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RESEARCH ARTICLE

Effects of A Small Dose of Biomass Smoke on The Morbidity of Community-acquired Pneumonia; A Multivariate Regression Analysis of 1600 Patients

Kembu Nakamoto, MD, PhD[1*], Sadahiro Kawamoto, MD, PhD[1]

[1]Division of General Thoracic Surgery and Division of Respiratory Medicine, Shunan Memorial Hospital, Kudamatsu, 744-0033 Japan

[*]nakamoto@hcsdojinkai.or.jp

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ABSTRACT

The annual number of deaths from pneumonia has markedly increased with the decrease in the prevalence of smoking since the 1980s in Japan. A small dose of biomass smoke functions as a disinfectant against droplet-borne biologic microbes, and smokeless residence has been proposed as one cause of the high incidence of pneumonia. The present study retrospectively investigated the effects of biomass smoke on the morbidity of pneumonia using a multivariate regression analysis.

This study analyzed 1600 patients with pneumonia or parapneumonic pleuritis between 2010 to 2021. A total of 181 health examinees for chest CT screening in 2019 were used as the control group. The following variables were selected for the analysis; sex, age, the smoking status, nursing care status, comorbidities, disease history, vaccination status, season, residence smoking status, pathogens, disease severity, medical interventions, and disease prognosis. The primary outcome was the morbidity of pneumonia, and the secondary outcomes were disease prognosis and the detection of pathogens.

Most pathogens were resident flora of droplet-borne contamination. A smokeless environment significantly increased the detection rate of pathogens associated with sever disease and a poor outcome. Patients with a low performance status had a significantly worse outcome. Indoor seasons and smokeless environments significantly increased morbidity, while vaccination and current smoking markedly reduced morbidity.

The main cause of pneumonia was speculated to be droplet-borne infection in smokeless community spaces. Smokeless residences appear to have increased the morbidity of pneumonia.

Introduction

The number of annual deaths from community-acquired pneumonia has markedly increased by 4- to 5-fold from the pre-1980s level of approximately 30,000 deaths in Japan, despite improvement in residential hygiene and a decrease in the prevalence of smoking[1,2]. Annual mortality is similar to that during Spain flu pandemics. We previously suggested that the risk of pneumonia was higher in a smokeless indoor communal environment than in an environment where smoking is permitted[3]. A low dose of biomass smoke, cigarette or incense smoke, functions as a disinfectant against droplets and air-borne pathogens, and facilitates the gulfing of tar-labelled microbes as carbonic foreign particles by macrophages[4,5]. Among current hygiene procedures, only biomass smoke disinfects room air contamination.

Therefore, the present study retrospectively investigated the effects of biomass smoke on the morbidity of community-acquired pneumonia using a multivariate regression analysis.

Patients and Methods

The Institutional Review Board of our hospital granted ethical approval for this study (IRB approval #02-02, SMH, Nov. 11, 2020). Informed consent was obtained from each patient. In this retrospective study, data were acquired from attending physicians and/or electronic medical records or through telephone inquiries or patient interview.

Patients with acute pneumonia, including parapneumonic pleuritis with effusion or empyema and excluding aspiration pneumonia with the clinical feature of dysphagia or food in the large airways on CT images, who were admitted to Shunan Memorial Hospital between January 1, 2010 to December 31, 2021, which corresponded to the end of the δ-variant COVID-19 pandemic and before smokeless cigarette era, were analyzed. Patients of readmission for recurrent pneumonias were excluded. Acute pneumonia was diagnosed by chest CT, clinical features, a hematological examination, a bacterial culture of sputum or effusion, and/or specific PCR examinations or immunological test for biological microbes. Health examinees who visited our hospital for lung cancer screening by chest CT between January 1 and December 31, 2019 were used as the control group because the majority were annual repeaters. A multivariate analysis of different categories of variables was performed. Host variables were as follows: sex, age, the current smoking status, performants status, Charlson's comorbidity score[6], respiratory comorbidities, such as COPD, chronic infections, immune diseases, and malignancies, nursing care services, and vaccination for pneumococcal pneumonia. Close conversations spreads millions of droplets-borne microbes in a short time, while contact or air-borne contamination contains a small number of microbes[4]. The elderly and individuals receiving nursing care services are routinely exposed to droplet-borne biocontamination due to the need for close conversations as a result of hearing impairments and contact nursing care. Individuals aged 75 years or older, those with hearing impairments, and those receiving nursing care services were defined as a high-risk group of droplets-born infections, while individuals younger than 75 years and not receiving nursing care services were defined as a low-risk group. The environmental variables included in the multivariate analysis were as follows: indoor seasons (December to March, July and August, representing cold, hot, and rainy periods, respectively); outdoor seasons (April to June, October and November); residence style (single or group living); residence type; and indoor smoking status (smoking allowed or smokeless). Based on the prevalence of smoking in the control group, approximately 70% of individual homes were presumed to have no smoking restrictions. In addition, about 30% of Japanese households are Buddhists who burn incense sticks twice daily[7], Consequently, approximately 80% of individual homes were speculated to have a smoke-exposed indoor environment. Welfare facilities and residences occupied by a single non-smoker or a couple with at least the male partner as a non-smoker were defined as having "smoking restrictions." Other individual homes were defined as "smoking allowance". The detection of pathogens, epidemic or resident flora, and drug resistance were selected as pathogen site variables. The positive detection of pathogens indicated exposed to a high pathogen load and negative detection as that to a low load[8]. Disease severity (lesions on CT images and CRP levels) and

the disease category of pneumonia or parapneumonic pleuritis were selected as disease site variables. Interventions with nasal or pleural space irrigation using electrolyzed saline, and respiratory rehabilitation, used as optional to conventional treatment, were selected as intervention variables. In-hospital prognosis was selected as the outcome variable of pneumonia. Explanatory variables with a small regression coefficient in the same category in the initial analysis were deleted from the final analysis for compact tables. The control group was used in the analysis of the morbidity of pneumonia.

Statistical analysis

A multivariate regression analysis was used in the present study[9]. Analyses were performed using Excel statistical software (Excel statistics 2020; Ekuseru-Toukei 2020, Social Survey Research Information Co. Ltd, Tokyo). The regression coefficients of the explanatory variables were interpreted as indicators of risk of the outcomes, including pneumonia morbidity. A regression coefficient with a p-value <0.05 was considered to be significant.

Results

A total of 1600 patients were admitted to Shunan Memorial Hospital under a diagnosis of acute pneumonia and 181 health examinees visited our hospital for lung cancer screening by chest CT.

Subjects' profiles (Table 1):

	Variables	Pneumonia group; n=1600 (Jan 2010-Desc 2021)	Control group; n=181 (Jan-Desc 2019)
Host	Sex; male/female	938/661	108/73
	Age; ≦75years / >75years	552/1048	150/31
	Smoking status; non-smoker/current smoker	1502/98	122/59
	Brinkman index (cigarettes/day× smoking year); 0/≦1000/>1000	1256/229/115	92/65/24
	Performance status; 0/1/2/3/4	747/258/138/457	177/4/0/0
	Nursing care service; no/yes	756/844	171/10
	Charlson's comorbidity score; free or mild/moderate or severe	1004/596	168/13
	Comorbid respiratory disease; no/yes	1000/600	153/28
	Droplet-borne infection; low- /high-risk group	446/1154	149/32
	Previous history of pneumonia; no/yes	1230/370	175/6
	Pneumococcal vaccination; finished/not yet	82/1518	53/128
Environment	Season; indoor/outdoor	1022/578	80/101
	Residence style; single/group	232/1368	28/153
	Residence; welfare facility/individual home	287/1313	4/177
	Smokeless residence/smoke allowed residence	811/789	35/146
Pathogen	Detection; negative / unknown / positive	346/524/730	
	virus/bacteria/fungus / not detected nor examined	104/603/23/870	
	epidemic/unknown/resident flora	121/582 /897	
	resistant/unknown/sensitive to drugs	271/ 638/691	
Disease	Affected lesions; single lobe or less / multiple lobes	743/857	
	Pneumonia / parapneumonic pleuritis	1425/175	
Prognosis	Hospitalized stay; ≦2wk/>2wk/death after2wk/death within2wk	672/701/125/102	

	Variables	Pneumonia group; n=1600 (Jan 2010-Desc 2021)	Control group; n=181 (Jan-Desc 2019)
	Discharged/hospital death	1373/227	
Intervention	Respiratory rehabilitation; no/yes/unknown	910/376 /314	
	Electrolyzed saline irrigation; no/yes	1510/90	

n: number of patients or examinee

Host site profile variables were as followed. In the patient group, approximately 67% of individuals were older than 75 years, whereas in the control group, approximately 83% were younger than 75 years. The sex ratio was similar between the two groups. The majority of females were housewives who had few opportunities to visit or stay in community places. The current smoker ratio was smaller in the patient group than in the control group. The prevalence of smoking in the control group was similar to that in the Japan Tobacco (JT) annual report[1]. The patient group had a poorer performance status, a higher Charlson's comorbidity score, and more frequently had comorbid respiratory diseases than the control group. The patient group also included a higher percentage of individuals at an increased risk for droplet-borne infections than the control group. Among pneumonia patients, 375 (23.1%) had a previous history of pneumonia. Furthermore, only 82 of 1,601 patients (5.4%) had completed pneumococcal vaccination upon admission, in contrast to 53 of 181 examinees (29.3%) in the control group. Environmental site profiles were as follows: approximately two-thirds of patients were admitted during indoor seasons, when more time is typically spent in air-conditioned communal spaces. Around 20% of patients were admitted from welfare facilities. Half of the residences had indoor smoking restrictions. Disease site profiles were as follows. Pneumonia lesions were in a single lobe or less in approximately half of patients, and in multiple pulmonary lobes in the remaining patients. CRP levels on hospital admission ranged 0.5 to 47 mg/dl (normal range: between 0.05 and 0.30 mg/dl). Among pneumonia patients, 501 (31.7%) received respiratory rehabilitation during their hospital stay. Eighty-three patients with parapneumonic empyema and COVID19 pneumonia received an intervention with electrolyzed saline irrigation of the intrapleural or naso-oral cavity. In total, 1372 patients discharged to their homes or transferred to other welfare facilities, while remaining 226 died in the hospital; therefore, the hospital mortality rate of pneumonia was 14.1%. Overall, 680 patients were discharged within 2 weeks and 694 after 2 weeks. A total of 124 patients died after a 2week hospital stay, while 109 patients had an early hospital death within 2 weeks.

Pathogens (Table 2):

detected/not detected/not examined	**745/346/524**				
Viral pneumonia	104	Bacterial pneumonia	738	Fungal pneumonia	23
Influenza virus	62	*Streptococcus Pneumoniae*	234(30)	*Aspergilluus*	4
δ variant COVID19	28	*Haemophilus Influenzae*	112(3)	*Candida*	16
Cytomegalovirus	13	MRSA	78	*Pneumocystis carinii*	3
Epstein Barr (EB)	1	*Staphylococci*	47(8)		
		Klebsiella pneumoniae	38(3)		
<Influenzas virus-induced pneumonia>		*Branhamella catarrhalis*	36(1)		
Streptococci	10	*Pseudomonas*	32		
Haemophilus influenza	4	*E. coli*	32(7)		
Branham cat	3	*Mycoplasma*	27		
Pseud	3	*Corynebacterium*	17(13)		

detected/not detected/not examined	745/346/524		
Klebs pn	1	*Acinetobacter baumannii*	10(4)
Acinetobacter	1	*Enterobacter*	12(5)
Enterobacter	1	*Serratia marcescens*	8(6)
Legionella	1	*Chlamydia pneumonia*	2
<EB-virus induced pneumonia>		*Legionella*	1
Streptococci pneumonia	1	miscellaneous	7
		<Parapneumonic empyema>	
		Fusobacterium	9
		Str intermed	9
		Str phary	5
		Stp aer	3
		Stp epid	3
		Streptococcus agalactiae	3
		Str anginosus	1
		misceraneous	12

n; number of detected pathogens, (n); number of drug-resistant pathogens

In total, 865 pathogens were detected in 745 patients, but not in 346. Microbiological examinations were not performed on 524 patients because symptoms were mild or difficulties were associated with sputum sampling. The primary pathogens were viral in 104 patients, bacteria in 695, and fungal in 23. The following epidemic pathogens were detected: Influenza virus in 62 patients, δ variant COVID19 in 28, and *Mycoplasma* in 27. Other pathogens were resident flora of air or droplet-borne contamination. The most frequently detected viral pathogen was *Influenza virus*. followed by bacterial pneumonia in 25 patients and *Cytomegalovirus* pneumonia in 13 patients. Rare pathogens of Epstein-Barr virus, *Pneumocystis pneumoniae* and *Legionella* were detected in 5 patients. The most frequently detected pathogens were bacteria. *Streptococcus pneumoniae*, *Hemophilus influenza* and methicillin-resistant *Staphylococcus aureus* (MRSA) were the main pathogens of bacterial pneumonia, followed by *Staphylococci*, *Klebsiella pneumoniae*, *Branhamella catarrhalis*, *Pseudomonas*, and *Escherichia coli*. All pathogens of empyema were pyogenic bacteria of the resident flora. Among bacterial pathogens, 190 exhibited drug resistance (27%) against more than 3 antibiotics.

Table 3. Multivariate regression analysis of the detection of pathogens

Objective variable; Detection of pathogens: negative (346) / unknown (524) / positive (730)					
			95% confidential interval		
Variables		regression coefficient	lower limit	upper limit	P value
Host site	Sex; female / male	0.0206	-0.0595	0.1007	0.6143
	Age; ≦75years / >75years	-0.0261	-0.1667	0.1145	0.7156
	Current smoker / non-smoker	-0.1002	-0.2660	0.0656	0.2361
	Brinkman Index; 0 / ≦1000 / >1000	-0.0133	-0.0292	0.0025	0.0995

	Objective variable; Detection of pathogens: negative (346) / unknown (524) / positive (730)				
	Nursing care service; yes / no	-0.0677	-0.1761	0.0406	0.2203
	Charlson's score; free, mild / moderate, sever	0.0786	-0.0095	0.1667	0.0802
	Comorbid respiratory disease; no / yes	-0.0208	-0.1021	0.0605	0.6157
	Droplet borne infection; low risk / high risk	0.0829	-0.0884	0.2542	0.3428
	Previous history of pneumonia; no / yes	0.0818	-0.0084	0.1720	0.0754
	Pneumococcal vaccine; not yet / finished	-0.1635	-0.3306	0.0035	0.0550
Environment	Season; outdoor / indoor	-0.0760	-0.1522	0.0001	0.0504
	Residence; welfare facility / individual home	0.0172	-0.0113	0.0457	0.2373
	Smokeless environment / smoke allowed environment	0.1476	0.0607	0.2344	P < 0.001**
Disease	Affected region; single lobe or less / multiple lobes	0.0823	0.0077	0.1569	0.0306*
	CRP 0.5~1.0 / 1.0~20 /> 20~	0.0322	-0.0280	0.0924	0.2940
	Disease category; Pneumonia / parapneumonic empyema	-0.1637	-0.2979	-0.0296	0.0168*
Prognosis	Hospital stay; ≦2wks / >2wks / hospital death after 2wks / hospital death within 2wks	0.0537	0.0040	0.1034	0.0341*

*; P<0.05, **; P<0.01, (n); number of patients

Patients from environments where smoking is permitted had a significantly lower detection rate of pathogens (p<0.001). This result suggests that biomass smoke may routinely suppress both epidemic and resident environmental pathogens. The affected lung regions were significantly smaller in patients with negative pathogen detection (p<0.05). In contrast, empyema was associated with a significantly higher rate of pathogen detection and a poorer prognosis. These results indicate that exposure to a high pathogen load is associated with more severe disease and worse outcomes. Therefore, airborne or droplet-transmitted pathogenic microbes appeared to be suppressed in environments where smoking is permitted.

Table 4. Multivariate regression analysis of outcome of pneumonia

Prognosis; hospital stay <2wks / >2wks / Hospital death after 2wks /Hospital death before 2wks : 672/701/125/102

Variables		regression coefficient	95% confidential interval lower limits	95% confidential interval upper limits	P value
Host	Sex; female / male	-0.1032	-0.1820	-0.0243	0.0104*
	Age; ≦75years / >75years	-0.2306	-0.3662	-0.0951	P<0.001**
	Current smoker / non-smoker	-0.0140	-0.1775	0.1495	0.8666
	Brinkman Index; 0 / ≦1000 / >1000	0.0150	-0.0007	0.0306	0.0604
	Nursing care service; no / yes	-0.2883	-0.3935	-0.1832	P<0.001**
	Charlson's score; free, mild / moderate, sever	0.2857	0.1999	0.3715	P<0.001**
	Comorbid respiratory disease; no / yes	-0.0718	-0.1520	0.0085	0.0795
	Droplet-borne infection; low risk / high risk	-0.0159	-0.1815	0.1497	0.8508
	Previous history of pneumonia; no / yes	-0.1244	-0.2133	-0.0355	0.0061**
	Pneumococcal vaccine; not yet / finished	0.0118	-0.1535	0.1771	0.8889
Environment	Season; outdoor / indoor	0.0125	-0.0628	0.0879	0.7444
	Residence; welfare facility / Individual homes	0.0553	0.0273	0.0833	P<0.001**
	Smoke allowed environment / smokeless environment	-0.0034	-0.0891	0.0823	0.9383
Pathogen	Detection; negative / unknown / positive	0.0151	-0.0358	0.0660	0.5606
	Epidemic / unknown / resident flora	-0.1152	-0.1747	-0.0556	P<0.001**
	Drug resistant / unknown / drug sensitive	0.1419	0.0898	0.1940	P<0.001**
Disease	Affected region; multiple lobes / single lobe or less	0.1727	0.1001	0.2453	P<0.001**
	Parapneumonic pleuritis / pneumonia	0.2522	0.1214	0.3830	P<0.001**
Intervention	Respiratory rehabilitation; yes / no	0.0300	-0.0168	0.0769	0.2088
	Electrolyzed saline irrigation; yes / no	-0.2121	-0.3957	-0.0286	0.0235*

*; P<0.05, **; P<0.01, n; number of patients

Pneumonia is a serious disease with a mortality rate of 14%. Most pathogens were resident flora of air or droplet-borne contamination. Drug-resistant pathogens were associated with significantly poor outcomes. Males, an advanced age, patients with a poor performance status and comorbidities, recurrent pneumonia, and residents of welfare facilities had significantly poor outcomes. Severe pneumonia and empyema were associated with a significantly poor outcome. Of the clinical interventions analyzed, electrolyzed saline irrigation for empyema and viral pneumonia, particularly δ variant COVID19 pneumonia, rapidly improved disease outcomes. The low contribution of variable for pathogen detection on the poor disease outcome may have been masked by the effects of electrolyzed saline, which was used only for the positive detection of pathogens.

Table 5. Multivariate regression analysis of the morbidity of pneumonia

Objective variable: pneumonia group (1600) / control group (181)

Variable		regression coefficient	95% confidential interval lower limits	95% confidential interval upper limits	P value
Host	Sex; female / male	-0.0145	-0.0416	0.0126	0.2948
	Age; ≦75years / >75years	0.0054	-0.0423	0.0532	0.8242
	Current smoker / non-smoker	-0.1582	-0.2074	-0.1091	P<0.001**
	Brinkman Index; 0 / ≦ 1000 / 1000<	0.0001	-0.0056	0.0057	0.9763
	Nursing care service; no / yes	-0.0331	-0.0695	0.0033	0.0749
	Charlson's score; free, mild / moderate, sever	0.0193	-0.0109	0.0494	0.2100
	Comorbid respiratory disease; no / yes	-0.0287	-0.0566	-0.0008	0.0439*
	Droplet-borne infection; low risk / high risk	-0.1294	-0.1866	-0.0722	P<0.001**
	Previous history of pneumonia; yes / no	0.0571	0.0255	0.0887	P<0.001**
	Pneumococcal vaccine; not yet / finished	0.2750	0.2291	0.3209	P<0.001**
Environment	Season; outdoor / indoor	-0.0697	-0.0951	-0.0443	P<0.001**
	Welfare facility / individual homes	0.0002	-0.0097	0.0102	0.9615
	Smoke allowed environment / smokeless environment	-0.0305	-0.0604	-0.0006	0.0455*

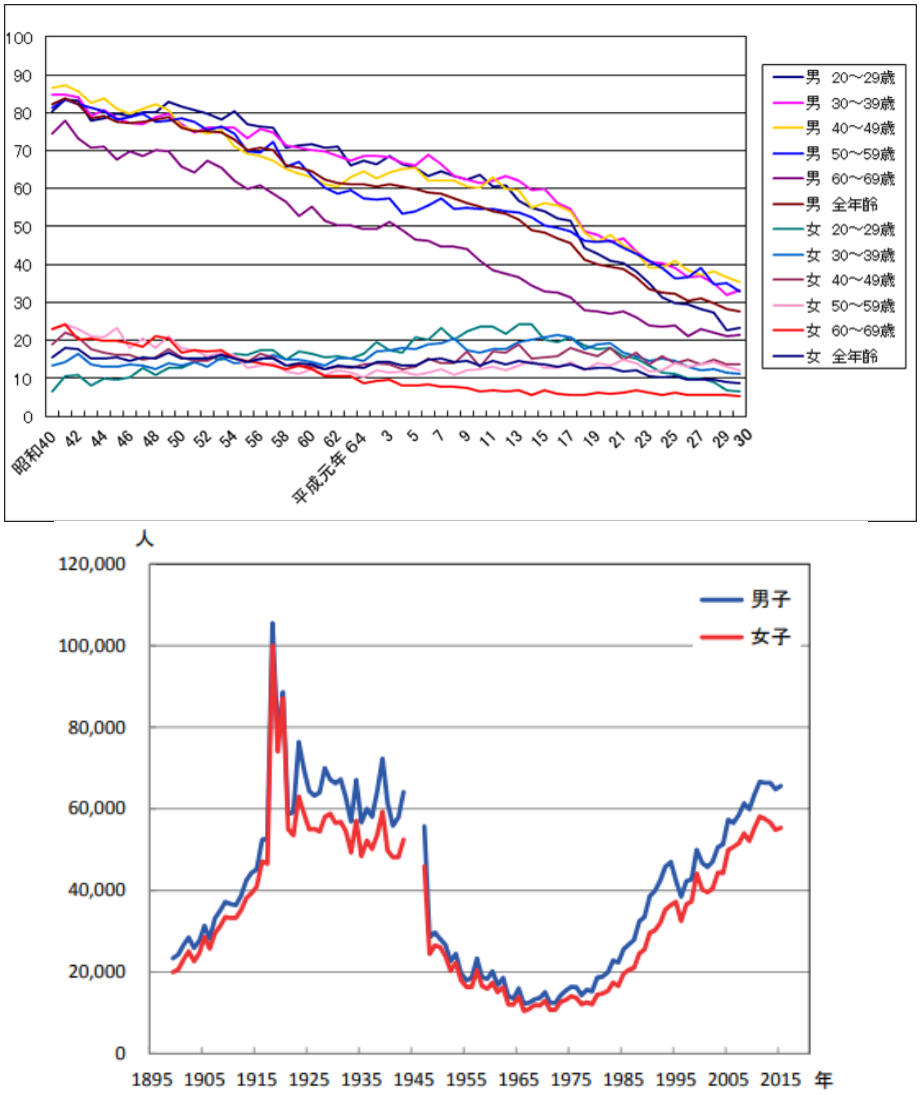
*: P<0.05 **: P<0.01, (n); number of patients or examinees

A multivariate regression analysis was used to investigate the effects of host and environmental site variables on the morbidity of pneumonia, including the control group. Vaccination resulted in the most significant decrease in the morbidity of pneumonia, followed by current smoking. The high-risk group for droplet-borne infections, individuals with a previous history of pneumonia, indoor seasons, and smokeless environments significantly increased the morbidity of pneumonia. Recurrent pneumonia may be attributed to continuous residence in a smokeless environment. High-risk factors for comorbid respiratory diseases may be chronic infection with *Pseudomonas* or MRSA in patients with bronchiectasis. Sex, age, and poor performance status were not associated with the morbidity of community-acquired pneumonia. Collectively, these results suggest that routine exposure to indoor smoke may reduce the morbidity of community-acquired pneumonia. In the present study, no adverse effects of so-called "passive smoking"[10] on pneumonia or comorbid respiratory diseases were observed, even though more than 95% of non-smokers in Japan are estimated to have been exposed to passive smoke from their parents or partners.

Discussion

The number of annual deaths from community-acquired pneumonia has markedly increased by 4- to 5-fold from the pre-1980s level of approximately 30,000 deaths in Japan, despite improvement in residential hygiene and a decrease in the prevalence of smoking.[1,2] Annual mortality is similar to that during Spain flu pandemics. (Figure 1)

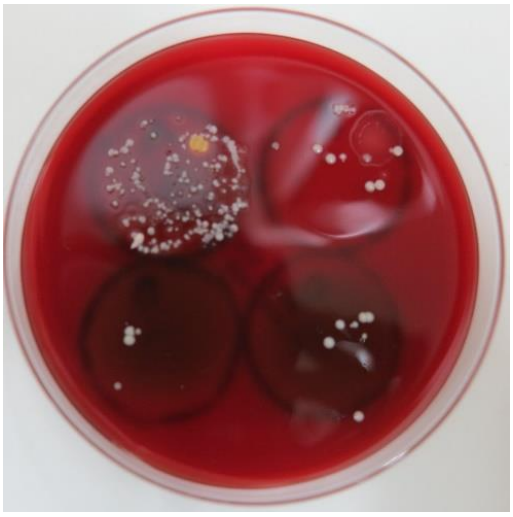
Figure 1. Annual changes in the prevalence of smoking and pneumonia mortality.



Upper; Annual changes in the prevalence of smoking according to sex and age (in decades) in1965-2018 in Japan[1] Lower; Annual changes in pneumonia mortality, excluding aspiration pneumonia, during 1895-2017 in Japan.[2] Blue: male, red: female.

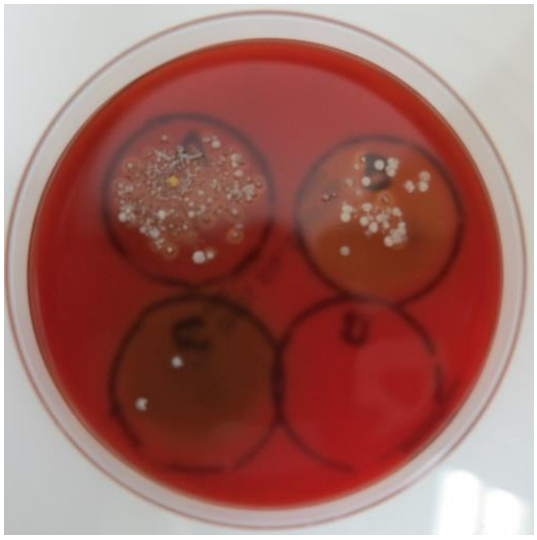
We previously suggested that the risk of pneumonia was higher in a smokeless indoor communal environment than in an environment where smoking is permitted.[3] A low dose of biomass smoke functions as a disinfectant against droplets and air-borne pathogens. (Figure 2,3)

Figure 2 Offensive disinfection activity of tobacco and incense stick smoke.



Stamp cultures of test plates on agar medium; ten vocalizations (“Ba, Bi, Bu, Be, Bo, Pa, Pi, Pu, Pe, and Po”) onto sterilized plastic plates placed 20~30cm from the mouth resulted in the formation of approximately 150CFU of bacteria and fungi. Biomass smoke from tobacco and incense sticks suppressed colony formation to a similar extent as electrolyzed saline. Top left, control; Top right, exposed to a single spray of electrolyzed saline mist; Bottom left, exposed to tobacco smoke for 5 minutes; Bottom right, exposed to the smoke of 6 incense sticks for 5 minutes[3].

Figure 3 Defensive disinfection activity of tobacco and incense stick smoke.



Vocalized droplets were deposited onto plastic plates that had been pre-exposed to biomass smoke in the same manner as Figure2. Top left, control; Top right, incense smoke; Bottom left, tobacco smoke[3].

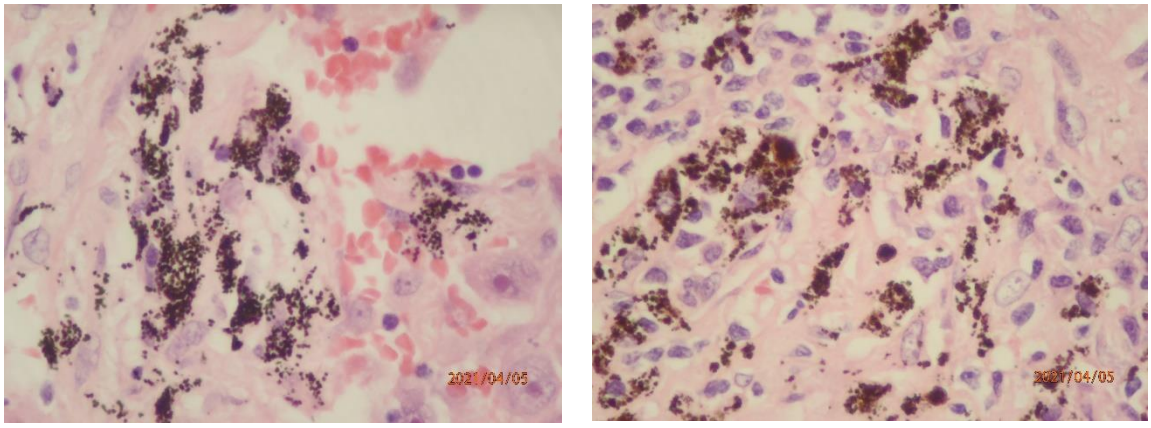
Biomass smoke has been shown to facilitate the engulfing of tar-labeled microbes, even novel viruses, as foreign particles by macrophages[4]. (Figure 4)

Figure 4. Gulfing activity of pulmonary macrophages against carbonic foreign particles.

Cut surfaces of a lung perfused with blood containing carbon black. Left; re-perfused isolated rabbit lung, control. Right; isolated rabbit lung designed to produce permeability edema by reperfusion. Leaked carbon black was promptly removed and transported to lobules by macrophages, producing leopard spots overnight[5].

Among current hygiene procedures, only biomass smoke disinfects room air contamination. The adverse effects of passive smoking are negligible, because quite small dose of smoke particle, diluted with air, is inhaled by passive smoking[11]. (Figure 5)

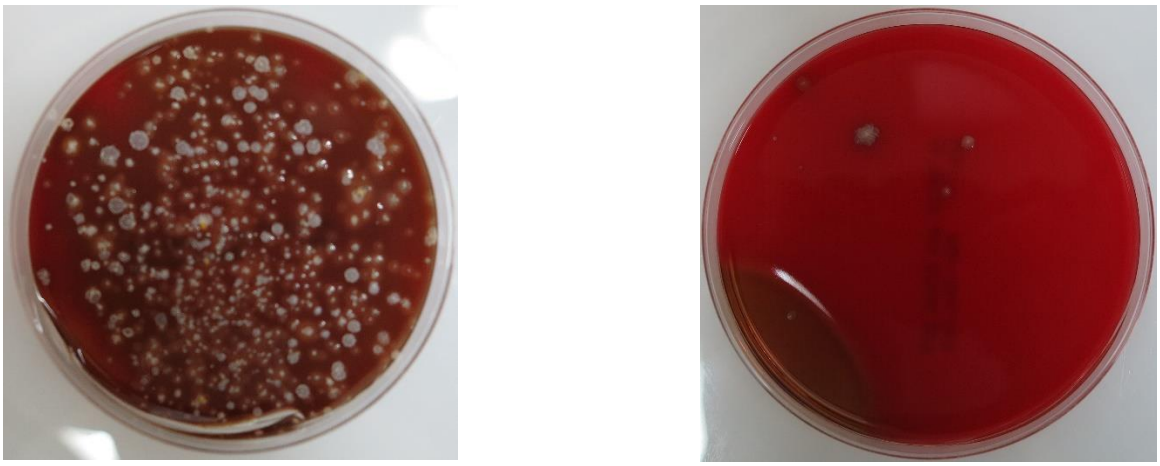
Figure 5. Pulmonary macrophages of early lung cancer.



Left: Early lung cancer specimen of a passive smoker whose parent and partner were heavy smokers. Macrophages are clear, while those in a heavy smoker (right) are stained in brown, smoke tar accumulation. Cabon particles in macrophages are vehicle tire dust.

Pneumonia is a serious disease, and its mortality rate was higher than that of COVID-19, reported in a mid-sized facility comparable our hospital[12]. A multivariate analysis of pathogens suggested that disease severity and outcomes depended on the pathogen load. The number of biological microbes was previously shown to be higher in vocalized droplets than in air[3]; therefore, the main cause of pneumonia was speculated to be droplet-borne infections. Among the host site variables examined in the present study, advanced age, serious comorbidities, and the use of nursing care services were the strongest risk factors for poor outcomes of pneumonia, followed by the presence of empyema, severe pneumonia, recurrent pneumonia, and drug-resistant pathogens. An intervention using electrolyzed saline was previously shown to significantly improve disease outcomes[13,14]. Electrolyzed saline is a novel disinfectant with broad-spectrum activity against viruses, bacteria, and fungi. It is currently considered a highly effective and versatile disinfectant that is non-toxic and easy to use[15]. (Figure 6). It may serve as a first-line barrier in future novel pandemics, but is currently available in Japan only.

Figure 6. Disinfection activity of electrolyzed saline gargling.



(A) Oral flora of vocalized droplets. (B) The oral flora of vocalized droplets was almost completely eliminated by electrolyzed saline gargling.

Vaccination and current smoking resulted in the greatest reductions in the rate of pneumonia. The outdoor season and environments where smoking is permitted may reduce both exposure to a high pathogen load and disease severity. An environment where smoking is permitted generally suppressed both epidemic and resident flora pathogens. Disease morbidity was significantly increased in the high-risk group for droplet-borne infections. Therefore, vaccination is strongly recommended for these patients. Disease morbidity was not dependent on the host-related variables of sex, age, and a poor performance status. Morbidity was mainly dependent on the resident environment. The present results showed that the main cause of community-acquired pneumonia was droplet-borne infections in indoor communal spaces with smoking restrictions. The results of the multivariate analysis support our hypothesis.

Biomass smoke has served as an incidental environmental disinfectant since humans began using biomass fuels for daily living[16]. The low incidence of COVID19 infections in refugee camps where biomass fuel was burned on the ground, one-sevenths that in the general population, provide support for the function of biomass smoke[17]. Biomass smoke exhibits both offensive and defensive antimicrobial activities against droplet-borne microbes[5]. (Figure 2, 3) Smoke particles kill microbes that adhere to the surface of the upper airway mucosa and may also intercept inhaled pathogens by attaching to the mucosal surface. Smoke tar has lipid affinity to the surface of microbes, which are covered with a lipid cell wall or envelope. Smoke particles are also charged with static electricity[18] and can readily capture negatively charged microbes.

Biomass smoke was eliminated from indoor spaces when biomass fuel was replaced with smokeless natural gas or electricity in the 1970s. Tobacco and incense smoke may represent the last remaining line of defense against biological contamination of indoor air in the absence of other alternatives. The hygiene level of environments where smoking is permitted is similar to that of semi-clean rooms[3]. The complete elimination of biomass smoke from community spaces has been suggested to increase the risk of transmission of pneumonia. Pure oxygen is toxic to biological tissues, whereas air, which comprises diluted oxygen and nitrogen gas, is essential for biological tissues. According to the same logic proposed by the toxicologist Paracelsus[19], a large amount of biomass smoke acts as an irritant to biological tissues; however, small doses may be essential for hygiene management in indoor communal spaces. Incidentally, the first outbreak of COVID19 in Japan was detected at the same time as legal smoking restrictions were initiated for indoor communal spaces on April 1, 2020[20]. The elimination of tobacco smoke from indoor communal spaces may decrease the security of public health and contribute to future novel pandemics.

Conclusions

The main cause of pneumonia is droplet-borne infections, which are suppressed in an environment where smoking permitted. The elimination of biomass smoke from indoor communal spaces may increase the morbidity of community-acquired pneumonia.

Conflict of Interest Statement:

None.

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