ORIGINAL RESEARCH

Serum nicotine level among various tobacco users

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ABSTRACT
Background: This study was conducted to evaluate serum nicotine levels in tobacco users. **Material and methods:** 100 subjects were recruited in all which were divided into two groups of 50 subjects each. Group A comprised of 50 tobacco users while Group B was the control group with 50 non users. For each participant, a questionnaire assessed tobacco use. Serum nicotine levels were measured. **Results**: Mean cotinine levels were 93.26 ± 16.73 and 26.54 ± 9.2 ng/ml among tobacco smokers and non-smokers, respectively. Cotinine ≥15 ng/ml was measured in 81% of smokers and in 23% of non-smokers. **Conclusion**: From the results, it was concluded that serum nicotine levels among tobacco users were higher in comparison to non-users.

Keywords: Tobacco, serum nicotine

INTRODUCTION
Nicotine is a natural ingredient acting as a botanical insecticide in tobacco leaves. It is the principal tobacco alkaloid, occurring to the extent of about 1.5% by weight in commercial cigarette tobacco and comprising about 95% of the total alkaloid content. Oral snuff and pipe tobacco contain concentrations of nicotine similar to cigarette tobacco, whereas cigar and chewing tobacco have only about half the nicotine concentration of cigarette tobacco. An average tobacco rod contains 10–14 mg of nicotine[1], and on average about 1–1.5 mg of nicotine is absorbed systemically during smoking.[2] Nicotine in tobacco is largely the levorotary (S)-isomer; only 0.1–0.6% of total nicotine content is (R)-nicotine.[3] Chemical reagents and pharmaceutical formulations of (S)-nicotine have a similar content of (R)-nicotine (0.1–1.2%) as impurity since plant-derived nicotine is used for their manufacture. Small amounts of the N′-methyl derivatives of anabasine and anatabine are found in tobacco and tobacco smoke. Several of the minor alkaloids are thought to arise by bacterial action or oxidation during tobacco processing rather than by biosynthetic processes in the living plant.[4]
Hence, this study was conducted to evaluate serum nicotine levels in tobacco users.

MATERIAL AND METHODS
In total, 100 subjects were chosen out of which 85 were males and 15 were females.The subjects were divided into 2 groups: tobacco group (A) and control group (B). Both the groups comprised of 50 subjects each. A survey was used to determine each participant's cigarette consumption. Nicotine levels in the blood were measured. Each participant filled out a questionnaire that measured their usage of tobacco, either through an interviewer or on their own. All participants' biological samples were also taken. The subjects ranged in age from 20 to 88 years old. The proportions of smokers and non-smokers with mean cotinine levels equal to or higher than our chosen cutpoints, 3 and 15 ng/ml, were compared using chi-square testing. To examine the relationship between cigarette usage and cotinine levels, we performed Mann-Whitney and Kruskal-Wallis tests. The analysis was carried out using the SPSS programme.

RESULTS
Mean cotinine levels were 93.26 ± 16.73 and 26.54 ± 9.2 ng/ml among tobacco smokers and non-smokers, respectively. Cotinine ≥15 ng/ml was measured in 81% of smokers and in 23% of non-smokers.

Table 1: mean serum nicotine levels among tobacco users and non-users.

Group	Mean serum nicotine levels (in ng/ml)
Group A (tobacco users)	93.26
Group B (control)	26.54

It was observed that serum nicotine levels were high in tobacco users in comparison to non-users.

Table 2: gender-wise distribution of subjects

Gender	Number of subjects	Percentage
Males	85	85%
Females	15	15%
Total	100	100%

DISCUSSION

Tobacco is a harmful, addictive chemical responsible for many oral diseases and adverse oral conditions.[5] It can be consumed in two forms, smoking and non smoking.[6] Tobacco consumption in any form is responsible for diseases like oral cancer, adult periodontal diseases, and congenital defects such as cleft lip and palate in children whose mother consumed tobacco during pregnancy.

Virtually all tobacco products contain nicotine in substantial concentration.[7] Cotinine, a major metabolite of nicotine, can be easily detected in various body fluids like blood, urine and saliva.[8] It is most commonly used marker to distinguish between tobacco users and non users because of its greater sensitivity and specificity than other biochemical tests.[9,10]

There is a high correlation between blood and saliva cotinine concentrations. A widely used biomarker is urine cotinine level since cotinine concentrations are four to six times higher in urine than that in blood or saliva.[10] This makes quantitative methods (like gas chromatography/mass spectrometry or high performance liquid chromatography, colorimetric assays and immunoassays) which measure urine cotinine more valid and reliable. The disadvantages of higher cost and time consumption have been addressed by recently developed semi quantitative methods.[11]

Hence, the current study was undertaken to assess the serum nicotine levels among tobacco users.

In most tobacco strains, nornicotine and anatabine are the most abundant of minor alkaloids, followed by anabasine. This order of abundance is the same in cigarette tobacco and oral snuff, chewing, pipe, and cigar tobacco. However, nornicotine levels are highest in cigar tobacco, anatabine levels are lowest in chewing tobacco and oral snuff, and anabasine levels are lowest in chewing tobacco.[12] Small amounts of the N′-methyl derivatives of anabasine and anatabine are found in tobacco and tobacco smoke. Several of the minor alkaloids are thought to arise by bacterial action or oxidation during tobacco processing rather than by biosynthetic processes in the living plant.[13] Quantitative aspects of the pattern of nicotine metabolism have been elucidated fairly well in people. Approximately 90% of a systemic dose of nicotine can be accounted for as nicotine and metabolites in urine. Based on studies with simultaneous infusion of labeled nicotine and cotinine, it has been determined that 70–80% of nicotine is converted to cotinine.[14]

JL Rapp et al[15] aimed to compare serum cotinine levels in e-cigarette and combustible cigarette smokers, in an attempt to quantify the potential chronic nicotine addiction risk that e-cigarettes pose. They analyzed 428 participants in 2015-2016 NHANES: 379 (87.03%) smoked combustible cigarettes alone and 49 (12.97%) smoked e-cigarettes. Serum cotinine levels were measured by isotope-dilution high-performance liquid chromatography/atmospheric pressure chemical ionization tandem mass spectrometric method with a detection limit of 0.015 ng/ml. Electronic cigarette smokers were younger than combustible cigarette smokers (mean age 36.79 versus 42.69 years, $P = 0.03$), more likely to be male (64.93% versus 48.32%, $P = 0.09$) and significantly less likely to live with other smokers (50.17% versus 90.07%, $P < 0.01$). Serum cotinine levels increased linearly with self-reported days of smoking in both electronic cigarette and combustible cigarette smokers, after accounting for living with a smoker. The analysis of the subgroup who reported daily use showed non-statistically significantly higher serum cotinine levels in electronic cigarette smokers versus combustible cigarette smokers (β adj = 52.50, $P = 0.10$).

CONCLUSION

It was observed that serum nicotine levels were significantly higher among tobacco users in comparison to non-users.

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