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Fine particle air pollution and secondhand smoke exposures and risks inside 66 US casinos

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ABSTRACT

Smoking bans often exempt casinos, exposing occupants to fine particles ($PM_{2.5}$) from secondhand smoke. We quantified the relative contributions to $PM_{2.5}$ from both secondhand smoke and infiltrating outdoor sources in US casinos. We measured real-time $PM_{2.5}$, particulate polycyclic aromatic hydrocarbons (PPAH), and carbon dioxide (CO_2) (as an index of ventilation rate) inside and outside 8 casinos in Reno, Nevada. We combined these data with data from previous studies, yielding a total of 66 US casinos with smoking in California, Delaware, Nevada, New Jersey, and Pennsylvania, developing $PM_{2.5}$ frequency distributions, with 3 nonsmoking casinos for comparison. Geometric means for $PM_{2.5}$ were 53.8 $\mu g/m^3$ (range 18.5–205 $\mu g/m^3$) inside smoking casinos, 4.3 $\mu g/m^3$ (range 0.26–29.7 $\mu g/m^3$) outside those casinos, and 3.1 $\mu g/m^3$ (range 0.6–9 $\mu g/m^3$) inside 3 nonsmoking casinos. In a subset of 21 Reno and Las Vegas smoking casinos, $PM_{2.5}$ in gaming areas averaged 45.2 $\mu g/m^3$ (95% CI, 37.7–52.7 $\mu g/m^3$); adjacent nonsmoking casino restaurants averaged 27.2 $\mu g/m^3$ (95% CI, 17.5–36.9 $\mu g/m^3$), while $PM_{2.5}$ outside the casinos averaged 3.9 $\mu g/m^3$ (95% CI, 2.5–5.3 $\mu g/m^3$). For a subset of 10 Nevada and Pennsylvania smoking casinos, incremental (indoor–outdoor) $PM_{2.5}$ was correlated with incremental PPAH ($R^2=0.79$), with ventilation rate-adjusted smoker density ($R^2=0.73$), and with smoker density ($R^2=0.60$), but not with ventilation rates ($R^2=0.15$). PPAH levels in 8 smoking casinos in 3 states averaged 4 times outdoors. The nonsmoking casinos' $PM_{2.5}$ ($n=3$) did not differ from outdoor levels, nor did their PPAH ($n=2$). Incremental $PM_{2.5}$ from secondhand smoke in approximately half the smoking casinos exceeded a level known to produce cardiovascular morbidity in nonsmokers after less than 2 h of exposure, posing acute health risks to patrons and workers. Casino ventilation and air cleaning practices failed to control secondhand smoke $PM_{2.5}$. Drifting $PM_{2.5}$ from secondhand smoke contaminated unseparated nonsmoking areas. Smoke-free casinos reduced $PM_{2.5}$ to the same low levels found outdoors.

1. Introduction

In 2009, there were 25 states with 487 commercial casinos, which were visited by 28% of the US adult population (61.7 million people), an average of 7 times per person per year, and which employed 328,277 workers (AGA, 2008–2010). In addition, 29 states had 456 tribal casinos, employing 204,000 persons (AGA, 2010; NIGA, 2010). An estimated 80% of casino patrons are nonsmokers (MMWR, 2009; Pritsos et al., 2008). Overall, 88% of US commercial casinos and 100% of tribal casinos are exempt from state clean indoor air laws, and very few are smoke-free, since the casino industry opposes smoking bans (AGA, 2009; ANR, 2009). By contrast, almost 74% of US restaurants and 63% of US bars are covered by smoke-free laws (ANR, 2010). For example, Nevada, which has 54% of all US commercial casinos (World Casino, 2009), prohibits smoking in casino restaurants, but permits smoking on the gaming floors.

Fine particulate matter ($PM_{2.5}$) is a harmful combustion source air pollutant with acute and chronic cardiovascular impacts (NAS, 2010; Pope and Dockery, 2006). The WHO 24-h $PM_{2.5}$ air quality

Abbreviations: ASC, active smoking count; ASP, active smoking percentage; ASHRAE, American Society of Heating, Refrigerating and Air-Conditioning Engineers; GM, geometric mean; GSD, geometric standard deviation; NHANES, National Health and Nutrition Examination Survey; NIOSH, National Institute for Occupational Safety and Health; USEPA, US Environmental Protection Agency; WHO, World Health Organization

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standard is 25 μg/m^3, and its annual standard is 10 μg/m^3, while the less stringent USEPA standard is 35 μg/m^3 averaged over 24 h, and the annual average is 15 μg/m^3 (USEPA, 2010). $PM_{2.5}$ causes adverse health effects on the respiratory and cardiovascular systems; the entire population is affected, but susceptibility to $PM_{2.5}$ pollution varies with age and health status (WHO, 2005). $PM_{2.5}$ risk increases with exposure, and there is little evidence to suggest a threshold below which no adverse health effects are anticipated. Adverse health effects may occur at $PM_{2.5}$ concentrations as low as 3–5 μg/m^3 (WHO, 2005). Secondhand smoke contains fine particulate matter ($PM_{2.5}$), its largest component by mass, as well as numerous gas and particulate-phase carcinogens and toxins (Hoffmann and Hoffmann, 1998; Repace, 2007). Secondhand smoke is a strong and often predominant source of indoor $PM_{2.5}$, adding to the outdoor $PM_{2.5}$ that infiltrates indoors (Surgeon General, 2006; Repace, 2007). By 2010, studies of $PM_{2.5}$ air pollution of casinos had been conducted in California (CA), Delaware (DE), Nevada (NV), New Jersey (NJ), and Pennsylvania (PA) (Jiang et al., 2010; York and Lee, 2010; Achutan et al., 2009, in press; Repace, 2009; Repace, 2004; Trout and Decker, 1996; Trout et al., 1998). Table 1 lists these previous studies of 59 casinos with smoking and 2 without smoking that comprised our initial database, reporting their locations and principal findings. We reviewed only studies reported in the peer-reviewed literature. Of the 61 casino studies listed in Table 1, 57 used gravimetrically calibrated, real-time particle monitors for 0.5–1-h sampling durations; while 4 casinos studied by NIOSH – 3 in Las Vegas, NV (Achutan et al., 2009, in press), and one in Atlantic City, NJ (Trout and Decker, 1996; Trout et al., 1998) – used integrated 8-h work shift average pump-and-filter gravimetric measurements. These studies of casinos in 5 states indicate that, relative to outdoors, high levels of indoor air pollution from $PM_{2.5}$ occur in this industry, but no previous study has brought these data together into a systematic framework.

In this work, we develop for the first time a comprehensive picture of air quality inside and outside a total of 66 US casinos with smoking, with 3 smoke-free casinos for comparison. We assess the contribution of secondhand smoke to indoor $PM_{2.5}$ and its potential for adverse health effects on casino patrons and workers. We hypothesize that secondhand smoke is the predominant source of incremental (indoor minus outdoor) $PM_{2.5}$ in casinos with smoking. Specifically, we (1) measure $PM_{2.5}$ and CO_2 inside and outside 8 casinos in Reno, NV; (2) assess the fraction of incremental $PM_{2.5}$ consisting of particulate polycyclic aromatic hydrocarbons (PPAH) in 5 of the 8 Reno casinos; (3) evaluate the relative effects of ventilation practices and smoker density on incremental $PM_{2.5}$ levels using data available from 10 NV and PA casinos; (4) combine data from 5 of our 8 Reno casinos with a study of 16 Las Vegas casinos (York and Lee, 2010) to compare casino smoking and nonsmoking areas with outdoors for 21 casinos in Nevada; (5) describe $PM_{2.5}$ and interpret dosimetry data from dealers in a NIOSH study of 3 Las Vegas casinos (Achutan et al., 2009, in press); (6) combine all studies to construct indoor and outdoor frequency distributions of $PM_{2.5}$ for 66 US smoking casinos in 5 states; (7) compare the 66 casino $PM_{2.5}$ levels with the levels measured in 3 smoke-free casinos in CA, DE, and NV; (8) estimate acute cardiovascular risks from secondhand smoke $PM_{2.5}$ for casino patrons and workers using a published clinical exposure–response relationship and discuss the chronic risk implications for casino workers.

Due to the rarity of smoke-free casinos, the high level of casino patronage, the large casino workforce, and the evidence quantitatively linking secondhand smoke exposure to heart disease, cancer, and respiratory disease, measuring concentrations of $PM_{2.5}$ and PPAH inside and outside casinos is critically important for assessing the risks to human health.

2. Methods

2.1. The Reno field study

There are 15 smoking casinos – 4 large (> 1400 slots), 5 medium (500–1400 slots), 6 small (< 500 slots) – in Reno, Nevada's second largest city, and one nonsmoking casino (< 500 slots) in neighboring Fernley. From these, we randomly sampled approximately half of the smoking casinos in each size category, yielding 2 large, 2 medium, and 3 small ones, as well as the only nonsmoking casino. We measured $PM_{2.5}$ for these 8 casinos and PPAH, and CO_2 as well for 5 of the remaining casinos on two weekends in March 2009, using concealed real-time air quality monitors to avoid disturbing normal casino activities.

Because indoor $PM_{2.5}$ and PPAH also may have indoor and outdoor sources other than secondhand smoke, such as cooking and diesel exhaust (Ott and Siegmann, 2006; Repace, 2004; Repace et al., 2006b), we compared indoor and outdoor measurements, and we searched for cooking sources in the casinos. In general, we had no information about the Reno casinos' mechanical ventilation systems or air cleaning devices. We measured smoker density, CO_2, PPAH, and $PM_{2.5}$ concentrations, plus indoor and outdoor temperature and relative humidity before and after each visit. During monitoring, we roamed through all gaming areas of the casinos, spending about 1 h in each casino, and we spent at least 1 h over dinner at a single location in each nonsmoking casino restaurant. Each investigator logged the locations visited and the times spent there. We used up to 7 battery-powered TSI SidePak® AM510 real-time aerosol monitors [TSI, Inc., Shoreview, MN] to measure and log 10-s average $PM_{2.5}$ concentrations on all visits. Prior to this field study, we gravimetrically calibrated our SidePak monitors in controlled experiments in a 3 m^3 automobile cabin using secondhand smoke from Marlboro cigarettes with a volunteer smoker. The individual SidePak custom calibration factors ranged from 0.245 to 0.305 and averaged 0.285; the average monitor precision was 3%; the calibration methods are described in Jiang et al. (2010).

We employed the mass-balance model to guide our sampling methodology (Ott, 1999, 2007; Repace, 2007). This model assumes that in a well-mixed volume, time-averaged $PM_{2.5}$ and PPAH concentrations from secondhand smoke are directly proportional to smoker density D_s (average active smoking count, $\overline{ASC}$, divided by the space volume, in units of 100 m^3) and inversely proportional to V_o,

Table 1
Casino measurement studies in the published literature: indoor $PM_{2.5}$ and PPAH results for 59 smoking casinos and 2 nonsmoking casinos.

Study	Ave. time measurement technique	Findings, mean $PM_{2.5}$ [PPAH] (ranges or SD)	Citation
3 NV casinos[a] (smoking)	8-h gravimetric	41 μg/m^3 (range 32–58 μg/m^3)	Achutan et al. (2009, in press)
35 CA casinos (smoking)	0.5–1 h real-time	63 μg/m^3 (range 21–183 μg/m^3)	Jiang et al. (2010)
1 CA casino (nonsmoking)	1 h real-time	5.4 μg/m^3 (SD 2.3 μg/m^3)	Jiang et al. (2010)
3 PA casinos (smoking)	1 h real-time	106 μg/m^3 (range 84–133 μg/m^3)	Repace (2009)
1 DE casino[b] (smoking)	0.5 h real-time	205 μg/m^3 (range 145–384 μg/m^3) [PPAH: 161 ng/m^3 (range 72–330 ng/m^3)]	Repace (2004)
1 DE casino[b] (nonsmoking)	0.5 h real-time	9 μg/m^3 (range 5–17 μg/m^3) [PPAH: 3.7 ng/m^3 (range 1–7 ng/m^3)]	Repace (2004)
1 NJ casino[a] (smoking)	8 h gravimetric	80 μg/m^3 (SD 12 μg/m^3)	Trout and Decker (1996)
16 NV casinos (smoking)	0.5 h real-time	48 μg/m^3 (range 20–73 μg/m^3)	York and Lee (2010)

[a] NIOSH study.
[b] The Delaware Park Casino, before and after a statewide smoking ban.

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Table 2
Reno casino parameters and $PM_{2.5}$ real-time air quality measurements[a].

1. Casino (maximum occupancy for space sampled)	2. Area (ft^2); ceiling height (ft)	3. Casino volume (m^3)	4. Ave. occupants (occupancy, %)	5. Ave. persons/ 1000 ft^2	6. ASC[b] = ave. # burning cigarettes	7. CO_2 indoors–outdoors (ppm)	8. Ave. $PM_{2.5}$ in gaming areas ($\mu g/m^3$)	9. Ave. $PM_{2.5}$ nonsmoking casino restaurant ($\mu g/m^3$)	10. Ave. $PM_{2.5}$ outdoors ($\mu g/m^3$)	11. V_o[c] (ventilation rate per person)	12. Est.[d] ASP (%)	13. D_s, active smoker density (ASC/ 100 m^3)	14. D_s/V_o[e] ventilation-adjusted smoker density
Atlantis (1478 slots)	87,909; 15.1	37,588	588 (40%)	6.7	55	456 − 344 = 112	32.6 (SD 12.4)	21.1 (SD 4.0)	1.8 (SD 1.1)	45	9.4	0.146	0.0032
Silver Legacy (1700 slots)	62,700; 31.5	55,926	610 (36%)	9.7	65	503 − 297 = 206	29.6 (SD 13.0)	13.9 (SD 1.7)	1.60 (SD 0.95)	24	10.7	0.116	0.0048
Grand Sierra (975 slots)	60,213; 21.7	36,999	339 (35%)	5.6	24	455 − 250 = 205	19.4 (SD 6.5)	22.5 (SD 3.9)	0.26 (SD 0.4)	24	7.1	0.0649	0.0027
Cal Neva[f] (390 slots)	14,210; 21.9	8812	157 (40%)	11.0	13.5	783 − 455 = 328	64.1 (SD 25.3)	100% smoking	1.3 (SD 1.0)	15	9.7	0.153	0.010
Harrah's[f] (597 slots)	27,407; 11.8	9158	303 (50%)	11.1	21.3	505 − 355 = 150	21.9 (SD 17.1)	5.4 (SD 3.7)	2.1 (SD 2.1)	33	7.6	0.232	0.007
Siena (423 slots)	19,752; 24.8	13,871	126 (30%)	6.4	16	–	60.1 (SD 13.0)	1.7 (SD 1.9)	1.1 (SD 3.7)	[g]	12.4	0.115	–
Tamarack (438 slots)	18,347; 24.4	12,676	133 (30%)	7.2	15.3	543 − 356 = 197	32.8 (SD 4.6)	21.5 (SD 6.5)	0.8 (SD 0.8)	25	10.3	0.121	0.0048
Fernley Nugget[h] (169 slots)	10,367; 12.8	3758	34 (20%)	3.3	0	553 − 368 = 185	0.6 (SD 1.4)	0.68 (SD 0.91)	1.4 (SD 0.9)	27	0.0	0.0	0.000
Mean—all smoking[j]	41,505; 21.6[i]	25,004	355 (38.5%)	8.5	–	–	37.2 (SD 17.8)	14.4 (SD 9.0)	1.28 (SD 0.63)	27.7	9.3 (2.1)	0.15 (SD 0.06)	0.005 (SD 0.003)

[a] Averaging time for indoor measurements: 1 h for casinos and restaurants; 20 min for outdoors (10 min before and after indoors).
[b] The active smoking count (ASC) was based on the combined count-weighted data from 2 observers.
[c] CO_2-estimated.
[d] ASP = active smoker percentage, (100 × no. of active smokers)/(no. of persons present).
[e] Ratio of active smoker density to ventilation rate per occupant.
[f] Only a portion of this casino was sampled.
[g] Monitor malfunction.
[h] Nonsmoking casino.
[i] Median area: 27,407 ft^2, median ceiling height: 21.9 ft.
[j] The total number of occupants of the 7 smoking casinos is 2256 persons.

the outdoor air ventilation rate per person (in units of liters per second per person) delivered by the ventilation system (Repace, 2007). Thus, by this model, average secondhand smoke pollutant concentrations should be proportional to and best explained by ventilation-adjusted smoker density, D_s/V_o.

We measured 1-h average PPAH, logged in 10-s intervals, with a factory-calibrated (Repace, 2004) real-time EcoChem PAS 2000CE® monitor (EcoChem Analytics, League City, TX) and CO_2 with an altitude-adjusted and 0- and 1000-ppm span-gas-calibrated TelAire® real-time CO_2 monitor (General Electric Sensing, Inc., CT) to assess ventilation rates. For the first 3 casinos listed in Table 2, a thermometer and hygrometer were not available; however, hourly outdoor temperature and relative humidity data were obtained later from Reno Airport data for the study dates (Weather Underground, 2009). For the remaining 5 casinos, we measured indoor and outdoor relative humidity and temperature using a Hygro-Thermometer (Sunleaves Inc., Bloomington, IN) or a HOBO Model U12-006 (Onset Computer Corporation, Bourne, MA). We estimated the casino floor areas using calibrated foot pacing, and we measured ceiling heights using a Zircon® DM S50L Sonic Measure (Campbell, CA). For the active smoking counts, one investigator counted persons and burning cigarettes in 10-min intervals, and another investigator counted them in 30-min intervals, each recording their entry and departure times.

We estimated the casinos' outdoor supply air ventilation rate per occupant V_o (L/s-person) from the casinos' indoor and outdoor CO_2 differences using an equation in ASHRAE Standard 62-2001 (ASHRAE, 2001)

$$V_o = G/(C_s - C_o) \quad (1)$$

where $G = 5000$ ppm-L/s-person is the generation rate of CO_2 for a sedentary person ($\sim$0.005 L of CO_2/s), C_s is the equilibrium CO_2 level in parts per million (ppm) in a building, and C_o is the outdoor air CO_2 concentration. For example, if $C_o = 400$ ppm is the default outdoor air CO_2 concentration and 733 ppm is the equilibrium CO_2 concentration inside the casino, then $V_o = \{5000/(733-400)\} = 15$ L/s-person, which was the ASHRAE-prescribed ventilation rate for a "well ventilated" smoking casino in 2001. The standard also noted that supplemental smoke-removal equipment was optional.

2.2. Data from previous casino studies

We incorporated data from all other published studies, and where data for outdoors was not measured, as in the NIOSH Las Vegas Health Hazard Evaluation study by Achutan et al. (2009, in press), we incorporated hourly data from the Las Vegas outdoor air quality network for the days and times on which the studies were conducted. There was only one casino – the NIOSH New Jersey study by Trout and Decker (1996) and Trout et al. (1998) – for which outdoor data was not measured and could not otherwise be obtained. For 2 studies, Achutan et al. (2009, in press) and York and Lee (2010), we obtained raw data from the investigators for individual casinos, and elsewhere we used outdoor data from nearby ambient monitoring networks.

2.3. Estimation of acute health risks

The evidence on the mechanisms by which smoking causes disease indicates that there is no risk-free level of exposure to tobacco smoke. Low levels of exposure, including exposures to secondhand tobacco smoke, lead to a rapid and sharp increase in endothelial dysfunction and inflammation, which are implicated in acute cardiovascular events and thrombosis (Surgeon General, 2010). In a clinical study, Argacha et al. (2008) exposed 11 healthy male nonsmokers, mean age 24.6 ± 3 yr, to 300 μg/m^3 $PM_{2.5}$ from the smoke of commercial cigarettes for 1 h, with control subjects exposed to non-tobacco smoke or normal air. Argacha et al. (2008) concluded that acute exposure to secondhand smoke alters both large and small arterial vascular functions, producing a marked change in aortic waveform, and impairs microvascular function, even after exposure ends.

We assessed the acute cardiovascular impact of secondhand smoke on casino patrons using a second clinical investigation of short-term secondhand smoke exposure and acute cardiac morbidity in nonsmokers by Pope et al. (2001). They exposed 16 adult nonsmokers, aged 22–76 (mean age 45 yr), for 1.75 h to an average increase of 53 μg/m^3 PM_3 (range 23–123 μg/m^3) due to secondhand smoke in airport smoking lounges. Their study found that secondhand smoke exposure at this level produced a robust 12% decrement in heart rate variability, which they estimated would produce a 12% increase in cardiac mortality risk. For secondhand smoke, a submicron aerosol, there is a negligible difference between $PM_{2.5}$ and PM_3 (Klepeis et al., 2003).

While there is no direct evidence that secondhand smoke exposure for periods less than 1 h could precipitate an acute coronary event, the circumstantial evidence is both compelling and biologically plausible (NAS, 2010), suggesting susceptible casino patrons are at acute risk even for brief visits.

2.4. Estimation of secondhand smoke exposures

We estimated secondhand smoke $PM_{2.5}$ exposure levels in US casinos for patrons from the differences between indoor and outdoor $PM_{2.5}$ concentrations for the 66 smoking casinos in 5 states, along with indoor–outdoor data from the 3 nonsmoking casinos in CA, NV, and DE. This metric may slightly overestimate the concentration of outdoor $PM_{2.5}$ penetrating indoors due to removal by active or passive filtration. Because casino workers are chronically exposed in close proximity to smokers, to put the magnitude of their exposure into perspective, we compared their cotinine and NNAL doses measured in NIOSH's studies of Las Vegas casino workers with national probability samples of the US adult nonsmoking population (CDC, 2005; Bernert et al., 2010), as well as with a NIOSH cotinine study of Atlantic City casino dealers by Trout and Decker (1996), Trout et al. (1998).

3. Results

3.1. Reno casinos

Table 2 gives values for the physical dimensions (area, ceiling height, and volume), relative occupancy (occupied slots, and total number of slots), and aerometric parameters (CO_2 and $PM_{2.5}$), plus computed values for smoker density D_s, ventilation rate V_o, and their ratio. $PM_{2.5}$ in the smoking gaming areas of 7 Reno casino averaged 37.2 μg/m^3 (SD 17.8 μg/m^3); nonsmoking restaurants in those casinos averaged 14.4 μg/m^3 (SD 9.0 μg/m^3), while outdoor levels averaged 1.28 μg/m^3 (SD 0.6 μg/m^3). By comparison, the $PM_{2.5}$ concentration in the nonsmoking Fernley Nugget averaged 0.6 μg/m^3 (SD 1.4 μg/m^3); its restaurant, showing no effect of cooking on $PM_{2.5}$ levels, averaged 0.7 μg/m^3 (SD 0.9 μg/m^3), and outdoor readings averaged 1.4 μg/m^3 (SD 0.9 μg/m^3). For all 8 casinos combined, the outdoor levels averaged 1.3 μg/m^3 (SD 0.6 μg/m^3); by comparison, the nearby Washoe County hourly ambient air monitoring levels for the corresponding dates and times averaged 1.6 μg/m^3 (SD 1.4 μg/m^3) (Peterson, CA, personal communication, 2009).

As Fig. 1 illustrates, there was considerable $PM_{2.5}$ variation in the nonsmoking restaurants among the 8 Reno casinos. Excluding Harrah's, whose restaurant was across the street from the casino floor and only remotely connected by an enclosed pedestrian overpass, and CalNeva, whose restaurant was in another building and was not sampled, the mean $PM_{2.5}$ in the gaming areas for the 5 remaining Reno smoking casinos was 33 μg/m^3 (SD 15 μg/m^3). By comparison, the mean $PM_{2.5}$ in the nonsmoking restaurants of these 5 casinos was 16 μg/m^3 (SD 9 μg/m^3), while the $PM_{2.5}$ mean for the air just outside those casinos was 1.1 μg/m^3 (0.6 μg/m^3). Table 3 shows the PPAH concentrations for the 5 Reno casinos, reporting relative indoor–outdoor statistics similar to those in Table 2 for $PM_{2.5}$. In the 4 smoking Reno casinos for which it was measured, PPAH averaged 17 ng/m^3 (SD 11 ng/m^3) in the smoking areas, 3.7 times as high as the 4.6 ng/m^3 (SD 0.5 ng/m^3) measured outdoors. Reno's nonsmoking casino restaurants averaged 3.5 ng/m^3 (SD 1.8 ng/m^3). The nonsmoking casino's indoor PPAH averaged 2.3 ng/m^3 (SD 1.5 ng/m^3), compared with an outdoor average of 2.2 ng/m^3 (SD 0.5 ng/m^3), while its restaurant averaged 2.2 ng/m^3 (SD 1.4 ng/m^3), showing no evidence of a cooking contribution.

For the 3 smoking casinos and one nonsmoking casino with CO_2 measurements, the correlation between ventilation-adjusted smoker density D_s/V_o and incremental PPAH in the gaming areas was $R^2 = 0.87$. Indoor temperature and relative humidity ranged from 21 to 24 °C and from 20% to 30%, respectively, while outdoor values ranged from 0 to 16 °C and from 16% to 75%, respectively. The temperature and humidity ranges were within the measured operating range of the SidePak monitor (Jiang, 2010). Using Eq. (1), V_o for the 8 Reno casinos ranged from 15 L/s-person to 45 L/s-person, and averaged 27.7 L/s-person (GM 26.2 L/s-person) (Table 2, Column 11).

Adult smoking prevalence is defined as the percentage of persons (aged 21 yr or more) who are smokers, which for Nevada in 2009 was 22% (95% CI, 19.5–24.5%); the US median was 20.6% (95% CI,

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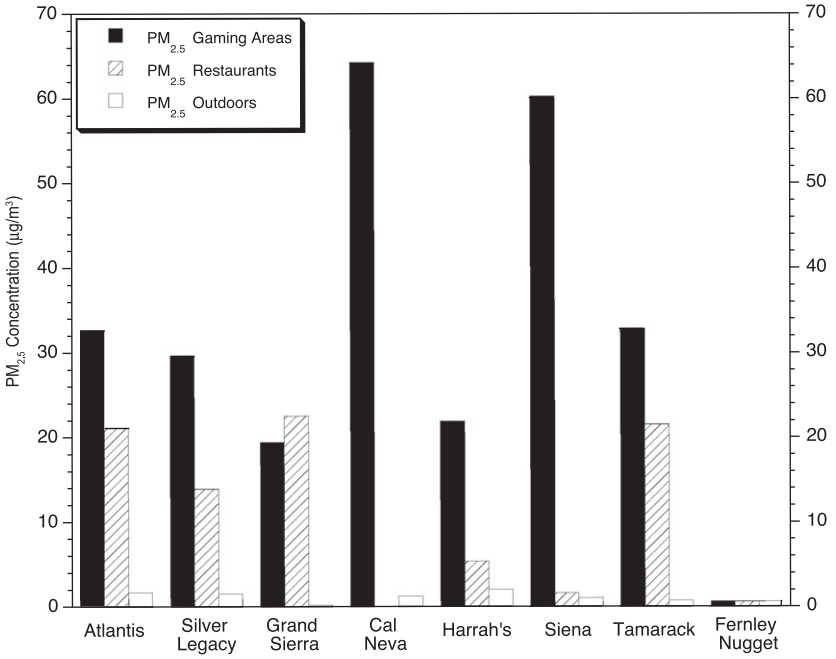

Fig. 1. $PM_{2.5}$ in Reno smoking casinos, nonsmoking restaurants in those casinos, and outside the casinos in outdoor air. Harrah's nonsmoking restaurant is across the street and is connected by an enclosed pedestrian overpass. CalNeva has no nonsmoking area, and the Fernley Nugget is a nonsmoking casino. Most nonsmoking areas in smoking casinos were polluted with secondhand smoke.

19.9–21.3%) (MMWR, 2009, 2010a, 2010b). The number of persons who are actively smoking in an area at any one time is defined as the *active smoking count* (ASC) (Repace, 2007). For the 7 smoking Reno casinos in Table 2, the *active smoking percentage* (ASP)=[100 times the ASC divided by the number of patrons]=9.3% (SD 2.1%) in our sample of 2256 persons (Table 2, column 12).

3.2. Results of previous studies of casinos

We now review all the casino studies previously published in the peer-reviewed literature. For all $PM_{2.5}$ measurement studies except Trout and Decker (1996) and Trout et al. (1998), we obtained the individual casino data from the investigators.

In a 3-day study of 18 Nevada casinos in 2006, Pritsos et al. (2008) observed an active smoking percentage of 7.2% in a sample of 4737 Reno/Sparks gamblers, 6.7% for 7633 Las Vegas gamblers, and 5.5% among 1682 Lake Tahoe gamblers. Overall, they observed 947 active smokers among 14,052 gamblers, yielding an active smoking percentage of 6.74%. Pritsos et al. (2008) corresponded this to an estimated smoking prevalence among Nevada casino patrons of 20.2% (95% CI 0.7%). By comparison, in 2006, the adult smoking prevalence in Nevada was 22.2% (95% CI 20.0–24.4%) and in the US at large the median was 20.8% (95% CI 20.1–21.5%) (MMWR, 2007).

In a survey of 36 of the 58 California Indian casinos, Jiang et al. (2010) measured $PM_{2.5}$ indoors and outdoors with gravimetrically calibrated real-time SidePak monitors. They reported 0.5–1 h average casino smoking area $PM_{2.5}$ concentrations of 63 $\mu g/m^3$ (range 21–183 $\mu g/m^3$), compared with 7 $\mu g/m^3$ outdoors (range 1–30 $\mu g/m^3$), and nonsmoking restaurants in these casinos averaged 29 $\mu g/m^3$ (range 1–81 $\mu g/m^3$), indicating migration of secondhand smoke from smoking to nonsmoking areas. They also noted that children were often present in these restaurants. The only nonsmoking CA casino, the Lucky Bear, averaged 4.8 $\mu g/m^3$ indoors and 4.9 $\mu g/m^3$ outdoors.

York and Lee (2010) measured real-time $PM_{2.5}$ inside and outside 16 smoking casinos in Las Vegas, NV, for an average of 34 min each (SD 7.3 min). The casino gaming area $PM_{2.5}$ averaged 48 $\mu g/m^3$ (SD 16 $\mu g/m^3$); the nonsmoking restaurant $PM_{2.5}$ averaged 31 $\mu g/m^3$ (SD 23 $\mu g/m^3$), while the outdoor $PM_{2.5}$ averaged 4.7 $\mu g/m^3$ (SD 3.1 $\mu g/m^3$). The $PM_{2.5}$ levels among casinos ranged from 20 to 73 $\mu g/m^3$, while nonsmoking casino restaurants ranged from 5 to 101 $\mu g/m^3$ (N. York, personal communication, 2010).

Repace (2009) measured real-time $PM_{2.5}$ concentrations inside and outside 3 PA casinos: 1-h real-time $PM_{2.5}$ concentrations inside the casinos averaged 106 $\mu g/m^3$ (SD 25 $\mu g/m^3$), 6 times the outdoor average of 18 $\mu g/m^3$. In a 17-min study of simultaneous $PM_{2.5}$ concentrations, one casino's smoking area averaged 201 $\mu g/m^3$; its equally large nonsmoking area on the floor above averaged 37 $\mu g/m^3$, and outdoor measurements averaged 13 $\mu g/m^3$. For all 3 casinos, PPAH concentrations averaged 20 ng/m^3 (SD 8 ng/m^3), 3.7 times the outdoor average of 5.4 ng/m^3. CO_2-estimated ventilation rates V_o were 16, 19, and 34 L/s-person for the 3 casinos.

Achutan et al. (2009, in press) performed integrated 8-h work shift average pump-and-filter gravimetric measurements in a 2007 NIOSH secondhand smoke complaint investigation of 3 smoking Las Vegas casinos—Bally's, Paris, and Caesar's Palace. NIOSH measured personal air and work-area exposure to the atmospheric markers respirable particles (essentially $PM_{2.5}$), nicotine, total PAH, and volatile organic compounds, as well as work shift changes in urine cotinine and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) in 114 casino dealers. NNAL is a metabolite of the particulate-phase tobacco-specific nitrosamine, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), a potent lung carcinogen (Hecht, 1999). Achutan et al. (2009, in press) reported total urine cotinine averaging 26.4 ng/mL (range 7.5–88 ng/mL) in 123 dealers reporting secondhand smoke exposure only at work (C. West, personal communication, 2009). Post-shift GM total cotinine increased from the pre-shift value of 22.25 ng/mL by 36% to the post-shift value of 30.37 ng/mL. Eliminating the 28% hydroxycotinine to convert total cotinine to free cotinine, we estimate the free urine cotinine median of $U=19.0$ ng/mL (range 5.4–64 ng/mL), calculated from the average of pre- and post-shift values. NIOSH also measured total NNAL concentrations ($n=114$); the pre-shift NNAL GM was 3.9 pg/L, and the post-shift NNAL GM increased by 77% over pre-shift values to 6.9 pg/mL. The average

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Table 3
Reno Casino 1-h $PM_{2.5}$ and PPAH measurements indoors and outdoors.

1. Casino, (maximum occupancy for space sampled)	2. Ave. # persons present (SD)	3. Ave. # persons per 1000 ft^2	4. Ave. number of burning cigarettes ($\overline{ASC}$)[a]	5. PPAH casino indoors (ng/m^3)	6. Ave. PPAH outdoor level, (ng/m^3)	7. PPAH non-smoking restaurant area (ng/m^3)	8. Ave. casino indoor $PM_{2.5}$ (μg/m^3)	9. Ave. outdoor $PM_{2.5}$ (μg/m^3)	10. Non-smoking restaurant $PM_{2.5}$ (μg/m^3)	11. Estimated active smoker percentage[b] (ASP%)	12. D_s, Active smoker density ($\overline{ASC}$ per 100 m^3)	13. V_o, ventilation rate per person (L/s-occupant)	14. D_s/V_o ventilation-adjusted smoker density
Siena	123.5	6.25	16	20.0	4.0	1.6	46.3	0.27	1.7	13	0.12	–	–
(423)	(SD 10.0)		(SD 2.2)	(SD 8.6)	(SD 4.0)	(SD 1.6)	(SD 17.3)	(SD 0.73)	(SD 0.95)				
Tamarack	131.5	7.17	15.5	5.7	5.0	3.6	30.9	0.19	21.6	11.8	0.12	25	0.005
(438)	(SD 44.6)		(SD 2.1)	(SD 2.6)	(SD 3.1)	(SD 2.4)	(SD 8.77)	(SD 0.52)	(SD 6.46)				
CalNeva	138.5	15.0	10	30.6	5.0	–	66.9	1.20	–	7.2	0.11	8.0	0.014
(390)	(SD 14.3)		(SD 4.8)	(SD 27.5)	(SD 5.0)		(SD 37.7)	(SD 1.82)					
Harrahs	314	11.5	20.5	11.6	4.5	5.18	18.8	1.5	5.40	6.5	0.22	33	0.007
(597)	(SD 17.3)		(SD 1.73)	(SD 6.3)	(SD 6.4)	(SD 4.8)	(SD 8.39)	(SD 1.63)	(SD 3.69)				
Fernley Nugget[c]	35.7	3.6	0	2.3	2.2	2.2	0.15	0.24	0.68	0	0	27	0.000
(169)	(SD 1.5)			(SD 1.5)	(SD 1.4)	(SD 1.4)	(SD 0.60)	(SD 0.55)	(SD 0.91)				
Mean, all smoking areas	–	–	–	17.0	4.62	3.46	40.7	0.79	9.57	9.63	0.14	–	–
				(SD 10.8)	(SD 0.48)	(SD 1.79)	(SD 20.8)	(SD 0.66)	(SD 10.6)	(SD 3.25)	(SD 0.05)		
Geometric means	–	–	–	14	4.6	2.8	36.6	0.55	5.8	–	–	–	–

[a] Average active smoking count ($\overline{ASC}$) = number of actively burning cigarettes counted every 10 min for 1 h and then averaged.
[b] Average active smoking count $\overline{ASC}$ divided by the average number of persons present.
[c] Nonsmoking casino.

of pre- and post-shift concentrations was 5.4 pg/mL. (*Note*: NIOSH did not release any personal data for individual casino workers.)

Achutan et al. (2009, in press) reported 8-h GM area $PM_{2.5}$ levels of 38, 33, and 56 μg/m^3 at stationary indoor locations for the 3 Las Vegas casinos investigated by NIOSH, yielding a combined GM of 41 μg/m^3 (n=22, range 23–86 μg/m^3). For dealers in these casinos, the personally monitored $PM_{2.5}$ GM was similar, at 42 μg/m^3 (n=33, range 13–140 μg/m^3), and personal nicotine was 5.3 μg/m^3 (n=107, range 0.6–17 μg/m^3) for dealers. Outdoor levels were not reported. NIOSH measured its casinos during the evening hours in January 2006 over 3 consecutive days. Outdoor $PM_{2.5}$ levels during NIOSH's study from Clark County's Sahara Avenue outdoor air monitoring site (2.4 km distant) averaged 7.7 μg/m^3 (SD 3.9 μg/m^3) for those times (Clark County, 2009). Our analysis of NIOSH data (C. West, personal communication) for Las Vegas casino dealers (n=28) yielded a paired personal incremental $PM_{2.5}$ to nicotine ratio of 9:1. NIOSH stated that both Bally's Las Vegas and Caesar's Palace casinos reported a low filtration efficiency, less than 30% for fine particles for those HVAC systems, while Paris reported a high efficiency rating (80–95%) for them. However, Bally's Las Vegas and Paris had similar $PM_{2.5}$ gaming area concentrations, while Caesar's Palace had nearly twice the concentration of the other two casinos, suggesting that additional air cleaning in the Paris casino did not result in lower relative concentrations. Achutan et al. (2009, in press) also reported that 24% (n=35) of the casino dealers had symptoms of work-related asthma.

Repace (2004) measured real-time indoor and outdoor $PM_{2.5}$ and PPAH for the Delaware Park casino before and after Delaware's 2002 statewide smoke-free workplace law. When this casino allowed smoking, mean 0.5-h $PM_{2.5}$ and PPAH levels averaged 205 μg/m^3 and 163 ng/m^3, respectively, while outdoor levels averaged 11 μg/m^3 and 27 ng/m^3, respectively. The indoor-to-outdoor PPAH ratio was 6:1. After the casino had become smoke-free, indoor levels averaged 9 μg/m^3 for $PM_{2.5}$ and 4 ng/m^3 for PPAH; while post-ban outdoor $PM_{2.5}$ and PPAH levels averaged 7 μg/m^3 and 8 ng/m^3, respectively. The pre-ban and post-ban indoor differences suggest that 96% of both $PM_{2.5}$ and PPAH were due to secondhand smoke. Indoor and outdoor differences prior to the ban suggest that at least 95% of $PM_{2.5}$ and 83% of PPAH were due to secondhand smoke.

Trout and Decker (1996) and Trout et al. (1998), in an earlier NIOSH secondhand smoke complaint investigation, measured 8-h average gravimetric area samples in the 80,000 ft^2 Bally's casino on the island of Atlantic City, NJ, in March 1996, yielding mean PM_{10} levels of 80 μg/m^3 (SD 12 μg/m^3) (n=5). The mean personal nicotine concentrations for nonsmoking dealers (n=17) averaged 9.5 μg/m^3 (SD 3.0 μg/m^3); the median was 10 μg/m^3. Outdoor levels were not measured. The casino had 17 air-handling units. CO_2 concentrations averaged 532 ppm (n=18) indoors (ranging from 425 to 850 ppm) and 300 ppm outdoors (n=3). In an analysis of Bally's Atlantic City CO_2 data, we used Eq. (1) to calculate an average value of 22 L/s-person for V_o. The maximum casino occupancy was 9560 patrons, plus 800 workers. Smoking was permitted throughout the casino floor. For nonsmoking workers exposed at work only (n=17), the post-shift GM serum cotinine was 1.82 ng/mL, a 36% increase over the 1.34 ng/mL pre-shift GM. The 1.58 ng/mL average of the pre-shift and post-shift GM concentrations exceeded the 0.318 ng/mL population GM for a national sample of US workers exposed only at work by a factor of 5 (Pirkle et al., 1996). Workers at nonsmoking tables did not experience less secondhand smoke exposure than those at smoking tables.

Both casino dealers and patrons absorb tobacco combustion products from casino air. NIOSH measured significant increases in dealers' cotinine levels at the end of work shifts in both the Las

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Vegas and Atlantic City studies, and in NNAL as well in the later Las Vegas study. Even greater increases in NNAL and cotinine have been found among casino patrons, who generally do not experience routine daily exposure to casino air. This likely occurs as a result of lower initial concentrations of these secondhand smoke biomarkers in their body fluids. Anderson et al. (2003) observed an 89% average increase in urine NNAL and a 4-fold increase in urine cotinine in casino patrons ($n=16$) exposed in a smoking Midwest casino for 4 h, while Repace (2009) observed a 19-fold average increase in urine cotinine in nonsmoking casino patrons ($n=7$) who visited 3 Pennsylvania casinos for 4 h.

3.3. Distributions of indoor and outdoor air pollution in 66 smoking casinos

We combined our data from the Reno field survey with the data from the previous CA, DE, NV, NJ, and PA casino studies to create indoor and outdoor frequency distributions for 66 smoking casinos in 6 locations (Fig. 2); the 3 nonsmoking casinos are not included. Indoor $PM_{2.5}$ concentrations in smoking casinos ranged from 18.5 to 205 μg/m^3 with a median of 57.2 μg/m^3; the outdoor $PM_{2.5}$ concentrations ranged from 0.26 to 29.7 μg/m^3 with a median of 4.5 μg/m^3. The vertical axis in Fig. 2 gives the $PM_{2.5}$ concentrations plotted on a logarithmic scale, while the horizontal axis gives the cumulative percentages of the data plotted on a scale based on the integral of the standard normal probability model. The loci of the data points for both indoor and outdoor casino measurements are relatively straight lines on this graph, indicating that both data sets fit the 2-parameter lognormal probability model well (Ott, 1996). For both data sets, the Shapiro–Wilk goodness-of-fit test rejects the normal probability model at the 95% level of confidence, while the lognormal probability model is not rejected even at the 99% confidence level.

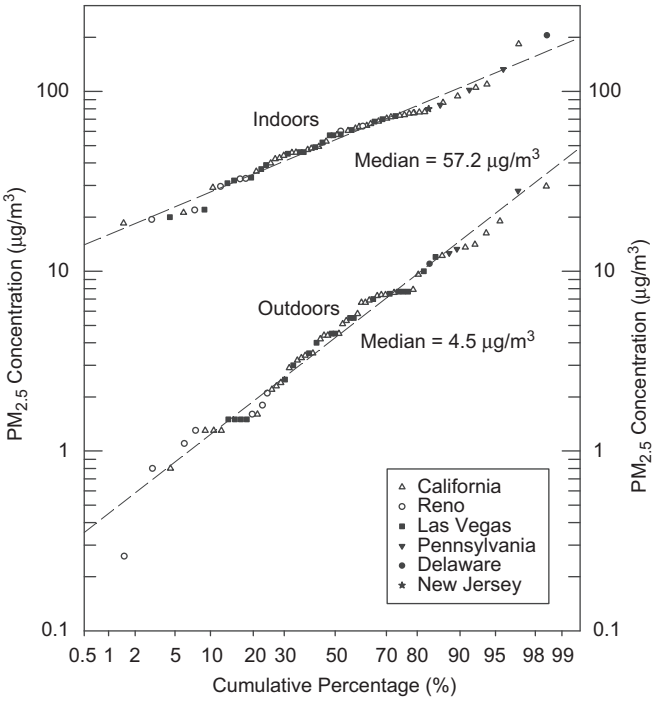

Fig. 2. A logarithmic probability plot showing the cumulative percentage of US casinos exceeding a given $PM_{2.5}$ level. Indoor ($n=66$) and outdoor ($n=65$) $PM_{2.5}$ concentrations for 66 visits to 66 smoking casinos in 5 states: 26 in Nevada (Reno and Las Vegas), 35 in California, 3 in Pennsylvania, and one each in Delaware and New Jersey. The graph shows that the indoor $PM_{2.5}$ concentrations in 93% of the US casinos exceeded 25 μg/m^3, the numerical value of the 24-h WHO $PM_{2.5}$ clean air guideline, and 80% of the casinos exceeded 35 μg/m^3, the numerical value of the USEPA 24-h $PM_{2.5}$ ambient air quality standard.

The frequency distributions plotted in Fig. 2 can be interpreted as follows: any point on the line gives the percentage of the casinos below a particular concentration on the horizontal axis. For example, to find the percentage of casinos with indoor concentrations above the level of WHO's 25 μg/m^3 24-h guideline, we find "25" on the vertical axis in μg/m^3, and then we read corresponding horizontal axis value, 7%. This result means that 100−7%=93% of the US casinos were at or above 25 μg/m^3. Similarly, 20% of the casinos were below 35 μg/m^3, so 80% were at or above 35 μg/m^3, the numerical value of EPA's 24-h standard. In this way, the entire frequency distribution of US indoor casino concentrations is available, with each datum displayed, and the percentage of the casinos at or above any concentration can be read directly from the graph by simple subtraction.

The parameters of the lognormal model were estimated by linear regression using Version 11 of Sigma-Plot® (Systat Software, San Jose, CA), yielding a 66-casino indoor GM of 53.8 μg/m^3 (GSD 1.7), more than 10 times as high as the outdoor GM of 4.3 μg/m^3 (GSD 2.6). The corresponding arithmetic means and standard deviations of the lognormal model of these distributions, which are useful for risk assessment, were 61.7 μg/m^3 (SD 34.5 μg/m^3) indoors and 6.8 μg/m^3 (SD 8.5 μg/m^3) outdoors. By comparison, $PM_{2.5}$ concentrations in the gaming areas of 3 nonsmoking casinos averaged only 4.9 μg/m^3 (SD 4.4 μg/m^3).

The individual 8-h gravimetric indoor $PM_{2.5}$ averages for all the 3 NIOSH-investigated casinos in Las Vegas ($n=24$) ranged from 19 μg/m^3 (the limit of detection of their samplers) to 86 μg/m^3, which correspond to the 2nd and 82nd percentiles of Fig. 2. These results suggest that the 0.5–1 h indoor measurements made by real-time studies, such as ours (with limits of detection of 1 μg/m^3), are consistent with NIOSH's indoor 8-h measurements.

3.4. Estimation of acute health effects from secondhand smoke in casinos

As discussed in Section 2.3, the exposure–response relationship of Pope et al. (2001) showed that an average exposure to 53 μg/m^3 of PM_3 from secondhand smoke for 1.75 h caused a decrement in heart rate variability of 2.3% per 10 μg/m^3 increase in PM_3. The difference between the indoor and outdoor medians of the $PM_{2.5}$ concentrations for the 66 smoking casinos in Fig. 2 was 57.2−4.5 μg/m^3=52.7 μg/m^3. Combining this result as an estimate of the median of $PM_{2.5}$ due to secondhand smoke with the acute exposure–response relationship of Pope et al. (2001), we find the estimated average decrement in heart rate variability for casino patrons and workers is (52.7 μg/m^3) (2.3%/10 μg/m^3)= 12% for 1–2 h of exposure. Therefore, about half the casinos were at or above this level. At the upper 95th percentiles of each distribution, the difference between the indoor and outdoor values is (127−21 μg/m^3)=106 μg/m^3, which corresponds to a decrement in heart rate variability of (106 μg/m^3) (2.3%/10 μg/m^3)= 24%. Thus, secondhand smoke poses an acute threat to the cardiovascular health of casino patrons and workers.

4. Discussion

4.1. Effect of smoking on indoor air pollution in casinos

In 2007, domestic US cigarette consumption was 360 billion cigarettes (CDC, 2010). In 2007, an estimated 19.8% (43.4 million) of US adults were current cigarette smokers (MMWR, 2008). Assuming 19.8% of the 61.7 million annual casino visitors (AGA, 2008) were smokers, we estimate that 12.2 million smoking

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patrons visited US casinos annually, and that the remaining 49.5 million nonsmoking patrons inhaled the smoke from their cigarettes. Similarly, an estimated 80.2% of the combined 532,277 commercial and tribal casino workers (AGA, 2010; NIGA, 2010) were nonsmokers, and thus we estimate that 427,000 nonsmoking casino workers breathed secondhand smoke at work.

Secondhand smoke is a prolific source of $PM_{2.5}$ in indoor air, with each cigarette emitting about 14 mg of $PM_{2.5}$. In addition, there are at least 172 toxic substances in tobacco smoke in both the gas and particulate phases, of which 33 are classified as hazardous air pollutants, 47 as hazardous wastes, 3 as criteria air pollutants, and 67 as known carcinogens (Repace, 2007). Of the latter, 20 are involved in lung carcinogenesis, and of these, PPAH (10 compounds) are among the most significant (Hecht, 1999).

The similarity of $PM_{2.5}$ in nonsmoking casinos' gaming floors with outdoor $PM_{2.5}$ levels, the correlation between incremental $PM_{2.5}$ and D_s/V_o on the gaming floors of smoking casinos, and the 10:1 ratio between $PM_{2.5}$ levels inside and outside casinos, taken together with the 9:1 ratio between personal incremental $PM_{2.5}$ and nicotine in the NIOSH Las Vegas study, suggest that, on average, at least 90% of the indoor $PM_{2.5}$ in smoking casinos is due to secondhand smoke. Even in the very large 37,000 m^3 dilution volume of the Grand Sierra casino in Reno with only a 35% occupancy (Table 2) and with the lowest ventilation-adjusted smoker density and lowest indoor $PM_{2.5}$ levels measured, the indoor $PM_{2.5}$ was 75 times the outdoor level, due to secondhand smoke. By contrast, the single Reno smoke-free casino had a level indoors that was lower than outdoors (Fernley Nugget, Table 2).

Most of the casino measurements in Fig. 2 are snapshots of concentrations averaging 0.5–1 h on a single visit to a casino, and a different average value might be encountered on another visit to the same casino on a different date. However, the range in 8-h averages measured by NIOSH in 4 casinos in Las Vegas and Atlantic City was similar to the range of short-term data for the remainder of the casinos of Fig. 2. Thus we believe the distribution we present accurately reflects the concentration range and percentiles expected for US casinos.

A total of 5 casinos in Reno from our field study and 16 casinos in Las Vegas studied by York and Lee (2010) (N. York, personal communication, 2009) had nonsmoking restaurant areas, allowing a comparison of the $PM_{2.5}$ concentrations in the smoking gaming areas with those in the nonsmoking restaurant areas for a 21-casino subset of the 66 casinos (Table 4). In these 21 Nevada casinos, $PM_{2.5}$ in smoking-permitted gaming areas averaged 45.2 μg/m^3 (95% CI, 37.7–52.7 μg/m^3), nonsmoking casino restaurants exposed to gaming areas averaged 27.2 μg/m^3 (95% CI, 17.5–36.9 μg/m^3), while $PM_{2.5}$ outside the casinos averaged 3.9 μg/m^3 (95% CI, 2.5–5.3 μg/m^3), indicating that although the restaurants were nominally "nonsmoking," they were not smoke-free. The ratio of the average $PM_{2.5}$ concentrations in nonsmoking restaurant areas to outdoors was 7:1; while the ratio of the average casino smoking area concentrations to outdoors was nearly 12:1, compared with that in the single nonsmoking casino in Reno, where the restaurant concentration ratio was 1:1 with outdoors.

These results suggest that secondhand smoke infiltrates into nonsmoking restaurants that are not isolated from the smoking areas, consistent with the findings of other studies (Repace and Lowrey, 1980; Repace, 2009; Jiang et al., 2010; Huss et al., 2010; York and Lee, 2010). Although Nevada's prohibition of smoking in casino restaurants reduced their secondhand smoke pollution by about 40% relative to the gaming areas, those restaurant areas remained 7 times as polluted as outdoors. PPAH levels in 8 smoking casinos in 3 states averaged 4 times outdoors. However, PPAH levels in the nonsmoking areas of 4 casinos were comparable to outdoors. These low PPAH levels may reflect the nearly double surface sorption rate measured for PPAH relative to $PM_{2.5}$ (Repace, 2004). The high PPAH deposition rate suggests additional potential for significant surface contamination and re-emission in casinos (Burton, 2011).

Table 4
Comparison of smoking, nonsmoking restaurant, and outdoor $PM_{2.5}$ concentrations from Las Vegas and Reno, NV[a].

	Casino	Smoking (μg/m^3)	Restaurant (μg/m^3)	Outdoors (μg/m^3)
1.	Ellis Island	57	26	7.0
2.	Terrible's	32	39	12.0
3.	Palace station	45	101	3.5
4.	4 Queens	49	22	4.5
5.	The Orleans	61	33	7.5
6.	Fiesta	39	7	3
7.	Sunset station	52	14	1.5
8.	MGM	70	50	1.5
9.	Luxor	46	43	1.5
10.	Monte Carlo	37	5	2.5
11.	Mandalay Bay	22	18	1.5
12.	NY NY	68	33	4
13.	Planet Hollywood	20	9	4.5
14.	Green Valley	57	23	10.0
15.	Circus Circus	73	38	5.5
16.	Riviera	46	30	5.5
17.	Atlantis	32.6	21.1	1.8
18.	Silver Legacy	29.6	13.9	1.6
19.	Grand Sierra	19.4	22.5	0.26
20.	Siena	60.1	1.7	1.1
21.	Tamarack	32.8	21.5	0.8
	Mean	45.2	27.2	3.9
	Std. dev	16.4	21.2	3.1
	95% CI about the mean	±7.5	±9.7	±1.4

[a] The first 16 casinos are in Las Vegas and are based on data from York and Lee (2010); while entries 17–21 are located in Reno and are based on our field survey.

4.2. Effect of ventilation on indoor air pollution in casinos

Linear regression analyses for 10 casinos, 7 in Reno plus 3 from Pennsylvania, showed a correlation between incremental $PM_{2.5}$ and ventilation-adjusted smoker density, D_s/V_o: incremental $PM_{2.5}=5702(D_s/V_o)+7.83$, with $R^2=0.73$. The regression for incremental $PM_{2.5}$ versus D_s alone gave $R^2=0.60$; while the incremental $PM_{2.5}$ versus V_o alone yielded only $R^2=0.15$, suggesting that smoker density is the primary determinant of secondhand smoke concentrations.

This conclusion is consistent with our CO_2 measurements. For 5 of the 6 smoking Reno casinos for which there were CO_2 measurements, our estimated V_o values exceeded the ASHRAE Standard 62-2001 recommended ventilation rate for casinos with smoking by 17–200%, indicating that the Reno casinos were well-ventilated by the engineering criteria of 2001. By contrast, using NIOSH work shift average carbon dioxide levels (C. West, personal communication, 2009) and Eq. (1), we calculated V_o values for the 3 Las Vegas casinos ranging from 9 to 13 L/s-person, or from 60% to 87% of the ASHRAE Standard 62-2001, indicating substandard ventilation. However, the difference in $PM_{2.5}$ means between the 7 Reno smoking casinos (37 μg/m^3, SD 18 μg/m^3) and the 3 Las Vegas Casinos (41 μg/m^3, SD 21 μg/m^3) was slight, suggesting that despite much higher ventilation in the Reno casinos compared with Las Vegas casinos, Reno did not have proportionally better air quality. Similarly, NIOSH's study of Bally's in Atlantic City, with V_o at 147% of the ASHRAE Standard, had much higher 8-h average PM_{10} and nicotine values of 80 and 11 μg/m^3, respectively, than the 41 and 5.3 μg/m^3 for the much more poorly ventilated Las Vegas casinos studied by NIOSH.

For the 14 casinos in DE, NJ, NV, and PA for which it was measured, V_o was approximately lognormally distributed with

GM=20 L/s-person (GSD 1.8), ranging from 9 to 45 L/s-person. Although these 14 casinos on average were well-ventilated according to ASHRAE Standard 62-2001, their GM $PM_{2.5}$ concentration (51.6 μg/m^3) was an order of magnitude higher than for nonsmoking casinos or for outdoors.

We have no information on the air cleaning devices for any casinos except those reported for the 3 casinos in the NIOSH Las Vegas study: Bally's Las Vegas had triple the filtration efficiency of either Paris or Caesar's Palace casino. However, when we normalized the $PM_{2.5}$ for both nicotine level and ventilation rate per occupant, their adjusted $PM_{2.5}$ concentrations were only marginally different: 31 μg/m^3 for Bally's Las Vegas versus 36 μg/m^3 for Paris and 32 μg/m^3 for Caesar's Palace, suggesting that air cleaning was not effective.

4.3. Effect of smoking on PPAH air pollution inside casinos

Secondhand smoke is a known human carcinogen (NIEHS, 2000), and PAHs are among a diverse group of gas- and particulate-phase carcinogens formed in the incomplete combustion of organic compounds in tobacco. PAHs induce respiratory tract tumors in animals upon inhalation (Hecht, 2003), and are implicated in cardiovascular disease as well (Glantz and Parmley, 1991). Particle-bound PAHs (PPAHs) are compounds with 4 or more benzene rings contained in tobacco smoke, as well as diesel exhaust, incense, wood smoke, and smoky candles. At least 10 PPAHs are carcinogens (Repace, 2004). Burning toast or frying hamburgers, likely common in casino restaurants, do not appear to be important sources of PPAH (Ott and Siegmann, 2006).

For the 5 Reno casinos in Table 3, the linear regression slope yielding the ratio of incremental PPAH to the incremental $PM_{2.5}$ was 0.048% (R^2=0.79). By comparison, this ratio was 0.049% for 6 Boston bars (Repace et al., 2006b); it was 0.049% for a casino, 6 bars, and a pool hall in Wilmington, DE (Repace, 2004), and 0.046% for 3 Pennsylvania casinos (Repace, 2009). The percentage of $PM_{2.5}$ consisting of PPAH for secondhand smoke from all 4 sets of data comprising these 21 indoor locations was 0.048% (SD 0.0014%), R^2=0.80. A controlled experiment with smolder-generated Marlboro cigarette smoke showed a similar ratio of 0.043% (Repace, 2004). For the 4 Reno smoking casinos in Table 3, the indoor-to-outdoor PPAH ratio was 3.7:1, while this ratio was 1.05:1 for the single Reno nonsmoking casino. By comparison, for 8 casinos, the weighted average indoor-to-outdoor PPAH ratio was 4:1 (3.7:1 for 4 casinos in Reno, 3.7:1 for 3 casinos in Pennsylvania (Repace, 2009), and 6:1 for the one in Delaware (Repace, 2004)).

4.4. Analysis of casino workers' secondhand smoke doses

To place the Las Vegas Casino dealers' cotinine doses from the NIOSH study (Achutan et al., 2009, in press) into perspective, we convert their median urine cotinine, U=19 ng/mL (range: 5.4–64 ng/mL), into an estimated serum cotinine dose P using the equation: $P=U/6.5=(19\ \text{ng/mL})/6.5=2.92$ ng/mL (range: 0.83–9.8 ng/mL) (Repace et al., 2006a). This high dose indicates a secondhand smoke exposure exceeding the 95th percentile (1.38 ng/mL; 95% CI, 1.11–1.84 ng/mL) measured for serum cotinine in a representative sample of nonsmoking US adults aged 20 yr or more in 2001–2002 in the NHANES study (CDC, 2005). This result is consistent with the earlier results of Trout et al. (1998) for Atlantic City casino workers. Further, Bernert et al. (2010) recently measured the urine concentrations of total NNAL in a representative sample of US adults in the NHANES survey, reporting a 75th percentile value of 2.0 pg/mL (95% CI 1.7–2.5 pg/mL) and a 90th percentile value of 8.1 pg/mL (95% CI 6.9–9.8 pg/mL) for nonsmokers. Thus, the average GM NNAL for Las Vegas casino dealers of 5.4 pg/mL (Achutan et al., 2009, in press) appears to be above the 80th percentile of the US population's exposure to this potent tobacco-specific lung carcinogen.

4.5. Health effect implications

Secondhand smoke exposure increases heart disease mortality: the California Environmental Protection Agency estimated that 46,000 excess ischemic heart disease deaths in US adults are annually attributable to secondhand smoke exposure at home and in the workplace (CalEPA, 2005). Lightwood et al. (2009) estimated that at early 21st Century exposure levels, secondhand smoke causes 60,000–204,000 deaths from ischemic heart disease, cardiac arrhythmias, heart failure, and myocardial infarction annually, with estimated treatment costs ranging from $1.8 to $6 billion annually.

Reducing secondhand smoke exposure reduces heart disease mortality: The National Academy of Sciences (NAS, 2010) concluded there is a causal relationship between smoking bans and decreases in acute myocardial infarctions. NAS (2010) estimated that smoking bans reduced acute myocardial infarctions by 6–47%. Meyers et al. (2009), in a meta-analysis of 11 studies, concluded that smoking bans in public places and workplaces are significantly associated with a 17% average reduction in acute myocardial infarction incidence; an incremental acute myocardial infarction reduction of 26% occurred for each year of ban implementation, with the greatest impact on younger nonsmokers. In the 10 months following the Scottish smoking ban, Pell et al. (2008) in a prospective study of hospital admissions for acute coronary syndrome (n=3235), found a 14% reduction in the number among smokers, a 19% reduction among former smokers, and a 21% reduction among persons who had never smoked. Overall, 67% of the decrease in coronary admissions occurred in nonsmokers.

The amount of secondhand smoke $PM_{2.5}$ associated with increases and reductions in heart disease mortality has not been quantified. However, Pope et al. (2009) suggest that $PM_{2.5}$ in the outdoor air and $PM_{2.5}$ from secondhand smoke appear to have similar toxicity. This analogy offers some insight in the potential magnitude of the effect of casino $PM_{2.5}$ on heart disease in casino workers. The pathophysiology of secondhand smoke induced cardiovascular disease is consistent with the known association between $PM_{2.5}$ and coronary heart disease from outdoor air pollution epidemiology (NAS, 2010). Long-term exposure to $PM_{2.5}$ increases cardiovascular mortality, biomarkers of cardiorespiratory risk, and subclinical atherosclerosis. Short-term $PM_{2.5}$ exposure is associated with cardiovascular and cerebrovascular mortality, increased hospital admissions, myocardial infarction, pulmonary and systemic inflammation, oxidative stress, altered cardiac autonomic function, and arterial vasoconstriction. The cardiopulmonary response to $PM_{2.5}$ on both daily and long-term exposures appears linear across the range of locally monitored outdoor air concentrations (Pope and Dockery, 2006; Pope et al. 2009).

As discussed in Section 3.4, applying the exposure–response relationship of Pope et al. (2001) to our data yielded an estimated decrement in heart rate variability ranging from about 12% to 24% for at least 50% of the smoking casinos. This range of estimates of increased acute morbidity from secondhand smoke in casinos is consistent with the contribution to acute cardiovascular morbidity from secondhand smoke exposure suggested by the public smoking ban studies by NAS (2010) and by Lightwood et al. (2009).

Chronically increased exposure to $PM_{2.5}$ is associated with significant increases in heart disease mortality. Quantitative estimates of exposure–response indicate that a daily increase of 10 μg/m^3 in outdoor $PM_{2.5}$ concentrations increased the risk of

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ischemic heart disease mortality by 18% (95% CI, 14–23%) in the American Cancer Society's Cancer Prevention Study II (time scale, 18 yr; cohort, >300,000 persons aged >30+ yr) (Pope et al., 2009). In the Women's Health Initiative study (93,676 women aged 50–75 yr), a daily increase of 10 μg/m^3 in outdoor $PM_{2.5}$ increased risk for cardiovascular disease mortality by 24% (95% CI, 9–41%), and by 28% (95% CI, 13–44%) in the extended Harvard 6-city study (time scale 1–8 yr; cohort, 8000 whites, aged 25–74 yr) (Pope et al., 2009). On average, 90% of the $PM_{2.5}$ in the smoking casinos is due to secondhand smoke. These findings suggest that in addition to short-term morbidity, the chronic exposure to the levels of $PM_{2.5}$ found in casinos with smoking poses long-term heart disease mortality risks for casino workers.

The high cotinine levels found in casino workers also suggest long-term increases in cardiovascular mortality. The cotinine levels found in the 114 casino dealers by Achutan et al. (2009, in press), with an estimated median serum cotinine equivalent of 2.92 ng/mL, and in the 17 casino dealers studied by Trout et al. (1998), with a measured median serum cotinine of 1.58 ng/mL, are in the range of chronic coronary disease increases reported for long-term (5–20 yr) exposure by Whincup et al. (2004). Whincup et al. (2004) examined the associations between serum cotinine concentration and fatal and non-fatal coronary heart disease in a 20-yr cohort study of 2000+ nonsmoking men aged 40–59 yr of age. Relative to the reference quartile (cotinine <0.7 ng/mL), men whose cotinine fell into successively higher quartiles had statistically significant increases in heart disease ranging from 45% (cotinine 0.8–1.4 ng/mL), to 49% (cotinine 1.5–2.7 ng/mL), to 57% (cotinine 2.8–14.0 ng/mL).

Moraros et al. (2010) reported that Delaware's comprehensive nonsmoking ordinance, which extended a decade-long workplace smoking ban to restaurants, bars, and casinos, was associated with statistically significant decreases in both acute myocardial infarction and asthma incidence in Delaware residents when compared with non-Delaware residents.

The California EPA has identified asthma induction and exacerbation in adults as among the respiratory health effects of secondhand smoke exposure (CalEPA, 2005). Jaakkola et al. (2003) reported that both cumulative lifetime and recent workplace secondhand smoke exposures increase the risk of adult-onset asthma [odds ratio 2.16 (95% CI, 1.26–3.72)]. These findings may explain the high 24% prevalence of occupational asthma symptoms NIOSH found among Las Vegas dealers (Achutan et al., 2009, in press), which was triple the 7.7% US adult asthma prevalence and the 8.3% Nevada adult asthma prevalence (CDC, 2009; American Lung, 2009).

Finally, the large increases in PPAH on the gaming floors of the smoking casinos compared with outdoors and nonsmoking casinos indicate that secondhand smoke contaminates casino air with high levels of carcinogens. Achutan et al. (2009, in press) observed that the increase in NNAL 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 large increase in NNAL levels in casino patrons and the high levels of NNAL in the body fluids of casino workers are significant because secondhand smoke has been identified as a cause of cancer of the lung, breast, and nasal sinus (Johnson et al., 2011; CalEPA, 2005).

5. Conclusions

Our study provides data on indoor and outdoor exposures for 0.5–8-h visits to 66 smoking casinos and 3 nonsmoking casinos, which constitute 7.6% of all commercial and tribal casinos in the US. Combining data from 66 different US casinos derived from 7 studies in 5 states shows that, due to smoking, indoor concentrations of $PM_{2.5}$ greatly exceed those found outdoors and in nonsmoking casinos, posing risks to both patrons and workers. Secondhand smoke in casinos readily migrates into nonsmoking areas, such as restaurants, not sealed off by closed doors and separate air handling systems. Our combined $PM_{2.5}$ data from 66 California, Delaware, Nevada, New Jersey, and Pennsylvania casinos shows that $PM_{2.5}$ in casino smoking areas averaged 10 times as high as outdoor $PM_{2.5}$. On the other hand, $PM_{2.5}$ concentrations in the smoke-free casinos were slightly less than the low levels measured outdoors. We conclude that eliminating smoking indoors in casinos reduces about 90% of indoor $PM_{2.5}$, bringing indoor levels down to outdoor levels. Similarly, based on data from 8 casinos in 3 states, making casinos smoke-free would reduce carcinogenic PPAH levels at least 80%.

We find that the primary determinant of $PM_{2.5}$ in the smoking areas of casinos is smoker density rather than ventilation. This result indicates that ventilation is an ineffective control for secondhand smoke, while reduction of smoker density is very effective. Our results are consistent with ASHRAE's conclusion that ventilation and air cleaning technologies cannot control the health risks from secondhand smoke in indoor spaces where smoking occurs (ASHRAE, 2008).

Air pollution from secondhand smoke contaminates casino air with high levels of cardiovascular, respiratory, and carcinogenic toxins. There are 49.5 million nonsmoking casino patrons and 427,000 nonsmoking casino workers exposed to secondhand smoke in US commercial and tribal casinos, placing them at increased risk of morbidity and mortality. Making casinos smoke-free will effectively eliminate these risks.

Disclosure statement

J.L. Repace has served as an international secondhand smoke consultant to governmental, non-profit, and private entities, and has been an expert witness in litigation involving casino workers injured by secondhand smoke, casinos, and the tobacco industry. The remaining authors declare no competing interest.

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