Environmental Tobacco Smoke Exposure Among Casino Dealers

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Objective: This study quantified casino dealers' occupational exposure to environmental tobacco smoke (ETS). **Methods:** We measured casino dealers' exposure to ETS components by analyzing full-shift air and preshift and postshift urine samples. **Results:** Casino dealers were exposed to nicotine, 4-vinyl pyridine, benzene, toluene, naphthalene, formaldehyde, acetaldehyde, solanesol, and respirable suspended particulates. Levels of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) in urine increased significantly during an 8-hour work shift both with and without adjustment for creatinine clearance. Creatinine-unadjusted cotinine significantly increased during the 8-hour shift, but creatinine-adjusted cotinine did not increase significantly. **Conclusions:** Casino dealers at the three casinos were exposed to airborne ETS components and absorbed an ETS-specific component into their bodies, as demonstrated by detectable levels of urinary NNAL. The casinos should ban smoking on their premises and offer employee smoking cessation programs.

Learning Objectives

- Discuss previous findings on occupational exposure to ETS in casino workers.
- Summarize the methods and findings of the new study, including evidence for exposure to and metabolism of ETS components in casino dealers.
- Discuss the study implications for occupational health and safety in casino workers.

Environmental tobacco smoke (ETS) is a complex array of approximately 5000 chemicals, of which 50 are known carcinogens. The association of ETS exposure in nonsmokers and an increased risk of cancer, cardiovascular disease, and respiratory distress has been well established.[1–7] As a result, many cities and states across the United States have enacted legislation banning cigarette smoking in indoor places.

Only recently has the question of occupational exposure of ETS in casino workers been examined. A study of nicotine levels in ambient air among hospitality venues, including casinos, found that ambient nicotine concentrations were 26 times higher than in office settings.[8] Trout et al[9] showed that Atlantic City casino workers were exposed to concentrations of respirable dust and nicotine well above those of a comparable working population and that levels of cotinine during a work shift were 300% to 600% higher than those in other smoking workplaces. A 2004 study showed that casinos in Delaware had levels of airborne particulates 6 times higher than in highways and city streets during rush hour traffic. Another study found a direct correlation between ETS exposure and DNA damage to casino workers in Reno and Las Vegas.[10] Studies have also shown a decrease in environmental particulate levels following implementation of smoke-free policies in casinos.[11,12]

This cross-sectional study assessing ETS exposure in 2006 was in response to National Institute for Occupational Safety and Health (NIOSH) health hazard evaluation requests received from casino dealers at three casinos in Las Vegas, NV. The purpose of this study is to quantify casino dealers' exposure to ETS.

METHODS

Description of Study Participants and Study Sites

Each of the three casinos offered table gaming such as blackjack, poker, roulette, and craps. Smoking was permitted for patrons throughout the gaming area of the three casinos except for a smoke-free poker room that was established by one of the casinos. At the time of our study, 1188 casino dealers were working. Most of the casino dealers worked throughout the gaming areas where smoking was permitted, except for a small percentage of dealers who worked in the poker room where smoking was not permitted. Work schedules were similar at the three casinos. The casinos were open 24 hours a day, and casino dealers primarily worked one of several 8-hour shifts.

Employees were selected as possible study participants if they reported on a screening questionnaire that they worked in a smoking area, did not use any tobacco products, and did not live with someone who smoked inside the home. The questionnaires were distributed to casino dealers and pit supervisors in their break room. The dealers and supervisors were instructed to return the completed questionnaires to the locked boxes NIOSH placed staff in the break rooms. Of the 412 employees who returned the screening questionnaires, 323 met these initial inclusion criteria.

Additional inclusion criteria for participants in the biological monitoring and environmental assessment phases included the following: (1) participants had to be casino dealers in an area where smoking was allowed, and (2) they had to work either the swing shift (starts between 6 pm and 9 pm) from Thursday through Saturday or the day shift (starts between 9 am and 12 pm) on Sunday, which are the busiest times of the week and when ETS levels were believed to be the highest.

Using these additional criteria, we identified 213 casino dealers as potential participants. Because of the limited number of personal sampling pumps available, we determined that we could evaluate 124 people. We chose a convenience sample of 124, ten of whom chose not to participate in the air sampling. The 124 participants broke down into approximately 10 employees per casino per day. Informed consent was obtained from all participants.

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Environmental Monitoring

In this study, vapor-phase nicotine, respirable suspended particulates (RSP), volatile organic compounds (VOCs), polynuclear aromatic hydrocarbons (PAHs), and aldehydes (ALDs) were monitored as marker substances for ETS. Full-shift area and personal breathing zone (PBZ) air samples for nicotine, 4-vinyl pyridine (4-VP), PAHs, VOCs, RSP, solanesol (SOL), toluene (TOL), total hydrocarbons (THC), and ALDs (formaldehyde [FLD] and acetylaldehyde [ACTLD]) were collected at each casino. Except for SOL, the sampling and analytical methods for these analytes were used as specified in the NIOSH *Manual of Analytical Methods*.[13] Nicotine and 4-VP were analyzed by NIOSH Method 2551, PAHs by NIOSH Method 5515, RSP by NIOSH Method 0600, VOCs by NIOSH Method 2501, and ALDs by NIOSH Method 2016. Solanesol was analyzed using a modification of ASTM Method 2004.[14] We collected two sets of area samples daily at each casino at the same height as the PBZ of the dealers. Ambient parameters, such as temperature, relative humidity (RH), and carbon dioxide (CO_2) concentration, as well as carbon monoxide (CO) concentration (found in ETS from tobacco combustion), were measured with a QTRAK Plus®, a direct reading instrument (TSI, St. Paul, MN).

Personal breathing zone air samples were collected on 113 casino dealers, including 110 who provided urine samples each day. If these dealers opted not to wear the sampling equipment, we attempted to recruit new nonsmoking casino dealers to replace them. Of the 10 casino dealers who participated each day, all who contributed a urine sample were asked to wear samplers for nicotine and 4-VP. Eight of the 10 casino dealers were also asked to wear an additional sampler that would sample for one of the following: RSP/SOL, PAH, and VOCs. The remaining two casino dealers were asked to wear a badge that passively measured ALDs in the air. In a few instances, volunteers wearing the samplers for RSP/SOL, PAH, and VOCs requested and were given passive badges to measure their ALD exposures.

Biological Monitoring

We chose a convenience sample of 124 participants for biological monitoring to match the limited number of PBZ air sampling pumps available. Fourteen casino dealers opted not to participate in the PBZ air sampling study. On the days of the exposure assessment, we asked about smoking status and exposure to ETS outside of work within the previous 4 days.

Casino dealers provided urine samples before the start of their work shift and at the end of it. Each sample was analyzed for urinary cotinine, total NNAL, and creatinine (used to adjust for urinary dilution). For casino dealers who reported nicotine replacement therapy use, analysis of biological monitoring was restricted to NNAL.

Participants were notified in writing of their individual biological monitoring results.

Sample Analyses

Urinary cotinine was analyzed using a solid-phase competitive chemiluminescent immunoassay. The assay's limit of detection (LOD) was 5 ng/mL, and the limit of quantification (LOQ) was 10 ng/mL. Analysis of total NNAL in urine samples was performed by hydrolyzing the sample with β-glucuronidase and then assaying NNAL by HPLC API-MS/MS combined with a novel sample cleanup using solid-phase extraction based on a molecularly imprinted polymer column developed specifically for this assay.[15] The LOD for this analysis was 0.0030 ng/mL (3 pg/mL). Urinary creatinine levels were measured by a commercial, automated colometric enzymatic method (Roche Creatinine Plus) procedure, using a Hitachi 912 analyzer.

Statistical Analysis

We used SAS version 9.1 (SAS Institute, Cary, NC) for all statistical analyses. We examined the differences between preshift and postshift NNAL, NNAL adjusted for creatinine, cotinine, and cotinine adjusted for creatinine using paired *t* tests or paired sign tests, depending on the distributions of the differences. Spearman's correlation coefficients were calculated among and between environmental and biological sampling results.

For environmental and NNAL samples with mass below the LOD, an estimate of the mass was obtained by dividing the LOD (obtained from the laboratory) by $\sqrt{2}$. Hornung and Reed have described this method of handling censored data.[16] For the environmental samples, this estimate was divided by the maximum sample volume to obtain the estimated sample concentration for the statistical analyses. Because the laboratory did not report COT measures below their LOQ, the LOQ divided by $\sqrt{2}$ was used in the statistical analyses.

RESULTS

Environmental Monitoring

Tables 1 and 2 present the geometric means (GMs), sample sizes, and ranges for the PBZ and area air samples by casino and for all casinos combined. The GMs of the area and PBZ sample concentrations were comparable within casinos for a particular analyte.

Volatile organic compounds: A VOC screen using thermal desorption tubes indicated the presence of approximately 98 chemicals per sample in the air; approximately 20 of these chemicals are commonly found in indoor environments. The samples were analyzed using gas chromatography with mass spectrometry detection. Figure 1 shows a typical profile of the VOCs in the casino air.

Using this profile, we chose benzene, TOL, *p*-dichlorobenzene, limonene, and THC for quantitative analysis on the basis of the relative amount present in the environment, their relative toxicities compared to other VOCs, and ability to separate them from the mixture. A trace concentration of *p*-dichlorobenzene was found in one PBZ sample at casino 1. Quantifiable concentrations of benzene were found in two PBZ samples, both at casino 3. The full-shift concentrations for both samples were 12 and 13 parts per billion (ppb). Quantifiable concentrations of limonene were found in two PBZ samples at casino 1 (0.6 and 1.3 mg/m^3) and in four PBZ samples at casino 3 (0.1 , 0.1 , 0.9 , and 1.0 mg/m^3).

Carbon monoxide: CO measures were unremarkable; almost all were below the instrument LOD of 0.1 ppm and well below occupational exposure limits (OELs).

Temperature, RH, and carbon dioxide: Temperature and RH values were within the American Society of Heating, Refrigerating and Air Conditioning Engineers, Inc. (ASHRAE), ranges for acceptable human comfort, except in one instance in casino 2, where the average temperature was below the ASHRAE indoor design guidance of 68.5 F for winter temperatures.[17] At each casino, maximum CO_2 concentrations were more than 700 ppm above the outdoor CO_2 concentrations, suggesting that not enough outdoor air may have been provided for acceptable odor control (body odor) and indicating the need for further evaluation of the ventilation systems. The outdoor CO_2 concentrations ranged from 250 to 300 ppm. Table 3 summarizes these results.

Biological Monitoring

Of 213 eligible casino dealers, we chose a convenience sample of 124 to participate in the biological monitoring phase of the evaluation. All stated that they were nonsmokers, and 107 (86%) reported exposure to ETS at work in the previous 4 days. Of the 483 urine samples (preshift: 123 cotinine and 122 NNAL; postshift: 119 cotinine and 119 NNAL), both NNAL and cotinine measures from three participants were excluded. Of these three, two were excluded because of reported exposure to tobacco smoke in settings outside of work 4 days before the study and one was excluded because of a cotinine value inconsistent with being a nonsmoker. One additional participant had only his or her cotinine measures excluded because of the reported use of nicotine replacement therapy. Other exclusions

TABLE 1. Geometric Means (Sample Sizes) and Ranges of Environmental Tobacco Smoke Components ($\mu g/m^3$) in Personal Breathing Zone Air Samples

	Casino 1		Casino 2		Casino 3		Combined	
	GM (*n*)	Range	GM (*n*)	Range	GM (*n*)	Range	GM (*n*)	Range
Acetylaldehyde	9.30 (11)	6.2–15	9.16 (12)	4.8–16	15.1 (6)	14–17	10.2 (29)	4.8–17
4-vinyl pyridine	0.867 (34)	ND–2.8	0.852 (35)	ND–1.7	1.33 (38)	0.57–2.5	1.00 (107)	ND–2.8
Formaldehyde	7.46 (11)	ND–22	7.75 (12)	ND–29	16.8 (6)	14–23	8.96 (29)	ND–29
Nicotine	4.60 (34)	1.6–12	4.03 (35)	0.58–10	7.80 (38)	3.9–17	5.32 (107)	0.58–17
Polynuclear aromatic hydrocarbon*	0.652 (12)	0.21–1.2	0.753 (11)	0.25–1.2	1.02 (11)	0.72–1.4	0.790 (34)	0.21–1.4
Respirable suspended particulates	43.0 (11)	22–78	31.6 (10)	ND–56	52.5 (12)	ND–140	42.1 (33)	ND–140
Solanesol	0.208 (11)	ND–0.82	0.145 (10)	ND–0.55	0.352 (12)	ND–1.1	0.226 (33)	ND–1.1
Total hydrocarbons	208 (6)	ND–390	425 (7)	130–1200	735 (9)	230–3100	438 (22)	ND–3100
Toluene	23.1 (6)	ND–290	6.29 (7)	ND–13	18.5 (9)	ND–180	13.9 (22)	ND–290

*The concentrations reflect the levels of naphthalene, the only polynuclear aromatic hydrocarbon detected in the samples.
GM, geometric mean.

TABLE 2. Geometric Means (Sample Sizes) and Ranges of Environmental Tobacco Smoke Components ($\mu g/m^3$) in Area Air Samples

	Casino 1		Casino 2		Casino 3		Combined	
	GM (*n*)	Range	GM (*n*)	Range	GM (*n*)	Range	GM (*n*)	Range
Acetylaldehyde	8.55 (5)	ND–19	9.30 (8)	6.9–14	15.4 (6)	7.6–20	11.0 (19)	ND–20
4-vinylpyridine	0.831 (8)	ND–1.8	1.10 (8)	0.72–1.6	1.76 (8)	0.97–3.4	1.20 (24)	ND–3.4
Formaldehyde	7.19 (5)	ND–36	6.70 (8)	ND–14	15.6 (6)	7.6–34	8.91 (19)	ND–36
Nicotine	5.31 (8)	ND–14	5.31 (8)	2.6–7.2	10.7 (8)	4.5–23	6.69 (24)	1.0–23
Polynuclear aromatic hydrocarbon*	0.607 (7)	ND–1.2	0.617 (8)	0.19–1.3	1.01 (8)	0.55–1.6	0.729 (23)	0.19–1.6
Respirable suspended particulates	38.1 (6)	23–77	32.6 (8)	25–44	56.1 (8)	40–86	41.4 (22)	23–86
Solanesol	0.300 (6)	ND–0.89	0.141 (8)	ND–0.36	0.359 (8)	0.10–0.99	0.242 (22)	ND–0.99
Total hydrocarbons	320 (8)	142–470	251 (8)	130–420	553 (8)	200–960	354 (24)	130–960
Toluene	18.7 (8)	ND–500	6.61 (8)	ND–12	9.56 (8)	ND–19	10.6 (24)	ND–500

*The concentrations reflect the levels of naphthalene, the only polynuclear aromatic hydrocarbon detected in the samples.
GM, geometric mean.

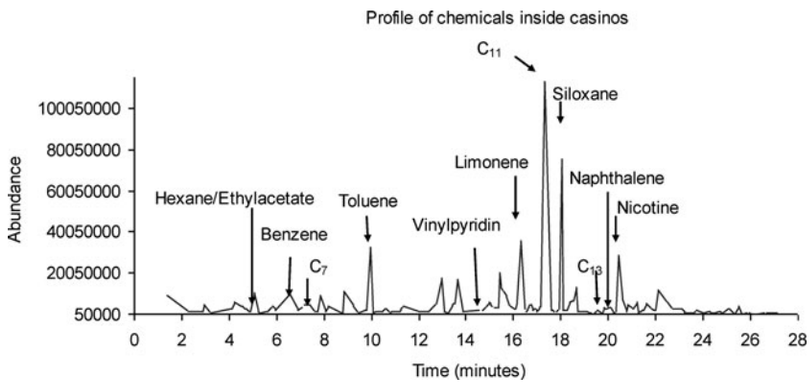

FIGURE 1. Profile of chemicals inside casinos.

included having only a preshift or a postshift sample but not both, and having insufficient urinary volume for analysis. For comparative analysis, these exclusions resulted in 114 preshift and postshift (paired) samples for unadjusted cotinine and NNAL, 112 preshift and postshift (paired) samples for creatinine-adjusted cotinine, and 113 preshift and postshift (paired) samples for creatinine-adjusted. Fourteen cotinine measures were below the LOQ of 10 ng/mL, and 93 NNAL measures were below the LOD of 0.0030 ng/mL. Of the 93 NNAL measures below the LOD, 63 (68%) were in preshift samples, and 30 (32%) were in postshift samples.

Table 4 presents the GM levels of preshift and postshift cotinine and NNAL for each casino and for all casinos combined unadjusted for creatinine. Table 5 presents the GM levels of preshift and postshift cotinine and NNAL adjusted for creatinine.

Because the pattern of biological monitoring results was similar across casinos, our analysis focused on the combined results. Urinary cotinine and NNAL levels unadjusted for creatinine for all three casinos combined increased significantly during an 8-hour shift ($P < 0.01$). The levels of urinary NNAL adjusted for creatinine for all three casinos combined increased significantly over an 8-hour shift

TABLE 3. Ranges of Indoor Environmental Quality Parameters

Casino	Temperature (°F)	RH (%)	CO_2 (ppm)
Casino 1	69.4–74.3	12.4–18.7	450–1311
Casino 2	60.5–74.7	8.3–18.9	286–1220
Casino 3	69.3–77.0	9.6–23.1	583–1244

TABLE 4. Geometric Means (and Sample Size) of Creatinine-Unadjusted ETS Components in Urine

	Cotinine (ng/mL)		NNAL (ng/mL)	
Casino	Preshift GM (*n*)	Postshift GM (*n*)	Preshift GM (*n*)	Postshift GM (*n*)
Casino 1	27.55 (34)	34.18 (34)	0.0053 (35)	0.0089 (35)
Casino 2	19.30 (35)	25.92 (35)	0.0042 (35)	0.0079 (35)
Casino 3	21.15 (45)	31.41 (45)	0.0029 (44)	0.0050 (44)
Combined	22.25 (114)	30.37 (114)	0.0039 (114)	0.0069 (114)

TABLE 5. Geometric Means (and Sample Sizes) of Creatinine-Adjusted ETS Components in Urine (in Milligrams of Creatinine)

	Cotinine (ng/mL)		NNAL (ng/mL)	
Casino	Preshift GM (*n*)	Postshift GM (*n*)	Preshift GM (*n*)	Postshift GM (*n*)
Casino 1	0.1706 (34)	0.1640 (34)	0.0270 (35)	0.0353 (35)
Casino 2	0.1302 (34)	0.1192 (34)	0.0236 (35)	0.0297 (35)
Casino 3	0.1947 (44)	0.1778 (44)	0.0226 (43)	0.0242 (43)
Combined	0.1655 (112)	0.1536 (112)	0.0242 (113)	0.0290 (113)

($P = 0.03$). Nevertheless, urinary cotinine levels adjusted for creatinine for all three casinos combined did not increase significantly over an 8-hour shift. It is possible that this reflected the relatively limited sensitivity of our cotinine assay. Nevertheless, the preshift unadjusted urinary cotinine was positively correlated with preshift urinary NNAL ($r = 0.53$; $P < 0.01$), and postshift urinary cotinine was positively correlated with postshift urinary NNAL levels ($r = 0.53$; $P < 0.01$). The net urinary unadjusted NNAL correlated with the net urinary unadjusted cotinine ($r = 0.44$; $P < 0.01$). The net levels of NNAL and cotinine corresponded to their preshift level subtracted from their postshift level.

Relationship Between Biological and Environmental PBZ Measures

No statistically significant positive correlations were found between the concentrations of unadjusted and adjusted net urinary NNAL and environmental PBZ measures (nicotine, 4-VP, RSP, SOL, PAH [naphthalene], TOL, THC, FLD, ACTLD) during the 4-day evaluation. No statistically significant positive correlations were found between unadjusted net urinary cotinine and environmental PBZ measures (nicotine, 4-VP, RSP, SOL, PAH [naphthalene], TOL, THC, FLD, ACTLD) over the 4-day evaluation. No statistically significant positive correlations were found between adjusted net urinary cotinine and most of the environmental PBZ air measures (nicotine, 4-VP, TOL, THC, FLD, ACTLD) except for the following environmental PBZ air measures: PAH ($r = 0.40$; $P = 0.02$), RSP ($r = 0.58$; $P < 0.01$), and SOL ($r = 0.42$; $P = 0.03$), where we found statistically significant positive correlations.

DISCUSSION

We found that casino dealers were exposed to workplace ETS, and that they absorbed detectable ETS-specific components, including a tobacco-specific carcinogen, NNAL, into their bodies. This study supports the current evidence that exposure to ETS at work places nonsmokers at risk for adverse health effects and that eliminating smoking at work is the only sure way to protect nonsmokers. Other methods to reduce exposure, such as separating smokers from nonsmokers, cleaning the air, and ventilating buildings, cannot eliminate occupational exposures to ETS.[6]

Environmental Monitoring

The findings from this evaluation are consistent with those found in other ETS exposure assessment studies. The range of PBZ nicotine air sample concentrations (0.58 to 17 $\mu g/m^3$) is comparable to past studies of workers in smoking environments,[18,19] including one study of casino dealers.[9] Likewise, the range of area air sample concentrations of nicotine was comparable to the data reported by Trout et al,[9] who reported PBZ nicotine concentrations between 6 and 15 $\mu g/m^3$ for casino dealers. The area air concentrations in this study were similar to the PBZ sample concentrations, indicating that they were representative of the atmosphere in the casino pits at the height of the breathing zone of dealers.

Historically, RSP has been used as a surrogate for the particulate phase of ETS. RSP is easy to quantify in the environment. A drawback is that it is not specific to ETS, thus possibly leading to an overestimation of ETS levels. The maximum area RSP level in this evaluation was 86 $\mu g/m^3$, which is similar to the 90 $\mu g/m^3$ reported by Trout et al.[9] The highest PBZ RSP concentration in this evaluation was 140 $\mu g/m^3$; Trout et al[9] did not measure PBZ RSP levels. In addition to RSP, SOL, a trisesquiterpenoid alcohol found in tobacco leaves, has been used as a surrogate for ETS components in the particulate phase.[20,21] Nevertheless, recent research suggests that SOL is degraded in indoor environments with ozone concentrations of 40 ppb.[22] Ozone levels were not measured as part of this study.

A surprising finding from this evaluation was that common PAHs, such as benzo-a-pyrene, anthracene, or pyrene, were not found in the PBZ and area air samples. These compounds, some of which are known lung carcinogens, have been found in previous studies of ETS in homes and restaurants.[23,24] Of the 16 PAHs evaluated, only naphthalene was found in quantifiable concentrations in both PBZ and area air samples. The EPA has classified naphthalene as a possible human carcinogen.[25] Overall, the concentrations of TOL, FLD, and ACTLD were lower than those reported in the scientific literature, although past ETS studies of these compounds were conducted in smaller spaces such as restaurants, automobiles, and residences.

Although the airborne components were below applicable regulatory and recommended OELs for these components individually, studies have shown that simultaneous exposure to multiple components (mixed exposures), even at low levels, can cause respiratory health effects, such as acute bronchitis and asthma.[26–28] These studies focused on health effects from air pollution from non-ETS fine particulates, but the mixed-exposure effects seen in these studies are still relevant to ETS exposure because many air pollutants (RSP, PAHs, VOCs, and ALDs) are also found in ETS. The regulatory and recommended OELs for these components are based on the individual component; the synergistic or additive properties are not considered.[29] OELs for some chemicals found in ETS (such as nicotine) were developed to protect workers whose exposure (route and quantity) to these chemicals are different from the ETS exposure route.

Most of the temperature and RH values fell within the acceptable ranges of thermal comfort recommended by ASHRAE. Each ventilation system reportedly was designed to comply with the relevant outdoor air recommendations in effect at the time of their installation. Nevertheless, our limited area air-monitoring data showed indoor concentrations of CO_2 to be more than 700 ppm relative to outdoors, suggesting the need to further evaluate the effectiveness of

the ventilation systems in maintaining acceptable odor control. (This guideline refers to the control of body odor from sedentary people and not odor from tobacco smoke.) Two of the casinos (casinos 2 and 3) had CO_2 sensors in the ductwork, but we do not know whether the relationships between duct concentrations and breathing zone concentrations have been evaluated or whether these sensors had been calibrated or maintained properly. The concentrations of ETS compounds could be expected to be higher when less outdoor air is brought into the casinos, and lower with more outdoor air to dilute these contaminants. ASHRAE has a position document stating that, although implementing engineering controls, such as current and advanced dilution ventilation, can reduce odors and some forms of irritation from ETS, they should not be relied upon to control health risks from ETS exposure in public spaces. ASHRAE concludes that the only means of eliminating the health risks associated with indoor ETS exposure is to ban all smoking.[30]

Biological Monitoring

The biological monitoring portion of the evaluation was designed to evaluate body burden of ETS metabolites; that is, whether the ETS components were absorbed in the body, metabolized, and detectable in the urine. Because cotinine is the major metabolite of nicotine, it has been extensively used in large epidemiologic studies to measure exposure to ETS. The tobacco-specific nitrosamine NNK, along with its urinary metabolite NNAL, are known pulmonary carcinogens,[31] and the presence of NNAL in urine links ETS exposure with an increased risk of lung cancer, a long-term outcome of ETS exposure.[6] NNAL is a tobacco-specific compound, so its detection specifically indicates ETS exposure.[31] A recent epidemiologic study showed that urinary levels of total NNAL were significantly associated with the risk of lung cancer.[32] The differences biologically in NNK and nicotine may help to explain the low correlation between their metabolites, NNAL and cotinine, seen in this evaluation.

We have documented an increase in urinary levels of NNAL over a work shift in casino dealers, which provides evidence that the increase is due to workplace ETS exposure. Our results are consistent with other studies that have shown an increase in levels of NNAL in bar and restaurant workers exposed to ETS over a work shift and casino patrons exposed over a 4-hour period.[33–35]

We have reported cotinine and NNAL levels with and without creatinine adjustment. In recently published ETS studies, researchers have chosen different approaches regarding creatinine adjustment.[9,33–37] Creatinine adjustment can account for potential dilutional effects when measuring biomarkers in spot urine samples. Nevertheless, according to Barr et al[38] and Boeniger et al,[39] creatinine adjustment of biomarkers in populations that vary by age, sex, and race may yield inaccurate results. Because these factors may have affected our results, we presented both methods. We found that the results, with or without adjustment for creatinine, were similar for NNAL but different for cotinine. Cotinine has a biological half-life of approximately 16 to 20 hours. In one study that quantified urinary levels of NNK and NNAL in cigarette smokers after smoking cessation, the researchers estimated that NNAL had an elimination half-life of 45.2 days,[40] which was later revised to approximately 26 days.[41] Goniewicz et al[42] suggested that it might be as low as 10 days. Nonetheless, it certainly would appear to be more than 1 week and thus much longer than the half-life of cotinine. No research is available describing the elimination of NNK and its metabolites as a result of exposure to ETS.

The GM values that we reported for unadjusted urinary cotinine are similar to those measured in other indoor environments, such as bars, casinos, and restaurants.[9,43–45] Trout et al,[9] for example, found a GM preshift level of 23.0 ng/mL and postshift level of 33.3 ng/mL with a significant increase in urinary levels of cotinine ($P < 0.01$) over the shift.

Given our findings that casino dealers are exposed to a potent carcinogen, legislative efforts should continue that extend smoke-free policies to state-regulated and tribal gaming establishments.

CONCLUSIONS

The increase in NNAL levels in the urine of most casino dealers at the end of their work shift demonstrates that casino dealers are exposed to a known tobacco-specific carcinogen at the casinos. The best means of eliminating workplace exposure to ETS is to ban smoking in the casinos. We recommend eliminating tobacco from the casinos and implementing smoking cessation programs.

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