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A Study on The Effect of Tobacco Smoking on Lipid Profile

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Abstract

Introduction: Tobacco smoking adversely affects lipid profile, increasing the risk of atherosclerosis coronary artery diseases. It has been found to alter lipid metabolism by stimulating sympathetic adrenal system leading to increased secretion of catecholamines.

Methods: Total of 50 healthy male smokers with smoking index more than 100. 50 healthy male nonsmokers age and body mass index matched selected from Patient's attendants and hospital staff.

Result: The quantity of cigarettes or beedies consumed by smokers in the age ranges of 10-13, 14-17, and 18-20 constituted 8%, 44%, and 48% respectively of the total consumption.

Conclusion: Lipid Profile Differences Between Smokers and Non-Smokers. Cigarette smoking is associated with dyslipidaemia among smokers. Total cholesterol and LDL may be considered as the main parameters that are affected by the heaviness of smoking. However, preventive strategies are needed to avoid the future cardiovascular diseases and in supporting the benefits of quit smoking.

Keywords: Tobacco, Smoking, Cardiovascular, Healthcare, Social Circles

Introduction

The human history, smoking tobacco has been a widespread activity, crossing cultural, ethnic, and economic boundaries. In numerous developing nations, tobacco-related diseases have become increasingly prevalent due to the rising rates of smoking. Ancient civilizations, such as those in the Americas, utilized tobacco in rituals and for medicinal purposes. However,

it wasn't until the 20th century that the extensive health and environmental risks associated with tobacco consumption became widely recognized[1].

In recent decades, statistics have highlighted the severity of the issue. For example, the World Health Organization (WHO) reports that over 8 million deaths annually are attributed to tobacco use, with more than 80% of these occurring in low- and middle-income countries. These alarming figures underscore the global health crisis posed by smoking. The addictive nature of nicotine is a significant factor in the widespread habit of smoking. This chemical compound, found in tobacco, stimulates the release of neurotransmitters like dopamine, which create pleasurable sensations and reinforce smoking behavior. Over time, this leads to dependency and makes quitting challenging for many individuals[1].

Healthcare professionals, especially doctors, are pivotal in smoking prevention efforts. They provide essential counseling to patients, offering guidance on the risks of smoking and strategies for cessation. Additionally, they promote healthy behaviors within communities through educational programs and support initiatives aimed at reducing tobacco use[2].

Nicotine's capacity to psychologically stimulate the central nervous system (CNS) plays a critical role in the progression from initial tobacco experimentation to habitual and addictive use. This transition occurs because nicotine activates reward pathways in the brain, releasing neurotransmitters like dopamine, which produce feelings of pleasure and relaxation, reinforcing the behavior and leading to dependency[2].

Tobacco use is a multifaceted learned behavior that becomes deeply integrated into daily routines and is intricately linked with a smoker's cognitive and perceptual framework. This habit forms through a complex interaction of actions, thoughts, and emotional states. Conditioned behaviors, driven by nicotine's neuroregulatory effects on neurotransmitters such as dopamine, serve as potent stimuli for continued smoking[3].

Various individual characteristics significantly influence tobacco use. Factors such as educational attainment, which affects awareness and comprehension of smoking-related health risks, self-efficacy in cessation attempts, and coping mechanisms for stress and anxiety, all play crucial roles. For instance, individuals with higher levels of education might possess greater knowledge regarding the detrimental health effects of smoking and thus exhibit a higher motivation to quit. Conversely, those with lower self-efficacy may encounter more substantial challenges in their efforts to cease smoking.

Contextual factors also heavily shape smoking behavior. Peer influence can either encourage or discourage smoking, depending on the prevalent attitudes within social circles. Workplace norms can create environments where smoking is either common and accepted or discouraged and restricted[4].

Smoking is associated with elevated serum levels of triglycerides, low-density lipoprotein (LDL) cholesterol, and total cholesterol, along with decreased levels of high-density lipoprotein (HDL) cholesterol, which possesses anti-atherogenic properties. Various studies have demonstrated a dose-dependent relationship between smoking and alterations in the lipoprotein profile. For instance, research has shown that each additional cigarette smoked per day correlates with higher LDL and lower HDL levels, exacerbating cardiovascular risks[4].

Despite significant advancements in understanding the causes and treatments of cardiovascular diseases, these conditions continue to affect people in developing countries like India. According to the World Health Organization, by 2020, coronary heart disease and stroke were projected to become the leading causes of disability and death, ranking first and fourth respectively. It was predicted that the number of coronary vascular disease cases would rise from 29 million in 2002 to approximately 64 million by 2015. Tobacco use is a cholesterol-dependent risk factor that works in conjunction with other factors to cause coronary heart disease. There is a notable synergistic interaction between high cholesterol levels and tobacco use in the development of coronary heart disease. The causes of coronary heart disease are multifaceted, including both preventable and non-preventable risk factors. Tobacco use contributes to the development of coronary heart disease through several mechanisms:

1. Carbon monoxide promotes the formation of atherosclerosis.
2. Nicotine increases adrenergic activity, leading to higher blood pressure and greater myocardial oxygen demand.
3. Disruption of lipid metabolism.

Tobacco smoke contains numerous harmful substances, including carbon monoxide, carbon dioxide, nitric oxide, nitrogen dioxide, dinitrogen trioxide, ammonia, hydrogen cyanide, volatile sulfur compounds, volatile aldehydes (such as formaldehyde, acetaldehyde, and acrolein), alcohols, ketones, and various nitrosamines[10].

This study aimed to investigate the specific lipoprotein patterns in healthy smokers and the changes in lipid profiles induced by smoking. Participants included smokers without any known cardiovascular diseases or other health conditions that could influence lipid levels. Fasting blood samples were collected to measure serum triglycerides, LDL cholesterol, total cholesterol, and HDL cholesterol and the significant impact of smoking on lipid metabolism and cardiovascular health. They emphasize the importance of smoking cessation interventions to mitigate these adverse effects and reduce the burden of smoking-related cardiovascular diseases.

Aims & Objectives

1. Quantitative estimation of total serum cholesterol, triglycerides, HDL-cholesterol and LDL-cholesterol in healthy male smokers.
2. Estimation of the serum lipid profile in age matched healthy nonsmoker individuals.
3. To find out the variable patterns of lipid profile in terms of duration of smoking and severity of smoking [smoking index].

Materials and Methods

Study population

Cases- total of **50** healthy male smokers with smoking index more than 100.

Controls - **50** healthy male nonsmokers age and body mass index matched selected from Patient's attendants and hospital staff.

Type of study: Case control study

Study place: Department of Medicine, Patna Medical College and Hospital, Patna.

Study period: January 2023 to June 2024.

Study Design

Type of Study: Case Control

Inclusion Criteria

- Healthy male smokers in 20-50 years age group
- Smoking index more than 100.

Exclusion Criteria

- People with Diabetes, Hypertension, obesity.

- People on drug interfering with lipid profile like beta blocker, thiazides.

After a detailed history followed by clinical examination, routine investigations like Hb, TC, DC, ESR, RBS, Serum creatinine, urine analysis and electrocardiogram, fundus examination will be done in all the cases. Venous blood was drawn after overnight fasting. Lipid was estimated which included total serum cholesterol, HDL-cholesterol, LDL-cholesterol and serum triglycerides.

Total cholesterol & amp; triglyceride level estimations are carried out using enzymatic end point kit method. HDL-C is estimated by precipitation of non-HDL lipoprotein & estimation done using supernant.

LDL-cholesterol was calculated from the formula:

LDL. C = Total cholesterol - (TGL / **5) -** HDL-C.

Statistical Analysis

Data was entered into Microsoft excel and analyses was done using the Statistical Package for Social Sciences (SPSS), Windows software (version **18.0).** The information collected regarding all the selected cases was recorded in the master chart.

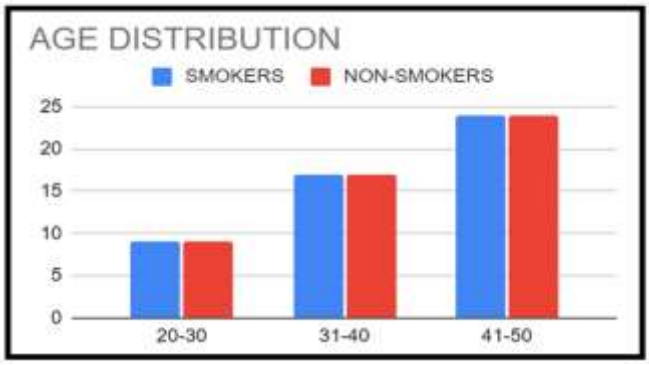
Data analysis was done with the help of computer using epidemiological information package.

Results

Table 1: Age Distribution

Age Distribution	Smokers	Percentage	Non-Smokers	Percentage	Total	Mean ± Sd
20-30	9	18%	9	18%	18	
31-40	17	34%	17	34%	34	39.68 ± 6.748
41-50	24	48%	24	48%	48	

Graph 1:

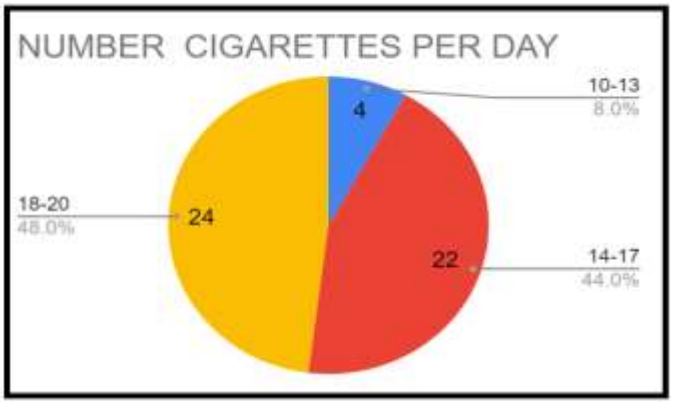


The age group of 41-50 years had the highest proportion of both smokers and non-smokers, making up 48% of the total. Conversely, the age group of 20-30 years had the lowest percentage of both smokers and non-smokers, totaling only 18% and the age group of 31-40 has 34% of smokers and nonsmoker.

Table 2: Number of Cigarettes Per Day

No. of Cigarettes	Smokers	Percentage
10-13	4	8
14-17	22	44
18-20	24	48

Graph 2:



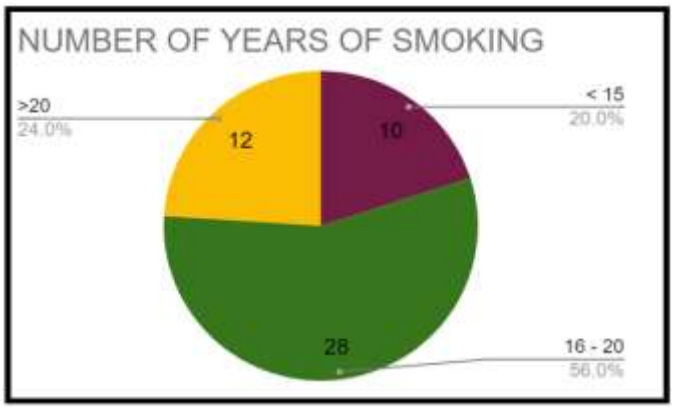
The quantity of cigarettes or beedies consumed by smokers in the age ranges of 10-13, 14-17, and 18-20 constituted 8%, 44%, and 48% respectively of the total consumption. This means that smokers in the 18-20 age group had the highest share of cigarette or beedie consumption, accounting for nearly half of the total,

while those in the 10-13 age range had the smallest share, making up only 8% of the total consumption.

Table 3: Duration of Smoking

Duration of Years	Smokers	Percentage
<15	10	20
16 - 20	28	56
>20	12	24

Graph 3:

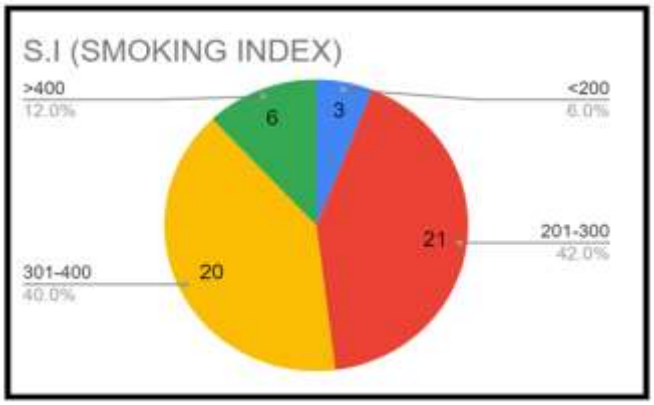


Among smokers, a breakdown based on the duration of smoking reveals that 20% smoked for over 15 years, 56% smoked for 16-20 years, and 24% smoked for more than 20 years. This distribution highlights that a majority of smokers, constituting 56%, had been smoking for 16-20 years, indicating a significant proportion with long-term smoking habits.

Table 4: Smoking Index

S.I (Smoking Index)	Smokers	Percentage
<200	3	6
201-300	21	42
301-400	20	40
>400	6	12

Graph 4:

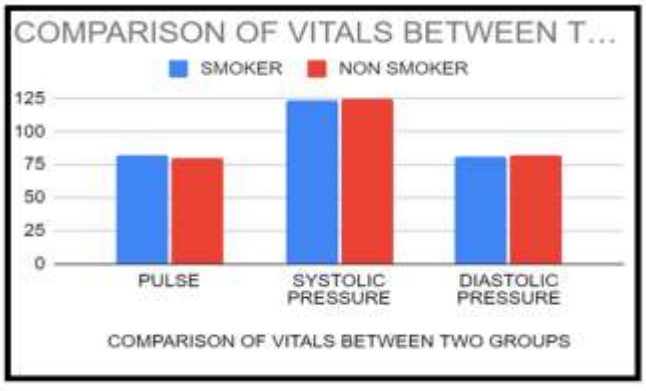


Among smokers, the distribution based on their smoking index is as follows: 3 (6%) had a smoking index of less than 200, 21(42%) had a smoking index between 201 and 300, 20 (40%) had a smoking index ranging from 301 to 400, and 6 (12%) had a smoking index exceeding 400. This breakdown illustrates that a substantial portion of smokers (40%) fell into the category of a smoking index between 301 and 400, indicating a significant level of smoking intensity within this group.

Table 5: Comparison of Vitals between Two Groups

Vital Signs	Smoker	Non Smoker
Pulse	82.32 ± 11.76719758	79.88 ± 6.714711233
Systolic Pressure	123.4 ± 5.760527187	124.72 ± 7.157242657
Diastolic Pressure	80.6 ± 4.785181207	81.88 ± 6.342728441

Graph 5:



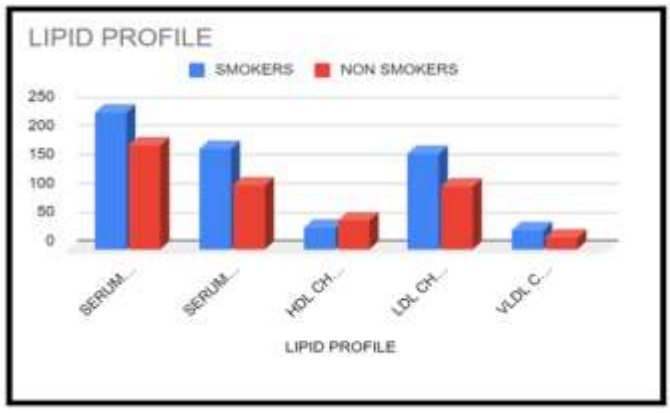
The comparison of vital signs between the two groups showed similarities in pulse rate and both systolic and diastolic blood pressure levels. This indicates that there

were no significant differences observed in these key cardiovascular measures between the two groups.

Table 6: Lipid Profile

Lipid Profile	Smokers (Mean ± Sd)	Non Smokers (Mean ± Sd)
Serum Cholesterol	237.36 ± 43.72257624	181.56 ± 15.25987135
Serum Triglycerides	173.84 ±45.85352011	110.26 ± 16.41031284
Hdl Cholesterol	36.66 ± 6.793183859	50.16 ± 7.775839085
Ldl Cholesterol	165.8 ± 40.68194204	109.34 ± 14.9962613
Vldl Cholesterol	33.96 ± 10.2339172	20.28 ± 5.707281422

Graph 6:

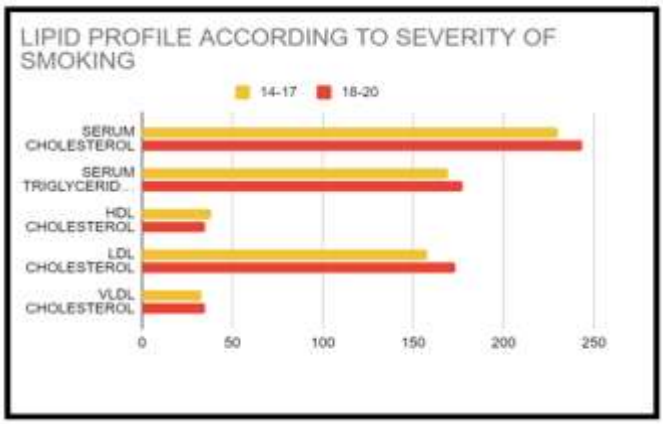


When analyzing the lipid profile of smokers versus non-smokers, certain lipid values such as serum cholesterol, triglycerides, LDL cholesterol, and VLDL cholesterol were found to be elevated in smokers. Conversely, smokers exhibited a reduction in HDL cholesterol levels compared to non-smokers. This comparison reveals a pattern of increased unfavorable lipid levels and decreased beneficial HDL cholesterol levels among smokers in contrast to non-smokers.

Table 7: Lipid Profile According to Severity of Smoking

Lipid Profile	14-17 Cigarettes Per Day (Mean ± Sd)	18-20 Cigarettes Per Day (Mean ± Sd)
Serum Cholesterol	230.045 ± 38.98287447	243.625 ± 45.97335003
Serum Triglycerides	169.54 ± 47.1711446	177.29 ± 40.72045977
Hdl Cholesterol	38.18 ± 8.098420561	34.95 ± 5.536276937
Ldl Cholesterol	157.77 ± 37.90958142	173.125 ± 43.55138145
Vldl Cholesterol	32.68 ± 9.915770813	34.91 ± 9.859520522

Graph 7:

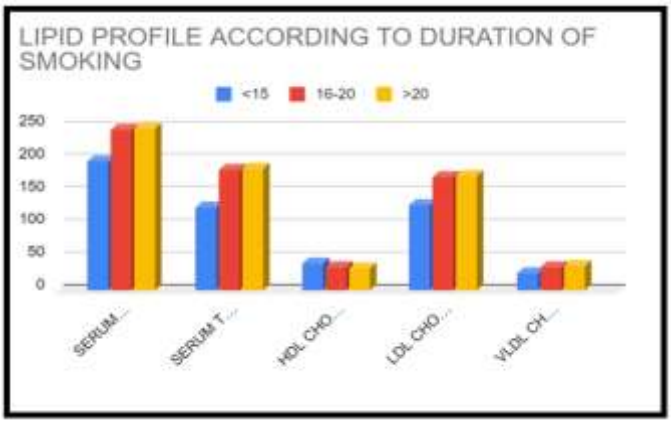


The study evaluated the severity of smoking based on the number of cigarettes smoked per day. It was found that as the severity of smoking increased, there was an associated rise in total serum cholesterol levels. Additionally, HDL-cholesterol levels decreased with increasing severity of smoking. Furthermore, LDL cholesterol, VLDL cholesterol, and triglyceride levels also increased as the severity of smoking increased. These findings indicate a direct relationship between the intensity of smoking and adverse changes in lipid profile, including elevated total cholesterol and triglycerides, along with decreased HDL cholesterol levels.

Table 8: Lipid Profile According to Duration of Smoking

Lipid Profile	<15 Years (Mean ± Sd)	16-20 Years (Mean ± Sd)	>20 Years (Mean ± Sd)
Serum Cholesterol	197.5 ± 38.16703057	247.2 ± 38.21513869	247.58 ± 43.97201245
Serum Triglycerides	127.8 ± 47.65804118	184.46 ± 40.91384875	187.41 ± 33.69504505
Hdl Cholesterol	40.8 ± 9.21110441	36.39 ± 5.533328552	33.83 ± 6.042776805
Ldl Cholesterol	131.1 ± 38.99415911	173.75 ± 34.1126945	176.16 ± 43.58029858
Vldl Cholesterol	26.5 ± 9.336309049	34.89 ± 9.405826743	38 ± 10.36602843

Graph 8:



The study assessed the duration of smoking based on the number of years individuals had been smoking. It was observed that as the duration of smoking increased, there was a corresponding increase in the mean levels of total cholesterol, LDL-cholesterol, and triglycerides. Conversely, HDL-cholesterol levels decreased with longer durations of smoking. These findings suggest that longer-term smoking is associated with adverse changes in lipid profile, characterized by higher total cholesterol, LDL-cholesterol, and triglyceride levels, coupled with lower HDL-cholesterol levels.

Discussion

In contrast to Western societies, cigarette smoking in Indian society tends to begin prominently during the third decade of life, accounting for the highest number of smokers. This trend often starts during late adolescence and continues into college life, where individuals are drawn to the allure of smoking for the "kick" it provides. Some individuals begin smoking to feel more adult-like, with a cigarette between their lips and smoke billowing from their nostrils. While many individuals eventually quit smoking, a minority may transition to more addictive substances like ganja.

In India, the type of smoking often correlates with social class. Bidi smoking, which is less expensive, is commonly seen among manual laborers. On the other hand, the elite class typically prefers filter cigarettes. This socioeconomic divide influences the choice of smoking materials within Indian society.

Numerous studies have explored the clinical, pathological, and biochemical aspects related to smoking, with the majority of research conducted abroad. However, there is a relative scarcity of such studies conducted in India. This current study, conducted as part of postgraduate training, seeks to quantitatively estimate lipid variables and compare the findings with existing published data.

Cigarette smokers face a higher risk of coronary heart disease compared to non-smokers. Several potential explanations have been proposed for this association, including changes in blood coagulation, reduced fibrinolysis, compromised arterial wall integrity, and alterations in blood lipid and lipoprotein levels. This study specifically investigates the relationship between smoking and changes in serum lipid and lipoprotein concentrations, analyzing the collected data to support this link.

In India, female smokers are relatively uncommon due to cultural taboos surrounding smoking for the majority of women. Consequently, this study predominantly focuses on male smokers to examine the impact of smoking on lipid profiles in this demographic.

Strength of the Study

- This study includes a well-defined cohort of male participants, which ensures homogeneity and reduces variability related to gender differences in lipid metabolism.
- The age distribution covers a broad range, allowing for the assessment of smoking effects across different life stages.

- The detailed analysis of various lipid parameters, including total cholesterol, HDL, LDL, and triglycerides, provides a comprehensive overview of the lipid profile in smokers versus non-smokers. The use of standardized methods for measuring lipid levels, such as the CHOD-PAP method for total cholesterol, ensures accuracy and reliability of the results.
- Additionally, the study's focus on newly diagnosed smokers provides insights into early lipid changes associated with smoking, potentially aiding in early intervention strategies.
- The comparison between different severities of smoking further adds depth to the understanding of how smoking intensity affects lipid levels.

Limitations of Study

- There are several potential confounders in the relationship between smoking and blood lipid-related indices that were not accounted for in this study, such as diet, education, occupation, and socio-economic status. Additionally, lifestyle factors like physical activity and alcohol consumption were not examined, which could influence lipid levels.
- Another limitation is that the study's cross-sectional design prevents the establishment of causality between smoking and changes in lipid-related indices.
- Furthermore, the reliance on self-reported data for smoking habits might introduce reporting bias.
- The sample size, although adequate, may not fully represent the broader population, limiting the generalizability of the findings.

Conclusion

Lipid Profile Differences Between Smokers and Non-Smokers.

In this study, healthy smokers exhibited significantly higher mean serum levels of low density lipoprotein-cholesterol (LDL-C) and triglycerides (TG), while the mean serum levels of high density lipoprotein-cholesterol (HDL-C) were significantly lower compared to non-smokers. These findings highlight distinct lipid profile differences associated with smoking status, suggesting potential cardiovascular risk factors in healthy smokers.

Association of Lipid Abnormalities with Smoking Duration and Severity

The study also revealed a linear relationship between lipid abnormalities (elevated LDL-C and TG, decreased HDL-C) and both the duration and severity of smoking. This suggests that longer duration and heavier smoking habits may contribute to worsening lipid profiles, posing increased cardiovascular risks among smokers. The observed lipid profile values in this study align with existing data published in India and internationally. This consistency underscores the generalizability and relevance of the study's findings, emphasizing the global impact of smoking on lipid metabolism and cardiovascular health outcomes.

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