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EFFECT OF CAFFEINE INTAKE AND ITS DURATION OF ACTION ON PULMONARY FUNCTION TEST: A RANDOMIZED CONTROLLED STUDY.

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Abstract

Background

Caffeine is a central nervous system stimulant with bronchodilatory effects that may influence pulmonary function. This randomized controlled study aimed to evaluate the effect of caffeine intake and its duration of action on pulmonary function tests (PFTs) in healthy adults.

Materials and Methods

One hundred healthy adults aged 18–35 years were randomly assigned to two groups: caffeine (200 mg oral caffeine, n=50) and control (no intervention, n=50). Pulmonary function parameters—Forced Expiratory Volume in 1 second (FEV_1), Forced Vital Capacity (FVC), FEV_1/FVC ratio, and Peak Expiratory Flow Rate (PEFR)—were measured at baseline and 1, 3, and 6 hours post-intervention.

Results

Participants had a mean age of 25.4 ± 4.8 years, balanced gender distribution (52% male, 48% female), and comparable body mass index (22.5 ± 2.2 kg/m²). The caffeine group showed a significant increase in FEV_1 (3.42 ± 0.39 L to 3.58 ± 0.40 L) and PEFR (7.6 ± 0.9 L/s to 8.1 ± 1.0 L/s) at 1-hour post-intake ($p < 0.05$). Improvements diminished by 3 hours and returned to baseline by 6 hours. The control group showed no significant changes.

Conclusion

Caffeine intake transiently improves pulmonary function, peaking at 1 hour and subsiding by 6 hours. Abstaining from caffeine for at least 6 hours before PFTs is recommended to avoid confounding results.

Recommendations

Individuals should abstain from caffeine-containing products for at least 6 hours before undergoing pulmonary function testing to ensure accurate and reliable results.

Keywords: *Caffeine, Pulmonary Function Test, Forced Expiratory Volume in 1 second (FEV_1), Peak Expiratory Flow Rate (PEFR), Bronchodilation, Respiratory Assessment.*
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Introduction

Caffeine, a naturally occurring methylxanthine present in beverages such as coffee and tea, is widely recognized for its stimulant effects on the central nervous system. In addition to enhancing alertness and cognitive performance, caffeine exerts notable pharmacological actions on the respiratory system [1]. As a non-selective antagonist of adenosine receptors, it promotes bronchodilation and increases respiratory drive—mechanisms that may influence pulmonary function [1,3].

Pulmonary Function Tests (PFTs), especially spirometry-based measurements like Forced Expiratory Volume in one second (FEV_1), Forced Vital Capacity (FVC), and Peak Expiratory Flow Rate (PEFR), are essential tools in evaluating

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respiratory health and diagnosing conditions such as asthma and chronic obstructive pulmonary disease (COPD) [2]. However, the acute bronchodilatory effects of caffeine may transiently enhance these measurements, potentially leading to inaccurate interpretation of a patient's baseline lung function [2,3].

To address this, clinical guidelines—including those from the American Thoracic Society—recommend avoiding caffeine before pulmonary function testing to prevent confounding test results [2]. Despite such recommendations, compliance among patients is inconsistent, and the optimal duration of abstinence to eliminate caffeine's influence remains unclear.

Several studies have highlighted caffeine's short-term benefits on pulmonary performance. For instance, it has been shown to improve FEV_1 and other spirometric parameters for up to four hours post-consumption [3,4]. These effects have been attributed to its action on adenosine receptors and phosphodiesterase inhibition, which enhances airway smooth muscle relaxation [3]. Furthermore, caffeine has been observed to alter breathing patterns during exercise, suggesting a potential role in modulating respiratory physiology under physical stress as well [5]. Despite these findings, there is a paucity of data quantifying the temporal profile of caffeine's influence on pulmonary function in healthy individuals.

The objective of this study is to evaluate how caffeine consumption influences lung function over time by analyzing changes in spirometry measurements taken at several post-intake intervals. By understanding the duration and magnitude of caffeine's impact on PFTs, we can better inform clinical practices regarding pre-test preparations and ensure an accurate assessment of respiratory function.

Methodology

Study Design and Duration

This was a randomized, controlled, prospective study conducted over three months from **January to March 2025** at the Department of Pulmonology, Alluri Sitarama Raju Academy of Medical Sciences (ASRAMS), Eluru, Andhra Pradesh, India. ASRAMS is a tertiary care teaching hospital affiliated with Dr. NTR University of Health Sciences, equipped with advanced diagnostic and research facilities.

Sample Size Determination

The sample size of 100 participants (50 per group) was calculated based on an expected effect size of 0.6 for the primary outcome (FEV_1), with 80% power and a significance level of 5%. Allowing for a 10% dropout rate, 100 healthy adult volunteers were recruited.

Study Population

Healthy adult volunteers aged 18 to 35 years were enrolled after screening. Inclusion criteria were: non-smokers or occasional smokers, no history of chronic respiratory or cardiovascular diseases, no medications influencing respiratory function, and willingness to abstain from caffeine for 12 hours before baseline testing. Exclusion criteria included known caffeine hypersensitivity, pregnancy or lactation, recent respiratory infections (within 4 weeks), and habitual caffeine intake exceeding 300 mg/day.

Randomization Procedure

Participants were randomly assigned in a 1:1 ratio into the Caffeine Group and the Control Group using a computer-generated random allocation sequence. Block randomization with blocks of size 10 was implemented to ensure balanced group sizes. The allocation sequence was generated by an independent statistician not involved in participant recruitment or assessment.

Allocation Concealment

The random allocation sequence was concealed using sequentially numbered, opaque, sealed envelopes prepared by the statistician. Envelopes were opened only after participant enrollment to assign the intervention.

Implementation

An independent study coordinator enrolled participants and assigned interventions according to the allocation sequence. The investigator performing the pulmonary function tests was blinded to group assignments to reduce assessment bias.

Interventions

Caffeine Group: Participants received a single oral dose of 200 mg of caffeine administered as a standardized caffeine tablet with 100 ml of water at the study center immediately after baseline pulmonary testing.

Control Group: Participants did not receive any intervention but were asked to remain under observation for the same duration.

Outcome Measures

Primary Outcome: Changes in Forced Expiratory Volume in 1 second (FEV_1) measured by spirometry.

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Secondary Outcomes: Forced Vital Capacity (FVC), FEV_1/FVC ratio, and Peak Expiratory Flow Rate (PEFR).

Pulmonary function tests were performed at baseline (pre-intervention), and 1, 3, and 6 hours post-intervention according to American Thoracic Society guidelines. Tests were conducted in a standardized manner by trained respiratory technicians blinded to group assignment.

Data Analysis

Continuous variables were expressed as mean ± standard deviation. Between-group comparisons at each time point were made using independent samples t-tests. Within-group changes over time were analyzed using repeated measures ANOVA. Statistical significance was set at $p < 0.05$. Analysis was performed using SPSS version XX (specify software).

Blinding

Outcome assessors conducting spirometry were blinded to participant group assignments. Participants and care providers were not blinded due to the nature of the intervention.

Ethical Considerations

The study was approved by the Institutional Ethics Committee of ASRAMS, Eluru. Written informed consent was obtained from all participants before enrollment by the Declaration of Helsinki.

Results

Participant Flow and Randomization

A total of 100 healthy adult volunteers were screened and successfully enrolled in the study. Participants were randomly assigned in equal numbers to either the caffeine group (n = 50) or the control group (n = 50) using a computer-generated randomization schedule. All participants completed the study protocol, and data from all 100 participants were included in the final analysis for the primary and secondary outcomes. There were no dropouts or exclusions post-randomization. Figure 1 presents the CONSORT flow diagram summarizing enrollment, allocation, follow-up, and analysis.

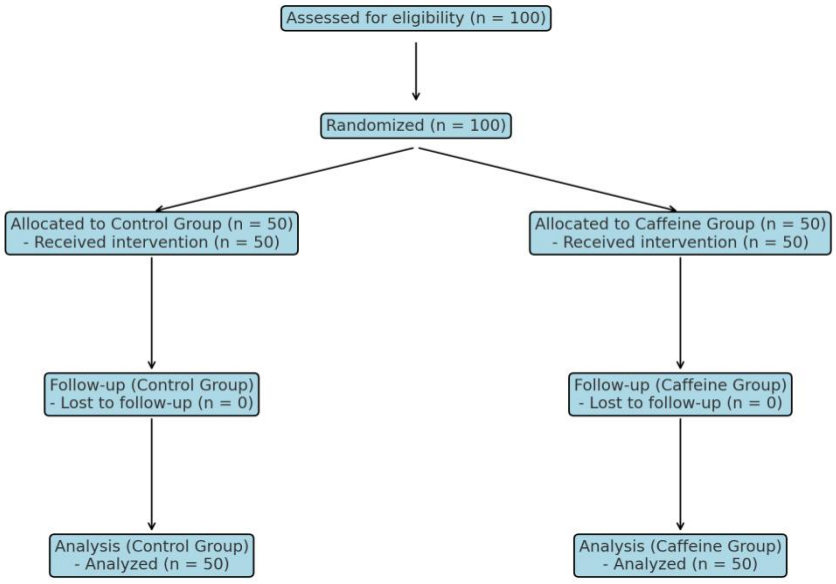

Figure 1: presents the CONSORT flow diagram summarizing enrollment, allocation, follow-up, and analysis.

Baseline Demographics and Clinical Characteristics

The demographic and baseline clinical profiles of participants were comparable between the two groups (Table 1). The overall mean age was 25.4 ± 4.8 years, with a balanced gender distribution (52% male, 48% female) in both groups. The mean BMI was 22.4 ± 2.3 kg/m² in the control group and 22.6 ± 2.1 kg/m² in the caffeine group. The majority of participants (85%) were non-smokers, and smoking status was evenly distributed across groups. No statistically significant differences were observed in baseline characteristics.

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Table 1: Demographic Characteristics of Participants (N = 100)

Variable	Control Group (n = 50)	Caffeine Group (n = 50)	Total (N = 100)
Age (years)	25.3 ± 4.9	25.5 ± 4.7	25.4 ± 4.8
Sex			
Male	26 (52%)	26 (52%)	52 (52%)
Female	24 (48%)	24 (48%)	48 (48%)
BMI (kg/m²)	22.4 ± 2.3	22.6 ± 2.1	22.5 ± 2.2
Smoker Status			
Non-smoker	42 (84%)	43 (86%)	85 (85%)
Occasional	8 (16%)	7 (14%)	15 (15%)

Baseline Pulmonary Function

Pulmonary function parameters measured at baseline—including Forced Expiratory Volume in 1 second (FEV_1), Forced Vital Capacity (FVC), FEV_1/FVC ratio, and Peak Expiratory Flow Rate (PEFR)—were statistically similar between the two groups, indicating that lung function was comparable before intervention (Tables 2 and 3).

Post-Intervention Pulmonary Function

Control Group:

Participants in the control group exhibited stable pulmonary function throughout the 6-hour observation period. Minor, non-significant changes were observed in FEV_1 (3.40 ± 0.38 L to 3.42 ± 0.39 L), FVC (4.20 ± 0.45 L to 4.22 ± 0.45 L), and PEFR (7.5 ± 0.9 L/s to 7.7 ± 0.9 L/s), suggesting no effect in the absence of caffeine (Table 2).

Table 2: Pulmonary Function Test Results – Control Group (n = 50)

Time Point	FEV_1 (L) ± SD	FVC (L) ± SD	FEV_1/FVC (%) ± SD	PEFR (L/s) ± SD
Baseline	3.40 ± 0.38	4.20 ± 0.45	81.0 ± 2.5	7.5 ± 0.9
1 Hour	3.40 ± 0.38	4.21 ± 0.45	80.8 ± 2.5	7.6 ± 0.9
3 Hours	3.41 ± 0.37	4.20 ± 0.45	81.2 ± 2.5	7.6 ± 0.9
6 Hours	3.42 ± 0.39	4.22 ± 0.45	81.0 ± 2.5	7.7 ± 0.9

Caffeine Group

In contrast, the caffeine group demonstrated statistically significant improvements in pulmonary function following caffeine intake. One hour post-consumption, FEV_1 increased from 3.42 ± 0.39 L to 3.58 ± 0.40 L ($p < 0.05$), and PEFR increased from 7.6 ± 0.9 L/s to 8.1 ± 1.0 L/s ($p < 0.05$). The FEV_1/FVC ratio also rose significantly from 81.2 ± 2.5% to 84.4 ± 2.5% ($p < 0.05$), indicating acute bronchodilation.

At 3 hours post-ingestion, the improvements slightly declined (FEV_1: 3.56 ± 0.39 L; PEFR: 7.9 ± 0.95 L/s), and by 6 hours, values returned close to baseline levels, indicating the temporary nature of the caffeine effect (Table 3).

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Table 3: Pulmonary Function Test Results – Caffeine Group (n = 50)

Time Point	FEV_1 (L) ± SD	FVC (L) ± SD	FEV_1/FVC (%) ± SD	PEFR (L/s) ± SD
Baseline	3.42 ± 0.39	4.21 ± 0.45	81.2 ± 2.5	7.6 ± 0.9
1 Hour	3.58 ± 0.40	4.24 ± 0.45	84.4 ± 2.5	8.1 ± 1.0
3 Hours	3.56 ± 0.39	4.23 ± 0.45	84.0 ± 2.5	7.9 ± 0.95
6 Hours	3.43 ± 0.40	4.22 ± 0.45	81.3 ± 2.5	7.6 ± 0.9

Duration of Action

The bronchodilatory effect of caffeine was most pronounced at 1 hour post-administration, partially sustained at 3 hours, and nearly diminished by 6 hours. This pattern supports a short-term pharmacodynamic window for caffeine's impact on airway function.

Adverse Events

No adverse events, side effects, or intolerances related to caffeine intake or the study procedures were reported by participants in either group throughout the study period.

Discussion

The findings indicate that caffeine consumption produces a temporary enhancement in lung function in healthy individuals, with marked elevations in FEV_1 and PEFR observed at the one-hour mark, followed by a progressive return to baseline levels within six hours. This time-dependent pattern reflects the pharmacokinetics of caffeine, as plasma levels typically peak between 30 and 120 minutes, and the average half-life is approximately five hours. Our findings align with prior evidence supporting the bronchodilatory potential of caffeine. A Cochrane review has documented that even low doses (<5 mg/kg) can elicit modest improvements in FEV_1 ranging from 5% up to 18% within the first two hours of ingestion. Mechanistically, caffeine acts as a non-selective adenosine receptor antagonist, inhibiting A_1 and A_2A receptor-mediated bronchoconstriction and inflammation. Additionally, caffeine's phosphodiesterase inhibition elevates intracellular cAMP, promoting relaxation of airway smooth muscle, similar to the action of β_2-agonists. It may also enhance bronchodilation indirectly by stimulating catecholamine release [6,7].

Recent investigations have reported similar outcomes, reinforcing caffeine's acute impact on lung function. Evidence supports its comparability to approximately 40% of the bronchodilatory effect produced by theophylline, with peak efficacy noted within 90 to 120 minutes. Caffeine's influence on pulmonary measures during both rest and exercise suggests potential benefits in various physiological states [7].

However, not all research has shown uniform effects. Some studies reported no significant changes in pulmonary function despite administering doses as high as 330 mg. Lifestyle factors such as smoking status and habitual caffeine consumption have been identified as possible confounders that could attenuate or obscure caffeine's respiratory benefits [8]. The relatively large sample size in our study (n = 100) may have increased the statistical power to detect subtle yet meaningful changes. Nevertheless, the lack of stratification based on caffeine tolerance or habitual intake remains a limitation and warrants consideration in future research.

Emerging population-based data have also suggested links between caffeine exposure and improved lung function, particularly in non-asthmatic individuals [9]. Additional evidence indicates that acute caffeine intake may enhance muscle oxygenation during physical exertion, potentially improving ventilatory efficiency [10]. Beyond adults, caffeine has shown beneficial respiratory effects in neonates, with improved breathing regulation in infant apnea and long-term pulmonary outcomes in low-birth-weight infants who received neonatal caffeine therapy [11,12].

From a clinical perspective, the timing of caffeine's peak effect, approximately one hour post-ingestion, can temporarily augment spirometry results. This may lead to misinterpretation in diagnosing airflow limitation or assessing disease severity. Conversely, caffeine withdrawal in regular users could yield suboptimal baseline values. These findings underscore the importance of consistent pre-test instructions regarding caffeine abstinence.

Current spirometry guidelines recommend avoiding caffeine for at least four hours before testing. Our study supports this recommendation, as the bronchodilatory effects had largely subsided by the 4–6 hour period. Ensuring a standardized abstinence window is essential for maintaining accuracy in pulmonary function assessment, especially in borderline or reversibility evaluations.

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Generalizability

These findings are generalizable to healthy young adults and may inform clinical practices and research protocols involving pulmonary testing across similar populations in diverse healthcare settings.

Conclusion

In summary, this study confirms that caffeine intake can produce a short-term bronchodilatory effect, with the most significant changes occurring one hour after ingestion and resolving within six hours. These results are consistent with earlier literature and can be explained by known pharmacological mechanisms involving adenosine receptor blockade, phosphodiesterase inhibition, and catecholamine release. Clinicians and pulmonary laboratories should account for recent caffeine intake when interpreting spirometry results.

Limitations

Despite the valuable insights offered, several limitations must be acknowledged. The study included only healthy adults, limiting generalizability to clinical populations such as individuals with asthma or COPD. Its single-center design and moderate sample size may also constrain external validity. Moreover, we did not stratify based on habitual caffeine use, nor did we assess long-term or cumulative effects. The absence of a placebo control precludes ruling out expectancy or diurnal effects. Additionally, only FEV_1 and PEFR were measured; mid-expiratory flows (e.g., $FEF_{25–75}$) and lung volumes were not assessed, limiting the completeness of pulmonary function profiling.

Recommendations

We recommend standardized patient instructions to abstain from caffeine at least four to six hours before testing to ensure reliable assessment. Further research in populations with respiratory disorders and studies exploring dose-response relationships and long-term effects would be valuable to refine these recommendations.

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List of Abbreviations

PFT – Pulmonary Function Test
FEV_1 – Forced Expiratory Volume in 1 Second
FVC – Forced Vital Capacity
PEFR – Peak Expiratory Flow Rate
FEV_1/FVC – Ratio of Forced Expiratory Volume in 1 Second to Forced Vital Capacity
COPD – Chronic Obstructive Pulmonary Disease
cAMP – Cyclic Adenosine Monophosphate
ATS – American Thoracic Society
BMI – Body Mass Index
SD – Standard Deviation
ABRAMS – Alluri Sitarama Raju Academy of Medical Sciences

Source of funding

The study had no funding.

Conflict of interest

The authors declare no conflict of interest.

Author contributions

BKC- Concept and design of the study, results interpretation, review of the literature, and preparation of the first draft of the manuscript. Statistical analysis and interpretation, revision of the manuscript. **MS**-Concept and design of the study, results interpretation, review of the literature, and preparing the first draft of the manuscript, revision of the manuscript.**JV**-Concept and design of the study, results interpretation, review of the literature, and preparing the first draft of the manuscript. Statistical analysis and interpretation, revision of the manuscript.**Review** of literature and preparing the first draft of the manuscript. Statistical analysis and interpretation

Data availability

Data Available

Author Biography

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