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Exposure of Casino Employees to Environmental Tobacco Smoke

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Abstract

Environmental and medical evaluations were performed to evaluate occupational exposure to environmental tobacco smoke (ETS) among casino employees. Air concentrations of both nicotine and respirable dust were similar to those published in the literature for other non-industrial indoor environments. The geometric mean serum cotinine level of the 27 participants who provided serum samples was 1.34 nanograms per milliliter (ng/mL)(pre-shift) and 1.85 ng/mL (post-shift). Both measurements greatly exceeded the geometric mean value of 0.65 ng/mL for participants in the Third National Health and Nutrition Examination Survey (NHANES III) who reported exposure to ETS at work. This evaluation demonstrates that a sample of employees working in a casino gaming area were exposed to ETS at levels greater than those observed in a representative sample of the US population, and that the serum and urine cotinine of these employees increased during the workshift.

In 1995 the National Institute for Occupational Safety and Health (NIOSH) received an employee request for a health hazard evaluation (HHE) concerning exposure to second-hand (environmental) tobacco smoke (ETS) among employees at a casino in Atlantic City, New Jersey. In response to this request, NIOSH performed a field study to evaluate the exposure of gaming floor employees to ETS using both environmental and biologic measures of exposure.1

Environmental Tobacco Smoke

Occupational exposure to ETS is recognized as an important public health issue.2,3 NIOSH has determined that ETS poses an increased risk of lung cancer, other lung disease, and possibly heart disease to occupationally exposed workers and recommends eliminating or restricting tobacco use in the workplace.4 Although many workplaces are adopting policies that restrict smoking, occupational exposure to ETS remains a concern among some of the 110 million Americans who work outside the home.5,6 Occupational ETS exposures have not been evaluated to the extent that home exposures have.4 In particular, there is very little information available concerning the exposure of casino employees in the United States to ETS.

In this survey, vapor-phase nicotine and respirable particulate were monitored as marker substances for exposure to ETS. Vapor-phase nicotine, which accounts for approximately 95% of nicotine in ETS, is currently a widely accepted marker for ETS exposure.7-9 One potential drawback of vapor-phase nicotine is that the physical properties of vapor-phase nicotine on indoor surfaces can alter (increase or decrease) its concentration relative to other ETS components.9 Respirable particulate has also been used as a marker of ETS, but it may be difficult to separate the ETS-associated particulate from that of other indoor sources.7-9 The concentrations of these markers in ETS are consistently lower than their respective occupational airborne exposure criteria, which were based primarily on acute effects. The NIOSH Recommended Exposure Limit(REL) and the American Conference of Governmental Industrial Hygienists'(ACGIH) threshold limit value (TLV) for nicotine, used primarily for exposure assessments in agriculture, are 500 micrograms per cubic meter of air($\mu g/m^3$), and are not applicable in evaluations of ETS exposure.10,11 A model used to derive a health-based standard for ETS has shown that an eight-hour, time weighted average exposure to 2.3 $\mu g/m^3$ of nicotine would correspond to 3 lung cancer deaths among 10,000 exposed over a working lifetime.12 The US Department of Labor, Occupational Safety and Health Administration (OSHA) general industry permissible exposure limit (PEL) for respirable particulate not composed of a substance that has its own PEL is 5000 $\mu g/m^3$ (the ACGIH TLV is 3000$\mu g/m;^3$ there is no REL).11,13 In contrast, the mean area air nicotine concentrations reported in ETS studies of public buildings have ranged from 0.7-37 $\mu g/m^3$, concentrations in restaurants and bars have ranged from 2.3-65.5 $\mu g/m^3$, and concentrations in gaming parlors and betting shops have ranged from 11-19$\mu g/m^3$.7,12 One study found that the median air nicotine concentration was 8.6 $\mu g/m^3$ in offices at worksites that allowed smoking.6 Respirable particulate measurements have ranged up to 115 $\mu g/m^3$ in office buildings and up to 843 $\mu g/m^3$ in restaurants.7

Biologic monitoring of exposure to ETS is most commonly conducted by measuring cotinine in the serum and/or urine of potentially-exposed persons.3,8,14-16 Cotinine, which is the major metabolite of nicotine, has a half-life of approximately 16-20 hours, reflecting exposure to nicotine from the previous one to two days.3 There are no NIOSH, ACGIH, or OSHA criteria for cotinine in blood or urine. Although studies reporting cotinine levels in non-smokers exposed to ETS have been summarized,12 differences in laboratory methods sometimes make it difficult to compare cotinine levels determined in different laboratories.17 A study of more than 600 non-smokers attending a medical clinic found a mean urine cotinine level of 8.8 ng/mL (range, 0-85), with increased levels correlating with reported exposures.14 Another study found a mean urine cotinine level of 9.2 ng/mL among non-smokers exposed to ETS at home or work.18 A recent US population survey measuring serum cotinine in over 2600 working adults reported the following geometric means by category: (1) no reported ETS exposure: 0.132 ng/mL;(2) reported ETS exposure at work: 0.318 ng/mL; (3) reported ETS exposure at home: 0.651 ng/mL; and (4) reported ETS exposure at home and work: 0.926 ng/mL.3

Although some foods, including tea, tomatoes, potatoes, and cauliflower, may contain nicotine in measurable quantities and therefore have been suggested as a source of cotinine in the body,19 the amount of cotinine in serum as a result of food intake has been shown to be extremely low relative to that resulting from ETS exposure.3

Workplace Description

The casino that was evaluated was constructed in 1979 and offers a variety of gaming activities, including slot machines, roulette, blackjack, baccarat, craps, and poker. The gaming floor has an area of 71,380 square feet(ft^2); a separate poker area has an area of 8679 $ft.^2$ Gaming activities are in operation 24 hours per day, seven days a week. The maximum occupancy of the casino is 9560 persons. The casino employs approximately 800 persons who work on the casino floor; approximately 330 are full-time dealers and approximately 180 are full-time dealer supervisors. Specific game or area assignments for dealers and supervisors were made at the start of each shift and changed daily (and sometimes within a given work shift). Other casino floor employees include waitresses, cashiers, and security personnel.

The heating, ventilating, and air-conditioning system was controlled by a Honeywell building management system (Honeywell Inc., Minneapolis, MN). There were 17 air handling units, each rated to supply 47,000 cubic feet per minute of conditioned air. Assuming a maximum casino capacity (9560 persons) and a reported minimum 30% outdoor air intake, an outside air rate of 25 cubic feet per minute per person (cfm/person) can be calculated. Although the ventilation system was not inspected, carbon dioxide (CO_2) measurements (which ranged from 425 to 850 parts per million) were consistent with the calculated outdoor air supply rates.

Tobacco smoking by customers is permitted throughout the casino floor; employees do not smoke while on duty. Although some gaming tables are designated as non-smoking, the non-smoking tables are generally located adjacent to tables where smoking is permitted. The employee cafeteria has smoking and non-smoking areas, but these areas are not physically partitioned, and tobacco smoke is evident in the non-smoking area. Employee lounges are designated non-smoking areas.

Methods

The field study was performed in March 1996 and consisted of environmental and medical evaluations. Employee representatives and management were notified in advance of the NIOSH site visit. The study population consisted of dealers and supervisors; there were 279 dealers and supervisors, including both smokers and non-smokers, scheduled to work the second shift(generally the busiest shift of the day) during the two days of the evaluation (Thursday and Friday nights). Dealers and supervisors were chosen as the study population because these were the only employees for whom adequate work schedule information was available. During the evening prior to the evaluation, NIOSH investigators were present in the casino cafeteria to distribute information sheets describing the HHE and to talk to employees. Although the goal was to contact and explain the HHE to each of the 279 employees who were non-smokers, the actual number of non-smokers in the population and the actual number contacted is unknown. Each non-smoking dealer and supervisor contacted was asked to participate in the HHE on one of the two nights (either Thursday or Friday night). Management presence at the time of employee recruitment and employee concern over management disapproval of the evaluation was likely an important factor negatively affecting employee participation. Participants were not paid.

Environmental

Personal breathing zone (PBZ) and general area (stationary) air samples for nicotine vapor were collected by drawing air through XAD-4 sorbent tubes(SKC® #22693; SKC Inc., Eighty Four, PA) with battery-powered SKC Pocket Pumps® at air flow rates of 150 milliliters per minute (mL/min) for personal samples and 200 mL/min for area samples. Sampling was conducted for approximately eight hours. The analyses for nicotine were conducted in the NIOSH laboratory using a modified version of American Society for Testing and Materials (ASTM) method D5075-90a, *Standard Test Method for Nicotine in Indoor Air*.1,20 The total mass of respirable particulate was collected according to NIOSH Method 0600 using pre-weighed polyvinyl chloride (PVC) filters installed in Dorr-Oliver (Milford, CT) nylon cyclones (which collect particulates less than ten microns in diameter).1,21 Sampling was conducted at a flow rate of 1.7 liters per minute (L/min) for approximately eight hours. Nine area samples were collected at the center tables in various gaming pits (locations in the casino gaming area are referred to as pits). Instantaneous measurements of CO_2 concentrations were obtained using a Gas Tech Model RI-411A Portable (direct reading) CO_2 monitor (Gas Tech Inc., Newark, CA). CO_2 measurements were obtained at various intervals and locations throughout the building.

Medical

The medical evaluation included a self-administered questionnaire and biologic monitoring for exposure to cigarette smoke. After giving informed consent and confirming that they did not currently use tobacco products, employees filled out a questionnaire that included questions on work history, tobacco use history, and exposure to ETS. Participants were asked to estimate the amount of time (hours/minutes) that they were exposed to ETS on the day of the evaluation and for the four previous days. The work practices and activities of all participants were observed by NIOSH personnel during the course of the evaluation.

Pre- and post-shift blood and urine specimens were collected from each participant. All samples were blind-coded and sent to the National Center for Environmental Health, Division of Environmental Health Laboratory Sciences. Serum cotinine was determined for each serum sample in duplicate by high-performance liquid chromatography/atmospheric-pressure chemical ionization tandem mass spectrometry (LC APCI MS/MS) according to a standard protocol.3 This method has been shown to be a specific and sensitive method for cotinine measurements.22 The limit of detection (LOD) was 0.050 ng/mL. The mean of two determinations is reported as the final result for all individual samples.

Urine cotinine analyses were made by using a similar LC tandem mass spectrometric procedure with the same LOD. However, for these samples, a preliminary hydrolysis of the cotinine glucuronides was carried out. Thus the urine cotinine results are the total (free cotinine + cotinine glucuronide) levels in the sample. The mean of two determinations is reported as the final result for all individual samples. Both serum and urine cotinine values are reported in units of ng/mL.23-25 Four samples of both serum and urine from each night of testing were split and sent to the laboratory as additional samples not identified as duplicates. Analysis of these samples indicated an overall method coefficient of variation of 2% for both the serum and urine assays in this study.

For the two sample *t* tests and correlations, serum and urine cotinine levels were log-transformed because of the skewness in their distributions. Statistical analyses were performed using Epi Info, Version 6 26 and SAS.27 A *P* value<= 0.05 was considered statistically significant.

Results

Environmental

Eighteen PBZ samples for nicotine and ten area samples each for nicotine vapor and respirable dust were collected. PBZ nicotine exposures for the Thursday evening monitoring ranged from 6-12 μg/m^3 (geometric mean, 8 μg/m^3), expressed as time-weighted averages (TWAs). The highest PBZ sample concentration (12 μg/m^3) was from a dealer working a poker game. Area TWA air concentrations (range, 6-12μg/m;^{3} geometric mean, 8 μg/m^3) were similar to the PBZ sample concentrations. For the Friday evening monitoring, the PBZ concentrations were slightly higher than those of Thursday evening, ranging from 4-15 μg/m^3 as TWAs (geometric mean, 10μg/m;^{3} $P = 0.11$). The highest PBZ exposure (15μg/m^3) was again found on a dealer working a poker game. TWA area air concentrations on Friday ranged from 8-16 μg/m^3(geometric mean, 11 μg/m^3). The two highest area air concentrations on each night were at poker registration and the poker tables. On both evenings, area air concentrations of respirable dust ranged from nondetected (detection limit, 20-30 μg/m^3) to 90μg/m^3. CO_2 concentrations ranged from 425-650 ppm(geometric mean, 527 ppm) on Thursday and from 475-850 ppm (geometric mean, 597 ppm) on Friday. Outdoor CO_2 measurements ranged from 275-300 ppm.

Medical

Twenty-nine persons (10% of the total number of dealers and supervisors[279] at work during the evaluation) participated in the evaluation, including 18 dealers and 11 supervisors. Of the 29 participants, 11 (38%) were supervisors; among the total number of full-time dealers and supervisors employed for all shifts at the casino, 180 (35%) were supervisors. Twenty of the 29 were men; the average age of all participants was 37 years (range, 21-53). No participants reported current tobacco use; 15 reported having never smoked cigarettes, 13 reported having their last cigarette more than 1 year prior to the evaluation, and one reported smoking a last cigarette two weeks prior to the evaluation. Seventeen (59%) of the participants reported no exposure to ETS outside the workplace over the four days prior to the evaluation. All participants provided pre- and post-shift urine samples; 28 provided pre- and post-shift blood samples. All participants were observed to perform their usual work duties during the course of the study.

Individual serum and urine cotinine levels, with the corresponding PBZ nicotine concentrations (when available), are presented in Tables 1 (for employees reporting exposure to ETS at work only) and 2 (for employees reporting exposure to ETS at work and outside of work). One participant (No. 8) was found to have cotinine levels approximately 100 times the levels of all other participants and above the 15 ng/mL serum level used as an indicator of active smoking 3; this person was therefore considered to be an active smoker, and the corresponding results were excluded from all analyses. The geometric means and standard deviations are presented in Table 3.

Participant No.	Job*	PBZ† Nicotine ($\mu g/m^3$)	Serum (ng/mL)		Urine (ng/mL)	
			Pre-Shift Cotinine	Post-Shift Cotinine	Pre-Shift Cotinine	Post-Shift Cotinine
1‡	D	7	2.74	2.62	159	197
2	D	NA§	1.19	1.45	37.7	54.4
3	D	10	1.58	2.22	16.7	39.1
4	S	6	0.885	1.36	21	28.4
5	S	NA	1.07	1.21	5.76	20.7
6	D	NA	0.967	1.32	23.7	26.7
7	S	NA	2.81	2.61	51.4	50.5
8	S	10	1.14	1.95	27.3	35.9
9	D	15	0.23	2.70	7.63	58.0
10	D	12	0.768	1.54	16.4	22.6
11	S	4	1.15	1.41	37.0	43.2
12	D	9	2.19	2.57	44.9	52.6
13	D	14	1.35	1.96	35.6	51.2
14	S	NA	2.38	2.56	26.8	31.2
15‡	D	NA	2.89	3.19	19.5	21.7
16	S	NA	0.659	0.917	23.0	24.1
17	D	NA	1.16	1.42	27.2	33.3

* Job titles: D, dealer; S, supervisor.
† Personal breathing zone sampling for nicotine vapor (time-weighted average).
‡ Some or all of workshift on day of sampling was spent at non-smoking table.
§ NA, test not performed.

TABLE 1 Serum and Urine Cotinine and Nicotine Air Sampling Results Among Casino Employees Reporting Exposure to Tobacco Smoke at Work Only

Participant No.	Job*	PBZ Nicotine† (μg/m³)	Serum (ng/mL)		Urine (ng/mL)	
			Pre-Shift Cotinine	Post-Shift Cotinine	Pre-Shift Cotinine	Post-Shift Cotinine
1	D	9	NA‡	NA	47.6	54.0
2	S	6	0.926	1.47	16.2	23.6
3§	D	9	2.72	2.56	21.2	45.3
4	D	12	2.78	2.91	42.4	58.6
5‖	D	6	113	73	4664	4137
6§	D	8	1.30	1.57	14	7.21
7	S	NA	4.24	3.52	61.1	59.3
8	D	10	1.37	1.77	28.4	33.9
9	D	11	1.39	1.16	23.4	25.3
10	S	NA	1.49	2.03	7.98	28.1
11	D	12	1.05	2.33	17.4	32.5
12	S	NA	0.516	0.959	2.54	3.87

* Job titles: D, dealer; S, supervisor.
† Personal breathing zone sampling for nicotine vapor (time-weighted average).
‡ NA, test not performed.
§ Some or all of workshift on day of sampling was spent at non-smoking table.
‖ Based on high cotinine levels, this participant was determined to be an active smoker; results are excluded from all analyses.

TABLE 2 Serum and Urine Cotinine and Nicotine Air Sampling Results Among Casino Employees Reporting Exposure to Tobacco Smoke at Work and Outside of Work

Parameter	Pre-Shift Cotinine GM* in ng/mL (GSD)²	Post-Shift Cotinine GM in ng/mL (GSD)	*P* Value (Paired *t* Test)
Serum	1.34 (1.9)	1.85 (1.4)	<0.01
Urine	23.0 (2.2)	33.3 (2.0)	<0.01

* GM, geometric mean; GSD, geometric standard deviation.

TABLE 3 Summary of Serum and Urine Cotinine Measurements of Casino Employees*

Post-shift cotinine levels for both serum ($P < 0.01$) and urine ($P < 0.01$) were significantly greater than pre-shift levels. Pre-shift serum and urine cotinine values were correlated with each other ($r = 0.63$, $P < 0.01$), as were post-shift serum and urine cotinine values ($r = 0.58$, $P < 0.01$). For workers who had PBZ air sampling performed during their shift, there were positive correlations (not statistically significant) between post-shift serum cotinine and the corresponding air nicotine concentration ($r = 0.43$, $P = 0.1$) and post-shift urine cotinine and the corresponding air nicotine concentration ($r = 0.05$, $P = 0.86$). The correlation between the cross-shift change in serum cotinine concentration and the PBZ air nicotine concentration was also not statistically significant($r = 0.45$, $P = 0.08$). There were no statistically significant differences between dealers and supervisors with respect to post-shift serum and urine cotinine levels.

Four persons worked all or part of their shift at non-smoking tables (see Tables 1 and 2). The post-shift serum cotinine concentrations of these four individuals (geometric mean, 2.41 ng/mL) were higher than the corresponding cotinine concentrations of those who worked at smoking tables (geometric mean, 1.77 ng/mL). However, those four individuals working at non-smoking tables began the shift with higher serum cotinine concentrations as well (geometric means, 2.30 ng/mL versus 1.22 ng/mL).

There was no significant difference in the mean post-shift serum cotinine values between those reporting ETS exposure at work only (Table 1-17 participants, geometric mean, 1.82 ng/mL) and those reporting ETS exposure at home and work (Table 2-10 participants, geometric mean, 1.91 ng/mL). There were no statistically significant relationships between cotinine levels and hours of reported exposure to ETS (both occupational and non-occupational, as reported in the questionnaire) on the day the sample was taken (r= .09 [post-shift serum cotinine]; r = .18 [post-shift urine cotinine]; mean exposure, 7 hours; range, 2-10 hours) or for hours of reported exposure to ETS on the day of collection and two days prior to the collection (r = -.18 [post-shift serum cotinine];r = -.23 [post-shift urine cotinine]; mean exposure, 17.6 hours; range, 6.5-24 hours).

Discussion and Conclusions

This evaluation demonstrates that a small sample of employees working in the gaming area of a large casino have greater ETS exposure than a representative sample of the US population, as measured in the Third National Health and Nutrition Examination Survey (NHANES III).3 The geometric mean serum cotinine levels of the casino employees in our evaluation were 1.34 (pre-shift) and 1.85 (post-shift) ng/mL. These levels are substantially higher than the geometric mean of 0.65 ng/mL for those participants of NHANES III reporting exposure to ETS at work and the geometric mean of 0.93 ng/mL for those reporting exposure to ETS at both home and work. A strength of our evaluation is that our laboratory analysis for serum cotinine was identical to that performed in the NHANES study, making such a comparison valid. The urine cotinine values in our evaluation are more difficult to compare with those in other studies since most methods for determining urine cotinine measure only free cotinine, whereas the method used in this study measured both free cotinine and cotinine glucuronide and can yield significantly higher values.

Five participants had a decrease in serum cotinine level from pre-shift to post-shift. None of these participants had a serum cotinine level less than 1.16 ng/mL, and four of the five had serum cotinine levels greater than 2.5 ng/mL in *both* their pre- and post-shift samples. Since individual exposure might be expected to vary from day to day within the workplace, it is conceivable that those whose cotinine levels declined slightly during the shift included people who-although exposed during the shift-were less exposed than on the previous day(s) and thus had somewhat lower serum cotinine levels at the end of their shifts than at the beginning. Three of these five participants reported exposure to ETS outside the workplace.

The airborne levels of nicotine and respirable particulates found in our evaluation are similar to those measured in other non-industrial indoor environments.6,7,13 Our evaluation of the ventilation system at this casino suggested that it would meet the American Society of Heating, Refrigeration, and Air-conditioning Engineers' (ASHRAE) recommended outside air ventilation rate for casinos of 30 cfm/person, except under conditions of maximal occupancy and extreme outdoor weather conditions(when the ventilation rate was estimated to be 25 cfm/person [see "Workplace Description"]). This is a greater ventilation rate than what is generally found in office spaces, where ASHRAE recommends 20 cfm/person.28 The CO_2 levels measured during our evaluation (geometric means, 527 and 597 ppm on the two days), which are well below the levels of 800-1000 ppm (levels used to indicate adequacy of fresh air intake),28,29 suggest that adequate outside air was being provided to the casino floor at the time of our evaluation. Our evaluation indicates that providing adequate ventilation in the workplace can help dilute air contaminants, including nicotine. Although there were anecdotal reports of an increased ventilation rate during the survey, it is not possible from the data we collected to accurately predict what affect this, or changes in other variables (such as occupancy rates) would have on measured levels of cotinine.

Based on both air and biological monitoring, employees working at the"non-smoking" tables did not have decreased exposure to ETS, compared with those working at smoking tables. This finding is not surprising since these non-smoking tables were generally located directly adjacent to other tables where smoking was allowed. Generalized exposure to ETS appears to occur throughout the gaming area, suggesting that other groups of casino employees not participating in this evaluation, such as waitresses, cashiers, and security personnel, are likely exposed to ETS at levels similar to the dealers and supervisors.

Similar post-shift serum cotinine values from employees reporting exposure to ETS at work only, compared with those reporting exposure both at home and at work, suggest that the ETS exposure among the group of participants is primarily work-related. This finding supports the findings of others who have demonstrated that occupational ETS exposure is comparable to domestic ETS exposure (which is the setting in which epidemiological evidence has demonstrated the adverse effects of ETS).6

In this small study we found positive, but not statistically significant, correlations between PBZ air nicotine concentration and both post-shift serum cotinine and cross-shift change in serum cotinine. The duration of ETS exposure reported in the questionnaires was not significantly correlated with serum or urine cotinine concentrations. This could be due to a number of factors, including the small number of persons evaluated, the relatively narrow range of cotinine levels, and the narrow range of hours of reported ETS exposure. Although the range of cotinine levels was narrow, the levels were high, compared with non-smokers in NHANES III who reported exposure to ETS at home and work.

A limitation of this study is that the percentage of the 279 dealers and supervisors working during the time of our evaluation who were non-smokers(and thus eligible to take part in the evaluation) is unknown; therefore the participation rate for our evaluation is unknown. Factors that likely affected the participation rate include active discouragement of employee participation by casino management, insufficient employee notification regarding the HHE, and concern over medical testing. Although we were not able to gather demographic or other information about non-participants, we have no reason to believe our participants differed from non-participants in any way that would have affected potential exposure to ETS at the work-place. For example, the wide age range of participants (21-53 years) and the fact that dealers and supervisors took part in numbers proportionate to the number of dealers and supervisors employed at the casino, suggests that a representative mix of employees took part in our evaluation.

There are more than 300,000 persons employed in approximately 450 large casinos in the United States (personal communication, American Gaming Association, October 1997); this figure does not include a potentially larger number of persons employed in smaller casino or gaming operations, as well as persons employed in casino or gaming operations operated on Native American property. The study described here provides the first quantitative data describing exposure to ETS among a small sample of workers in this industry. Further study is needed to determine how generalizable the exposures observed in this study are to the gaming industry as a whole. In the meantime, NIOSH recommends that workers not be involuntarily exposed to tobacco smoke.4 The best method for controlling worker exposure to ETS is to eliminate tobacco use from the workplace and to implement a smoking cessation program for employees. The "non-smoking" tables, as currently situated, did not measurably decrease employee exposure to ETS. Until tobacco use can be completely eliminated, employers should make efforts to protect employees from ETS by isolating areas where smoking is permitted. Separate smoking areas with dedicated ventilation are a means to accomplish this. Restricting smoking to the outdoors (away from building entrances and air intakes) is another method to protect employees from ETS.

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IMAGE GALLERY

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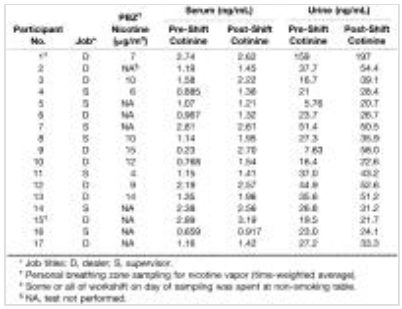
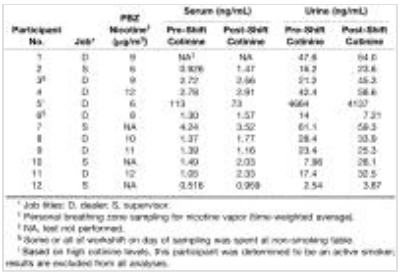


Participant No.	Job*	PBZ† Nicotine (µg/m³)	Serum (ng/mL) Pre-Shift Cotinine	Serum (ng/mL) Post-Shift Cotinine	Urine (ng/mL) Pre-Shift Cotinine	Urine (ng/mL) Post-Shift Cotinine
1‡	D	7	2.74	2.62	159	197
2	D	NA§	1.19	1.45	37.7	54.4
3	D	10	1.58	2.02	16.7	39.1
4	S	6	0.885	1.36	21	26.4
5	S	NA	1.07	1.21	5.76	20.7
6	D	NA	0.987	1.32	23.7	25.7
7	S	NA	2.61	2.61	51.4	50.5
8	S	10	1.14	1.55	27.3	35.9
9	D	15	0.27	2.76	7.83	38.0
10	D	12	0.788	1.84	16.4	27.6
11	S	4	1.19	1.41	37.9	43.2
12	D	9	2.19	2.57	44.8	52.6
13	D	14	1.35	1.98	35.6	51.2
14	S	NA	2.38	2.56	26.8	31.2
15‡	D	NA	2.89	3.19	19.5	21.7
16	S	NA	0.659	0.917	23.0	24.1
17	D	NA	1.18	1.42	27.2	33.3

* Job titles: D, dealer; S, supervisor.
† Personal breathing zone sampling for nicotine vapor (time-weighted average).
‡ Some or all of workshift on day of sampling was spent at non-smoking table.
§ NA, test not performed.

Table 1



Participant No.	Job*	PBZ† Nicotine (µg/m³)	Serum (ng/mL) Pre-Shift Cotinine	Serum (ng/mL) Post-Shift Cotinine	Urine (ng/mL) Pre-Shift Cotinine	Urine (ng/mL) Post-Shift Cotinine
1	D	9	NA‡	NA	47.6	64.0
2	S	6	0.926	1.47	19.2	23.6
3§	D	9	2.72	2.66	21.9	48.3
4	D	12	2.78	2.91	42.4	58.6
5‖	D	6	113	73	4664	4137
6‖	D	9	1.30	1.57	14	7.31
7	S	NA	4.24	3.52	61.1	59.3
8	D	10	1.37	1.77	28.4	30.9
9	D	11	1.39	1.16	23.4	25.3
10	S	NA	1.49	2.03	7.96	28.1
11	D	12	1.05	2.33	17.4	32.5
12	S	NA	0.516	0.968	2.54	3.87

* Job titles: D, dealer; S, supervisor.
† Personal breathing zone sampling for nicotine vapor (time-weighted average).
‡ NA, test not performed.
§ Some or all of workshift on day of sampling was spent at non-smoking table.
‖ Based on high cotinine levels, this participant was determined to be an active smoker; results are excluded from all analyses.

Table 2

Parameter	Pre-Shift Cotinine GM* in ng/mL (GSD†)	Post-Shift Cotinine GM in ng/mL (GSD)	P Value (Paired *t* Test)
Serum	1.24 (1.9)	1.85 (1.4)	<0.01
Urine	23.0 (2.2)	33.3 (2.0)	<0.01

* GM, geometric mean; GSD, geometric standard deviation.

Table 3

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