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Original Research Article

Comparative Study of Cardiovascular Reactivity to Cold Pressor Test in Smokers vs Non-Smokers

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Abstract

Introduction:

Cigarette smoking is associated with autonomic dysregulation, endothelial dysfunction, and increased cardiovascular risk. The cold pressor test (CPT) is a validated method for assessing sympathetic cardiovascular reactivity. This study compared cardiovascular responses to CPT between smokers and non-smokers and evaluated post-stimulus recovery patterns.

Methods:

A comparative cross-sectional study was conducted among 60 apparently healthy male participants aged 18–35 years, including 30 smokers and 30 non-smokers. Baseline heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were recorded. Participants underwent CPT by immersing the non-dominant hand in ice water (4–6°C) for one minute. Peak cardiovascular responses and recovery parameters at 1 and 3 minutes post-test were assessed. Data were analysed using Student's t-test and Pearson correlation analysis.

Results:

Baseline demographic and cardiovascular characteristics were comparable between groups. Following CPT, smokers demonstrated significantly greater cardiovascular reactivity than non-smokers, with higher increases in HR (13.9±4.5 vs. 8.8±3.6 beats/min), SBP (21.3±5.8 vs. 15.4±4.9 mmHg), and DBP (14.6±4.7 vs. 9.8±3.9 mmHg) (all $p<0.001$). Smokers also exhibited significantly higher HR and blood pressure values during the recovery phase, indicating delayed autonomic recovery. Smoking duration showed a positive correlation with SBP reactivity ($r=0.42$, $p=0.021$) and HR response ($r=0.37$, $p=0.039$).

Conclusion:

Smokers exhibit exaggerated cardiovascular responses and delayed recovery following cold pressor stimulation, suggesting enhanced sympathetic activity and impaired autonomic regulation. These findings indicate early subclinical cardiovascular dysfunction in apparently healthy smokers and underscore the importance of smoking cessation for cardiovascular risk reduction.

Keywords:

Smoking; Cold Pressor Test; Cardiovascular Reactivity; Autonomic Nervous System; Sympathetic Activity.

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Introduction

Smoking remains one of the leading preventable causes of cardiovascular morbidity and mortality worldwide. Chronic exposure to nicotine and other tobacco constituents has been associated with altered autonomic nervous system activity, endothelial dysfunction, and exaggerated sympathetic responses, all of which may adversely affect cardiovascular regulation [1,2]. The cold pressor test is a simple, non-invasive autonomic function test that evaluates cardiovascular reactivity by inducing sympathetic stimulation through cold-induced nociceptive stress [3]. During the test, immersion of the hand in ice-cold water produces a transient rise in heart rate and blood pressure mediated primarily through sympathetic activation and peripheral vasoconstriction [4]. Exaggerated cardiovascular responses to stress have been linked with increased future risk of hypertension and cardiovascular disease [5]. Although smoking-related autonomic alterations are well recognized, comparative assessment of cardiovascular reactivity among smokers and non-smokers in young adults remains insufficiently explored in many populations. The present study was therefore undertaken to evaluate and compare cardiovascular responses to cold pressor testing in smokers and non-smokers.

METHODOLOGY

Study Design and Setting

This comparative cross-sectional analytical study was conducted in the Human Physiology Laboratory of the Department of Physiology at a tertiary care teaching institution over a period of six months. The laboratory environment was standardized with respect to ambient temperature (22–24°C), humidity, lighting, and noise levels to minimize external autonomic influences on cardiovascular parameters. All recordings were performed during the morning hours (09:00 AM–12:00 PM) to reduce circadian variability in cardiovascular reactivity.

Study Population

Apparently healthy adult male volunteers aged between 18 and 35 years were enrolled in the study and categorized into two groups comprising smokers and non-smokers. Smokers were defined as individuals with a history of regular cigarette smoking for at least one year with a minimum consumption of five cigarettes per day, whereas non-smokers had no history of active smoking or use of tobacco products in any form. Only male participants were included in order to eliminate the confounding influence of menstrual cycle–related hormonal fluctuations on autonomic cardiovascular reactivity.

Sample Size and Sampling Technique

Sample size estimation was performed using previously reported differences in systolic blood pressure response to cold pressor testing between smokers and non-smokers, assuming a confidence interval of 95%, statistical power of 80%, and a moderate effect size. The minimum required sample size was calculated to be 27 participants in each group. To account for potential attrition and incomplete recordings, 30 subjects were recruited in each group, yielding a total sample size of 60 participants.

Participants fulfilling the eligibility criteria were recruited by purposive sampling from students, hospital staff, and healthy attendants after detailed clinical screening and informed consent.

Inclusion Criteria

Participants satisfying all of the following criteria were included:

- Male subjects aged between 18 and 35 years
- Apparently healthy on clinical evaluation

- Resting blood pressure within normotensive range (<140/90 mmHg)
- For smokers:
  - Active cigarette smoking for ≥1 year
  - Consumption of ≥5 cigarettes/day
- For non-smokers:
  - No current or past history of smoking or tobacco consumption

Exclusion Criteria

Participants with any of the following conditions were excluded:

- History of hypertension, ischemic heart disease, arrhythmia, peripheral vascular disease, diabetes mellitus, bronchial asthma, endocrine disorders, or autonomic dysfunction
- Acute febrile illness within the preceding two weeks
- Current use of medications affecting cardiovascular or autonomic function, including antihypertensives, beta-blockers, antidepressants, bronchodilators, or sympathomimetic agents.
- Alcohol intake within 24 hours prior to testing
- Consumption of caffeine-containing beverages within 12 hours before the procedure
- Engagement in strenuous physical activity within 24 hours before testing
- Obesity (body mass index ≥30 kg/m²)
- Use of smokeless tobacco, vaping devices, or recreational substances

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee prior to commencement. Written informed consent was obtained from all participants after explaining the objectives, procedures, potential discomfort, and voluntary nature of participation. Confidentiality and anonymity of participant data were maintained throughout the study in accordance with the principles outlined in the Declaration of Helsinki.

Study Instruments and Equipment

Cardiovascular parameters were recorded using a calibrated mercury sphygmomanometer or a validated automated blood pressure monitor in accordance with standard international recommendations. Heart rate was assessed manually by radial pulse palpation over one minute. Water temperature during the cold pressor test was continuously monitored using a calibrated clinical thermometer, and the ice water bath temperature was maintained between 4°C and 6°C throughout the procedure.

Anthropometric measurements including height and weight were recorded using standardized techniques, and body mass index was calculated as weight in kilograms divided by height in meters squared.

Pre-Test Standardization

Participants were instructed to:

- Abstain from smoking for at least 2 hours prior to testing to avoid acute nicotine-induced hemodynamic fluctuations while preserving chronic autonomic effects.
- Avoid caffeine-containing beverages, alcohol, and heavy meals for at least 12 hours before the test.
- Refrain from vigorous physical activity for 24 hours prior to assessment

Upon arrival at the laboratory, participants were allowed to acclimatize for 15 minutes in a quiet sitting environment before initiation of recordings.

Baseline Measurements

Anthropometric measurements including height and weight were recorded using standardized techniques, and body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters.

Thereafter, baseline cardiovascular parameters were assessed with the participant in a relaxed seated posture:

- Resting heart rate (beats/minute)
- Systolic blood pressure (SBP, mmHg)
- Diastolic blood pressure (DBP, mmHg)

Two readings were obtained at an interval of two minutes, and the average value was considered for analysis.

Cold Pressor Test Procedure

The cold pressor test was performed according to standardized autonomic function testing protocols. Participants were instructed to immerse their non-dominant hand up to the wrist in an ice-water bath maintained at 4–6°C for a duration of one minute. Uniform immersion depth and water temperature were ensured for all subjects.

Heart rate was recorded at baseline, at 30 seconds during immersion, and immediately at the end of one minute. Systolic and diastolic blood pressure were measured immediately following completion of the immersion period. Recovery measurements of heart rate and blood pressure were subsequently recorded at one minute and three minutes after removal of the hand from cold water in order to assess post-stimulus autonomic recovery.

Participants unable to tolerate the cold exposure for the prescribed duration were excluded from the final analysis.

Outcome Measures

The primary outcome measures included changes in systolic and diastolic blood pressure in response to cold pressor stimulation. Secondary outcome measures included alterations in heart rate and recovery characteristics following cessation of the cold stimulus.

Cardiovascular reactivity indices were derived using the following calculations:

- ΔSBP = Peak systolic blood pressure during test − Baseline systolic blood pressure
- ΔDBP = Peak diastolic blood pressure during test − Baseline diastolic blood pressure
- ΔHR = Peak heart rate during test − Baseline heart rate

Data Management and Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS version 26.0.

Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were represented as frequencies and percentages. Normality of distribution was assessed using the Shapiro–Wilk test.

For intragroup comparison of baseline and post-stimulus cardiovascular parameters, paired Student's t-test was used. Intergroup comparisons between smokers and non-smokers were performed using independent Student's t-test for normally distributed variables. Pearson correlation analysis was applied to assess the relationship between smoking duration/intensity and cardiovascular reactivity parameters where appropriate.

A two-tailed p-value <0.05 was considered statistically significant.

Measures to Minimize Bias and Confounding

Several methodological precautions were adopted to improve internal validity and reproducibility of the study. All recordings were obtained under uniform environmental conditions by the same investigator using standardized equipment and testing procedures. Testing time was kept constant for all participants to reduce circadian variability. Strict pre-test restrictions were implemented to minimize the acute influence of caffeine, nicotine, alcohol, and physical exertion on autonomic cardiovascular responses. Additionally, exclusion of individuals with comorbid illnesses or medications affecting autonomic function helped reduce physiological confounding.

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RESULTS

A total of 60 participants were enrolled in the study, comprising 30 smokers and 30 non-smokers. All participants completed the cold pressor test protocol successfully, and no adverse events were recorded during the procedure. The demographic characteristics of the study population are summarized in Table 1.

Table 1. Baseline demographic and cardiovascular characteristics of study participants

Parameter	Smokers (n=30)	Non-smokers (n=30)	p-value
Age (years)	24.8 ± 3.6	23.9 ± 3.2	0.312
Body Mass Index (kg/m²)	24.1 ± 2.8	23.5 ± 2.6	0.418
Baseline Heart Rate (beats/min)	78.6 ± 6.9	75.9 ± 6.1	0.118
Baseline Systolic BP (mmHg)	121.4 ± 7.8	118.2 ± 6.9	0.094
Baseline Diastolic BP (mmHg)	78.1 ± 5.6	75.8 ± 5.1	0.102

The mean age and body mass index were comparable between smokers and non-smokers, and the differences were not statistically significant. Baseline heart rate, systolic blood pressure, and diastolic blood pressure were marginally higher among smokers; however, these differences did not reach statistical significance, indicating reasonable baseline homogeneity between the two groups.

Following cold pressor stimulation, both groups demonstrated significant increases in cardiovascular parameters from baseline values. However, the magnitude of cardiovascular response was considerably greater among smokers. Comparative cardiovascular reactivity during the cold pressor test is presented in Table 2.

Table 2. Cardiovascular responses during cold pressor test among smokers and non-smokers

Parameter	Smokers (n=30)	Non-smokers (n=30)	p-value
Peak Heart Rate (beats/min)	92.5 ± 8.3	84.7 ± 7.2	<0.001
Δ Heart Rate (beats/min)	13.9 ± 4.5	8.8 ± 3.6	<0.001
Peak Systolic BP (mmHg)	142.7 ± 9.4	133.6 ± 8.1	<0.001
Δ Systolic BP (mmHg)	21.3 ± 5.8	15.4 ± 4.9	<0.001
Peak Diastolic BP (mmHg)	92.7 ± 6.8	85.6 ± 5.9	<0.001
Δ Diastolic BP (mmHg)	14.6 ± 4.7	9.8 ± 3.9	<0.001

Smokers exhibited significantly exaggerated cardiovascular reactivity during cold exposure compared with non-smokers. The increase in systolic blood pressure among smokers was markedly higher, with a mean rise of 21.3 ± 5.8 mmHg compared to 15.4 ± 4.9 mmHg in non-smokers. Similarly, diastolic blood pressure response was significantly augmented among smokers.

Heart rate response during cold stimulation also demonstrated a significantly greater rise in smokers than in non-smokers, reflecting enhanced sympathetic cardiovascular activation.

Recovery of cardiovascular parameters following cessation of cold exposure was assessed at one minute and three minutes post-test. Smokers demonstrated delayed recovery compared to non-smokers, as shown in Table 3.

Non-smokers demonstrated relatively rapid normalization of cardiovascular parameters following withdrawal of the cold stimulus. In contrast, smokers continued to exhibit persistently elevated heart rate and blood pressure values during the recovery phase, suggesting impaired autonomic recovery and sustained sympathetic predominance.

Correlation analysis performed among smokers revealed a modest but statistically significant positive association between smoking duration and cardiovascular reactivity indices. Duration of smoking positively correlated with rise in systolic blood pressure ($r=0.42$, $p=0.021$) and heart rate response ($r=0.37$, $p=0.039$), indicating progressively exaggerated sympathetic cardiovascular responses with increasing smoking exposure.

Overall, the study findings demonstrated that smokers exhibit significantly heightened cardiovascular reactivity to cold pressor stimulation compared with non-smokers, characterized by exaggerated rises in heart rate and blood pressure along with delayed post-stress recovery. These findings are suggestive of altered autonomic cardiovascular regulation and enhanced sympathetic responsiveness among smokers.

Table 3. Post-test recovery cardiovascular parameters following cold pressor test.

Parameter	Smokers (n=30)	Non-smokers (n=30)	p-value
Heart Rate at 1 min recovery (beats/min)	84.8 ± 7.2	78.6 ± 6.4	0.001
Systolic BP at 1 min recovery (mmHg)	129.8 ± 7.6	122.1 ± 6.8	<0.001
Diastolic BP at 1 min recovery (mmHg)	82.9 ± 5.4	77.1 ± 4.8	<0.001
Heart Rate at 3 min recovery (beats/min)	80.2 ± 6.5	76.4 ± 5.8	0.021
Systolic BP at 3 min recovery (mmHg)	124.7 ± 6.9	119.4 ± 6.1	0.003
Diastolic BP at 3 min recovery (mmHg)	79.8 ± 5.1	76.3 ± 4.5	0.008

DISCUSSION

The present study was conducted to evaluate whether chronic cigarette smoking influences cardiovascular autonomic reactivity during sympathetic stress induced by the cold pressor test.

The findings demonstrated that smokers exhibited significantly greater increases in systolic blood pressure, diastolic blood pressure, and heart rate in response to cold stimulation compared with non-smokers. In addition, smokers showed delayed recovery of cardiovascular parameters following withdrawal of the cold stimulus. These

observations suggest altered autonomic cardiovascular regulation with sympathetic predominance among apparently healthy young smokers.

The cold pressor test remains one of the most widely accepted physiological methods for assessing sympathetic cardiovascular reactivity under controlled experimental conditions [3]. Exposure to cold nociceptive stimulation activates peripheral afferent pathways, leading to hypothalamic and medullary autonomic activation with subsequent catecholamine release, peripheral vasoconstriction, and cardiovascular stimulation [4]. Earlier work by Hines and Brown established the utility of the cold pressor test in identifying exaggerated vascular reactivity associated with future hypertension risk [3]. Subsequent physiological studies further confirmed that the pressor response during cold exposure is mediated predominantly through sympathetic neural activation and increased peripheral vascular resistance [4,6].

In the present study, baseline cardiovascular parameters were marginally higher among smokers, although the differences were not statistically significant. Similar findings have been reported in previous studies involving young adult smokers with relatively short smoking duration [2,7]. Chronic nicotine exposure is known to stimulate sympathetic ganglia and adrenal catecholamine release, resulting in persistent low-grade sympathetic activation even during resting conditions [2]. Nicotine additionally contributes to endothelial dysfunction, oxidative stress, and impaired nitric oxide–mediated vasodilatation, all of which may predispose smokers to heightened cardiovascular responses during physiological stress [7,8].

The most notable finding of the present study was the significantly exaggerated blood pressure response to cold pressor stimulation among smokers. Smokers demonstrated substantially greater rises in both systolic and diastolic blood pressure compared with non-smokers. These observations are in agreement with previous investigations demonstrating enhanced vasoconstrictor responsiveness among chronic smokers [6,9]. Cryer et al. reported that cigarette smoking causes marked activation of the sympathetic nervous system with associated elevation in plasma norepinephrine levels and increased vascular tone [9]. Similarly, Narkiewicz et al. demonstrated that smokers exhibit augmented sympathetic nerve activity and impaired autonomic modulation, contributing to abnormal cardiovascular reactivity during stress exposure [10].

The exaggerated pressor response observed in smokers may be explained by several interrelated mechanisms. Chronic tobacco exposure has been associated with increased arterial stiffness, endothelial injury, reduced vascular compliance, and enhanced alpha-adrenergic sensitivity [2,8]. Under conditions of sympathetic stimulation such as cold exposure, these vascular alterations may amplify peripheral vasoconstriction, thereby producing a disproportionately elevated blood pressure response. The findings of the present study therefore support the concept that smoking induces subclinical vascular and autonomic dysfunction even in otherwise healthy young adults.

Heart rate response during cold pressor stimulation was also significantly greater among smokers in the present study. This observation is consistent with previous evidence suggesting impaired sympatho-vagal balance among chronic smokers [10,11]. Lucini et al. demonstrated reduced vagal modulation and increased sympathetic predominance in smokers using autonomic function analysis and heart rate variability assessment [11]. Similarly, Karakaya et al. reported that smoking adversely affects autonomic cardiac control by suppressing parasympathetic activity while simultaneously enhancing sympathetic

cardiovascular drive [12]. The significantly greater increase in heart rate among smokers observed in the present study therefore likely reflects autonomic imbalance characterized by vagal withdrawal and exaggerated sympathetic activation during stress exposure.

An important observation in the current study was the delayed recovery of heart rate and blood pressure among smokers following cessation of cold exposure. Non-smokers showed relatively rapid return of cardiovascular parameters toward baseline values, whereas smokers continued to demonstrate persistently elevated heart rate and blood pressure during the recovery phase. Recovery following sympathetic stimulation depends predominantly on efficient parasympathetic reactivation and restoration of baroreflex sensitivity [13]. Impaired recovery observed among smokers may therefore indicate diminished vagal responsiveness and persistent sympathetic excitation.

Previous studies have similarly identified impaired autonomic recovery as an early marker of cardiovascular dysregulation. Cole et al. demonstrated that delayed heart rate recovery following stress or exercise is associated with increased cardiovascular mortality and autonomic dysfunction [13]. In another study, Mourot et al. observed altered cardiac autonomic control during recovery from cold pressor stimulation, particularly in individuals with sympathetic predominance [14]. The delayed cardiovascular normalization observed in smokers in the present study therefore may represent an early subclinical manifestation of autonomic impairment preceding overt cardiovascular disease.

The present study additionally demonstrated a positive correlation between smoking duration and cardiovascular reactivity indices, indicating progressively exaggerated sympathetic responsiveness with increasing smoking exposure. Similar findings have been reported by Grassi et al., who demonstrated that chronic smoking produces sustained sympathetic overactivity proportional to cumulative tobacco exposure [15]. Long-term nicotine exposure has been shown to impair arterial baroreceptor sensitivity and alter central autonomic regulation, thereby contributing to exaggerated cardiovascular responses during physiological stress [15,16].

The findings of the present study are also consistent with broader evidence linking exaggerated cardiovascular stress reactivity with future hypertension and cardiovascular morbidity. Matthews et al. previously demonstrated that individuals exhibiting excessive cardiovascular responses to stress are more likely to develop sustained hypertension later in life [5]. More recent evidence suggests that sympathetic hyperreactivity identified during autonomic stress testing may serve as an early predictor of endothelial dysfunction, vascular injury, and adverse cardiovascular outcomes [16,17]. The exaggerated cardiovascular responses observed among smokers in the present study may therefore have important long-term clinical implications despite the relatively young age and apparently healthy status of the participants.

Although most previous studies support enhanced cardiovascular reactivity among smokers, certain discrepancies exist regarding the magnitude of heart rate responses during cold pressor testing [14,18]. Variability in study findings may be attributable to differences in participant age, smoking intensity, duration of abstinence prior to testing, autonomic assessment techniques, and cold pressor protocols. Ethnic and environmental differences may also influence autonomic cardiovascular responsiveness. In some studies, acute nicotine withdrawal before testing has been shown to transiently modify sympathetic responses, thereby contributing to inter-study variability [18].

CONCLUSION

In conclusion, the present study demonstrated that smokers exhibit significantly exaggerated cardiovascular responses to cold pressor stimulation compared with non-smokers, characterized by greater elevations in systolic blood pressure, diastolic blood pressure, and heart rate along with delayed post-stress recovery. These findings indicate altered autonomic cardiovascular regulation with sympathetic predominance among smokers and suggest the presence of early subclinical cardiovascular dysfunction even in apparently healthy individuals. Identification of such autonomic alterations at an early stage may have preventive implications in reducing future cardiovascular risk among chronic smokers. The present study possesses several methodological strengths. Strict standardization of laboratory conditions, uniform timing of testing, exclusion of systemic illnesses, and controlled pre-test behavioural restrictions helped minimize confounding influences on autonomic cardiovascular responses. The inclusion of recovery phase analysis further strengthened the physiological assessment by providing additional insight into autonomic restoration following sympathetic stress.

Nevertheless, certain limitations should be acknowledged. The study included only young adult males, which may limit generalizability to females and older populations. Smoking exposure was assessed using self-reported history without biochemical validation through serum cotinine estimation. Furthermore, direct autonomic assessment methods such as heart rate variability analysis, plasma catecholamine measurement, or microneurography were not performed. Future studies incorporating larger multicentric cohorts and advanced autonomic evaluation techniques may provide more comprehensive characterization of smoking-related autonomic dysfunction.

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