Mentholated cigarette smoking and brachial artery, carotid artery, and aortic vascular function

Mentollü sigara içilmesi ve brakiyal arter, karotis arter, aort damar işlevleri

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Objectives: We investigated possible acute effects of mentholated versus nonmentholated cigarette smoking on vascular functions and left ventricular diastolic functions in otherwise healthy young smokers.

Study design: The study included 20 otherwise healthy smokers (6 women, 14 men; mean age 25.6 years) and 22 healthy nonsmokers (12 women, 10 men; mean age 25.1 years). Ultrasound and echocardiographic examinations were performed to determine baseline characteristics for the brachial artery, aorta, and carotid artery, including brachial flow-mediated dilation (FMD), aortic and carotid stiffness index (SI), distensibility, and elastic modulus (EM). On day 2, each subject smoked either two mentholated or nonmentholated cigarettes and ultrasound and echocardiographic examinations were repeated. The procedure was repeated 15 days later with each subject smoking the other type of cigarette.

Results: From the baseline level of 14.0±9.0%, FMD decreased significantly to 8.3±3.2% (p=0.012) and to 9.8±5.5% (p=0.025) after smoking mentholated and nonmentholated cigarettes, respectively. Increase in systolic blood pressure was significant only with mentholated cigarettes (p=0.003). Increases in heart rate and rate-pressure product were significant in both groups, being more prominent with mentholated cigarettes. Both types of cigarettes resulted in significant prolongation of mitral E deceleration time and decrease in mitral E/A ratio. Changes in aortic SI and EM were significant only with mentholated cigarettes, while changes in carotid SI and EM were significant in both groups. Menthol-associated changes in systolic blood pressure, heart rate, rate-pressure product, carotid strain, and carotid SI parameters differed significantly from those seen after nonmentholated cigarette smoking (p=0.027, p<0.001, p<0.001, p=0.037, and p<0.001, respectively).

Conclusion: Our findings show that mentholated cigarettes are not safer than nonmentholated cigarettes and that menthol-associated acute impairment is more severe in many parameters of elasticity and stiffness.

Key words: Aorta; blood pressure; brachial artery; carotid arteries; elasticity; hemodynamics; menthol/adverse effects; smoking.

Amaç: Bu çalışmada, başka açıdan sağlık sorunu olmayan gençlerde mentollü ve mentollü olmayan sigara kullanımının vasküler fonksiyonlara ve sol ventrikül diyastolik fonksiyonlara etkisi araştırıldı.

Çalışma planı: Çalışmaya sigara içen 20 (6 kadın, 14 erkek; ort. yaş 25.6) ve sigara kullanmayan 22 (12 kadın, 10 erkek; ort. yaş 25.1) sağlıklı kişi alındı. Deneklerin başlangıç özelliklerini belirlemek için ultrason ve ekokardiyografik incelemeler yapıldı; brakiyal arter, aort ve karotis arter incelemelerinden brakiyal akım aracılı genişleme (FMD), aort ve karotis arterler için sertleşme indeksi (Sİ), distensibilite ve elastik modulus (EM) ölçüldü. İkinci gün, her bir gönüllüye iki adet mentollü ya da mentolsüz sigara içirilerek ultrason ve ekokardiyografik incelemeler tekrarlandı. On beş gün sonra, her bir deneğe ilk ölçümde kullanılan sigara yerine diğeri içirilerek ölçümler tekrarlandı.

Bulgular: Mentollü ve mentolsüz sigara içilmesinden sonra FMD değerleri %14.0±9.0'dan sırasıyla %8.3±3.2 (p=0.012) ve %9.8±5.5'e (p=0.025) geriledi. Sistolik kan basıncında anlamlı artış sadece mentollü sigara içimiyle görüldü (p=0.003). Kalp hızı ve hız-basınç ürünü değerleri, mentollü sigarayla daha belirgin olmak üzere, iki grupta da anlamlı artış gösterdi. İki tür sigarayla da, mitral E yavaşlama zamanında anlamlı artış ve mitral E/A oranında anlamlı düşüş gözlendi. Aorta ait Sİ ve EM değerleri sadece mentollü sigara kullanımı sonrası anlamlı değişim gösterirken, karotis Sİ ve EM değerleri iki tür sigara ile de anlamlı değişim gösterdi. Sigara içiminden sonraki değerlerin karşılaştırılmasında, mentollü sigara kullanımında, sistolik kan basıncı, kalp hızı, hız-basınç ürünü, karotis gerinimi ve karotis Sİ parametrelerinde, mentolsüz sigara içimine göre anlamlı farklılık saptandı (sırasıyla, p=0.027, p<0.001, p<0.001, p=0.037 ve p<0.001).

Sonuç: Bulgularımız mentollü sigaranın mentolsüz sigaradan daha az zararlı olmadığını; hatta, mentollü sigaranın elastisite ve sertlikle ilgili parametrelere daha ciddi akut zararlar verdiğini göstermektedir.

Anahtar sözcükler: Aort; kan basıncı; brakiyal arter; karotis arter; elastisite; hemodinami; mentol/zararlı etki; sigara içme.

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Cigarette smoke contains thousands of toxic components. It has been suggested that the endothelium is the main target of these toxic compounds.[1] One of them is nicotine and it causes increased endothelial cell proliferation, intimal hyperplasia, and increased serum carbon monoxide levels. Even smaller amounts of nicotine than those contained in cigarette smoke can cause acute endothelial dysfunction. Free radicals in cigarette smoke tar can damage the vascular endothelium.[1]

Commercially available for a long-time, mentholated cigarettes are increasingly consumed.[2,3] Menthol has numerous biological actions. Its cooling effect may contribute to the intensity of smoking (deeper inhalation and/or more prolonged breath holding) resulting in greater exposure to tobacco smoke toxins. The effect of menthol through increasing the permeability of cell membranes results in a greater absorption of smoked toxins.[2,3] Ahijevych and Garrett[2] reported higher cotinine levels in mentholated cigarettes compared to nonmentholated cigarettes, suggesting greater nicotine absorption per cigarette. Clarke et al.[3] found that mentholated cigarette smoking was associated with higher serum cotinine and carbon monoxide levels per cigarette.

These reports are consistent with the concept that menthol increases inhalation and/or absorption of more than 4,000 toxic ingredients of tobacco smoke. Additionally, mentholated cigarette smoking significantly inhibits the metabolism of nicotine.[2,3]

Impaired flow-mediated dilation (FMD) of the brachial artery is a diffuse disease process resulting in abnormal regulation of blood vessel tone and loss of several atheroprotective effects of the normal endothelium; thus, it may be a marker of increased risk for cardiovascular disease.[4]

Arterial stiffening is an important cardiovascular risk factor and an independent predictor of all-cause and cardiovascular death.[5] Increased aortic stiffness index (AoSI) or elastic modulus (AoEM) and/or decreased aortic distensibility (AoD) may reflect the widespread nature of the atherosclerotic process. Since atherosclerosis can affect the aorta and the coronary arteries simultaneously, aortic stiffness and distensibility may predict cardiovascular events.[5] In acute and chronic smokers, it has been shown that compliance of large- and medium-sized arteries decreases after smoking one cigarette.[6] Even in the absence of atherosclerosis of the vessels, active or passive smoking is associated with increased arterial stiffness.[6-8] Based on these findings, it may be suggested that the majority of the effects of smoking on arterial stiffness are mediated by smoking-related endothelial dysfunction.[8] It is known that the adverse effect of smoking on the endothelial functions is the same regardless of the number of cigarettes smoked a day.[9]

The type of the cigarette smoked affects lung cancer risk.[10] To date, however, there has been no study that has comprehensively investigated the cardiovascular effects of mentholated cigarettes. In the present study, we aimed to compare possible acute effects of mentholated versus nonmentholated cigarette smoking on vascular functions and left ventricular diastolic functions in otherwise healthy young smokers.

PARTICIPANTS AND METHODS

Study population. Twenty healthy smokers (6 women, 14 men; mean age 25.6±6.4 years) and 22 healthy nonsmoking volunteers (12 women, 10 men; mean age 25.1±4.2 years) from our hospital staff were consecutively enrolled in the study. Inclusion criteria were being a chronic smoker, being free of coronary risk factors other than smoking and, for women, being on a regular menstrual cycle. Exclusion criteria included the presence of any disease associated with left ventricular diastolic dysfunction or impairment in the elastic properties of the aortic and carotid artery or brachial FMD (eg, hypertension, diabetes mellitus, or family history of coronary artery disease), alcohol use, and obesity (body mass index greater than 30 kg/m^2). Subjects taking any vasoactive drug and those exhibiting electrocardiogram changes implicating coronary heart disease were excluded. Women were evaluated within the follicular phase of their menstrual cycle.

A complete physical examination was performed. We checked peripheral arterial pulses and searched for carotid bruits. Blood pressure was recorded in the sitting position. Each subject was questioned about alcohol consumption and major cardiovascular risk factors. Blood glucose, total cholesterol, high-density lipoprotein, and low-density lipoprotein cholesterol levels were measured in venous blood samples after at least 12-hour fasting. Plasma high-sensitivity C-reactive protein levels were measured by a highly sensitive sandwich ELISA technique. Each subject underwent echocardiographic examination to measure left ventricular diastolic function, AoSI, AoD, AoEM, carotid artery stiffness index (carotid SI), carotid artery distensibility (carotid D), carotid artery elastic modulus (carotid EM), and brachial FMD during reactive hyperemia measurement.

The study was conducted in compliance with the Declaration of Helsinki on Biomedical Research Involving Human Subjects. The institutional ethics committee approved the study protocol and written informed consent was obtained from each subject.

Study design. We compared the acute effects of mentholated (0.9 mg nicotine, 11 mg tar, 12 mg carbon monoxide) and nonmentholated (0.9 mg nicotine, 12 mg tar, 12 mg carbon monoxide) cigarettes on the FMD of the brachial artery and on elastic properties of the carotid and aortic arteries in otherwise healthy smokers. Initially, each subject underwent ultrasound and echocardiographic examinations of the brachial artery, aorta, carotid artery, whereby FMD, AoSI, AoEM, AoD, carotid SI, carotid D, carotid EM, and left ventricular diastolic functions were measured after 12-hour fasting to determine the individual baseline characteristics for atherosclerotic predictors.

On the second day, each subject was asked to smoke, in a closed room, either two mentholated (0.9 mg nicotine, 11 mg tar, 12 mg carbon monoxide) or nonmentholated (0.9 mg nicotine, 12 mg tar, 12 mg carbon monoxide) cigarettes within 15 minutes. Then, within 20 to 30 minutes, each subject underwent vascular ultrasound and echocardiographic examinations. Fifteen days later, the procedure was repeated with the subject smoking the type of cigarette different from the former.

Measurement of brachial FMD. Flow-mediated dilation of the brachial artery following transient ischemia was evaluated as previously described.[4] A high-resolution 7.5-MHz linear array ultrasound transducer (attached to a Hitachi EUB 6500, Japan) was used. All the measurements were made by the same investigator blinded to the clinical data. All subjects were examined after a 12-hour fasting, and during abstinence from caffeine or xanthine-derivative-containing drinks for at least 12 hours. Women were evaluated during the follicular phase of their menstrual cycle. The brachial artery was scanned in the longitudinal section 3 to 5 cm above the antecubital fossa. Three consecutive measurements (obtained through consecutive cardiac cycles) were averaged and recorded. After baseline measurements of the brachial artery, the cuff was placed proximal to the section of the brachial artery and inflated to 250 mmHg (or 50 mmHg higher than the systolic blood pressure) and kept in the same pressure for 4.5 minutes to induce forearm ischemia. Subsequently, the cuff was deflated, and the arterial diameter was measured 60 seconds after cuff release.

All recordings were stored on a videotape to be analyzed later. At the end of the study, two specialists, unaware of the clinical data, analyzed the recordings independently. Endothelium-dependent dilatation was expressed as the percent change in the internal diameter of the brachial artery from baseline *(i)* to subsequent reactive hyperemia and *(ii)* to subsequent sublingual nitroglycerine administration.

The interobserver and intraobserver concordance correlation coefficients were 0.913 and 0.923 for the brachial artery measurements, 0.941 and 0.952 for flow-mediated dilatation, 0.935 and 0.947 for carotid strain, 0.922 and 0.931 for carotid distensibility, 0.924 and 0.937 for aortic stiffness, 0.928 and 0.943 for aortic elastic modulus, respectively.

Echocardiographic examination. Each subject was examined using the Acuson Sequoia C256 echocardiography system (Acuson Corp, Mountain View, CA, USA) equipped with a 3V2c broadband transducer for second harmonic imaging. Two-dimensional, M-mode, and subsequent standard and pulsed tissue Doppler echocardiographic examinations were performed in the lateral decubitus position. The echocardiographic tracings were recorded on VHS videotapes. All measurements were performed by M-mode imaging.

The pulsed Doppler sample volume was positioned at the tip of the mitral leaflets. Early diastolic peak flow velocity (E), late diastolic peak flow velocity (A), E/A ratio, and E wave deceleration time were measured by transmitral Doppler imaging.

Left ventricular mass determination. Left ventricular mass (LVM) was calculated from M-mode records taken on parasternal long-axis images according to the formula defined by Devereux and Reichek.[11] The left ventricular mass index (LVMI) was expressed as LVM per meter squared of the body surface area.

Measurements of aortic distensibility and stiffness.Patients were examined in the left lateral decubitus position. The operator measured the internal dimensions of the transverse aortic arch in at least three consecutive cardiac cycles using the Acuson Sequoia C256 echocardiography system equipped with a broadband transducer with second harmonic capability (3V2c). The measurements were carried out in the proximal ascending aorta, 3 cm from the origin of the aorta. Aortic strain, AoD, AoSI, AoEM were calculated using the following formulas:[5]

Table 1. Baseline demographic and biochemical characteristics smokers and nonsmokers

	Smokers (n=20)	Nonsmokers (n=22)	*p*
Age (years)	25.6±6.4	25.1±4.2	0.176
Body mass index (kg/m^2)	23.8±3.5	23.9±3.3	0.934
Systolic blood pressure (mmHg)	119.5±11.4	112.9±13.3	0.162
Diastolic blood pressure (mmHg)	69.5±9.4	72.0±7.7	0.230
Heart rate (bpm)	69.8±8.4	71.9±11.0	0.337
Glucose (mg/dl)	87.3±4.6	88.0±5.1	0.762
Total cholesterol (mg/dl)	158.4±33.7	161.5±35.8	0.734
Triglyceride (mg/dl)	104.7±71.2	105.7±71.9	0.821
HDL-cholesterol (mg/dl)	42.8±8.0	44.4±7.9	0.917
LDL-cholesterol (mg/dl)	92.9±20.3	96.1±21.7	0.685
High-sensitivity C-reactive protein (mg/l)	1.57±1.2	1.76±1.4	0.563
Left ventricular mass index (g/m^2)	76.3±12.6	75.1±14.4	0.936
Mitral E max (cm/sec)	84.0±13.1	82.7±10.0	0.705
Mitral A max (cm/sec)	50.9±8.8	54.1±7.9	0.225
Mitral E/A ratio	1.7±0.3	1.6±0.3	0.124

- Aortic/carotid strain: $100 \times (D_s\text{-}D_d)/D_d$.
  (D_s and D_d: aortic/carotid diameter at systole and diastole, respectively)
- Aortic root/carotid distensibility:
  $2 \times [(D_s\text{-}D_d)/D_d]/dP$ ($cm^2 \times dyn^{-1} \times 10^{-6}$).
  (dP: Systolic-diastolic pressure change)
- Aortic/carotid stiffness index:
  Ln $(P_s/P_d)/[(D_s\text{-}D_d)/D_d]$.
  (Ln: Logarithm base n; P_s: Systolic blood pressure; P_d: Diastolic blood pressure)
- Aortic/carotid pressure elastic modulus:
  $dP/[(D_s\text{-}D_d)/D_d]$ ($cm^2 \times dyn^{-1} \times 10^{-6}$).

Statistical analyses. Statistical analyses were performed using the SPSS 10.0 (SPSS for Windows, version 10.0, Chicago, IL, USA) software package. Numeric values were expressed as mean ± standard deviation (SD). Statistically different parameters were determined by the Friedman test. Measurements (including changes) at baseline and after smoking mentholated and nonmentholated cigarettes were compared using the Wilcoxon signed-rank test. A *p* value of less than 0.05 was considered significant.

RESULTS

There were no differences between the smokers and nonsmokers with regard to age, body mass index, blood pressure, heart rate, and the levels of glucose, cholesterol, and high-sensitivity C-reactive protein (Table 1). Basal and hyperemic brachial artery diameters were similar after mentholated and nonmentholated cigarette smoking. Compared to baseline levels, both groups had significantly lower FMD values after smoking (Table 2). However, FMD values after smoking mentholated and nonmentholated cigarettes were similar ($p>0.05$). Mitral A velocities, mitral E deceleration time and mitral E/A ratios were similar between the two groups (Table 2).

Systolic blood pressure, heart rate, and rate-pressure product values increased after smoking both mentholated and nonmentholated cigarettes. The increase in systolic blood pressure after smoking a nonmentholated cigarette did not reach a statistical significance. Albeit not significantly, diastolic blood pressure increased after smoking mentholated cigarettes, but did not change after nonmentholated cigarette smoking. After smoking mentholated cigarettes, the measurements of heart rate, and rate-pressure product were statistically different from the values obtained after smoking nonmentholated cigarettes. (Table 2).

Compared with baseline measurements, smoking both types of cigarettes resulted in significant prolongation of mitral E deceleration time (mentholated $p=0.015$; nonmentholated $p=0.020$), and significant decreases in mitral E/A ratio (mentholated $p=0.021$; nonmentholated $p=0.025$) (Table 2).

The elastic properties of the aortic and carotid arteries changed with increased stiffness 20 to 30 minutes after smoking of two cigarettes. However, changes in AoD and carotid strain did not reach statistical significance for both types of cigarettes ($p>0.05$). After smoking nonmentholated cigarettes, AoSI and AoEM slightly increased compared with baseline ($p=0.062$ and $p=0.059$, respectively).

Menthol-associated changes in systolic blood pressure, heart rate, rate-pressure product, carotid strain,

Table 2. Hemodynamic, echocardiographic, and flow-mediated dilation findings, and elastic properties of the aortic and carotid artery in mentholated/nonmentholated cigarette smokers

		After smoking		*p*		
	Baseline (1)	Mentholated (2)	Nonmentholated (3)	1 *vs.* 2	1 *vs.* 3	2 *vs.* 3
Systolic blood pressure (mmHg)	117.75±15.34	130.67±20.83	118.00±17.35	**0.003**	0.793	**0.027**
Diastolic blood pressure (mmHg)	71.25±9.85	73.47±8.75	70.00±11.24	0.547	0.283	0.155
Heart rate	69.00±9.90	101.20±10.91	82.05±13.48	**<0.001**	**0.001**	**<0.001**
Brachial artery diameter (mm)						
Baseline	3.82±0.53	3.71±0.47	3.78±0.54	0.777	0.961	0.765
Hyperemic	4.33±0.51	4.01±0.49	4.12±0.45	0.117	0.398	0.401
Flow-mediated dilation (FMD)						
In millimeters	0.51±0.28	0.30±0.12	0.35±0.15	**0.007**	**0.011**	0.499
In percentage	14.01±9.04	8.29±3.17	9.77±5.50	**0.012**	**0.025**	0.550
Mitral						
E max (cm/sec)	87.50±17.04	83.73±8.63	90.75±13.75	0.390	0.225	**0.044**
A max (cm/sec)	50.90±8.84	57.67±12.64	60.25±12.50	**0.040**	**0.014**	0.616
E deceleration time (msec)	178.30±27.50	202.80±36.86	194.15±22.42	**0.015**	**0.020**	0.546
E/A ratio	1.76±0.42	1.52±0.38	1.57±0.40	**0.021**	**0.025**	0.421
Rate-pressure product	8147.0±1628.9	13274.9±2840.0	9734.8±2521.2	**<0.001**	**0.033**	**<0.001**
Systolic aortic diameter (mm)	26.40±2.14	28.20±3.30	27.88±6.89	**0.034**	0.590	0.121
Diastolic aortic diameter (mm)	23.50±1.93	25.90±3.74	25.50±6.57	**0.020**	**0.030**	0.222
Aortic						
Strain (%)	12.47±4.98	9.28±3.2	9.58±3.62	**0.021**	**0.009**	0.845
Distensibility (cm^2 $dyne^{-1}$ 10^{-6})	4.61±3.00	3.51±1.87	4.13±1.60	0.433	0.970	0.078
Stiffness index	5.05±3.47	7.63±4.58	6.28±3.19	**0.005**	0.062	0.260
Elastic modulus (cm^2 $dyne^{-1}$ 10^{-6})	4.61±3.00	7.65±4.78	5.71±2.75	**0.002**	0.590	0.071
Carotid						
Strain (%)	11.55±4.30	10.80±3.31	8.92±3.75	0.526	0.052	**0.037**
Distensibility (cm^2 $dyne^{-1}$ 10^{-6})	5.25±2.46	4.12±1.93	3.84±1.65	**0.040**	**0.015**	0.758
Stiffness index	1.64±0.59	5.69±1.81	2.16±0.72	**<0.001**	**0.010**	**<0.001**
Elastic modulus (cm^2 $dyne^{-1}$ 10^{-6})	4.65±2.05	5.81±2.56	6.15±2.55	**0.035**	**0.021**	0.745

and carotid SI parameters differed significantly from those occurred after smoking nonmentholated cigarettes ($p=0.027$, $p<0.001$, $p<0.001$, $p=0.037$, and $p<0.001$, respectively; Table 2).

DISCUSSION

This study demonstrated that either mentholated or nonmentholated cigarettes had acute detrimental effects on the arterial system. Smoking mentholated cigarettes impairs FMD, AoSI, AoD, and AoEM, left ventricular diastolic functions in a similar manner as does smoking nonmentholated cigarettes. Both kinds of cigarettes acutely impaired FMD and the elastic properties of the aortic artery. Systolic blood pressure, heart rate, rate-pressure product, carotid strain, and carotid SI values indicated that smoking mentholated cigarettes impaired vascular elastic properties to a greater degree than did smoking nonmentholated cigarettes. Our study is the first to demonstrate that smoking mentholated and nonmentholated cigarettes has similar hazardous acute effects on the cardiovascular system.

The market share of mentholated cigarettes has increased substantially over the past decades.[12] Mentholation of cigarettes has aroused considerable concern because of the high rates of lung cancer in African-American smokers, most of whom smoke mentholated cigarettes, compared to whites, who predominantly prefer nonmentholated cigarettes.[11,13] Menthol is known to stimulate cold receptors and to produce a cooling sensation as well as local anesthesia. In animals, menthol inhalation resulted in longer air retention time in the lungs.[14,15] Furthermore, many African-Americans report ease of inhalation and ability to inhale more deeply as reasons for smoking mentholated cigarettes.[16] It is therefore reasonable to infer that mentholation of tobacco might increase the depth of inhalation and/or the duration of smoke retention in the lungs, resulting in greater exposure to carcinogens. The knowledge that menthol enhances dermal absorption of various drugs[17,18] has raised concern about its contribution to enhance lung permeability to toxic chemicals in tobacco smoke. Additionally, menthol was reported to alter hepatic drug-metabolizing enzyme levels in rats.[19]

MacDougall et al.[20] reported that menthol inhibited nicotine metabolism in human microsomes. Menthol inhibited the metabolism of nicotine and cotinine.[21] Menthol could affect human nicotine metabolism and, therefore, smoking behavior could affect the metabolic activation or detoxification of toxic tobacco-related compounds. Compared to that of nonmentholated cigarettes, the cooler taste of mentholated cigarettes might contribute to a false psychological perception of safety.[12]

Neunteufl et al.[22] showed that nicotine caused acute endothelial dysfunction in long-term smokers, suggesting that there might be some contribution of ingredients of cigarette smoke to this adverse effect. Our study provided evidence that acute smoking of mentholated cigarettes was associated with impairment of endothelial function 20 to 30 minutes after smoking.

Evidence from both animal and human studies suggests that the endothelium is an important regulator of arterial stiffness, both functionally and structurally.[5] A recent study demonstrated increased aortic stiffness in patients with hypertension, end-stage renal disease, Marfan syndrome, and obesity.[23] In our study, smoking mentholated and nonmentholated cigarettes resulted in increased AoEM and AoSI. We found significant impairments after smoking mentholated versus nonmentholated cigarettes in blood pressure, heart rate and, therefore, rate-pressure product, carotid strain, and carotid SI (Table 2). This could be explained by acute increments in blood nicotine levels caused by mentholated cigarette-induced activation of the sympathetic nervous system, resulting in greater increments in systolic blood pressure, heart rate, and rate-pressure product after smoking mentholated cigarettes. Benowitz et al.[21] reported that blood nicotine levels were slightly higher while smoking mentholated cigarettes.[21]

Our study showed that smoking mentholated and nonmentholated cigarettes has similar acute detrimental effects on endothelial function and the elastic properties of the aortic artery. Considering the elastic properties of the aortic artery and rate-pressure product, acute detrimental effects may be aggravated by smoking mentholated cigarettes by means of sympathetic activation.

In conclusion, our findings show that smoking mentholated and nonmentholated cigarettes has a similar negative effect on the arterial system. On the other hand, compared with nonmentholated cigarettes, mentholated cigarettes might cause more prominent unfavorable acute effects on the elastic properties of the carotid artery. Further studies are needed to investigate the possible chronic effects of smoking mentholated cigarettes on the cardiovascular system.

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