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Menthol and Nonmenthol Cigarettes and Smoke Exposure in Black and White Women

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AHIJEVYCH, K., J. GILLESPIE, M. DEMIRCI AND J. JAGADEESH. *Menthol and nonmenthol cigarettes and smoke exposure in black and white women.* PHARMACOL BIOCHEM BEHAV **53**(2) 355-360, 1996.—Purposes of this investigation were to compare smoke constituent exposure (CO and nicotine boosts) and smoking topography parameters between black and white women, and between women regularly using menthol or nonmenthol cigarettes. A two-factor factorial design with a sample of 37 women stratified by race and menthol or nonmenthol cigarette use was implemented. There were significant main and interaction effects of race and menthol/nonmenthol use on CO boost. Black women had a mean CO boost of 10.1 ppm vs. 7.2 ppm for white women, while women using nonmenthol cigarettes had a higher CO boost (mean = 10.6 ppm) compared to those regularly using menthol cigarettes (mean = 6.5 ppm). White menthol smokers had the lowest CO boost of all subgroups. There was a trend for black women to have higher nicotine boost than white women (21.4 ng/ml vs. 15.9 ng/ml). Black women had nonsignificantly higher puff volumes compared to white women (mean = 48.4 vs. 43.5 ml), while nonmenthol smokers had nonsignificantly higher puff volumes than menthol smokers (mean = 48.5 vs. 42.7 ml). Lower CO boost with mentholated cigarettes suggests factors beyond mentholation may affect elevated smoke constituent exposure among black women.

Menthol Topography Women Race Cigarettes Nicotine Carbon monoxide

SMOKING-RELATED disease results in more deaths among African Americans than those from homicide, accidents, drug abuse, and AIDS combined (3). Further, cigarette smoking intensifies a number of serious health problems that disproportionately affect African Americans, including hypertension, diabetes, low birth weight, and infant mortality (17). African American women smokers are at higher risk for lung cancer in relation to other race-gender groups of smokers (12). In addition, there is a lower quit rate among black women ever-smokers compared to white women ever-smokers (28). Although blacks report smoking fewer cigarettes, they tend to smoke brands with higher nicotine and tar yields, especially mentholated brands (30). Based on survey data, Orleans et al. (30) proposed that low-rate smokers of high nicotine menthol cigarettes may achieve higher than assumed levels of nicotine intake and dependency, and tobacco smoke exposure. Wagenknecht et al. (42) indicated that mentholated cigarettes, smoked by 89% of black smokers in their study, as compared to only 30% of white smokers, may yield greater nicotine exposure because of the anesthetic effect of menthol on depth of inhalation (42). A higher mean cotinine level in young adult black women (251 ng/ml) compared to that in white women (176 ng/ml) of similar age was reported (42).

Cigarette smoking topography parameters provide information regarding smoke constituent exposure through puff volume, puff duration, and lung exposure duration measures. Although there are a number of cigarette smoking topography studies that included women in the samples (5,13-15,18,19, 21,26), only one of these presented descriptive smoke exposure and topography data by sex (18). In addition, several topography studies included only men as participants (4,6,32), and one study focused on only women subjects (27). Race or ethnicity of participants was reported in only a few studies as 100% (13) and 40% of subjects as white (6). In summary, an intentional focus on smoke constituent exposure and topography in black women has not been reported to date, and these areas are inadequately understood. Information about smoke constituent exposure and topography may provide insights regarding nicotine dependence, lower smoking cessation rates, and reported higher cotinine levels among black women.

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In addition, reports describing the effect of mentholated cigarettes on respiratory parameters in humans are limited (6). Higher nicotine delivery is associated with menthol brands, compared with counterpart nonmenthol cigarettes (7), and it is possible that anesthetizing effects of menthol additives enable the smoker to tolerate deeper or more frequent inhalations (30), or greater lung exposure through prolonged exhalation. Animal studies provide potential support for this hypothesis. *l*-Menthol constituted a specific stimulant of laryngeal cold receptors in anesthetized dogs (34), suggesting less laryngeal irritation with mentholated cigarettes. In the presence of menthol, prolonged expiratory duration was observed in guinea pigs (29) and newborn dogs (35), indicating a potential increase in lung exposure duration with menthol cigarettes. Because black women predominantly choose mentholated cigarettes, it is important to examine the effect of this additive on smoking topography parameters and plasma nicotine and expired air carbon monoxide (CO) levels. Examining race and menthol/nonmenthol preference may contribute additional information to explicate higher cotinine levels in black women.

The purpose of this study was to determine if there were differences in smoke constituent exposure by race and menthol/nonmenthol use in women. In addition, smoking topography parameters were compared by race and menthol/nonmenthol use. The relationships of CO and nicotine boosts to smoking topography parameters, nicotine dependence, and smoking beliefs were also investigated. The following hypotheses were posited for testing: 1) menthol smokers compared to nonmenthol smokers will have higher CO and nicotine boost and smoking topography measures; 2) black women compared to white women will have higher CO and nicotine boost and smoking topography measures; and 3) there will be an interaction of race and menthol/nonmenthol use on smoke constituent exposure.

METHOD

Subjects

Participants were 37 women recruited through neighborhood and campus advertisement and worksite postings for a study on cigarette smoking. Recruitment stratified by race and usual menthol or nonmenthol use resulted in 18 black women and 19 white women participants. Approximately one-half of each group regularly smoked menthol cigarettes ($n = 8$ in blacks, $n = 10$ in whites). Participants were limited to those self-reporting ≤ 20 cigarettes per day and no other forms of nicotine intake. Each woman was paid $40 for participation. Participants were limited to women to reduce variance related to gender in a small sample (37) and to address the gap in reported smoke constituent exposure and topography research concerning women.

Procedure

A two-factor factorial design with 37 women stratified by race and menthol/nonmenthol use was implemented. Women were admitted to the Clinical Research Center, acclimated to the unit, and provided with an unopened pack of their usual brand of cigarettes, as up to 25% loss of menthol has been reported within 1 day of pack opening (38). Following written informed consent, a carbon monoxide in expired air sample was obtained 2 min before smoking one of her usual cigarettes. One minute before subject's smoking, a blood sample for nicotine and baseline cotinine analysis was obtained via an indwelling venous catheter. Subjects' smoking and respiratory topography were measured as described below (43). One minute after the cigarette was completed, a blood sample for nicotine analysis was obtained, followed by a CO sample 2 min post cigarette. These measures yielded nicotine and CO boost variables.

Puffing topography measures were determined by a flowmeter cigarette holder (Arthur D. Little, Inc., Cambridge, MA) attached to a differential pressure transducer (BEC Controls Corp., Davenport, IA). Transducer selection was based on pressure differences detected by the holder as measured by a water manometer. The holder was calibrated by obtaining voltage output for 38 flow rates over the desired range by attaching a steppor motor (Harvard Apparatus, Dover, MA) to the plunger of a 400 ml syringe. Ten custom-designed graduated shafts and four steppor motor settings were used to generate flow rates from 1.5 ml/s to 82 ml/s. A curve-fitting program resulted in $< 5\%$ error on known volumes. During the experiment, analog data at 100 Hz from the transducer were converted by an AD board (National Instruments, Austin, TX), and displayed via LabVIEW software (National Instruments, Austin, TX) on a 486DX2 EISA computer (Comtrade, City of Industry, CA). C++ language was used to program peak detection and interface with LabVIEW software.

Respiratory variables of inhalation and exhalation volumes and duration associated with each cigarette puff were obtained by Respigraph inductive plethysmography (NonInvasive Monitoring Systems, Miami, FL) calibrated with a dry rolling seal spirometer (S&M Instruments Co. Inc., Doylestown, PA). A 3 liter syringe (Hans Rudolph, Inc., Kansas City, MO) was used daily to validate spirometer readings.

Exposure to carbon monoxide was assessed in a sample of expired air measured in parts per million (ppm). Ecolyzer 2000 equipment (National Draeger, Pittsburg) was calibrated daily with a standard CO sample of 50 ppm. Participants were instructed in the correct technique to provide an alveolar air sample for analysis (20).

Plasma nicotine and cotinine were assayed by high-performance liquid chromatography (11). The assay was standardized daily, using a series of nicotine and cotinine standards from values of 1.56 to 100 ng/ml and 3.9 ng/ml to 1000 ng/ml, respectively. In our laboratory, average extraction efficiency was 89% and average interassay coefficient of variation was 4.9% (2).

Nicotine dependence was measured via self-report with nine items assessing automatic and dependent smoking behavior (40). Convergent construct validity of the nine-item nicotine dependence instrument was supported by a moderate correlation ($r = 0.49$) with the Fagerstrom Tolerance Questionnaire (8,40). A four-point Likert response yielded a score range of 0 to 27 for the instrument, with higher scores indicating more nicotine dependence. A score ≥ 19 was considered high dependence (40). In a previous study, internal consistency reliability for the nicotine dependence scale was $\alpha = 0.87$ among 187 black women (1). A second measure of nicotine dependence was self-reported time (in minutes) to first cigarette of the day (TTF), because latency to first cigarette was identified as an important predictor of degree of dependency (16). Heatherton and others (16) reported TTF explained 30% of variance in cotinine levels, compared to 11% variance explained by number of cigarettes per day. Pomerleau and others (33) also identified latency to first cigarette as a strong predictor of cotinine level.

Pros and cons of smoking beliefs were assessed with a 20-item instrument based on a decision-making model (41).

Principal components analysis had yielded two orthogonal components labeled as positive and negative beliefs about smoking with internal consistency reliabilities of 0.87 and 0.90, respectively (41). Possible scores range from 10 to 50, with higher scores indicating stronger beliefs.

Because this study was naturalistic in design, women were instructed to smoke as they usually would during the day prior to the experiment. There were no significant differences on time of data collection by race or menthol/nonmenthol cigarette use. While appointment times ranged from 0800 to 1700 h, the mean appointment times for blacks, whites, menthol and nonmenthol users ranged from noon to 1325 h by group. Time since last cigarette was determined by self-report, and there were no significant differences by race or menthol/nonmenthol cigarette use. There were no significant relationships between time since last cigarette and CO boost ($r = -0.14$, $p = 0.4$) or nicotine boost ($r = 0.03$, $p = 0.9$).

Data Analysis

In group comparisons of subject characteristics, *t*-tests were used for means of continuous variables and χ^2 tests were used for categorical variables. Two-way ANOVAs were used to compare plasma nicotine and expired air carbon monoxide boosts between black vs. white women, between menthol vs. nonmenthol use, and the interaction of race and menthol status. These analyses were accomplished using appropriate SAS (Statistical Analysis System, SAS Institute, Inc., Carey, NC) programs for an unbalanced design. Multiple regression analyses were used to determine explanatory variables for CO and nicotine boost.

RESULTS

All subjects ($n = 37$), age 19 to 59 years (mean = 35.4), reported an average 14.7 cigarettes per day, regular smoking for a mean of 15.0 years, and cigarette brands with FTC nicotine yield from 0.4 to 1.7 mg (mean = 0.92). Education ranged from 10 to 17 years (mean = 14.1) and 41% of the sample reported an annual household income < $15,000. Sixty percent were employed full time and 19% were married. Of those with partners ($n = 24$), 62% of partners were smokers. Sixty-two percent of the sample reported that one-half or more of their friends were cigarette smokers. Mean scores on beliefs about the positive and negative aspects of cigarette smoking were 26.4 and 32.4, respectively.

There were significant ($p < 0.05$) differences between the white and black participants on age, with blacks being older (mean = 39.1) than whites (mean = 31.9), and on education, with whites (mean = 14.8 years) reporting more education than blacks (mean = 13.3 years). In addition, cigarettes used regularly by black women were significantly higher in 1991 FTC nicotine yield (mean = 1.04 vs. 0.80 mg), tar (mean = 14.2 vs. 9.9 mg), and carbon monoxide (mean = 13.9 vs. 10.8 mg), than cigarettes smoked by white women. Baseline plasma cotinine was significantly ($p = 0.04$) higher in black women (mean = 273 ng/ml vs. 188 ng/ml). The ratio of plasma cotinine to number of self-reported cigarettes per day provided an indicator of tobacco consumption over time (31). Black women had significantly ($p = 0.001$) higher ratios than white women at 21.1 ng/ml cotinine/cigarette vs. 12.7 ng/ml cotinine/cigarette, respectively. Women did not differ significantly by race on other sociodemographic or smoking history variables, such as income, partner's smoking status, years of regular smoking, cigarettes per day, nicotine dependence score (10.8 in black women and 11.0 in white women), or time to first cigarette (27 min in blacks and 37 min in whites).

There were no significant differences on sociodemographic or smoking history variables between women regularly smoking menthol cigarettes and those smoking nonmenthol cigarettes. Baseline cotinine was nonsignificantly higher in nonmenthol smokers compared to menthol smokers (254 ng/ml and 204 ng/ml, respectively). Cotinine/cigarette ratios were nonsignificantly higher in nonmenthol smokers as well (18.1 vs. 15.3 ng/ml cotinine/cigarette).

CO Boost

On CO boost there was a significant main effect for race with blacks higher than whites, $F(1, 33) = 4.98$, $p = 0.03$, and a main effect for menthol/nonmenthol use with nonmenthol smokers higher than menthol smokers, $F(1, 33) = 9.40$, $p = 0.004$. Black women had a mean CO boost of 10.1 ppm compared to 7.2 ppm for white women, while women regularly using nonmenthol cigarettes had a higher mean CO boost (10.6 ppm) compared to those smoking menthol cigarettes (mean = 6.5 ppm). There were no race-menthol preference interaction effects (Figs. 1 and 2).

To examine whether CO content of cigarettes influenced CO boost, multiple regression analyses were conducted. The variables menthol/nonmenthol use and cotinine/self-reported cigarette ratio explained 31.5% of adjusted variance in CO boost, $F(2, 34) = 9.27$, $p = 0.006$. Greater CO boost occurred in women who regularly smoked nonmenthol cigarettes and among women with higher cotinine/cigarette ratios. Interestingly, CO content of cigarettes and puffing topography

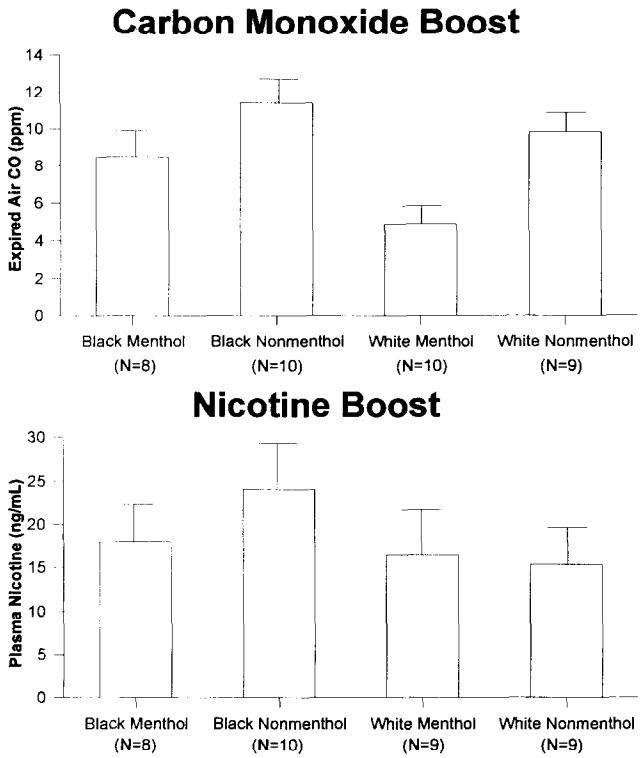

FIG. 1. Mean CO and nicotine boost by race and menthol/nonmenthol use.

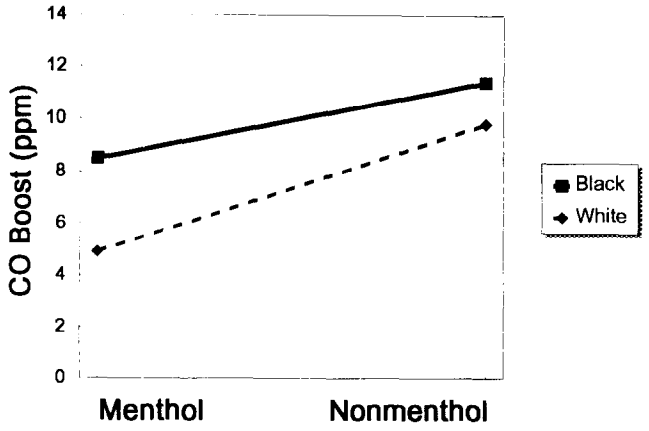

FIG. 2. Effect of race and menthol preference on mean CO boost.

parameters were not significant explanatory variables of CO boost. When menthol/nonmenthol use was not included in the model, 35% of CO boost adjusted variance was explained by the variables cotinine/cigarette ratio, time to first cigarette of the day, and years of regular smoking, $F(3, 33) = 7.43$, $p = 0.0006$. Higher cotinine/cigarette ratio, shorter time to first cigarette, and fewer years of regular smoking were associated with higher CO boost values.

Nicotine Boost

There were no significant differences in nicotine boost by race and/or menthol/nonmenthol use. However, there was a trend for black women to have higher nicotine boost (21.4 ng/ml vs. 15.9 ng/ml) than white women. Nonmenthol and menthol cigarette smokers had similar mean nicotine boosts at 19.9 and 17.2 ng/ml, respectively (Fig. 1). FTC nicotine yield of cigarette explained 13.5% of the variance in nicotine boost, $F(1, 34) = 5.3$, $p = 0.03$. No other study variables contributed significantly to this explanation.

Puffing and Respiratory Parameters

There were no significant differences by race or menthol/nonmenthol use in puffing and respiratory parameters as evidenced in Table 1. There were, however, significant relationships between topography parameters and behavioral and biochemical measures. Counterintuitively, mean puff duration was negatively associated with positive beliefs about smoking ($r = -0.47$, $p = 0.005$). Baseline ratio of cotinine/self-reported cigarette was related to both puff duration ($r = 0.42$, $p = 0.01$), and CO boost ($r = 0.44$, $p = 0.006$).

DISCUSSION

Contrary to our hypothesis, menthol smokers did not have higher smoke constituent exposure compared to nonmenthol smokers. In fact, nonmenthol smokers experienced significantly higher CO boost and nonsignificantly higher puff volumes. The second hypothesis regarding race and smoke constituent exposure was partially supported in that black women had significantly higher CO boost, and nonsignificantly higher nicotine boost and puff volumes. Finally, while white women menthol smokers had the lowest CO boost of all race-menthol preference subgroups, there were no significant race-menthol preference interaction effects.

Anticipated anesthetic effects of menthol cigarettes on smoke constituent exposure and topography did not occur in this study. Similar findings were reported by Tashkin and others (39) in that significantly greater mean puff volume and total puffs occurred with nonmenthol vs. menthol cigarettes. However, they observed a greater COHb boost with menthol cigarettes in a crossover design including 10 black and 10 white smokers, with gender unreported (39).

Several investigators reported complex effects of menthol, which may explain conflicting results regarding mentholated cigarettes. Naito et al. (25) reported that although menthol lozenges yielded an increased sensation of nasal patency by 15 adults, there was no significant difference in actual nasal resistance. Green (9,10) described a complicated series of effects of menthol on the perception of oral warming and cooling related to menthol concentrations (10). Also, at higher concentrations, menthol was perceived as irritating or bitter (9). In addition, the temporal course of menthol pretreatment affected thermal modulation (9). Time to complete smoking one cigarette may influence menthol pretreatment effects. However, in the current study there were no significant differences by race or menthol/nonmenthol use in total cigarette duration or lung exposure duration.

Sant'Ambrogio (35) noted that a thick mucus layer may reduce the effect of menthol on mucosal cold nerve endings. Because cigarette smokers experience increased airway mucus production, the anesthetic effects of menthol may be diminished. In addition, menthol pyrolyses conducted at the reported burning temperature of a cigarette, resulted in unreacted menthol accounting for only 16% of the neutral fraction (36). Thus, the amount of menthol in mainstream smoke may be reduced by pyrolysis, as compared to studies previously cited using direct menthol application. Although this reasoning may address the lack of an anticipated menthol anesthetic effect, there is no clear explanation for higher CO boost with nonmenthol cigarette use.

The main effect of race on CO boost is intriguing, with black women being significantly higher than white women. FTC cigarette CO content was not a significant variable explaining CO boost in multiple regression analysis. Significant explanatory variables of CO boost included higher cotinine/cigarette ratios, a measure of consumption, and shorter TTF, an indicator of nicotine dependence, which are logical factors in understanding elevated CO boost. This finding, however,

TABLE 1

MEAN PUFFING AND RESPIRATORY PARAMETERS BY RACE AND MENTHOL PREFERENCE*

	Black $n = 16$	White $n = 17$	Menthol $n = 15$	Nonmenthol $n = 18$
Mean puff duration (s)	1.8	1.5	1.70	1.66
Mean puff volume (ml)	48.4	43.5	42.7	48.5
Mean interpuff interval (sec)	20.7	19.8	22.6	18.2
Total puffs	13.8	15.8	13.6	15.8
Total puff duration	24.7	23.7	22.9	25.3
Total puff volume	660.4	666.1	578.7	733.9
Inhalation duration (s)	1.8	2.1	2.2	1.8
Exhalation duration (s)	3.8	3.3	3.5	3.6

*Topography data for four subjects not available (one black menthol, one black nonmenthol, two white menthol).

contradicts Caskey's (6) report of no difference in CO boost by race among men in a crossover design where each subject smoked menthol and nonmenthol cigarettes.

In the current study, lower CO boost with mentholated cigarettes suggests that factors beyond mentholation may affect differences in cotinine levels in black and white women. Nicotine dependence may be an important variable in increased smoke constituent exposure, as evidenced by higher cotinine/cigarette ratios and shorter latency to first cigarette associated with elevated CO boost. Black women had significantly higher cotinine/cigarette ratios than white women in this study and were higher in every smoking category compared to Mexican American women in a previous study (2). Gender and age have been reported to influence smoke constituent exposure (22,37), and ethnicity encompasses lifestyle considerations such as socioeconomic factors, diet patterns, and metabolic issues that have been identified as influencing nicotine consumption and metabolism (24,37).

Effects of race and menthol on smoke constituent exposure and smoking topography parameters require further study in a larger group of women with increased variability on self-reported cigarettes per day. Further research can examine nicotine dependence, inter- and intraindividual nicotine metabolism variation, as well as lifestyle influences of socioeconomic issues and diet on smoke constituent exposure.

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