Medical Research Archives

European Society of Medicine

OPEN ACCESS

Published: March 31, 2024

Citation: Repace JL, 2024. Forensic Analysis of Lung Cancer from Secondhand Smoke Exposure of a Motel Worker, Medical Research Archives, [online] 12(3). https://doi.org/10.18103/mra.v12i3.5120

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DOI
https://doi.org/10.18103/mra.v12i3.5120

ISSN: 2375-1924

CASE REPORT

Forensic Analysis of Lung Cancer from Secondhand Smoke Exposure of a Motel Worker

James L. Repace, MSc.

Email: repace1@verizon.net

ABSTRACT

Background: In July 2018, Ms. PJM, a 50-year-old lifelong nonsmoker, was diagnosed with primary Stage IV lung adenocarcinoma with secondary metastasis to the brain, after being hospitalized for a fall in the motel where she worked, in Los Angeles, California. She had been employed as a maid cleaning rooms in this motel for 20 years, where she was exposed to secondhand and thirdhand tobacco smoke daily. Her death certificate, dated June 2021, declared metastatic lung cancer to be the cause of her death at age 53. Occupational disability and death claims filed by her attorney were contested by her employer.

Aims: To conduct a forensic analysis using mathematical modeling to quantify Ms. PJM's toxic secondhand and thirdhand smoke exposures, in order to estimate her risk of lung cancer from exposure to tobacco combustion products and other potential carcinogenic agents to which she might have been exposed.

Methods: A quantitative analysis employing assessment of Hazard, Exposure, Dose, Dose-Response, and Risk plus a Discussion of Uncertainty. It is routinely used by U.S. federal regulatory agencies, including the Occupational Safety and Health Administration (OSHA), and the Environmental Protection Agency (EPA).

Results: Ms. PJM's modeled exposure to fine particulate matter from secondhand smoke ranged from the *Hazardous* to *Significant Harm* Levels of the EPA Air Quality Index for fine particles ($PM_{2.5}$). Her modeled dose of serum cotinine ranged from the 90th to beyond the 95th percentile of nonsmokers' dose, measured in a statistical sample of the U.S. nonsmoking population. Her estimated risk exceeds OSHA's *Significant Risk of Material Impairment of Health* Level by a factor of three. She is estimated to have been exposed to the thirdhand smoke of at least 1.4 million cigarettes outgassing from room surfaces during her 20 years of labor. As for potential confounders, there were no known carcinogens in any of the cleaning agents she used; there is no evidence that she was exposed to asbestos, and she resided in a low-radon area of Los Angeles.

Conclusions: As a result of her occupational exposure to secondhand and thirdhand smoke, Ms. PJM lost an estimated 33 years of life expectancy. The State of California has been remiss in failing to extend its workplace smoking ban to hotels and motels, leaving their workstaff at grave risk of the manifold diseases of passive smoking.

Introduction

This is a case study of an occupational exposure to secondhand smoke that caused the premature death from lung cancer and brain cancer in a 53 year old motel maid, Ms. PJM. It demonstrates the forensic utility of quantitative risk assessment in evaluating an occupational injury claim. It discusses the accidental discovery of the lung tumor and its subsequent diagnosis by several physicians. After an occupational death claim was filed by the plaintiff's attorney, the claim was disputed by the defendant's expert medical witness on two grounds: was the tumor a primary or was it a secondary metastasis, and whether the cancer was caused instead by plaintiff's exposure to carcinogens such as asbestos, radon, or cleaning agents she used in her job?

Accordingly, this paper addresses the role of secondhand smoke in carcinogenesis in nonsmokers, using quantitative risk assessment. Quantitative risk assessment is composed of five elements: hazard identification, exposure assessment, dose, dose-response, and risk characterization, plus a discussion of uncertainty. Exposure to secondhand smoke produces an elevation in the risk of lung cancer in the exposed population. The degree of risk elevation for individuals depends upon both the magnitude and duration of their exposure. Ms. PJM's exposure to secondhand smoke while employed by the CI motel, and the probability that Ms. PJM's lung cancer and brain cancer were caused by exposure to secondhand smoke are quantified.

Exposure is quantified by mathematical modeling of the density of fine particulate matter ($PM_{2.5}$) from secondhand smoke (the smoke emitted from the burning end of a cigarette, pipe, or cigar) to which the subject was exposed. Thirdhand smoke arises from the outgassing of secondhand smoke deposits on room surfaces. Dose is quantified by modeling the level of the nicotine metabolite, cotinine, in blood serum. Dose-response is expressed in units of lung cancer deaths per hundred thousand person-years per milligram of tobacco tar inhaled daily. Risk is quantified in units of mortality probability at the age of death, and is compared to federal statistical compilations of mortality from various cancers by age, gender, and ethnicity. Finally, loss of life expectancy is estimated from life tables compiled for the general U.S. population by ethnicity, gender, and age.

Background

In July 2018, Ms. PJM, aged 50 years, employed as a maid cleaning motel rooms in the CI Motel in Los Angeles, CA (Figure 1), was diagnosed with primary Stage IV adenocarcinoma of the lung, with secondary metastasis to the brain. Ms. PJM had been employed there for 20 years. The motel was in the neighborhood of the Los Angeles Airport and also was near the SoFi Sports & Entertainment Stadium, host to the Los Angeles Rams and Chargers football teams. As such, it hosted many out-of-town guests, leading to many transient visitors over the course of a single day, who comprised a majority of daily guests. Room occupancy ranged from 50 to 90 percent. Often rooms were rented by cardplayers who smoked while Ms. PJM cleaned the room. Initially, all rooms were smoking; later it changed to 50% smoking, 50% nonsmoking. Accordingly, many of the motel rooms were contaminated with both secondhand and thirdhand tobacco smoke. So Ms. PJM was exposed to the products of tobacco combustion over a period of 20 years. In June 2021, when Ms. PJM was 53 years old, subsequent to courses of radiotherapy and chemotherapy, she died from primary lung cancer with metastasis to the brain. An occupational injury claim filed before The Workers' Compensation Appeals Board Of The State of California by her attorney in 2018, subsequently morphed into an occupational death claim on behalf of her surviving family. The author was retained as an expert witness in this case by the law firm representing the plaintiff.

PLAINTIFF'S MEDICAL HISTORY:

On December 24th 2017, at age 49, Ms. PJM underwent a CT scan for back pain as a result of a fall at work. The scan was comprehensive, resulting in the discovery of a (3.8 x 3.2 cm) brain tumor compatible with metastatic disease, and a possible lung cancer (suprahilar lung mass of 5.9 cm^3) by Dr. Cynthia Lloyd of the Little Company of Mary Medical Center in Torrance, CA. On 1/26/18, Dr. James J. Yeh of Harbor UCLA Medical Center stated in a progress note, "Patient reports that she had an accident at work where she tripped and fell down. This led to an ED visit where a chest x-ray was done and she was found to have a lung tumor. Which then led to CT of chest, abdomen and pelvis and MRI brain and she was found to have brain metastasis. … Dx: "Patient with new diagnosis of biopsy proven stage IV adenocarcinoma with suprahilar lung mass and right occipital parietal mass, status post subtotal resection at Little Company of Mary. Tx plan: Ordered labs, CT of abdomen, chest and pelvis. F/u in 2 weeks."

Figure 1: The CI Motel, Where Ms. PJM Worked in Los Angeles, California.

On 2/16/18, Dr. An. N. Uche of Harbor UCLA Hematology/Oncology reported that Ms. PJM had a diagnosis of "Dx: Biopsy proven stage IV adenocarcinoma with suprahilar lung mass and right occipital parietal mass." On 3/30/2018, Ms. PJM was diagnosed by Dr. Rex Hoffman of the Disney Family Cancer Center as "A 49-year-old female with metastatic lung cancer to brain for which she is status post subtotal resection of the right parieto-occipital brain metastasis." On 7/03/2018, Ms. PJM, born 4/15/68, was diagnosed as "Permanently and totally disabled" at age 50 by the UCLA Harbor Medical Center. On 12/11/2018, Ms. PJM's medical history was reported by Dr. Venus Vakhshori of the LAC/USC Medical Center as follows: "History of present illness: A 50-year-old female with past medical history of lung cancer with brain metastases status post subtotal resection of brain mass currently on chemotherapy presenting with 3 years of left hip pain acutely worsened over the past 3-4 days. Reported a fall at work, but no new falls."

In December 2019, Dr. Mark M. Ngo, an Agreed Medical Evaluator by both parties to the litigation, reported that Ms. PJM had lytic lesions in the left femur and greater trochanter and underwent a prophylactic nail fixation of her hip due to the risk for pathologic fracture. At some point, she was switched to Tagrisso [a chemotherapeutic agent for non-small-cell lung carcinoma or NSCLC] with subsequent progression of her carcinoma in April 2021. She received radiation therapy with Dr. Chad Sila in May 2021. Ms. PJM's death certificate, dated June 5, 2021, indicated that the primary cause of her death was metastatic lung cancer, and that secondhand smoke was implicated in its causation. At the time of her death, she was 53 years old.

THE MEDICAL-LEGAL DISPUTE:

Attorneys for CI contested the PJM cancer claim. Ms. PJM's medical records were reviewed by the defendant's Medical Expert, Dr. Michael Bronshvag, a medical examiner qualified in internal medicine, who had examined the patient prior to her death. In a report dated April 23, 2019, Dr. Bronshvag noted that: "[The Defendants attorney's] letter takes note of her job duties. It was stated that the claimant developed lung cancer which metastasized to her brain (second-hand smoke). It was stated in the referral letter that the claimant is a 'non-smoker' with 'no family history of cancer.' The motel in question was largely composed of 'smoking rooms.' Her second-hand smoke exposure included cleaning rooms while the occupant was smoking. Exposure to chlorine, Fabulosa, Kaboom, and a pink liquid are mentioned. She cleaned heaters, filters, and fan equipment."

Dr. Bronshvag's review expressed skepticism. He stated that "It is at this time concluded (? correctly?) that the left lung lesion is the 'primary,' and the brain lesion and the left hip lesion are the 'metastases.' Since the issue here is causation of the primary tumor, the issue becomes whether this is indeed a lung primary or a primary 'somewhere else.' " His commentary continued: "The claimant gives at least 'some' if not 'more than some' evidence of exposure to second-hand smoke and other agents. The description of 'adenocarcinoma' requires further contemplation as well." Dr. Bronshvag concluded at that time: "As all will note, the pertinent issues are: 1. Adenocarcinoma rather than squamous cell cancer. 2. Is the lung the 'primary' for the NSCLC? 3. Then, the role of the mentioned toxins, including cigarette smoke, and let's not forget asbestos." The "cancer claim" is currently an issue before the Worker's Compensation Appeals Board of the State of California."

However, Dr. Bronshvag's opinion concerning the brain, hip and lung lesions conflicted with the diagnoses by the four physicians who treated Ms. PJM, as well as raising the issue of other carcinogenic agents. Accordingly, Ms. PJM's attorney requested the Author, a secondhand smoke consultant, to evaluate the probability that Ms. PJM developed this cancer as a result of her exposure to tobacco smoke in her workplace or from other carcinogens. For over 40 years, as a biophysicist, I have conducted original research measuring of, modeling human exposure to, and risk from, secondhand smoke in a wide variety of locations where people live and work. I have explored a diverse set of projects on human exposure to secondhand smoke, ranging from exposure and doses of flight attendants, workers in restaurants, bars, offices, factories, casinos, and in nonsmokers' homes, to levels of smoke in outdoor cafes, on cruise ships at sea, and on college campuses.[1-13] I have also served as an expert witness in other secondhand smoke injury cases.

METHODS:

The methods involve the use of quantitative risk assessment to evaluate this occupational death. It has seven components: determination of hazard, exposure, dose, dose-response, risk, control, and discussion of uncertainty.[6]

Hazard: Secondhand smoke is the combined effluent from the burning ends of a cigarettes, pipes, or cigars, and the smoke exhaled from the smoker. The average cigarette smoker smokes two cigarettes per hour over the course of the day[1,3,4] Cigarette smoke, both actively and passively inhaled, is a known lung carcinogen. The tobacco smoke aerosol is a mixture of more than 4,000 chemical by-products of tobacco combustion, 500 of which are in the gas phase. Of these secondhand smoke byproducts, 172 are known toxic substances, many of which are regulated — except in the nonindustrial indoor air environment, where most exposure takes place. secondhand smoke includes 3 criteria air pollutants and 33 hazardous air pollutants regulated under the Clean Air Act, 47 pollutants that are classified as hazardous wastes whose disposal in solid or liquid form is regulated by the Resource Conservation and Recovery Act, 67 known human or animal carcinogens, and 3 industrial chemicals regulated under the Occupational Health and Safety Act.[3]

Secondhand smoke is a potent and pervasive lung carcinogen. A body of evidence on the health risks of secondhand smoke has been compiled by environmental, occupational, and public health authorities, summarizing research conducted over the past three decades which connects secondhand smoke exposure to premature death from lung cancer and other diseases.

In 2015 The U.S. National Institute for Occupational Safety and Health (NIOSH) issued Current Intelligence Bulletin CIB 67, *Promoting Health and Preventing Disease and Injury through Workplace Tobacco Policies*[24]. NIOSH stated that "there is no risk-free level of exposure to secondhand smoke. Secondhand smoke exposure causes more than 41,000 deaths each year among U.S. nonsmokers. Among exposed adults, there is strong evidence of a causal relationship between exposure to secondhand smoke and a number of adverse health effects, including lung cancer, heart disease (including heart attacks), stroke, exacerbation of asthma, and reduced birth weight of offspring (due to Secondhand smoke exposure of nonsmoking pregnant women). In addition, there is suggestive evidence that exposure to secondhand smoke causes a range of other health effects among adults, including other cancers (breast cancer, nasal cancer), asthma, chronic obstructive pulmonary disease (COPD), and premature delivery of babies born to women exposed to secondhand smoke.

The U.S. National Toxicology Program voted 13:0 to list secondhand smoke as a known carcinogen in its 9th Report on Carcinogens.[25] In 1997, the Environmental Protection Agency of the State of California (CalEPA)[26], in a scientific report reviewing new data since the 1992 EPA report on passive smoking[28], and considering public comments from individuals from federal, state, and local government agencies, universities, and various

research organizations, as well as from the tobacco industry, concluded that in adult nonsmokers, Secondhand smoke exposure in the U.S. population causes lung cancer, plus fatal heart disease and nasal sinus cancer as well[26].

Exposure: Ms. PJM reported that she was a lifelong nonsmoker, and had not been exposed to secondhand smoke as a child, or in her marriage. Her nephew, who lived in a detached building on the same lot was a smoker, but Ms. PJM reported that he was forbidden from smoking in her home due to her son's asthma. However, Ms. PJM asserted that she was occupationally exposed to secondhand smoke and thirdhand smoke in the CI motel where she was employed for 20 years.

In assessing the degree of her secondhand smoke exposure in her workplace, the exposure concentration is estimated using the mass-balance model of Repace et al.[1,3] In this model, which posits the exposure concentration from the ratio of the smoker density to the room air exchange rate, the important variables are the number of habitual smokers N_{hs}, the space volume V (in units of m^3) and the air exchange rate C_v, in units of air changes per hour (hr^{-1}). Habitual smokers are defined as smoking at the national average rate of 2 cigarettes per hour, confirmed by measurements.[3,4] The ratio 100 n_{hs}/V is defined as the habitual smoker density D_{hs}, in units of habitual smokers per hundred cubic meters. Then **R**, the concentration of uniformly-diluted ETS-RSP generated is given by Equation 1, whose units are micrograms per cubic meter ($\mu g/m^3$)

$$\mathbf{R = 217\ D_{hs}/C_v\ ,\ (\mu g/m^3)\ Equation\ 1.}$$

Air exchange rates: According to the California Energy Code, section 120.1, ventilation requirements for hotel guest rooms <500 ft^2 are 30 ft^3/min per room. This is equivalent to (30 ft3/min) (60 min/hour) = 1800 ft^3/hour. The typical room area (double Queen and King Spa) in the CI is 340 ft^2, and with an 8 ft ceiling, the volume is 2720 ft^3, or in metric units, 77 cubic meters (m^3). The area of the smallest King room is 336 ft^3, for a volume of 2688 ft^3, or 76 m^3. Thus the air exchange rates for those rooms would range from C_v = (1800 ft^3/hour)/(2720 ft^3)= 0.66 air changes per hour (h^{-1}) to (1800 ft^3/hour)/(2688 ft^3) = 0.67 h^{-1}. Ms. PJM stated however, that the AC units only recirculated air (PJM, personal communication, 8/1/2019), so this air exchange rate is a conservative value.

Habitual Smoker density: The minimum number of smoking occupants of a smoking room in the CI is Ns = 1. According to Ms. PJM, when there were parties, which occurred four to five times per month, there were up to 6 smokers in a room, N_s = 6. Ms. PJM stated that she would be required to routinely clean 20 rooms daily, of which only 5 were nonsmoking, and on some days had to clean as many as 25 rooms. Her daily hours were 8 per day 5 days per week. Often Ms. PJM cleaned rooms while smokers were still occupying them and actively smoking. She stated that when she entered such a room with active smokers, she would open the door and windows to let the accumulated clouds of smoke out, but the smokers would soon close them, allowing the smoke to build up again (PJM, personal communication, 8/1/2019). When there were no smokers present, Ms. PJM would be exposed to thirdhand smoke evaporating from the contaminated room surfaces.

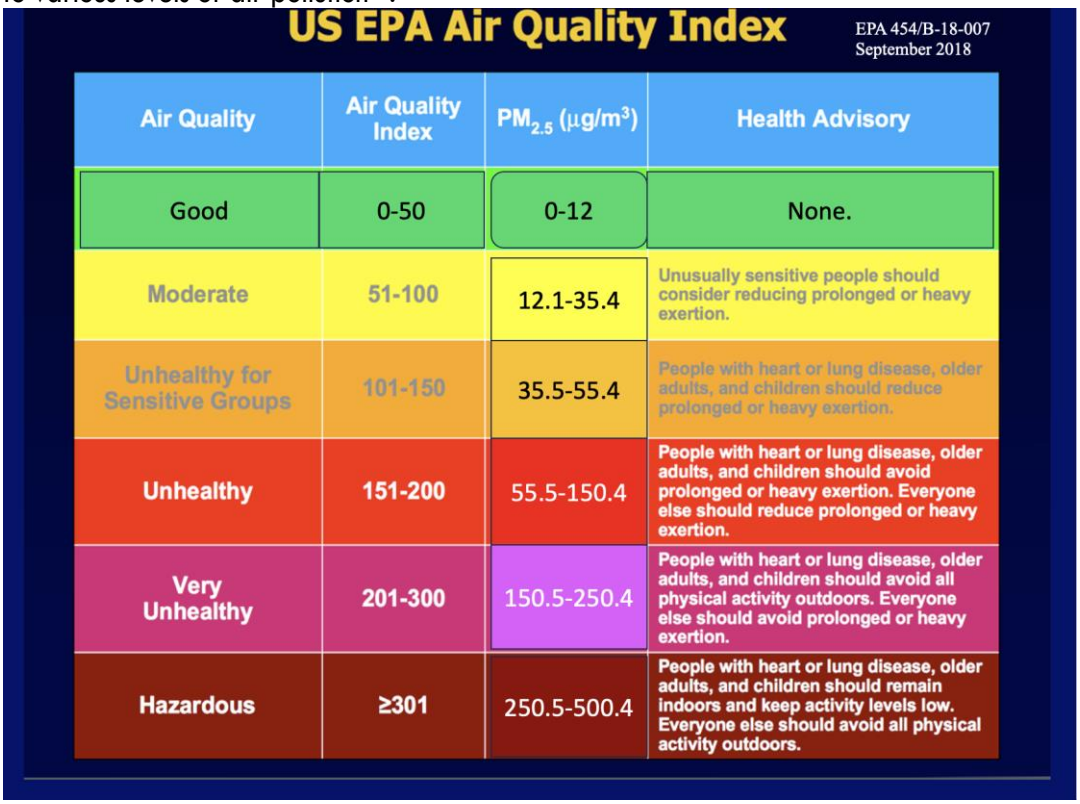
Exposure Duration: Ms. PJM reported that she worked 8 hours per day cleaning 15 smoking and 5 nonsmoking rooms with no breaks. Thus, 75% of the time (15/20)(8hr) = 6 h/d or 30 hours per week, she often worked directly in smoking rooms when people were smoking.

Estimated respirable particulate (RSP) concentration from secondhand smoke: For a single smoker, the smoker density in the 340 ft^2 room of the CI would be Dhs =100 n_{hs}/V = (100)(1)/(77) = 1.30 habitual smokers per 100 m^3. And in the 336 ft^2 room, the smoker density would be Dhs =100 N_{hs}/V = (100)(1)/(76) = 1.32 habitual smokers per 100 m^3. Thus, for the larger room,

R_{340} = 217(1.30)/(0.66) = 427 $\mu g/m^3$, and essentially the same
for the smaller room, R_{336} = 217(1.32)/(0.67) = 428 $\mu g/m^3$.
For six smokers, then, R = (6)(427) = 2562 $\mu g/m^3$.

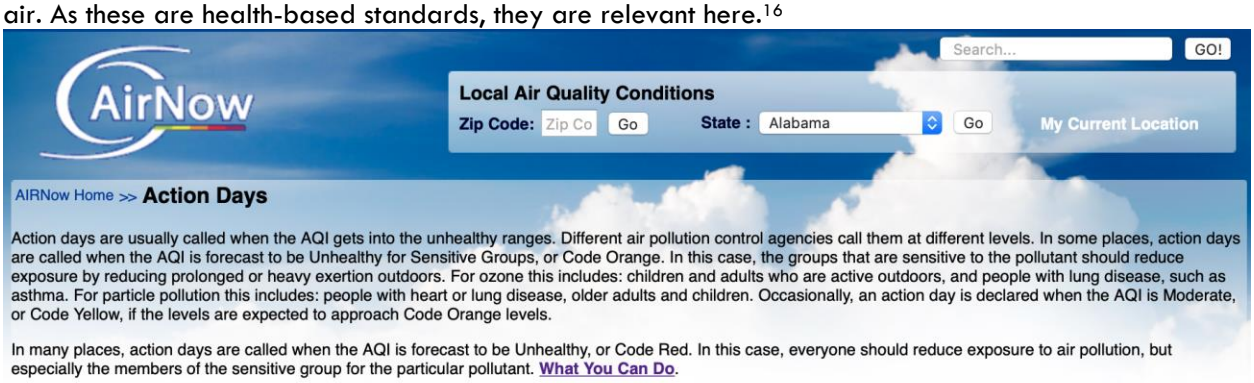
To put these estimated concentrations into perspective, consider the US Air Quality Index for Fine Particulate matter ($PM_{2.5}$), which is a regulated outdoor air pollutant. This is shown in Figure 2: estimated levels of secondhand smoke ($PM_{2.5}$) in CI's smoking guest rooms ranged from 427 to 2562 $\mu g/m^3$. The lower concentration corresponds to Hazardous Air Quality and the upper one to EPA's *Significant Harm* level https://archive.epa.gov/epa/aboutepa/epa-defines-air-pollution-danger-levels.html.

Figure 2. The 2018 U.S. EPA Air Quality Index for $PM_{2.5}$ showing the range of fine particles that correspond to various levels of air pollution[15].



In perspective, as Figure 3 shows, Action Days are usually called when the air quality is Code Orange or beyond. It is clear that the air in the CI's smoking rooms during smoking was polluted to levels that an Alert Day would have been declared in the outdoor air.

Figure 3. Action days are declared when the AQI ascends to the Unhealthy range or beyond in the outdoor air. As these are health-based standards, they are relevant here.[16]



Thirdhand Smoke Exposure:

Thirdhand Smoke is a term describing the residual tobacco smoke pollutants that remain on surfaces and in dust after tobacco has been smoked, are re-emitted into the gas phase, or react with oxidants and other compounds in the environment to yield secondary pollutants. Constituents of this toxic mixture include nicotine, 3-ethenylpyridine (3-EP), phenol, cresols, naphthalene, formaldehyde, and tobacco-specific nitrosamines -- including some not found in freshly emitted tobacco smoke. Building materials and furnishings essentially operate as sinks, reservoirs, or sources for these chemicals. Thirdhand Smoke compounds can remain for extended periods in all indoor environments in which tobacco smoke has been produced. The persistence of thirdhand Smoke in real-world residential settings has been demonstrated based on nicotine and 3-EP concentrations in air, dust, and surfaces in the days, weeks, and months after the last smoking has taken place. Further support comes from quantitative measurements of ultrafine

tobacco smoke particles resuspended after their deposition on household surfaces.[17]

Indoor environments that frequently change ownership or occupancy present the highest risk of involuntary exposure to thirdhand Smoke pollution for occupants. Such environments include hotel rooms, rental apartments, condominiums and houses, and rental and used cars. Because thirdhand smoke exposure is low-level cumulative exposure over long periods, health risks some of the known thirdhand smoke components could affect human health. The chemicals that mediate adverse health consequences can be considered in categories such as irritants, carcinogens, and mutagens (e.g., tobacco-specific nitrosamines, polycyclic aromatic hydrocarbons, heavy metals, nicotine). These may include some of those compounds in secondhand and mainstream smoke as well as new ones not yet directly associated with tobacco smoke. Some of the known carcinogens identified by the International Agency for Research on Cancer (IARC) that are found in mainstream and sidestream smoke are continuously or intermittently present in thirdhand smoke. Tobacco-specific nitrosamines, such as NNK, are potent lung carcinogens, and some of these form from nicotine on indoor surfaces through chemical reactions.[17]

Matt et al.[18] measured thirdhand smoke in a broad range of randomly selected San Diego, CA hotels from March 2009 to February 2010, including: budget small, budget large, midscale small and midscale large. The sample of 30 smoking hotels consisted of 8 budget small, 8 budget large, 7 midscale small and 7 midscale large hotels. The sample of 10 non-smoking hotels consisted of 3 budget small, 3 budget large, 2 midscale small and 2 midscale large. At each smoking hotel, reservations were made for one smoking room and one non-smoking room. At each non-smoking hotel, a reservation was made for one nonsmoking room. Hotels with and without complete smoking bans were investigated to determine whether non-smoking guests staying overnight in these hotels were exposed to tobacco smoke pollutants. Levels of thirdhand smoke in hotels without complete smoking bans had geometric mean levels of air nicotine in smoking rooms (n=28) of 452.4 ng/m^3, ranging from 1.4 to 4302.2 ng/m^3; while nonsmoking rooms (n=28) had geometric mean air nicotine levels of 28.9 ng/m^3, ranging from 0.0 to 463.5 ng/m^3.[18]

When nonsmokers were assigned to designated smoking rooms, a one-night stay led to significantly higher exposure to nicotine as measured by its metabolite, cotinine, found in urine collected during the following day. Urine cotinine levels among nonsmoking confederates staying in a hotel guestroom had (n=28) geometric mean levels of 0.10 ng/ml (range 0.0 -- 0.41 ng/ml) if they stayed in a nonsmoking room, and (n=28) 0.63 ng/ml (range 0.0 – 2.64 ng/ml) if they stayed in a smoking room. Thus, third-hand smoke permeated both smoking and nonsmoking rooms. Matt et el.[18] concluded that: designated smoking rooms are highly polluted with thirdhand smoke and lead to tobacco smoke exposure, including exposure to the potent tobacco-specific lung carcinogen NNK (assessed through measuring its metabolite NNAL in urine).

Matt et al.[18] observed that "The research findings suggest that the existing smoke-free exemptions in California hotels make it virtually impossible to protect a non-smoking guest who stays in a designated smoking room from tobacco smoke exposure—even if no one smokes during their stay. This is because smoking hotel rooms become reservoirs of tobacco smoke toxicants that accumulate in carpets, dust, upholstery, mattresses, curtains and furniture, penetrate wallpaper and paint, and are even stored in drywall. Existing exemptions even make it difficult to protect non-smoking guests who stay in a non-smoking room of a hotel that allows smoking in other rooms. This is because similar to a multi-unit housing building, tobacco smoke cannot be confined to a hotel room but may spread to adjacent and more distant non-smoking rooms, hallways, ventilation systems, windows and utility ducts." This has adverse implications for hotel workers such as maids.

Sheu et al.[57] measured real-time thirdhand smoke off-gassing from smokers themselves (exhaled breath and clothing) into a large nonsmoking movie theater using high-resolution mass spectrometry. Prominent emission events of thirdhand smoke tracers (e.g., 2,5-dimethylfuran, 2-methylfuran, and acetonitrile) and other tobacco-related volatile organic compounds (VOCs) coincided with the arrival of certain moviegoers and left residual contamination. These VOC emissions exposed occupants to the equivalent of 1 to 10 cigarettes of secondhand smoke, including multiple hazardous air pollutants (e.g., the carcinogens benzene and formaldehyde) at parts-per-billion concentrations. Nicotine and related intermediate-volatility nitrogen-containing compounds vaporized from clothes/bodies and recondensed, comprising 34% of the measured organic aerosol. Sheu et al.[57] concluded that exposure to thirdhand smoke VOC emissions would be considerably enhanced in poorly ventilated or smaller spaces, amplifying

concentrations and potential impacts on health and indoor chemistry.

According to the California Office of Health Hazard Evaluation,[19] "The smoke from tobacco products sticks to indoor surfaces such as walls, windows, furniture, and floors. It does not simply blow away. Thirdhand tobacco smoke residue remains in indoor environments, reacts with air to make additional pollutants, and re-emits from surfaces back into the air. It is a distinct public health problem. Thirdhand smoke sticks to skin, hair, and clothing, and can be transferred into environments where smoking is not allowed. Employees and customers in environments where smoking is allowed (such as hotels, casinos, or long-term health facilities that allow smoking indoors) are more likely to be exposed to thirdhand smoke. In hotels with only partial smoking restrictions, thirdhand smoke has been found in both smoking and non-smoking rooms. You can be exposed to thirdhand smoke if you touch a surface on which thirdhand smoke has accumulated, because it can be absorbed through your skin or inhale thirdhand smoke in the air. Some of the chemicals in thirdhand smoke are different from those found in fresh smoke because thirdhand smoke changes over time, becoming progressively more toxic. Thirdhand smoke is a source for long-term exposure to harmful pollutants, which have been shown to damage human cells and DNA, and may be associated with short- and long-term health problems such as asthma and cancer."

Ms. PJM's Estimated occupational exposure to Thirdhand Smoke

Check in time at the CI motel is 3 PM, and check-out time is noon the next day. In the course of this 21-hour stay, it is reasonable to assume that each smoker smokes for 6 hours in the room, at a rate of 2 cigarettes per hour, liberating 14 mg of tobacco tar for each cigarette smoked.[3] Thus, over the course of 6 hours, a dozen cigarettes would be smoked. Then (2 cig/hr)(6 hr)(14 mg/cig) = 168 mg of tobacco tar would be emitted into the room. Over the course of a year, assuming an 80% occupancy rate, (168 mg/day)(0.80)(365 days) = 49,056 mg of tobacco tar, or 49 g, would be emitted into the air of each room occupied by a single smoker. Over the course of the 20 years that Ms. PJM worked, an estimated (20 yr)(49g/yr) = 981 grams of carcinogenic tobacco tar would be released into the air of each smoking room.

And over the course of a year, at 12 cigarettes per day per smoker at an 80% occupancy rate times 365 days yields (12 cig/day)(0.80)(365 days) = 3504 cigarettes would be smoked in that room. Over a 20 year time span, (3504 cig/yr)(20 yr) = 70,080 cigarettes would be smoked per smoker per room. Assuming 65% of the 52 rooms are smoking, as permitted by California law, then approximately (52)(.65) = 34 rooms would be available for smoking. At 70,080 cigarettes per room per smoker times 34 rooms, 2,382,720 cigarettes would have been smoked in the CI motel. And Ms. PJM, who cleaned a minimum of 15 smoking rooms per day, would have been exposed to the thirdhand smoke of at least (20 smoking rooms/34 smoking rooms)(2,382,720 cigarettes) = 1,401,600 cigarettes over the course of 20 years, conservatively excluding her finite exposure in nonsmoking rooms.

At 0.66 room air changes per hour, for a 77 m^3 room, (0.66)(77 m^3) = 50.8 m^3 of air would flow out of the room to be replaced by tobacco-smoke-free outdoor air. Thus, (50.8/77)(981 g) = 674 g of tobacco tar would be lost due to air exchange, and 981 – 674 = 307 g of tobacco tar would be deposited on the walls, floors, and ceilings of that room per smoker, not counting the toxic volatile and semi-volatile organic chemicals in tobacco smoke. If there were no smokers in the room while Ms. PJM cleaned it, she would have breathed just the thirdhand smoke emissions deposited on the room surfaces.

Ms. PJM's exposure to both secondhand and thirdhand smoke while performing her duties 8 hours a day, 5 days a week cleaning smoking rooms in the CI motel exposed her to hazardous levels of air pollution and to potent tobacco-specific carcinogens.

DOSE. Repace et al.[12] developed mathematical models to directly compare secondhand smoke atmospheric markers to each other and to secondhand smoke dosimetric biomarkers, permitting intercomparison of clinical and atmospheric studies. They used atmospheric and pharmacokinetic models for the quantitative estimation of secondhand smoke exposure and dose for infants, children, and adults, based on building smoker density and air exchange rate, and from exposure duration, default pharmacokinetic parameters, and respiration rates. These "Rosetta Stone" Equations allow the secondhand smoke atmospheric markers, respirable particles, nicotine, and carbon monoxide, to be related to the secondhand smoke biomarkers, cotinine in blood, urine, and saliva and nicotine in hair, permitting intercomparison of clinical and atmospheric studies of secondhand smoke dose and exposure. The Rosetta Stone Equations are summarized in Figure 4.

Figure 4. The Rosetta Stone Equations.[12]

JOEM • Volume 48, Number 2, February 2006

TABLE 3

Rosetta Stone Conversion Equations for SHS Atmospheric Biomarker Estimation for Adults (as a Function of Respiration Rate ρ, Daily Hours of Exposure, *H*) and Between Hair Nicotine and Urine Cotinine in Infants

SHS Marker, Units	Conversion Equation
R = RSP, $\mu g/m^3$	R = 10 N
N = Nicotine, $\mu g/m^3$	N = 21.7 D_{hs}/C_v
CO = Carbon monoxide, ppm_{mass}	CO = 0.004 R
PPAH = Particulate polycyclic aromatic hydrocarbons, $\mu g/m^3$	PPAH = R/2000
P = Plasma (Serum) cotinine, ng/mL	*P* = 0.006 ρHN
S = Saliva cotinine, ng/mL	S = 1.16 P
U = Urine cotinine, ng/mL	U = 6.5 P
$\Omega_{infants}$ = Hair nicotine, ng/mg	$\Omega_{infants} \approx 0.7\ U_{infants}$

Note that the units of N in the cotinine equations are $\mu g/m^3$.

D_{hs} indicates smoker density (no. of habitual smokers smoking 14 mg SHS-RSP/cigarette at the rate of 2 cigarettes/hr in the micro environment per 100 m^3 of space volume); C_v, air exchange rate of space volume in air changes per hour (hr^{-1}).

The nicotine metabolite cotinine in blood, urine, and saliva is the pre-eminent biomarker for tobacco smoke exposure. Using the Rosetta Stone Equations, cotinine in blood serum, saliva, and urine can be compared and corresponded with daily exposure to tobacco tar (RSP) from secondhand smoke. For example, the range of saliva cotinine from the Mulcahy study[20] of Irish hotel workers yielded a median dose of 1.6 ng/mL of saliva cotinine with a 25% to 75% range of 0.85 ng/mL to 2.6 ng/mL respectively. Using the equation S = 1.16 P, the serum cotinine level P equivalent to a saliva cotinine level S is given by the equation P = S/1.16 = 0.862 S (ng/mL). Thus the Irish hotel worker serum cotinine equivalent median would be P = (0.862)(1.6 ng/mL) = 1.38 ng/mL, with a 25% to 75% range of (0.862)(0.85 ng/mL) = 0.73 to (0.862)(2.6 ng/mL) = 2.24 ng/mL respectively.

Ms. PJM's equivalent serum cotinine exposure in CI's smoking rooms during smoking from 1 to 6 smokers can be estimated as follows from her modeled exposure to $PM_{2.5}$ as calculated above, which ranged from R = 427 to 2562 $\mu g/m^3$. Using the Rosetta Stone Equation P = (0.006) ρHN, where N=R/10 yields P = (0.006) ρHR/10, where H is the daily duration of exposure, and ρ (rho) is the respiration rate of Ms. PJM during exposure. Her respiration rate can be estimated from Table 1 in Repace et al.[12] which is replicated below as Figure 5. The respiration rate during light activity is 1.0 m^3/hr.

Figure 5. Pharmacokinetic Parameters for the Rosetta Stone Equations (Table 1 in Repace et al.) [12]

TABLE 1

Serum, Urine, and Saliva Cotinine Pharmacokinetic Parameters for Adults, Children, and Infants

Parameter	Adults (19–65+ Years)	Children 6–8 Years	Infants 0–2 Years
α, nicotine lung absorption*	0.71	0.71	0.71
δ_r, renal cotinine clearance*	5.9 mL/min	5.9 mL/min	5.9 mL/min
δ_t, total cotinine clearance*	64 mL/min	64 mL/min	64 mL/min
ϕ, nicotine-cotinine conversion efficiency†	0.78	0.78	0.78
ρ, respiration rate (sedentary)‡	0.5 m^3/hr	0.4 m^3/hr	0.28 m^3/hr
ρ, respiration rate (light activity)‡	1.0	1.0	0.38 m^3/hr
ρ, respiration rate (long-term exposures)‡	females 11.3 m^3/d males 15.2 m^3/d	10 m^3/d	4.5–6.8 m^3/d
V_u, daily urine volume§	1300 mL	800 mL	544 mL
Saliva/Serum conversion efficiency*	1.16	1.16	1.16

Mulcahy et al.[20] investigated the secondhand smoke exposures of Irish hospitality workers, before and after the smoking ban in a cohort of 35 workers from a sample of 15 city hotels (n = 15). The workers were tested for saliva cotinine concentrations and completed questionnaires. Pre-ban Cotinine concentrations for 11 hotel workers who were not waiters or in management (Category Other) had a geometric mean saliva cotinine level of 1.6 nanograms per milliliter (ng/mL) and their 25th to 75th dose percentiles ranged from 0.85 ng/mL to 2.6 ng/mL. Median self-reported exposure to secondhand smoke at work was 30 hours per week. The serum cotinine equivalent of the Irish hotel workers can be calculated from Table 3 in Figure 4: S= 1.16 P, or P = 1.6 ng/mL/1.16 = 1.38 ng/mL. By comparison, from the table in Figure 6, this is between the 90th and 95th percentile for U.S. adults aged over 20 years.

The range of serum cotinine equivalents for Ms. PJM during exposure to secondhand smoke can be calculated as follows. P = (0.006) ρHR/10. Assuming a respiration rate of ρ =1 m^3/h, and H = 6 h/d, then Ms. PJM's estimated cotinine dose corresponding to $PM_{2.5}$ ranging from 427 $\mu g/m^3$ to 2562 $\mu g/m^3$ would be: P = (0.006) ρHR/10 = (0.006)(1)(6)(427/10) = 1.54 ng/mL for exposure to a single smoker, and for exposure to 6 smokers, P = (0.006)ρHR/10 = (0.006)(1)(6)(2562/10) = 9.22 ng/mL. This is put into perspective as follows: The U.S. Centers for Disease Control, Figure 6, published tables of serum cotinine for the U.S. nonsmoking population from 1999 to 2010.[21] As Figure 6 shows, the 95th percentile of dose for US. Females in 1999 was 1.85 ng/mL (95% CI 1.33-2.45), and in 2010 was 1.03 ng/mL (95% CI 0.720-1.61). So Ms. PJM's estimated cotinine dose from secondhand smoke, ranging between 1.53 ng/mL to 9.22 ng/mL. By comparison, the 90th percentiles of serum cotinine for the U.S. population during 1999-2010 range from 0.850 ng/mL to 0.380 ng/mL. Figure 7 shows the percent of the U.S. nonsmoking population at a given cotinine dose from a national survey (NHANES III) with Ms. PJM's estimated dose range indicated. In other words, Ms. PJM's secondhand smoke exposure while cleaning in a room full of smokers was extreme and placed her at great risk of the diseases of secondhand smoke exposure, or passive smoking. Epidemiological studies tend to underestimate the risk of secondhand smoke due to the paucity of truly unexposed persons, as shown in Figure 7.[1,3,4]

DOSE-RESPONSE. Repace and Lowrey[6] developed a phenomenological exposure-response relationship, 5 lung cancer deaths(LCDs) per year per 100,000 persons exposed, per mg daily tar exposure. This relationship yielded modeled lung cancer mortality rates and mortality ratios for a U.S. cohort within 5% with the results of two large prospective epidemiological studies of passive smoking and lung cancer in the United States and Japan.

This exposure-response relationship can be applied to estimate the risk increase that Ms. PJM experienced while cleaning multiple rooms in the motel that were occupied by a single active smoker over a 6-hour exposure day. As discussed earlier, the secondhand smoke concentration she would have encountered was 427 micrograms per cubic meter ($\mu g/m^3$). Over a 6 hour period, with a respiration rate of 1 m^3/hr, Ms. PJM would have inhaled a dose of tobacco tar of D = (6 hr/d)(1m^3/hr)(427 $\mu g/m^3$) = 2.562 mg/day. Applying this to the exposure-response relationship of Repace and Lowrey (1985) yields an excess LCD risk due to secondhand smoke exposure of (5 x10^{-5})[LCDs/yr-mg/day] x 2.562 [mg/day] = 12.81 x 10^{-5} LCDs/yr. Since Ms. PJM was exposed for 20 years, her estimated risk increase over this period is (12.81 x 10^{-5} LCDs/yr)(20 yr) = 2.6 x10^{-3}. By comparison, the background risk of lung cancer for Hispanic Females Birth to Age 49 from Figure 8, Table 2, is 1/1208 = 0.8 x 10^{-3}. In other words, Ms. PJM's risk increase from exposure to the smoke of a single smoker 6 hours daily for 20 years while cleaning rooms of active smokers is tripled over background: (2.6 x 10^{-3}/0.8 x 10^{-3}) = 3.25.

RISK. In 1994, The U.S. Occupational Safety and Health Administration (OSHA, 1994) proposed a rule to eliminate nonsmokers' secondhand smoke exposures in the workplace.[27] OSHA estimated that as many as 722 U.S. workers would die annually from passive-smoking-induced lung cancer. In 1992, the U.S. Environmental Protection Agency[28] declared secondhand smoke to be a known human lung carcinogen, causing conservatively 3000 lung cancer deaths (LCDs) annually. In 1991, NIOSH (CIB 54) "considers secondhand smoke to be a potential occupational carcinogen and recommends that exposures be reduced to the lowest feasible concentration. All available preventive measures should be used to minimize occupational exposure to secondhand smoke. NIOSH urges employers to disseminate this information to employees[27]."

Figure 6. Serum Cotinine Levels For U.S. Nonsmokers. Fourth Report on Environmental Chemicals, US Centers For Disease Control & Prevention, Updated Tables, 2019.[21]

Serum Cotinine (1999 – 2010)

Metabolite of nicotine (component of tobacco smoke)

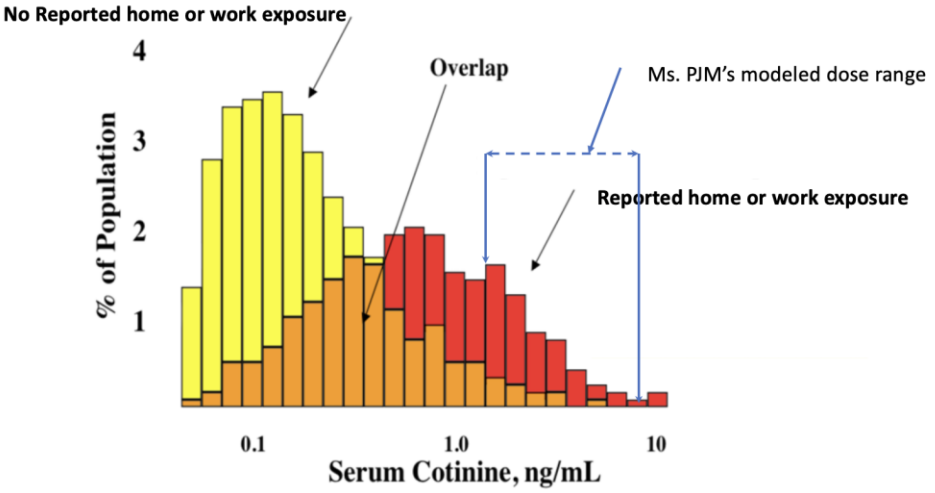
Geometric mean and selected percentiles of serum concentrations (in ng/mL) for the ***non-smoking** U.S. population from the National Health and Nutrition Examination Survey.

	Survey years	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval) 50th	75th	90th	95th	Sample size
Total	99-00	*	**.060** (<LOD-.080)	**.240** (.190-.302)	**1.02** (.770-1.28)	**1.96** (1.60-2.62)	5999
	01-02**	**.062** (.050-.077)	**< LOD**	**.160** (.120-.220)	**.930** (.740-1.17)	**2.20** (1.83-2.44)	6819
	03-04	**.071** (.057-.089)	**.050** (.040-.070)	**.210** (.140-.310)	**.990** (.740-1.30)	**2.17** (1.81-2.54)	6320
	05-06	**.054** (.047-.061)	**.040** (.030-.040)	**.120** (.100-.150)	**.630** (.460-.870)	**1.47** (1.15-1.92)	6347
	07-08	**.057** (.048-.068)	**.040** (.030-.040)	**.130** (.100-.180)	**.760** (.550-1.09)	**1.81** (1.45-2.43)	6197
	09-10	**.041** (.037-.046)	**.030** (.020-.030)	**.070** (.070-.090)	**.450** (.350-.580)	**1.29** (1.04-1.61)	6678
Age group							
3-11 years	99-00	**.164** (.115-.234)	**.110** (.066-.188)	**.500** (.260-1.16)	**1.88** (.997-3.44)	**3.44** (1.42-4.79)	1174
	01-02**	**.110** (.076-.160)	**.070** (<LOD-.130)	**.570** (.310-1.00)	**2.23** (1.63-2.78)	**3.23** (2.53-4.01)	1415
	03-04	**.137** (.088-.213)	**.120** (.060-.220)	**.620** (.310-1.20)	**2.04** (1.38-2.94)	**3.35** (2.12-4.68)	1252
	05-06	**.078** (.062-.097)	**.050** (.040-.070)	**.220** (.160-.350)	**1.22** (.880-1.82)	**2.42** (1.63-3.46)	1296
	07-08	**.095** (.067-.136)	**.060** (.040-.100)	**.380** (.170-.840)	**1.67** (1.10-2.54)	**2.81** (2.26-3.54)	1337
	09-10	**.060** (.047-.075)	**.040** (.030-.050)	**.160** (.110-.250)	**.920** (.560-1.58)	**2.15** (1.35-3.00)	1355
12-19 years	99-00	**.163** (.142-.187)	**.110** (.080-.163)	**.540** (.428-.660)	**1.66** (1.50-1.95)	**2.62** (2.09-3.39)	1773
	01-02**	**.086** (.059-.126)	**.050** (<LOD-.110)	**.350** (.190-.580)	**1.53** (1.09-2.12)	**3.12** (2.47-3.99)	1902
	03-04	**.110** (.087-.139)	**.080** (.060-.120)	**.510** (.350-.670)	**1.55** (1.21-1.93)	**2.68** (1.96-4.02)	1783
	05-06	**.074** (.060-.092)	**.050** (.040-.060)	**.230** (.150-.350)	**1.16** (.860-1.69)	**2.26** (1.69-2.72)	1714
	07-08	**.081** (.061-.106)	**.050** (.030-.070)	**.350** (.170-.550)	**1.25** (.930-1.99)	**2.54** (2.04-2.94)	934
	09-10	**.056** (.044-.072)	**.030** (.020-.040)	**.130** (.080-.230)	**.980** (.510-1.80)	**2.49** (1.31-3.65)	1042
20 years and older	99-00	*	**.050** (<LOD-.061)	**.167** (.140-.193)	**.630** (.533-.820)	**1.50** (1.28-1.66)	3052
	01-02**	**.052** (<LOD-.063)	**< LOD**	**.110** (.090-.150)	**.630** (.470-.790)	**1.42** (1.14-1.89)	3502
	03-04	**.058** (.047-.071)	**.040** (.030-.050)	**.140** (.100-.200)	**.630** (.480-.840)	**1.54** (1.26-1.92)	3285
	05-06	**.047** (.042-.053)	**.030** (.030-.040)	**.100** (.080-.120)	**.440** (.310-.620)	**1.14** (.870-1.41)	3337
	07-08	**.049** (.043-.057)	**.030** (.030-.040)	**.100** (.080-.120)	**.490** (.390-.640)	**1.37** (.970-1.70)	3926
	09-10	**.037** (.034-.040)	**.020** (.020-.030)	**.070** (.060-.070)	**.310** (.240-.390)	**.990** (.700-1.24)	4281
Gender							
Males	99-00	**.124** (.106-.145)	**.080** (.060-.110)	**.308** (.220-.410)	**1.20** (.950-1.49)	**2.39** (1.66-3.22)	2789
	01-02**	**.075** (.059-.094)	**.050** (<LOD-.070)	**.230** (.160-.320)	**1.17** (.960-1.49)	**2.44** (2.23-2.99)	3152
	03-04	**.087** (.070-.108)	**.060** (.040-.080)	**.280** (.190-.360)	**1.23** (.910-1.68)	**2.63** (2.09-3.19)	2937
	05-06	**.064** (.055-.074)	**.040** (.040-.050)	**.150** (.120-.190)	**.720** (.550-1.07)	**1.85** (1.42-2.24)	2922
	07-08	**.068** (.056-.081)	**.040** (.040-.050)	**.170** (.120-.240)	**1.07** (.690-1.39)	**2.52** (1.84-2.92)	2948
	09-10	**.046** (.042-.051)	**.030** (.030-.030)	**.080** (.070-.100)	**.550** (.410-.700)	**1.60** (1.19-2.38)	3181
Females	99-00	*	**< LOD**	**.180** (.148-.230)	**.850** (.600-1.14)	**1.85** (1.33-2.45)	3210
	01-02**	**.053** (<LOD-.066)	**< LOD**	**.120** (.090-.180)	**.710** (.540-.990)	**1.77** (1.32-2.20)	3667
	03-04	**.060** (.047-.077)	**.040** (.030-.060)	**.160** (.110-.260)	**.860** (.580-1.15)	**1.76** (1.32-2.22)	3383
	05-06	**.047** (.040-.054)	**.030** (.030-.040)	**.100** (.070-.130)	**.510** (.300-.830)	**1.23** (1.04-1.52)	3425
	07-08	**.050** (.042-.059)	**.030** (.030-.040)	**.110** (.080-.150)	**.630** (.400-.900)	**1.40** (1.09-1.87)	3249
	09-10	**.037** (.033-.042)	**.020** (.020-.030)	**.070** (.050-.080)	**.380** (.270-.570)	**1.03** (.720-1.61)	3497

Limit of detection (LOD, see Data Analysis section) for Survey years 99-00, 01-02, 03-04, 05-06, 07-08, and 09-10 are 0.05, 0.05, 0.015, 0.015, 0.015, and 0.015 respectively. .
< LOD means less than the limit of detection, which may vary for some chemicals by year and by individual sample.
* Not calculated: proportion of results below limit of detection was too high to provide a valid result.
**In the 2001-2002 survey period, 83% of measurements had an LOD of 0.015 ng/mL, and 17% had an LOD of 0.05 ng/mL.
***Non-smoking is defined as a serum cotinine concentration of 10 ng/mL or less.

Biomonitoring Summary: http://www.cdc.gov/biomonitoring/Cotinine_BiomonitoringSummary.html
Factsheet: http://www.cdc.gov/biomonitoring/Cotinine_FactSheet.html

Figure 7. Frequency Histogram of Serum Cotinine for the U.S. Population in 1996.[3,22] Ms. PJM's modeled dose ranges from 1.53 to 9.22 ng/mL, beyond the 90th percentile of the cotinine distribution for U.S. nonsmokers. The boundary between passive and active smoking is 10 ng/mL. This figure also illustrates why some of the early epidemiological studies of lung cancer from passive smoking failed to detect statistically significant results, due to the large fraction, 40%, of nonsmokers who reported having "no exposure" to secondhand smoke on a questionnaire, but manifesting finite cotinine doses.



Repace and Lowrey[29] reviewed ten published risk assessments of SHS and lung cancer in the peer-reviewed scientific literature, and reported that the estimates averaged 5000 ± 2500 LCDs annually, a contribution to cancer risk which is 57 times greater than the impact of all regulated outdoor air pollutants combined, and 25% higher than indoor radon gas. In 1986, both the Surgeon General and the National Research Council issued comprehensive reports indicating that secondhand smoke was a cause of lung cancer in nonsmokers. In 1985, Repace and Lowrey[6] estimated that "Aggregate exposure to ambient tobacco smoke is estimated to produce about 5000 lung cancer deaths per year in U.S. nonsmokers aged ≥35 yr, with an average loss of life expectancy of 17 ± 9 yr per fatality. The estimated risk to the most-exposed passive smokers appears to be comparable to that from pipe and cigar smoking. Mortality from passive smoking is estimated to be about two orders of magnitude higher than that estimated for carcinogens currently regulated as hazardous air pollutants under the federal Clean Air Act." Repace and Lowrey estimated that one-third of lung cancer deaths were attributable to passive smoking.[29]

To put the lung cancer estimate of Repace and Lowrey[6] in perspective, according to Thun et al.[23] (2006), who analyzed data from two large American Cancer Society Cancer Prevention Study cohorts during 1959 – 1972 (CPS-I) and 1982 – 2000 (CPS-II), approximately 85% – 90% of all lung cancer deaths in the United States have been caused by active cigarette smoking. The remaining 10% – 15% represent between 17 000 and 26 000 deaths annually, a number that would rank among the six to eight most common fatal cancers in the United States if considered as a separate category. An estimated 15 000 lung cancer deaths caused by factors other than active cigarette smoking occur in lifelong non-smokers; the rest are combined with and statistically indistinguishable from the much larger number caused by cigarette smoking among current and former smokers. Known causes of lung cancer other than cigarette smoking include secondhand smoke, active smoking of other tobacco products, and exposure to other carcinogens such as asbestos, radon, radiation therapy, combustion products, and various other exposures in occupational, environmental, and/or medical settings."

Cancer incidence and mortality for Hispanics by gender in the US estimated for 2018 is given in Table 8. These are not differentiated by smoking status. Brain cancer in Latinas is not frequent enough to be listed separately (Figures 8a,b).[30] Figure 8a shows 5,000 deaths in 2018 for Hispanic females, 6% of total estimated new cases.

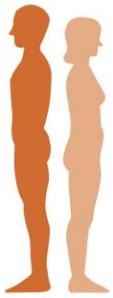
Figure 8b shows that the probability of developing lung cancer for a 49 yr. old Hispanic female is 1 in 1,208, or 0.08%.

Figure 8a, ACS Cancer Facts & Figures for Hispanics/Latinos 2015-2017.[30]

Figure 2. Leading Sites of New Cancer Cases and Deaths Among Hispanics – 2018 Estimates

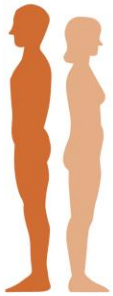
Estimated New Cases*

Males			Females		
Prostate	13,900	21%	Breast	24,000	29%
Colon & rectum	7,900	12%	Thyroid	6,800	8%
Lung & bronchus	5,600	8%	Uterine corpus	6,700	8%
Kidney & renal pelvis	4,800	7%	Colon & rectum	6,500	8%
Liver & intrahepatic bile duct	4,500	7%	Lung & bronchus	5,000	6%
Non-Hodgkin lymphoma	3,900	6%	Non-Hodgkin lymphoma	3,500	4%
Leukemia	3,200	5%	Kidney & renal pelvis	3,200	4%
Urinary bladder	2,900	4%	Leukemia	2,500	3%
Pancreas	2,300	3%	Ovary	2,500	3%
Stomach	2,200	3%	Uterine cervix	2,400	3%
All sites	67,400	100%	All sites	81,700	100%



Estimated Deaths

Males			Females		
Lung & bronchus	3,500	16%	Breast	3,200	16%
Liver & intrahepatic bile duct	2,700	12%	Lung & bronchus	2,600	13%
Colon & rectum	2,400	11%	Colon & rectum	1,800	9%
Prostate	2,000	9%	Pancreas	1,600	8%
Pancreas	1,700	8%	Liver & intrahepatic bile duct	1,300	6%
Stomach	1,100	5%	Ovary	1,100	5%
Leukemia	1,000	5%	Uterine corpus	1,000	5%
Non-Hodgkin lymphoma	1,000	4%	Leukemia	900	4%
Kidney & renal pelvis	900	4%	Stomach	800	4%
Brain & other nervous system	700	3%	Non-Hodgkin lymphoma	700	3%
All sites	22,300	100%	All sites	20,400	100%



*Estimates are rounded to the nearest 100 and exclude basal and squamous cell skin cancers and in situ carcinoma except urinary bladder. Rankings are based on modeled projections and may differ from the most recent observed data. Estimates exclude Puerto Rico.

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Figure 8b, ACS Cancer Facts & Figures for Hispanics/Latinos 2015-2017.[30]

Table 1. Probability (%) of Developing Invasive Cancer among Hispanics/Latinos during Selected Age Intervals by Sex, US, 2010-2012*

		Birth to 49	50 to 59	60 to 69	70 and Older	Birth to Death
All sites†	Male	2.6 (1 in 39)	4.6 (1 in 22)	11.3 (1 in 9)	32.8 (1 in 3)	38.8 (1 in 3)
	Female	4.4 (1 in 23)	4.8 (1 in 21)	8.0 (1 in 13)	24.2 (1 in 4)	34.4 (1 in 3)
Breast	Female	1.5 (1 in 68)	1.8 (1 in 55)	2.6 (1 in 38)	5.1 (1 in 20)	9.8 (1 in 10)
Colon & rectum	Male	0.3 (1 in 386)	0.6 (1 in 175)	1.1 (1 in 87)	3.7 (1 in 27)	4.7 (1 in 21)
	Female	0.2 (1 in 416)	0.5 (1 in 219)	0.8 (1 in 131)	3.1 (1 in 32)	4.1 (1 in 24)
Liver & intrahepatic bile duct	Male	0.1 (1 in 1,009)	0.4 (1 in 228)	0.7 (1 in 149)	1.4 (1 in 69)	2.2 (1 in 44)
	Female	<0.1 (1 in 3,466)	0.1 (1 in 1,078)	0.2 (1 in 456)	0.8 (1 in 124)	1.0 (1 in 97)
Lung & bronchus	Male	0.1 (1 in 1,406)	0.3 (1 in 370)	0.9 (1 in 107)	4.6 (1 in 22)	4.8 (1 in 21)
	Female	0.1 (1 in 1,208)	0.2 (1 in 421)	0.7 (1 in 143)	3.0 (1 in 33)	3.6 (1 in 28)
Non-Hodgkin lymphoma	Male	0.2 (1 in 443)	0.3 (1 in 383)	0.5 (1 in 191)	1.8 (1 in 54)	2.4 (1 in 42)
	Female	0.2 (1 in 604)	0.2 (1 in 488)	0.4 (1 in 254)	1.4 (1 in 69)	2.0 (1 in 50)
Prostate	Male	0.2 (1 in 574)	1.4 (1 in 73)	4.7 (1 in 21)	9.9 (1 in 10)	13.0 (1 in 8)
Stomach	Male	0.1 (1 in 1,099)	0.2 (1 in 644)	0.3 (1 in 294)	1.5 (1 in 68)	1.7 (1 in 59)
	Female	0.1 (1 in 1,117)	0.1 (1 in 923)	0.2 (1 in 471)	0.9 (1 in 108)	1.2 (1 in 83)
Thyroid	Male	0.1 (1 in 886)	0.1 (1 in 1,100)	0.1 (1 in 787)	0.2 (1 in 482)	0.5 (1 in 210)
	Female	0.7 (1 in 154)	0.3 (1 in 304)	0.3 (1 in 315)	0.5 (1 in 211)	1.7 (1 in 60)
Uterine cervix	Female	0.3 (1 in 324)	0.2 (1 in 639)	0.2 (1 in 619)	0.3 (1 in 298)	0.9 (1 in 111)

*For those who are free of cancer at beginning of each age interval. †All sites excludes basal and squamous cell skin cancers and in situ cancers except urinary bladder.

Source: DevCan: Probability of Developing or Dying of Cancer Software, Version 6.7.3. Statistical Research and Applications Branch, National Cancer Institute, 2015. http://surveillance.cancer.gov/devcan/.

American Cancer Society, Inc., Surveillance Research, 2015

As Figure 9 indicates, there is a huge difference in lung cancer mortality between female smokers and female nonsmokers. This is an indication of the profound carcinogenicity of tobacco smoke exposure. There is also substantial evidence that the dose-response curve from passive to active smoking is non-linear and very steep at low doses.[5] The lung cancer vs. age data for nonsmokers is plotted below in Figure 10 from NCI data[31].

Discussion:

I have analyzed Ms. PJM's lung cancer as a function of her employment at CI in three ways: in terms of exposure, dose, and risk from secondhand smoke. I have modeled Ms. PJM's secondhand smoke exposure concentrations in the rooms where she worked while anywhere from one to as many as six smokers were actively smoking. My estimates range from 427 µg/m^3 to 2562 µg/m^3. To put this in perspective, judged by the US Air Quality Index for $PM_{2.5}$, this ranges from Hazardous to Significant Harm levels of fine particle air pollution. Repetitive exposures of this nature to such high levels of air pollution are dangerous.

Travers et al.[48] measured RSP in 14 smoky bars and restaurants in Western New York State in 2003. The mean before a state-wide smoking ban was 412 µg/m^3. Post-ban, the levels had declined to 27 µg/m^3, indicated that 90% of the RSP was due to smoking. So one smoker in the small volume of a poorly ventilated motel room was as polluted as a smoky bar or restaurant.

Figure 9. Mortality from lung cancer as underlying cause of death among lifelong nonsmokers and current cigarette smokers: Women (1982-2000), Cancer Prevention Study II, Smoking and Tobacco Control Monograph No. 8, National Cancer Institute, Chapter 5, Table 6.[31]

Smoking and Tobacco Control Monograph No. 8

Table 6

Mortality from lung cancer as underlying cause of death among lifelong nonsmokers and current cigarette smokers: Women, Cancer Prevention Study II

Age Specific

	Nonsmokers		Current Cigarette Smokers			
Age	Deaths	Rate[a]	Deaths	Rate[a]	RR	RD[a]
35-39	1	2.0	1	4.0	–	2.0
40-44	0	–	4	8.9	–	8.9
45-49	4	1.9	43	42.4	22.1	40.5
50-54	18	5.8	93	64.7	11.3	58.9
55-59	25	7.2	175	119.9	16.6	112.7
60-64	42	12.3	215	176.6	14.3	164.3
65-69	47	16.7	232	286.3	17.1	269.5
70-74	63	30.5	142	310.0	10.2	279.5
75-79	44	32.5	77	400.0	12.3	367.5
80+	41	57.6	24	417.6	7.3	360.0
Total	285		1,006			

Age Standardized to 1980 U.S. Population

	Nonsmokers	Current Cigarette Smokers
Death Rate[a]	8.6	101.3
Rate Ratio	1.0	11.9
(95% CI)	–	(10.1-13.7)
Rate Difference[a]	–	92.7
(95% CI)	–	(83.7-101.7)

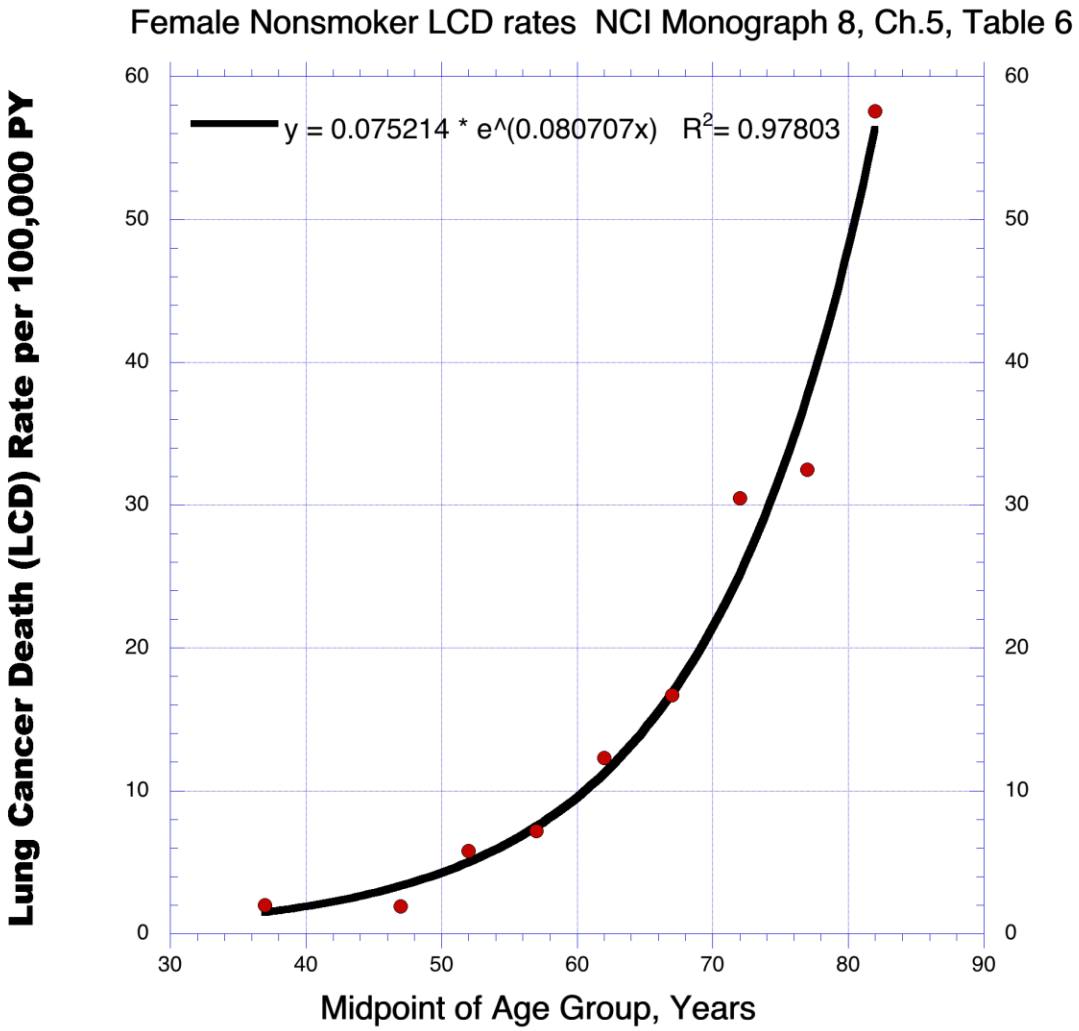
[a] Death rate and rate difference per 100,000 person-years.

Key: RR = rate ratio; RD = rate difference; CI = confidence interval.

The graph in Figure 10 shows that the probability of a 49 year-old nonsmoking woman of contracting lung cancer is very small, of approximately 4 cases per 100,000 women.[31]. How might this baseline risk be increased as a result of Ms. PJM's secondhand and thirdhand smoke exposure?

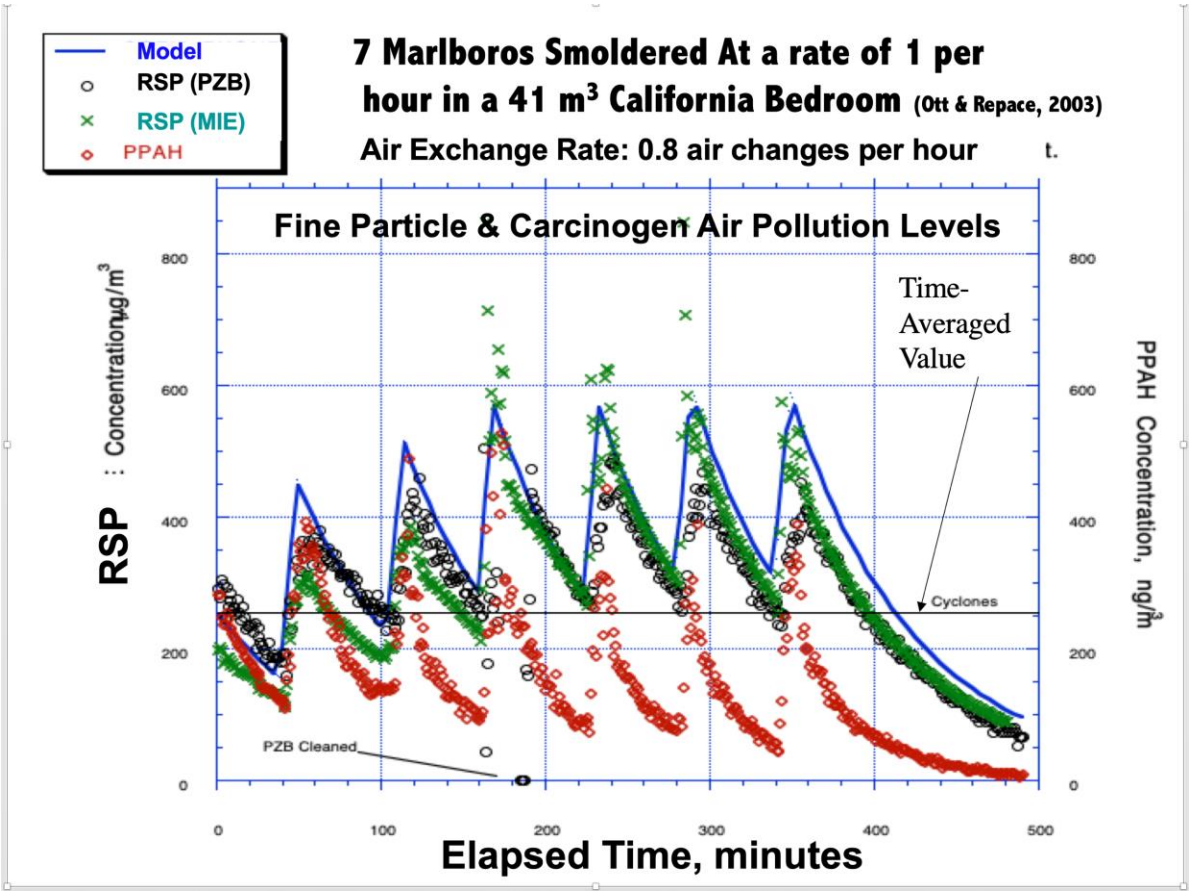
Real-time measurements of RSP and carcinogenic particle-bound polycyclic aromatic hydrocarbons (PPAH) from 7 Marlboro cigarettes smoked in a 43 m^3 room at the rate of 1 per hour, half of the average rate for a smoker. Levels of RSP peaked at over 600 µg/m^3 and averaged out at about 250 µg/m^3 above background, while carcinogenic particulate polycyclic aromatic hydrocarbons (PPAH) averaged about 250 ng/m^3 (Figure 11).[3] Repace[2] measured RSP in a casino, six bars, and a pool hall in Wilmington DE before and after a state-wide smoking ban. Secondhand smoke contributed 90 to 95% of the RSP and carcinogenic PAH.

Figure 10. The graph indicates that the rate of lung cancer for the age group 45-49 is about 4 per 100,000 person-years (data source: NCI Monograph 8.)[30]



At the upper limit with 6 smokers in the same 340 m^2 (77 m^3) CI room, I estimated a concentration of 2562 µg/m^3. Repace and Lowrey[1] (1980) measured real-time RSP in a 113 m^3 mechanically ventilated office building conference room with 4 chain smokers at nearly 2000 µg/m^3, with a non-smoking background of 57 µg/m^3. In a controlled experiment, Dacunto et al.[34] conducted real-time measurements of RSP from 6 Marlboro cigarettes at a rate of one cigarette per hour, generating secondhand smoke in a 77 m^3 Pacific Inn motel room in Redwood City, CA, as shown in Figure 12. Dacunto et al. (unpublished report) measured secondhand smoke RSP in at levels approaching 2000 µg/m^3, within 10 minutes after doors were closed (Figure 12). The air exchange rate was 0.263 h^{-1}. Thus, the modeled level for the CI motel room in which Ms. PJM worked is realistic.

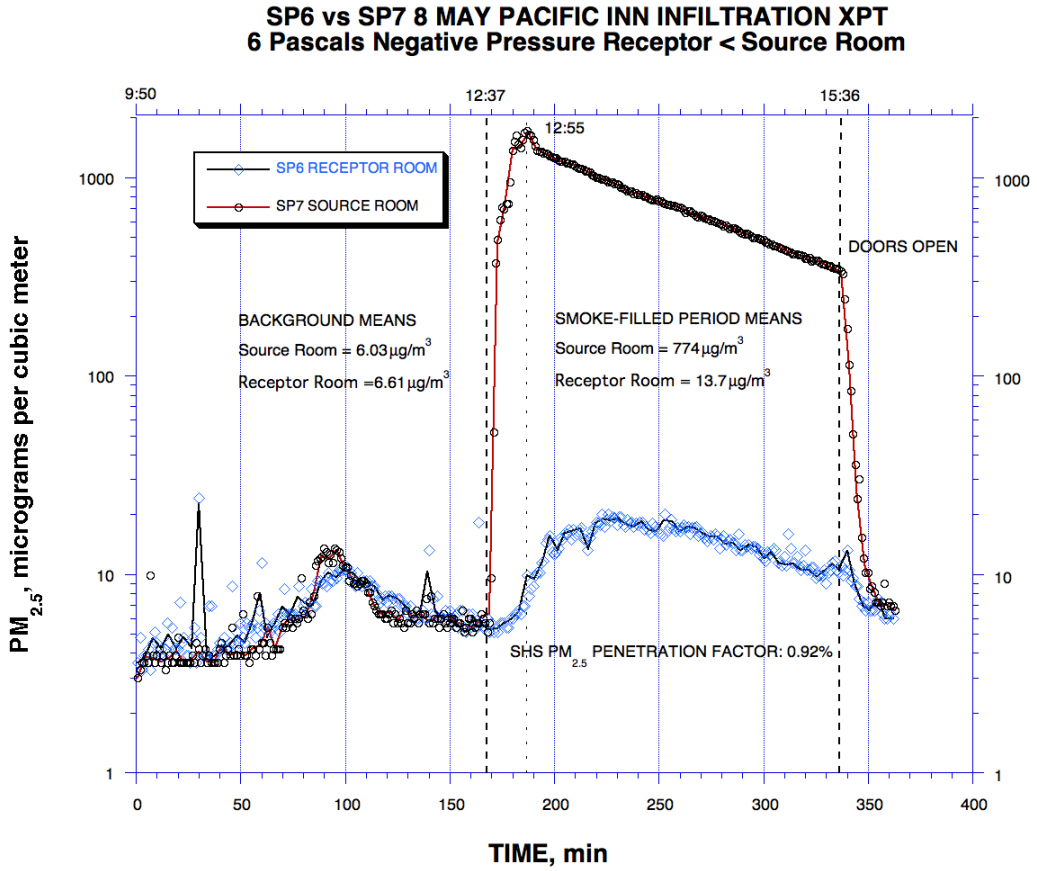
Figure 11. Respirable particles (RSP)and carcinogenic polycyclic aromatic hydrocarbons (PPAH) from 7 Marlboro cigarettes smoked at the rate of 1 per hour in a 43 m^3 room with an air exchange rate of 0.8 h^{-1}. A typical cigarette smoker smokes at the rate of 2 cig/hr. [1,3]



Further, Ms. PJM was exposed to thirdhand smoke in every room that she cleaned. Thirdhand smoke contains potent tobacco-specific nitrosamines and other toxic and carcinogenic chemicals, some of which are not present in SHS. In other words, from either direct secondhand smoke or indirect thirdhand smoke, Ms. PJM inhaled tobacco smoke carcinogens in every room that she cleaned every working day for 20 years. As the California Office of Health Hazard Assessment has stated[119], "Thirdhand smoke (THS) is a source for long-term exposure to harmful pollutants, which have been shown to damage human cells and DNA, and may be associated with short- and long-term health problems such as asthma and cancer."

Figure 12 shows actual measurements of secondhand smoke in a California motel room where 6 cigarettes were smoked in 3 hours (source room), and secondhand smoke infiltrated into a neighboring nonsmoking room. The air exchange rate was 0.263 h^{-1}, and scaled to the air exchange rate estimated for the Crystal Suites of 0.665 h^{-1}, produces an estimated SHS $PM_{2.5}$ concentration of (774) (0.263/0.665) = 306 µg/m^3 with one smoker smoking 2 cigarettes per hour, and 1836 µg/m^3 with 6 smokers puffing away.

Figure 12. $PM_{2.5}$ vs. time, respectively in adjacent smoking and nonsmoking rooms in the Pacific Inn in Redwood City, CA. The receptor room is under 6 Pascals of negative pressure; SHS is elevated in Receptor room for about 3 hours after a 6-cigarette smoking episode. Peak level of tobacco tar particles ($PM_{2.5}$) was attained after 10 minutes of smoking. The air exchange rate in this 77 m^3 room was 0.263 h^{-1} (J.L. Repace, unpublished data from Dacunto et al.[34] This is the same volume as many of the CI's motel rooms. Six pascals is a typical pressure difference encountered in residential housing.



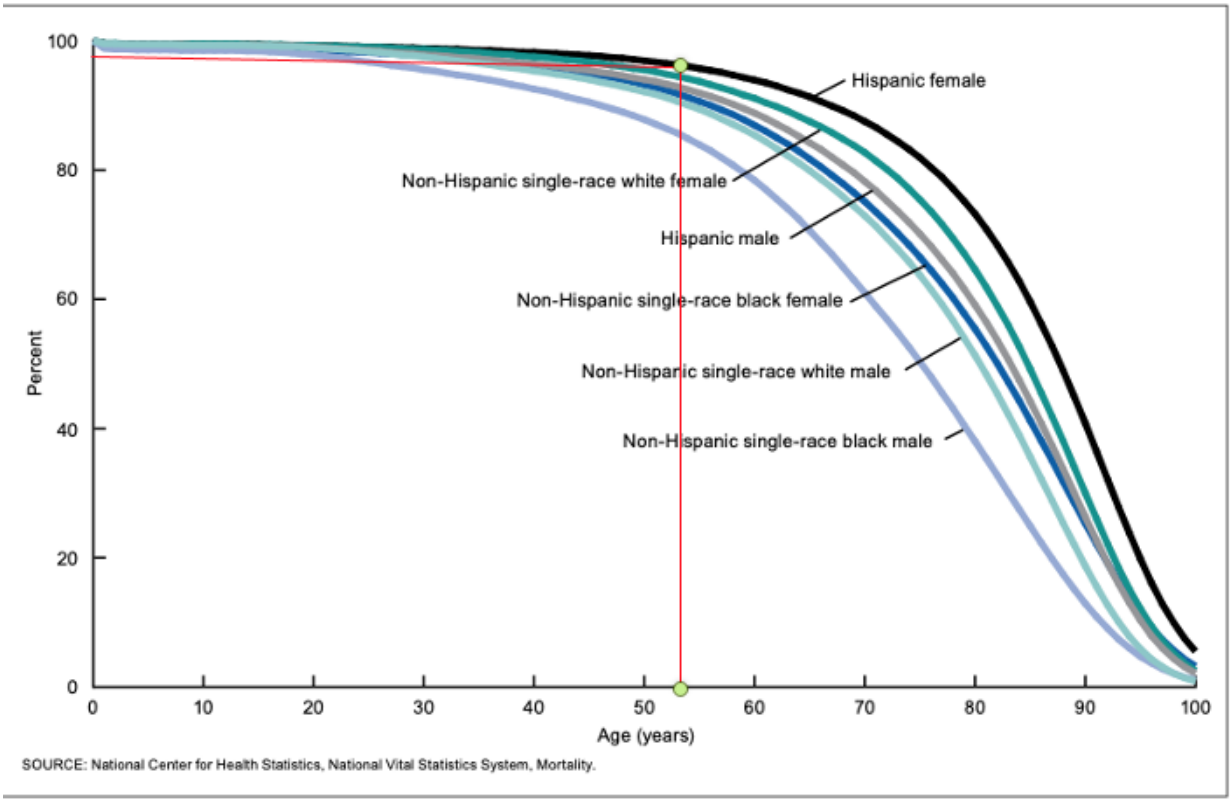
Using atmospheric and pharmacokinetic models, I have estimated Ms. PJM's daily dose of the nicotine metabolite, cotinine, from secondhand smoke. For exposure to a single smoker while cleaning each smoking room over a 6 hour day, her estimated serum cotinine equivalent from secondhand smoke would have exceeded the 90th percentile of U.S. nonsmokers' dose from secondhand smoke. On days when she was cleaning up during smoking at parties, it would have ranged beyond the 95th percentile. To put this in perspective, Ms. PJM's serum cotinine dose due to exposure to a single smoker, 1.53 ng/mL, corresponds to a urine cotinine concentration using Table 3 in Figure 4, of U = 6.5 P = (6.5)(1.54) = 10 ng/mL. By comparison, Toronto Bartenders exposed to secondhand smoke had urine cotinine levels of 10.3 ng/mL before a smoking ban.[11]

The third way of assessing Ms. PJM's risk from secondhand smoke at the CI motel and its role in the genesis of her Stage IV lung cancer, was that her estimated risk from job-related secondhand smoke exposure corresponds to an OSHA-standard working lifetime risk of approximately 3 per 1000. This is 3 times OSHA's Significant Risk of Material Impairment of Health.

The most frequently used life table statistic is life expectancy (*ex*), which is the average number of years of life remaining for persons who have attained a given age (*x*). Life expectancy and other life table values for each age in 2018 are shown for the total population and by Hispanic origin, race, and sex in Tables 1–1.[32]. Life expectancy is summarized by age, Hispanic origin, race, and sex in Table A. The U.S. Life Tables[32] indicate that at age 53 Ms. PJM had only a 2% mortality probability, as Figure 13 shows, with ~98% of the Hispanic female cohort remaining alive, and that a Hispanic white female at age of 53 would have about a 33 year life expectancy remaining (Table 9). Thus, Ms. PJM lost 33 years of life due to workplace exposure to secondhand smoke.

Figure 13. 98% of Hispanic Females remain alive @ Age 53).[32]

Figure 4. Percentage surviving, by Hispanic origin and race, age, and sex: United States, 2018



Control.

When Ms. PJM complained to the motel manager about the smoke, he told her to use a "vacuum machine" to vent the smoke outdoors. However, this would not have made a significant difference: The American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) has issued a series of position Documents on environmental tobacco smoke, issued periodically since 2005: ASHRAE "concludes that[36]:

- It is the consensus of the medical community and its cognizant authorities that ETS is a health risk, causing lung cancer and heart disease in adults, and exacerbation of asthma, lower respiratory illnesses and other adverse effects on the respiratory health of children.
- At present, the only means of effectively eliminating health risk associated with indoor exposure is to ban smoking activity.
- Although complete separation and isolation of smoking rooms can control ETS exposure in non-smoking spaces in the same building, adverse health effects for the occupants of the smoking room cannot be controlled by ventilation.
- No other engineering approaches, including current and advanced dilution ventilation or air cleaning technologies, have been demonstrated or should be relied upon to control health risks from ETS exposure in spaces where smoking occurs. Some engineering measures may reduce that exposure and the corresponding risk to some degree while also addressing to some extent the comfort issues of odor and some forms of irritation. However, the public now expects smoke-free air which cannot be accomplished with any engineering or other approaches."

Table 9. A 50-year-old U.S. Hispanic Female has approximately 33 years of life expectancy remaining (CDC Life Tables).[32]

Table A. Expectation of life, by age, Hispanic origin, race for the non-Hispanic population, and sex: United States, 2018

	All origins			Hispanic[1]			Non-Hispanic white[1]			Non-Hispanic black[1]		
Age	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female
0	78.7	76.2	81.2	81.8	79.1	84.3	78.6	76.2	81.1	74.7	71.3	78.0
1	78.2	75.7	80.7	81.2	78.5	83.7	78.0	75.6	80.4	74.5	71.2	77.7
5	74.3	71.8	76.7	77.3	74.6	79.8	74.1	71.7	76.5	70.7	67.3	73.8
10	69.3	66.8	71.8	72.3	69.6	74.8	69.1	66.7	71.5	65.7	62.4	68.9
15	64.3	61.9	66.8	67.4	64.6	69.9	64.1	61.8	66.6	60.8	57.4	64.0
20	59.5	57.1	61.9	62.5	59.8	64.9	59.3	57.0	61.6	56.0	52.8	59.1
25	54.8	52.4	57.0	57.7	55.1	60.1	54.5	52.3	56.8	51.4	48.3	54.3
30	50.1	47.8	52.2	53.0	50.5	55.2	49.9	47.7	52.0	46.9	43.9	49.5
35	45.4	43.3	47.5	48.2	45.8	50.3	45.2	43.2	47.3	42.3	39.5	44.8
40	40.8	38.7	42.7	43.5	41.2	45.5	40.6	38.7	42.6	37.8	35.1	40.2
45	36.2	34.2	38.1	38.8	36.5	40.7	36.1	34.2	37.9	33.5	30.9	35.7
50	31.7	29.9	33.5	34.2	32.0	36.0	31.6	29.8	33.3	29.2	26.7	31.4
55	27.4	25.7	29.0	29.8	27.7	31.4	27.3	25.7	28.9	25.2	22.9	27.2
60	23.3	21.8	24.8	25.5	23.6	27.0	23.3	21.7	24.7	21.4	19.3	23.2
65	19.5	18.1	20.7	21.4	19.7	22.7	19.4	18.1	20.6	18.0	16.1	19.5
70	15.8	14.6	16.8	17.5	16.0	18.6	15.7	14.5	16.6	14.9	13.3	16.0
75	12.3	11.3	13.1	13.9	12.6	14.7	12.2	11.3	13.0	11.9	10.5	12.7
80	9.2	8.4	9.8	10.5	9.4	11.1	9.1	8.4	9.7	9.2	8.1	9.8
85	6.6	6.0	7.0	7.6	6.7	8.0	6.5	5.9	6.9	6.9	6.1	7.3
90	4.5	4.1	4.8	5.3	4.6	5.5	4.5	4.0	4.7	5.0	4.5	5.2
95	3.1	2.8	3.2	3.6	3.1	3.7	3.0	2.7	3.1	3.7	3.3	3.7
100	2.2	2.0	2.2	2.5	2.2	2.5	2.1	1.9	2.2	2.7	2.5	2.7

[1]Life tables by Hispanic origin are based on death rates that have been adjusted for race and ethnicity misclassification on death certificates. Updated classification ratios were applied; see Technical Notes.

SOURCE: National Center for Health Statistics, National Vital Statistics System, Mortality.

Discussion of Uncertainty:

Cancer diagnosis: With respect to the issue of adenocarcinoma. Fontham et al. (1994) studied the relative risk of lung cancer in lifetime never smokers in five metropolitan areas in the U.S., including Los Angeles. Lifelong nonsmoking women exposed to secondhand smoke had a 30% excess risk of lung cancer from all types of primary lung carcinoma (OR=1.29 P<0.05), including pulmonary adenocarcinoma (OR = 1.28 P<0.05). The excess risk of lung cancer for women ever exposed to secondhand smoke during adult life was 24% in the household and 39% in the workplace. At the highest level of exposure, there was a 75% increased risk. Fontham et al. (1994) concluded that exposure to secondhand smoke in adult life increases the risk of lung cancer in lifetime nonsmokers. Both EPA and OSHA used the Fontham report to quantitatively estimate the risk of lung cancer in the U.S. population and in the workplace respectively. Thus adenocarcinoma of the lung is related to secondhand smoke exposure. Further, four of Ms. PJM's physicians plainly wrote that Ms. PJM's Stage IV lung cancer was the primary and the brain tumor was a secondary metastasis from that site.

Brain cancer. According to Vida et al.[3], "The incidence of primary intracranial tumours in Western Europe, North America, and Australia ranges from 4 to 11 per 100,000 population per year. This is approximately four times the incidence reported in the lowest risk regions of the world, although some of this variability may be due to differences in access to diagnostic services. In the United States the age-adjusted incidence increased by about 19% in men and 27% in women between 1973 and 2003. Glioma and meningioma are the two most common types of brain tumours, comprising approximately 75% of all brain tumours. Gliomas are more common in men; in the US, they are more common among whites than among blacks and Hispanics. The median age at diagnosis is 53 years. For the most part, the etiology of brain tumours is not understood; in particular it is not known if exogenous chemicals are capable of causing human brain tumours.

In a case-control study of 166 subjects with glioma and 93 with meningioma compared to 648 population controls, Vida et al.[33] reported that "Except for an unexplained and possibly artefactual excess risk in one population subgroup, we found little or no evidence of an association between smoking and either glioma or meningioma." Overall, the incidence of brain and other nervous system cancer in California was 6 per 100,000 in 2011-2015, compared to 43.4 per 100,000 for lung and bronchus.[30] (ACS, 2019). It

appears that Hispanics are at lower risk of brain cancer than other general population subgroups, and that lung cancers are seven times more likely than brain cancers in the general population. This further suggests that Ms. PJM's brain cancer was a secondary metastasis from a lung tumor, as her physicians diagnosed.

Radon and Asbestos: Radon gas in homes can cause lung cancer. However, Ms. PJM resided in Iglewood California 90304. According to the California Geological Survey,[53] Inglewood is in a zone which has "Low potential for Indoor Radon levels above the EPA standard of 4 picocuries per liter. Asbestos is a known lung carcinogen. However, there is no evidence that Ms. PJM has been occupationally exposed to asbestos or otherwise. Her occupation as a maid would seem to preclude asbestos exposure.

Ms. PJM was occupationally exposed to cleaning agents including Fabuloso and Kaboom and chlorinated disinfectants. Fabuloso contains sodium dodecyl benzene sulfonate (linear). According to the Material Safety Data Sheet (MSDS) for this chemical, "No component of this product present at levels greater than or equal to 0.1% is identified as probable, possible or confirmed human carcinogen by the International Agency for Research on Cancer (IARC). No component of this product present at levels greater than or equal to 0.1% is identified as a carcinogen or potential carcinogen by OSHA. And No component of this product present at levels greater than or equal to 0.1% is identified as a known or anticipated carcinogen by the National Toxicology Program (NTP). With respect to Kaboom, the active ingredient is Urea monohydrochloride. The MSDS states with respect to carcinogenicity, that "No ingredients listed by ACGIH, IARC or NTP."

The MSDS's for sodium hypochlorite bleach or toilet bowl cleaner likewise do not indicate any carcinogenicity.

Model Uncertainty: In 1986, the National Research Council[51] (NRC) reviewed the literature on environmental tobacco smoke (ETS), and concluded in agreement with our findings, that "ETS is the dominant contributor to indoor levels of RSP." The NRC also concluded that our model for ETS-RSP "predicted RSP values reasonably well over a wide range of values of input parameters.[51]" In 1987, the International Agency for Research on Cancer[47] published an authoritative peer-reviewed monograph on passive smoking which contained two chapters which featured my methods for modeling (Chapter 3) and measuring (Chapter 10) ETS concentrations. Ott et al.[50] concluded that our ETS-RSP model is a special case of a more general time-averaged model which they derived and validated. In 1986, Weiss[45] reviewed the Repace and Lowrey model of lung cancer from passive smoking, observing that "Repace and Lowrey's figures remain the best current estimates of lung cancer deaths from passive smoking." Similarly, Zitting et al.[44] noted that the lung cancer risk model of Repace and Lowrey was being used in both Finland and Norway. Kolb et al.[56] in implementing German regulations on assessing cumulative SHS exposure of hospitality workers, concluded that "the model developed by Repace and Lowrey was considered appropriate. It offers the possibility of retrospectively assessing exposure with existing parameters (such as environmental dimensions, average number of smokers, ventilation characteristics and duration of exposure). The relative risk of lung cancer can then be estimated based on the individual cumulative exposure of the worker."

The exposure modeling methods in this paper are extensively discussed in *Exposure Science,* a textbook in the Stanford University Department of Civil and Environmental Engineering.[46] Uncertainty in modeling exposure and dose is discussed in Repace et al.,[3] where it is noted that the exposure model applied to predict the weekly average concentration of secondhand smoke RSP yield results within 14% of measurements. The model was used to predict serum cotinine dose in 40 nonsmoking adults to steady-state secondhand smoke in a chamber for 4 hours. Model predictions compared very well with observations.

Ms. PJM reported that although she initially opened windows and doors when she cleaned the smoking rooms, but when active smokers were present, they quickly closed them. Based on experiments with cigarette smoking in the Pacific Inn motel in Redwood City CA, as shown in figure 12, the level of smoke would increase rapidly within 10 minutes to closed-door levels. Finally, the calculations of Ms. PJM's exposure to thirdhand smoke in nonsmoking rooms was not considered, further suggesting that her estimated exposure is conservative.

In summary: considering potential confounders, it does not appear that exposure to cleaning chemicals, asbestos, or radon caused Ms. PJM's lung cancer. Her baseline risk of lung cancer for her age, gender, and ethnic group, is only 0.08%. The exposure, dose and risk models employed suggest that the most probable cause of Ms. PJM's lung cancer is her 20-year-long exposure to secondhand

and thirdhand smoke at work cleaning smoking rooms in the motel.

Conclusions:

Ms. PJM, a lifelong nonsmoker, was diagnosed with primary Stage IV adenocarcinoma of the lung, with secondary metastasis to the brain, causing her premature death at age 53, losing 33 years of life expectancy. Adenocarcinoma of the lung is strongly correlated with secondhand smoke exposure. Her probability of contracting lung cancer at age 50 was less than 0.08%. She was exposed to both secondhand and thirdhand smoke for 20 years while cleaning smoking rooms in the motel, which comprised 75% of the rooms she cleaned. Her modeled exposure to fine particle air pollution from secondhand smoke ranged from the *Hazardous* to the *Significant Harm* Levels of the EPA Air Quality Index.

Ms. PJM's modeled level of serum cotinine ranged from the 90th to beyond the 95th percentile of a probability sample of U.S. female nonsmokers. In the course of her work, Ms. PJM is estimated to have been exposed to the thirdhand smoke of 70,000 cigarettes per year, and over a 20-year period, to the thirdhand smoke of nearly a million and a half cigarettes. The California Dept. of Health has declared that thirdhand smoke pollutants can enter the body by inhalation and dermal contact, that employees of hotels are at risk from exposure, and that such exposure can damage DNA, causing cancer.

Ms. PJM's estimated risk from passive smoking on the job is three times OSHA's Significant Risk of Impairment of Health level of 1 death or irreversible injury per 1000 workers. Her exposure to cleaning chemicals such could not have caused her cancer, since there are no known carcinogens in either product. She lived in a nonsmoking home, there is no evidence that she was exposed to asbestos, and she lived in a low-radon area of Inglewood. Thus, Ms. PJM's occupational injury and death claim is justified.

As a result of California's exemption of hotel and motel rooms from its state-wide workplace smoking ban, those workers continue to be exposed to toxic and carcinogenic secondhand and thirdhand smoke. Further, hotels and motels that are not 100% smoke-free have been found to have a 35% higher rate of complaints from guests ($p<0.05$).[55]

References

1. Repace JL, and Lowrey AH. Indoor Air Pollution, Tobacco Smoke, and Public Health. SCIENCE 208: 464-474 (I980).
2. Repace JL. Respirable Particles and Carcinogens in the Air of Delaware Hospitality Venues Before and After a Smoking Ban. Journal of Occupational and Environmental Medicine, 46:887-905 (2004).
3. Repace JL. Human Exposure to Secondhand Smoke. Chapter 9, In: Human Exposure Analysis, W Ott, L Wallace, and A Steinemann (Eds.), CRC Press (2006).
4. Repace JL, and Lowrey AH. Tobacco Smoke, Ventilation, and Indoor Air Quality. ASHRAE TRANSACTIONS 88: Part I,895 (I982).
5. Repace JL. Is the Dose-Response curve between Tobacco Smoke Exposure and Lung Cancer Linear from Active to Passive Smoking? *Environment International* 18: 427-429 (1992).
6. Repace, JL, and Lowrey, AH (1985a). A quantitative estimate of nonsmokers' lung cancer risk from passive smoking. Environment International 11: 3-22.
7. Repace JL, and Lowrey AH. An enforceable indoor air quality standard for environmental tobacco smoke in the workplace." Risk Analysis, 13:463-475 (1993).
8. Repace JL, Jinot J, Bayard S, Emmons K, and Hammond SK. Air nicotine and saliva cotinine as indicators of passive smoking exposure and risk. Risk Analysis 18: 71-83 (1998).
9. Repace JL, Hyde JN, Brugge D. Air Pollution in Boston Bars Before and After a Smoking Ban. BMC Public Health 2006, 6:266 (27 Oct 2006). Open Acess, on-line journal: <http://www.biomedcentral.com/1471-2458/6/266>.
10. Repace JL, Jiang RT, Cheng K-C, Acevedo-Bolton V, Klepeis NE, Ott WR, and Hildemann LM. Fine Particle and Secondhand Smoke Air Pollution Exposures and Risks Inside 66 US Casinos. Environmental Research 111 (2011) 473–484.
11. Repace J, Zhang B, Bondy SJ, Benowitz N, Ferrence F. Air Quality, Mortality, and Economic Benefits of a Smoke-Free Workplace Law for Non-Smoking Ontario Bar Workers. Indoor Air 2013; 23: 93–104.
12. Repace JL, Al-Delaimy WK, Bernert JT. Correlating Atmospheric and Biological Markers in Studies of Secondhand Tobacco Smoke Exposure and Dose in Children and Adults. JOEM 48: 181-194 (2006).
13. Repace JL. Secondhand Smoke in Pennsylvania Casinos: A Study of Nonsmokers' Exposure, Dose, and Risk. American Journal of Public Health 99: 1478–1485 (2009).
14. Repace J, Zhang B, Bondy SJ, Benowitz N, Ferrence F. Air Quality, Mortality, and Economic Benefits of a Smoke-Free Workplace Law for Non-Smoking Ontario Bar Workers. Indoor Air 2013; 23: 93–104.
15. *EPA Technical Assistance Document for Reporting of Daily Air Quality – the Air Quality Index* (AQI). EPA 454/B-18-007, September 2018. https://nepis.epa.gov/exe/zypdf.cgi/p100w5ug.pdf?dockey=p100w5ug.pdf
16. Air Now. Action Days. https://airnow.gov/index.cfm?action=airnow.actiondays.
17. Matt GE, Matt GE, Quintana PJE, Zakarian JM et al. Tobacco Control 2011 20: e1, doi: 10.1136/tc.2010.037382.
18. Matt GE, Quintana PJE, Fortmann AL, et al. Thirdhand smoke and exposure in California hotels: non-smoking rooms fail to protect non-smoking hotel guests from tobacco smoke exposure. Tobacco Control 2014;23:264–272.
19. Tobacco Smoke. Proposition 65 Warnings, Office of Environmental Health Hazard Assessment, OEHHA. https://www.p65warnings.ca.gov/sites/default/files/downloads/factsheets/tobacco_smoke_fact_sheet.pdf
20. Mulcahy M, Evans DS, Hammond SK, Repace JL and Byrne M. Secondhand smoke exposure and risk following the Irish smoking ban: an assessment of salivary cotinine concentrations in hotel workers and air nicotine levels in bars. Tobacco Control 14: 384-388 (2005).
21. U.S. Centers for Disease Control & Prevention. Fourth National Report on Human Exposure to Environmental Chemicals Updated Tables, January 2019, Volume One (2019).
22. Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the population to environmental tobacco smoke: the third national health and nutrition examination survey, 1988 to 1991. JAMA 275: 1233- 1240 (1996).
23. Thun MJ, Henley SJ, Burns D, Jemal A, Shanks TG, Calle EE. Lung cancer death rates in lifelong nonsmokers. J Natl Cancer Inst. 2006 May 17;98(10):691-9. doi: 10.1093/jnci/djj187. PMID: 16705123.
24. Niosh Current Intelligence Bulletin #54. Environmental Tobacco Smoke in the Workplace, Lung Cancer and Other Health Effects. U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, Cincinnati, OH June 1991.
25. National Toxicology Program. 9th Report on Carcinogens 2000. U.S. Dept. of Health & Human

Services, National Institute of Evironmental Health Sciences, Research Triangle Park, NC.
26. California Environmental Protection Agency. Health Effects of Exposure to Environmental Tobacco Smoke, Final Report 1997.
27. U.S. Dept. of Labor, Occupational Safety & Health Administration. 29 CFR Parts 1910, 1915, 1926, and 1928 Indoor air quality, proposed rule Fed Reg 59 # 65, Tues April 5, 1994, 15968-16039.
28. U.S. Environmental Protection Agency. Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders. EPA/600/6-90/006F.
29. Repace JL, and Lowrey AH. Risk Assessment Methodologies in passive smoking-induced lung cancer. RISK ANALYSIS, 10: 27-37, (1990).
30. American Cancer Society (2015). Cancer Facts & Figures for Hispanics/Latinos, 2015-2017; American Cancer Society. ACS cancer-facts-and-figures-for-hispanics-and-latinos-2018-2020.
31. National Cancer Institute. Smoking and Tobacco Control Monograph No. 8: Changes in Cigarette-Related Disease and their Implications for Prevention and Control. (1996).
32. Arias A, Xu J. United States Life Tables, 2018. National Vital Statistics Reports 99: 12, Nov. 17, 2020
33. Vida S. et al. Environmental Health 2014, 13:55 Brain tumours and cigarette smoking: analysis of the INTERPHONE Canada case–control study.
34. Dacunto PJ, Cheng K-C, Acevedo-Bolton V, Jiang R-T, Klepeis NE, Repace JL, Ott WR, Hildemann LM. Identifying and quantifying secondhand smoke in source and receptor rooms: logistic regression and chemical mass balance approaches. Indoor Air 2013, Apr 30. doi: 10.1111/ina.12049.
35. American Cancer Society. California CANCER SCREENING AND RISK FACTOR PREVALENCE, 2018.
36. ASHRAE Position Document on Environmental Tobacco Smoke. American Society of Heating Refrigeration and Ventilating Engineers (2013).
37. Cal/OSHA Consultation Service, AB-13 FACT SHEET - CALIFORNIA WORKPLACE SMOKING RESTRICTIONS (1997).
38. CalEPA, 2006. California Environmental Protection Agency, Air Resources Board Office of Environmental Health Hazard Assessment, State of California. Proposed Identification of, Environmental Tobacco Smoke as a Toxic Air Contaminant As Approved by the Scientific Review Panel, on June 24, 2005.
39. Cancer in Los Angeles Trends by Race/Ethnicity 1976-2012 County. Liu et al. Los Angeles Cancer Surveillance Program USC/Norris Comprehensive Cancer Center The Keck School of Medicine of the University of Southern California.
40. Los Angeles County. Lung and Bronchus Cancer Incidence Rate by Race/Ethnicity County: Los Angeles.
41. MMWR. Age-adjusted Death Rates from Lung Cancer by Race/Ethnicity. National Vital Statistics System, United States, 2001-2013. https://www.cdc.gov/mmwr/volumes/67/wr/mm6730a8.htm.
42. Msds fabuloso all purpose cleaner liquid lavender. Colgate Palmolive Co.
43. MSDS Kaboom Shower Tub & Tile Cleaner. Orange Glo International.
44. Zitting A, Husgafvel-Pursiainen K, Rantanen J. Environmental tobacco smoke—a major preventable cause of impaired health at work. *Scandinavian Journal of Work, Environment, & Health* (Vol. 28, supplement 2, 2002).
45. Weiss, ST. Passive Smoking, what is the risk? Am Revs Resp Dis 133:1-3, 1986.
46. Ott WR, Wallace LA, and Steinemann A. Human Exposure Analysis, (Eds.), CRC Press (2006).
47. Repace JL. Indoor concentrations of environmental tobacco smoke: field surveys. Ch. 10, IARC Scientific Publications no. 81, Environmental Carcinogens--Selected Methods of Analysis--Volume 9 Passive Smoking; I.K. O'Neill, K.D. Brunnemann, B. Dodet & D. Hoffman, International Agency for Research on Cancer, World, Health Organization, United Nations Environment Programme, Lyon, France, (1987).
48. Travers MJ, KM Cummings, A Hyland, J Repace, S Babb, T Pechacek, PhD, R Caraballo. Indoor Air Quality in Hospitality Venues Before and After Implementation of a Clean Indoor Air Law — Western New York, 2003. MMWR Vol. 53 / No. 44 1038-104, November 12, 2004.
49. Repace JL. Is the Dose-Response Curve between tobacco smoke exposure and lung cancer linear from active to passive smoking? Environment International 18: 427-429 (1992).
50. Ott WR. Mathematical models for predicting indoor air quality from smoking activity. Environmental Health Perspectives 107, suppl 2, 375-381 (1999).
51. National Research Council (1986). Environmental tobacco smoke -- measuring exposures and assessing health effects. National Academy Press, Washington, DC.
52. Thun MJ, Henley SJ, Burns D, Jemal A, Shanks TG, Calle EE. Lung cancer death rates in lifelong nonsmokers. J Natl Cancer Inst. 2006 May

17;98(10):691-9. doi: 10.1093/jnci/djj187. PMID: 16705123.

53. Churchill, January 2005. California Geological Survey Radon Potential Zone Map for Southern Los Angeles County, California.

54. Jacob P 3rd, Benowitz NL, Destaillats H, Gundel L, Hang B, Martins-Green M, Matt GE, Quintana PJ, Samet JM, Schick SF, Talbot P, Aquilina NJ, Hovell MF, Mao JH, Whitehead TP. Thirdhand Smoke: New Evidence, Challenges, and Future Directions. Chem Res Toxicol. 2017 Jan 17;30(1):270-294. doi: 10.1021/acs.chemrestox.6b00343. Epub 2016 Dec 21. PMID: 28001376; PMCID: PMC5501723. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5501723/.

55. Weigel EA, Matt GE. When Hotel Guests Complain About Tobacco, Electronic Cigarettes, and Cannabis: Lessons for Implementing Smoking Bans. Tob Use Insights. 2022 Sep 5;15:1179173X221124900. doi: 10.1177/1179173X221124900. PMID: 36090650; PMCID: PMC9449509.

56. Kolb, S., Brückner, U., Nowak, D. et al. Quantification of ETS exposure in hospitality workers who have never smoked. Environ Health 9, 49 (2010). https://doi.org/10.1186/1476-069X-9-49.

57. Sheu R, Stönner C, Ditto JC, Klüpfel T, Williams J, Gentner DR. Human transport of thirdhand tobacco smoke: A prominent source of hazardous air pollutants into indoor nonsmoking environments. Sci Adv. 2020 Mar 4;6(10):eaay4109. doi: 10.1126/sciadv.aay4109. PMID: 32181345; PMCID: PMC7056301.