INDOOR AIR POLLUTION BY TOBACCO SMOKE: MODEL STUDIES ON THE UPTAKE BY NONSMOKERS

Dietrich Hoffmann, Klaus D. Brunnemann, John D. Adams, and Nancy J. Haley
Naylor Dana Institute for Disease Prevention
American Health Foundation
Valhalla, New York, 10595, U.S.A.

Abstract

For a small room (16 m^3), continously polluted with sidestream smoke from 2, 3 or 4 cigarettes, the highest pollution levels recorded were: CO, 25 ppm; NO_x, 0.91 ppm; HCN, 56 $\mu g/m^3$; formaldehyde, 1.6 $\mu g/m^3$; nicotine, 288 $\mu g/m^3$; and particulate matter, 4,600 $\mu g/m^3$. During each session, the nonsmokers stayed in the room for 80 minutes. Saliva, blood and urine, collected before, during and 5 hrs following exposure, were analyzed for nicotine, cotinine, and thiocyanate. Blood was analyzed also for COHb. The highest levels of nicotine or cotinine in the physiological fluids of the passively exposed individuals did not exceed 3% of the mean per person recorded for 450 smokers of $\geq$20 cigarettes/day. Nitrosoproline (NPRO) in urine, serves as an indicator for endogenous nitrosamine formation upon exposure to nitrosating agents. First data after passive smoke exposure do not indicate increased urinary excretion of NPRO.

Introduction

Epidemiological studies have indicated an increased risk for lung cancer in passive smokers (2,5,6,13). Weak asssociations have also been reported for passive smoking and nasal sinus cancer and for childhood brain tumors in the offspring of women passively exposed to sidestream smoke during pregnancy (6,11). However, a number of other studies failed to confirm such associations (3,8,9). This discrepancy is not surprising because of the relatively small increased risk for cancer in passive smokers and because of the many potential sources of bias and confounders inherent in epidemiological studies on weak associations (12). An elucidation of the significance of the association requires epidemiological studies with different population groups, a better understanding of the physicochemical nature of sidestream smoke and polluted indoor air and quantitative comparisons of the uptake of smoke constituents by active and passive smokers.

Sidestream Smoke and Indoor Pollution

Sidestream smoke (SS) is a composite of effluents generated in different ways during the burning of a tobacco product. The peak temperature in the burning cone of a cigarette during puffing while mainstream smoke (MS) is being formed, reaches ≈900°C; during puff intervals it is ≈600°C. This is an important factor for the divergence of certain physicochemical parameters and toxic agents in mainstream and sidestream smoke (14; Table 1). SS releases also significantly higher quantities of certain components than does MS. This applies especially to CO, nicotine pyrolysis products and those vapor phase components for which the tobacco nitrate serves as a precursor (14).

Table 1. Comparisons of Mainstream and Sidestream Smoke of Cigarettes.

Parameters	MS	SS
Peak temperature during formation (°C)	≈900	≈600
Particle Size (µm)	0.1 - 1.0	0.01 - 0.1
pH of smoke (blended cigarette)	5.8 - 6.1	6.9 - 8.0
Smoke dilution (Vol.%)*		
Carbon monoxide	3 - 5	≈1
Carbon dioxide	8 - 11	≈2
Oxygen	12 - 16	16 -20
Hydrogen	3 - 15	≈0.5

*10 mm away from the burning cone.

It needs to be noted that the release of particulate matter, of nicotine and of many other smoke components is significantly decreased in the MS of low-yield and ultra low-yield cigarettes, but that the SS yields remain relatively unchanged (10). The reported values for CO and other toxic agents for smoke polluted indoor environments range between 5-110 ppm depending on room size and degree of ventilation and on the number of cigarettes smoked (10). In respect to carcinogenesis, the high concentrations of volatile N-nitrosamines (up to 0.24 µg/m^3) and of NO_x (up to 0.5 ppm) which may contribute to the endogenous formation of nitrosamines are of special interest (1,7).

generated
duct. The
ring puff-
es ≈900°C;
ant factor
meters and
Table 1).
rtain com-
, nicotine
for which

moke of

SS
≈600
0.01 - 0.1
- 8.0
≈1
≈2
6 -20
≈0.5

matter, of
cantly de-
igarettes,
(10). The
e polluted
ng on room
cigarettes
concentra-
and of NO_x
formation

Inhalation of Polluted Air by Nonsmokers: A Model Study.

Two or 3 male nonsmokers were placed for 80 min in a small room (≈16 m^3) which was continuously polluted with SS of 2, 3 or 4 cigarettes, respectively. SS was generated by a smoking machine, the MS from these cigarettes was directed outside the room. Table 2 presents data pertaining to characteristics and the environment of the test chamber.

Table 2. Test Laboratory.

Size: 16.3 m^3		
Temperature: 22 ± 1°C		
Air Exchanges: 6 x per hour		
Pollutants:	Sidestream Smoke of 4 concurrently smoked 1R1 Cigarettes	
Indoor Pollution:	Particulate Matter	4,600 $\mu g/m^3$
	Nicotine	280 $\mu g/m^3$
	Hydrogen Cyanide	56 $\mu g/m^3$
	Carbon Monoxide	25 ppm
	NO_x	0.91 ppm
	Formaldehyde	1.6 mg/m^3

Before passive smoke exposure, an indwelling catheter was inserted in the antecubital vein of the 10 volunteers, permitting blood sampling before, during, and after exposure. Duplicate blood samples were drawn at 10 minute intervals up to 80 minutes of exposure and, subsequently, at larger intervals up to 5 hrs post exposure.

Neither thiocyanate in saliva, serum and urine nor carboxyhemoglobin in serum (<2.0%) were significantly elevated. The average nicotine values in saliva increased significantly, reaching maxima of 430, 840 and 880 ng/ml after 60-80 min exposure for the 3 degrees of pollution. Upon leaving the room, the nicotine values decreased rapidly, reaching background levels after 2-3 hrs. Cotinine levels in saliva up to 5 ng/ml were recorded 2 hrs after leaving the room. Nicotine in serum was not significantly different from the background whereas cotinine reached 4 ng/ml 3 hrs after the volunteers had left the room. The most significant nicotine-cotinine results were observed in the urine analysis. A dose response for nicotine was indicated

Table 3. Nicotine and Cotinine Levels In Saliva, Serum And Urine of Volunteers
(Summary of Average Values)

Time** (min)	Saliva (ng/mg)				Serum (ng/ml)	Urine (ng/mg Creatinine)					
	Nicotine			Cotinine	Cotinine	Nicotine			Cotinine		
	2*	3*	4*	4*	4*	2*	3*	4*	2*	3*	4*
Baseline	8	1	3	1.0	0.9	24	20	17	14	14	14
I 40	350	719	830	1.1	0.9						
I 60	430	830	880	2.1	1.2	26	34	84	16	21	28
O 30	76	157	148	1.7	1.8						
O 120	6	17	23	2.5	2.9	40	94	100	21	34	46
O 240	8	2	3	2.0	3.3						
O 300	7	7	7	3.5	3.4	51	58	48	21	38	55

*Numbers represent room pollution by smoke of 2, 3 or 4 cigarettes.

**I = Inside exposure room during pollution; O = outside exposure room after leaving the room.

**I = Inside exposure room during pollution; O = outside exposure room after leaving the room.

within 2 hrs and for cotinine within 2-5 hrs after exposure (Table 3).

In a preliminary study, we measured the 24 hr excretion of N-nitrosoproline (NPRO) in the urine of 4 male nonsmokers who had been in the exposure room polluted by SS of 4 cigarettes on 2 consecutive days three times daily for 80 minutes. NPRO excretion did not increase significantly (average 2.7 to 3.9 µg/24 hr).

Discussion

This model study has shown that nicotine in saliva is the best immediate indicator of passive smoke exposure whereas cotinine (and possibly nicotine) in urine appears to be the most reliable marker of such exposure during the following 24 hrs. In our study of 450 active smokers, we found a plasma cotinine plateau at 300 ng/ml which was reached after about 20 cigarettes (4). The highest value recorded in this study of passive smoke exposure was 6 ng/ml. This indicates that the nicotine (and smoke) uptake from passive exposure amounts to merely a small percentage of the uptake from active cigarette smoking. However, this conclusion requires confirmation by field studies.

The preliminary study on the endogenous formation of NPRO in 4 passive smokers did not demonstrate significant increases of urinary NPRO excretion (2.7 to 3.9 µg/24 hr) compared to the average NPRO excretion in cigarette smokers (11.7 µg/24 hr; 7). However, this model study requires confirmation with data from a larger group of volunteers and also from field studies with passively exposed subjects.

Reference

(1) Brunnemann, K.D. and Hoffmann, D. Analysis of volatile nitrosamines in tobacco smoke and polluted indoor environments. Intern. Agency Res. Cancer Sci. Publ. 1978, No. 19, 343-356.

(2) Correa, P., Pickle, L.W., Fontham, L., Lin, Y. and Haensel, W. Passive smoking and lung cancer. Lancet, 1983, 12, 595-597.

(3) Garfinkel, L. Time trends in lung cancer mortality among nonsmokers and a note on passive smoking. J. Natl. Cancer Inst., 1981, 66, 1061-1066.

(4) Hill, P., Haley, N.J., and Wynder, E.L. Cigarette smoking: carboxyhemoglobin, plasma nicotine, cotinine and thiocyanate vs. self-reported smoking data and cardiovascular disease. J. Chron. Dis., 1983, 36, 439-449.

(5) Hirayama, T. Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. Brit. Med. J., 1981, 282, 183-185.

(6) Hirayama, T. Passive smoking and lung cancer, nasal sinus cancer and ischemic heart disease. Proc. 5th World Conference on Smoking and Health. In print.

(7) Hoffmann, D. and Brunnemann, K.D. On the endogenous formation of N-nitrosoproline in cigarette smokers. Cancer Res., 1983, 43, 5570-5574.

(8) Koo, L.C., Ho, J.H-C., and Saw, D. Active and passive smoking among female lung cancer patients and controls in Hong Kong. J. Exptl. Clin. Cancer Res., 1983, 4, 367-375.

(9) Kabat, G.C. and Wynder, E.L. Lung cancer in nonsmokers. Cancer, 1984. In print.

(10) Laboratory of the Government Chemist, Report 1979 - Smoking and Health, London, 1980, pp. 1709-183.

(11) Preston-Martin, S., Yu, M.C., Benton, B., and Henderson, B.E. N-Nitroso compounds and childhood brain tumors: a case control study. Cancer Res., 1982, 42, 5240-4245.

(12) Schlesselman, J. and Wald, N. (eds.). Weak associations in epidemiology and their interpretation. Prev. Med., 1982, 11, 464-476.

(13) Trichopoulos, D., Kalandidi, A., and Sparros, L. Lung cancer and passive smoking: conclusion of Greek study. Lancet, 1983, 12, 677-678.

(14) U.S. Surgeon General "Smoking and Health" - Involuntary smoking, 1979, DHEW Publ. NO (PHS) 79-50066 11, 1-41.

Our studies are supported by National Cancer Institute Grant PO1-CA-29580.

Proceedings of the 3rd International Conference on Indoor Air Quality and Climate

held in Stockholm
August 20–24, 1984

Under the High Patronage of
His Majesty King Carl XVI Gustaf

Organized by
The Karolinska Institute and
The National Institute of Environmental Medicine
with the cosponsorship of
The World Health Organization

Sponsored by

Commission of the European Communities
Swedish Ministry of Health and Social Affairs
U S Environmental Protection Agency
Swedish Council for Building Research
Swedish Work Environment Fund
National Institute of Radiation Protection (Sweden)
Representatives of European Heating and Ventilating Associations
American Society of Heating, Refrigerating and Air-Conditioning Engineers
Electric Power Research Institute (USA)
Gas Research Institute (USA)
Ruhrgas AG (FRG)
Kawasaki Ltd (Japan)
Drägerwerk AG (FRG)
Fläkt AB (Sweden)
Bahco AB (Sweden)
Bruel & Kjaer (Denmark)

INDOOR AIR

Volume

2

Radon, Passive Smoking, Particulates and Housing Epidemiology

Editors

BIRGITTA BERGLUND
Department of Psychology, University of Stockholm

THOMAS LINDVALL
Karolinska Institute and the
National Institute of Environmental Medicine

JAN SUNDELL
National Board of Occupational Safety and Health

Swedish Council for Building Research
Stockholm, Sweden 1984