aging level; SpO_2 – saturation of partial pressure arterial oxygen.

smoked were assigned to the experimental group. All subjects were asked to complete a survey regarding their sex, age, hypertension, and drinking. In addition, their blood vessel condition, SpO_2, and stress were measured. To ensure stable condition, the test started after having the subjects rest for about 30 min after they arrived. For the pre-test of smokers as the experimental group, based on the average 40 min half-life of nicotine (24~84 min) [23], blood vessel condition, SpO_2, and stress were measured on the condition that the examinees did not smoke at least 2 h before the test. For the post-test, 20 min after the pre-test, the experimental group was asked to smoke a given cigarette (raison, KT & G) down to 1 cm from the filter. The experimental group was tested again within 5 min after smoking. To control the influence of type of cigarette on the results, the same type of cigarette with the same amount of tar and nicotine was given to all smokers. The pre-test for the control group was carried out in the same way as the experimental group. For the post-test, the control group was tested again after having a rest by sitting for 20 min. In measuring blood vessel condition and stress, the room temperature was maintained at 23~25°C and the test was conducted in a well-lit and quiet room in order for the autonomic nervous system not to be affected by the external environment. The subjects were tested while lying comfortably supine with casual clothes on.

Statistical analysis

The data was analyzed by using SPSS version 18 (SPSS, Inc., an IBM Company, Chicago, Illinois, USA). The homogeneity test for the common characteristics of the non-smoking group and the smoking group was analyzed by chi-square test. The effect of before/after-smoking on blood vessel condition, SpO_2, and stress was analyzed by paired t-test. Finally, the difference between before and after smoking was analyzed by independent samples t-test.

Results

The effect of smoking on health condition of blood vessels

First, the result of BVOP for blood vessel condition showed that the non-smoking group had no significant difference in BVOP between the pre-test (–70.00±11.10) and the post-test (–71.79±18.63), (t=0.71, *p*=.481). Conversely, the BVOP of the smoking group showed that there was a statistically significant decrease from the pre-test (–69.43±13.04) to the post-test (–61.27±16.02), (t=–3.63, *p*=.001). The BVOP of pre-/post-tests differed significantly between the non-smoking group and the smoking group (t=2.95 *p*=.004) (Table 2).

BVT did not differ significantly for both the non-smoking group and the smoking group. The BVT of the non-smoking group was –17.56±13.41 in the pre-test and –15.16±7.90 in the post-test, (t=–1.11, *p*=.273). In the smoking group, the BVT was –23.17±12.68 in the pre-test and –24.15±13.13 in the post-test, (t=0.38, *p*=.710), but without significant difference between groups. In addition, there was no significant difference between pre-/post-tests of both the non-smoking group and the group (t=–1.01, *p*=.317) (Table 2).

RBV did not differ in the non-smoking group between the pre-test (–39.45±13.62) and the post-test (–36.35±12.41), (t=–1.84, *p*=.074). Conversely, RBV in the smoking group increased significantly from the pre-test (–38.77±15.76) to the post-test (–45.76±12.77), (t=2.69, *p*=.012). Comparison of the RBV of pre-/post-tests between the non-smoking group and the smoking group showed a statistically significant difference (t=–3.38 *p*=.001) (Table 2).

Finally, BVAL did not differ significantly in the non-smoking group and had no significant difference between the pre-test

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(3.49±1.02) and the post-test (3.32±1.08) (t=0.86, *p*=.394). Conversely, the BVOP of the smoking group increased significantly from the pre-test (3.79±1.32) to the post-test (4.72±1.79) (t=–3.55, *p*=.001). The difference in the aging phase of blood vessels between the pre-test and the post-test for the non-smoking group and the smoking group showed a statistically significant increase (t=3.48 *p*=.001) (Table 2).

The effect of smoking on SpO_2

SpO_2 for both groups did not differ significantly. In particular, the SpO_2 of the non-smoking group was 96.93±1.41% in the pre-test and 97.20±0.93% in the post-test. The SpO_2 of the smoking group was 96.75±1.29% in the pre-test and 96.95±1.00% in the post-test. SpO_2 between the pre-test and the post-test for both groups did not differ significantly (t=–0.35, *p*=.732) (Table 2).

The effect of smoking on stress

Stress resistance of the non-smoking group did not differ significantly between the pre-test (107.30±49.70) and the post-test (109.46±50.88) (t=–0.43, *p*=.672). Stress resistance of the smoking group also did not differ significantly from the pre-test (126.55±53.74) to the post-test (130.00±53.18), (t=–1.00, *p*=.326). Moreover, the difference between the pre-test and the post-test for both non-smoking group and the smoking group was not statistically significant, (t=0.20, *p*=.844) (Table 2).

The stress scores in the non-smoking group tended to decrease from the pre-test (74.32±17.09) to the post-test (73.24±10.02); however, there was no statistically significant difference (t=0.36, *p*=.720). Similarly, the smoking group did not differ significantly between the pre-test (72.76±20.51) and the post-test (73.45±20.05), (t=–1.00, *p*=.326). In addition, the difference between the pre-test and the post-test for both the non-smoking group and the smoking group was not statistically significant, (t=0.51, *p*=.609) (Table 2).

Discussion

This study aimed to investigate the harmfulness of smoking and change misconceptions about smoking by examining the effects of smoking on blood vessel condition, SpO_2, and stress. Of the residents who received health services from community health care clinics in Eumseong-gun in Chungcheongbuk-do, the people who usually smoked were assigned to the smoking group and the people who have never smoked were assigned to the non-smoking group. Blood vessel condition, SpO_2, and stress were measured for smokers before and after they smoked and for non-smokers at the beginning and end of a 20-min interval.

The result showed that the BVOP for the smoking group decreased from pre-smoking to post-smoking, while the RBV and BVAL significantly increased from pre-smoking to post-smoking.

Nevertheless, SpO_2, stress resistance, and stress scores did not decrease. Furthermore, comparison of the difference of pre-/post-tests for non-smoking group and smoking groups showed that the BVOP of the smoking group was statistically lower than in the non-smoking group, while both RBV and BVAL of the smoking group were statistically higher. Therefore, as there were no statistically significant differences in SpO_2, stress resistance, and stress scores between the 2 groups, it seems that smoking did not weaken blood vessel condition and stress.

Moreover, we compared the difference in pre-/post-tests between non-smoking group and smoking groups. Of the blood vessel conditions, BVOP was significantly lower in the smoking group than the non-smoking group. Both RBV and BVAL for the smoking group were higher than for the non-smoking group. This finding indicates that smoking weakens blood vessel function. On the other hand, there was no statistically significant difference in BVT. This result is similar to that of Han's [26] study investigating the effect of smoking on BVT for male adults in their 20s. By measuring BVT by using an arterial compliance tester in smokers and non-smokers, the study reported that there was no statistical difference. However, this result is different from that reported by Kim, et al. [27], performance. They reported that the smoking group had loss of or a problem with the expansion of vessel diameter. Also, they found that vasodilation was inversely proportional to the amount of smoking. The problem with the expansion of vessel diameter in smokers is caused when nicotine in tobacco interacts with the nicotinic cholinoceptor and leads to the hyperactivity of sympathetic nerves by facilitating the secretion of neurotransmitters. It then stimulates lipoprotein lipase, which is important for the metabolism of cholesterol and triglycerides, to increase free fatty acids and VLDL, while it reduces HDL and vascular compliance [28]. Nevertheless, such changes result from the long-term effect of smoking. In light of this, Han's [26] study and the present study did not show significant differences because the former study focused on people in their 20s whose smoking history was short, and because the present study measured the change of vascular compliance after smoking just once. However, this study found a decrease of BVOP and increase of RBV. Moreover, BVAL, estimated by analyzing the waves presented by BVOP and RBV, increased. This is due to vessel contraction, increased heart rate, and increased blood pressure, all of which result from the temporary increase of the concentration of nicotine in blood due to smoking [4,5].

As this study found that there was no statistically significant difference in SpO_2 between before/after smoking, this result is

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the same as in Han's [26] study reporting no statistically significant difference in SpO_2 depending on smoking. However, this differs from Cho's [29] study, which reported that the level of carboxyhemoglobin for the experimental group of smokers suffering from variant angina pectoris decreased from 3.9% to 1.8% after they successfully quit smoking through a smoking cessation program. In Cho's [29] study, the carboxyhemoglobin level was lower after the smoking cessation program than before. When CO is absorbed into the body during smoking, it increases the carboxyhemoglobin level by combining with red corpuscles. However, as the smokers began quitting smoking after the smoking cessation program, CO combined with hemoglobin is discharged, and the carboxyhemoglobin level decreases [26].

Smokers often report that they smoke to relieve stress [13,14]. Smokers also recognized that smoking helped relieve stress [14]. In light of this, this study investigated whether smoking relieves stress objectively by analyzing stress resistance and stress scores using autonomic nervous system (ANS) activity before and after smoking. The result showed that there was no statistically significant difference before and after smoking. This result is the same as in Parrott and Murphy's [16] study, which reported that smoking helps relaxation when the smokers are anxious because of cessation symptoms due to the shortage of nicotine in the body. However, the smoking itself does not help relieve stress. Previous studies [13,17] confirmed that the high stress groups smoke more. Nevertheless, there is no clear evidence that smoking reduces stress; it is verified only by an association between smoking and stress. Thus, it is difficult to set the directionality between 2 variables. For the smoking cessation program, therefore, the results from this study can be used as evidence in a program to change the misconception that a purpose of smoking is to relieve stress.

Conclusions

This study investigated the effects of smoking on blood vessel condition, SpO_2, and stress. The people who had smoked for more than 1 year were assigned to the smoking group and the people who have never smoked were assigned to the non-smoking group. The blood vessel condition, SpO_2, and stress were measured for smokers before and after they smoked. We found that smoking led to decreased BVOP, whereas it increased RBV and BVAL. However, there was no difference in SpO_2 and stress before and after smoking. This result implies that smoking increases the risk of cardiovascular disease by aggravating blood vessel condition. At the same time, smoking is not an effective way to relieve stress. In this study, the long-term variables that do not change over a short time period did not show a statistically significant difference because this study measured the change after smoking just once. To measure these variables, it is necessary to evaluate the long-term effects of smoking. Moreover, this study measured stress by using autonomic nervous system (ANS) activity even though the stress was a subjective variable. In light of this, further research is needed to conduct a comparative analysis of the effect by using an instrument to assess stress subjectively.

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