LUNG CENTER OF THE PHILIPPINES

Scientific Proceedings

Volume 14 No. 1 | January-June 2026 Issue | ISSN 0117-9322

WHAT'S INSIDE

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CLINICAL RESEARCH DEPARTMENT

The Clinical Research Department (CRD) oversees all research projects at the Lung Center of the Philippines (LCP). It receives, evaluates and coordinates all research activities. It establishes policies and guidelines for the development, writing, presentation and approval of research proposals. Thru its Technical Review Board (TRB), it provides guidance and technical expertise on protocol development, including sample size calculation and statistical analysis plan. It spearheads institutional researches and coordinates with other national and international agencies for clinical trials, student undergraduate and graduate research, and collaborative research. It runs the TB Research Team at the LCP's National Center for Pulmonary Research (NCPR) as well as spearheads the Lung Cancer Registry to gather and collate the comprehensive local data on pulmonary tuberculosis and lung cancer, respectively. It maintains the Clinical Research Facility (CRF), an establishment that provides room, space and storage facilities for clinical trials and research.

The CRD publishes the Scientific Proceedings, the official journal of the LCP, to share local relevant educational material in the field of pulmonary medicine. The Scientific Proceedings Journal publishes original clinical investigations, epidemiological studies, case reports, review articles, evaluation of diagnostic and surgical techniques, and latest updates on management guidelines.

In 2019, the CRD started to align with the vision and strategic direction of the LCP on research. The current challenges involve providing resources to support priority programs and projects with other departments to undertake institutional research on advanced procedures to support new clinical pathways, programs and policies and contribute to impact healthy lungs and healthy environment.

The department likewise is aligned with the National Unified Health Research Agenda 2021–2025 on [1] responsive health system [2] research to enhance and extend healthy lives [3] holistic approaches to health and wellness [4] health resiliency [5] global competitiveness and innovation in health and [6] research in equity and health.

In order to achieve these proposed strategic directions, the CRD reviews its accomplishment using the perspectives of the Balanced Scorecard in [1] learning and growth [2] internal business processes [3] customer satisfaction and [4] financial perspective. From these perspectives, the CRD hopes to monitor the outcomes of all action plans and to evaluate the implementation of such plans.

LISTING AND BRIEF DESCRIPTION OF AVAILABLE SERVICES

Registration of researches to be conducted at the LCP : processes all applications for Institutional Research, Clinical Trials, Student Undergraduate Research, Graduate Research and Collaborative Research.

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Clinical Research Facility: provides rental for room space, investigational product storage and archiving of completed research.

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Officer-in-Charge
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VERGEL A. AGUSTIN

CATHERINE G. MAGAS
MICHAELLA S. ROQUE
CESYL O. BRINQUIS

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Scientific Proceedings

Volume 14 No. 1 | January-June 2026 Issue | ISSN 0117-9322

Contact Information:
Clinical Research Department,
Lung Center of the Philippines,
Quezon Avenue Extension, Diliman,
Quezon City 1100
LCP Trunkline: (632) 8924-6101
LCP GSM Gateway SIM: 0917-837-9602
0998-964-5748

Extension Numbers: 4051 / 4052
E-mail: scientificproceedings@lcp.gov.ph
Website: lcpscientificproceedings.com

The **Scientific Proceedings**, the official journal of the Lung Center of the Philippines, is an open-access, English language, medical science journal, published biannually by the Lung Center of the Philippines. The journal intends toshare local relevant scientific findings in the field of respiratory medicine through publication of high quality original clinical investigations, epidemiological studies, case reports, review articles, evaluations of diagnostic and surgical techniques, and the latest updates on management guidelines. The journal's target audience are clinicians, surgeons, specialists, laboratorians, scientists, and researchers working on pulmonary medicine. The Scientific Proceedings does not charge any subscription, review, or manuscript processing fees.

The Scientific Proceedings is guided by the International Committee of Medical Journal Editors (ICMJE) "Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly Words in Medical Journals". (https://icmje.org)

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The Scientific Proceedings is a proud member of the Philippine Association of Medical Journal Editors with the aim of raising the quality of medical journal publishing in the country. (https://pamje.org)

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Scientific Proceedings

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MESSAGE FROM THE EXECUTIVE DIRECTOR

It is with renewed resolve that I welcome you to this latest issue of the *Scientific Proceedings of the Lung Center of the Philippines*. This publication reflects our institution's enduring commitment to advancing scientific knowledge, fostering research excellence, and improving respiratory health through evidence-informed clinical practice.

At the Lung Center of the Philippines, research is more than an academic pursuit. It is a catalyst for innovation, informed clinical decision-making, and better patient outcomes. Each contribution to this volume reflects the dedication, scholarly rigor, and collaborative spirit that define our scientific community. Through their work, our researchers, clinicians, educators, and healthcare professionals continue to advance the frontiers of knowledgAe and address the evolving challenges in respiratory health and patient care.

As the country's premier institution for pulmonary health, we recognize our responsibility to cultivate a vibrant research culture founded on perseverance, ethical integrity, and innovation. We remain steadfast in empowering investigators, forging strategic partnerships, and providing a trusted platform for the dissemination of high-quality research that advances national health priorities while contributing to the global body of medical knowledge. We cannot be THE Apex Center for Lung Health without contributing to our national agenda for research especially in the respiratory field. I am also excited to witness the take off of our translational research initiatives.

As we continue to build upon the legacy of the *Scientific Proceedings of the Lung Center of the Philippines,* may this publication inspire curiosity, and uphold the highest standards of scientific inquiry. Together, let us generate knowledge that advances respiratory medicine, useful to improve health policy and ultimately benefitting the patients and communities we are privileged to serve.

On behalf of the Lung Center of the Philippines, I extend my heartfelt gratitude to our authors, peer reviewers, members of the Editorial Board, and the many healthcare professionals whose expertise, dedication, and unwavering commitment have made this publication possible. Your shared commitment uphold our institution's mission.

Rest assured of the administration's strong and consistent support by providing necessary funding every year and crafting policies needed to further institutionalize research initiatives.

Dr. Jubert P. Benedicto
Executive Director
Education, Training and Research Services

MESSAGE FROM THE DEPUTY EXECUTIVE DIRECTOR

It is with great pride and enthusiasm that I welcome you to the latest issue of the Lung Center of the Philippines *Scientific Proceedings*, the official journal of the Lung Center of the Philippines.

This issue highlights the breadth and relevance of research being conducted within our institution and among our collaborators, addressing important questions that directly impact respiratory health and patient care in the Philippines. The featured studies reflect our continuing commitment to evidence-based medicine, scientific inquiry, and innovation.

The investigation on respiratory symptoms, lung function abnormalities, and cardiac conditions among jeepney drivers underscores the significant health consequences of environmental and occupational exposures in our communities. The study on ventilator-associated pneumonia provides valuable insights into risk factors that may guide preventive strategies and improve outcomes among critically ill patients. Likewise, the evaluation of pleural fluid adenosine deaminase in the diagnosis of tuberculous pleural effusion contributes meaningful local evidence to support clinical decision-making in tuberculosis care.

Complementing these original researches is a timely feature article on biobanking, which emphasizes the growing importance of preserving biological resources to advance translational research, precision medicine, and future scientific discoveries.

I commend our investigators, mentors, reviewers, and editorial team for their dedication to advancing knowledge and fostering a culture of research excellence. May this issue inspire continued collaboration, innovation, and the pursuit of scientific endeavors that ultimately improve the health and well-being of the Filipino people.

Dr. Virginia S. delos Reyes
Deputy Executive Director
Education, Training and Research Services

EDITORIAL

For the researchers of the Lung Center of the Philippines (LCP), the *Scientific Proceedings* represents more than just a repository of data; it is the foundational soil where a sustainable culture of publication takes root.

In the academic and medical community, the pressure to "publish or perish" often distorts the true purpose of scholarly writing. While high-impact factors and institutional prestige are frequently cited as the benchmarks of success, we must recalibrate our focus. The core of research is not the enhancement of a resume or the elevation of a center's ranking; it is the purposeful dissemination of scientific findings. A healthy research environment is one where writing is driven by the desire to share findings that could improve patient care.

However, with the privilege of sharing comes the weight of responsibility. Dissemination must be grounded in publication ethics, vigilance to "predatory journals," and responsible use of artificial intelligence: must be viewed as a competent aid to enhance and not to replace the critical thinking that defines a true researcher.

As we navigate the modern landscape, may the Scientific Proceedings serve a dual purpose: to provide authors with a dedicated space in the global scientific community to share their expertise and research findings, while simultaneously cementing the center's mandate as a training and research center for respiratory care. By starting here, we do more than just record data–we begin a legacy of transparency, ethical rigor, and a commitment to the global exchange of knowledge. Let us write not just to be known, but to be understood and to be useful.

Dr. Racquel C. Ibañez
Editor-in-Chief

EDITORIAL POLICIES

ABOUT THE JOURNAL

The **Scientific Proceedings**, the official journal of the Lung Center of the Philippines, is an open-access, English language, medical science journal, published by the Lung Center of the Philippines. Its policies are guided by the latest version of the International Committee of Medical Journal Editors (ICMJE) **"Recommendations for the Conduct, Reporting, Editing, and Publication of Scholarly Work in Medical Journals."**

FOCUS AND SCOPE

The **Scientific Proceedings** intends to share local relevant scientific findings in the field of respiratory medicine through publication of high quality original clinical investigations, epidemiological studies, case reports, review articles, evaluations of diagnostic and surgical techniques, and the latest updates on management guidelines. The journal's target audience are clinicians, surgeons, specialists, respiratory therapists, laboratorians, scientists, researchers working on pulmonary medicine, and policy makers.

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ORIGINAL ARTICLE

PREVALENCE OF RESPIRATORY SYMPTOMS, LUNG FUNCTION ABNORMALITIES, AND CARDIAC CONDITIONS IN JEEPNEY DRIVERS EXPOSED TO DIFFERENT LEVELS OF BLACK CARBON

Luisito F. Idolor, MD, Vincent M. Balanag Jr.*, MD, MSc, Mylene B. Cayetano**, PhD, Qi Zhao, MMD, PhD****
*Tamara Schikowski, PhD, MPH****

** National Center for Pulmonary Research, Lung Center of the Philippines*
*** Institute of Environmental Science and Meteorology, University of the Philippines–Diliman*
**** IUF–Leibniz Research Institute of Environmental Medicine, Duesseldorf*

ABSTRACT

Background & Aim: The jeepney, a traditional mode of transportation in the Philippines since the end of World War II, is a major source of black carbon (BC) in Metro Manila's air. Chronic exposure to black carbon may constitute a significant health hazard for jeepney drivers. This study assessed the adverse effects of BC exposure on the prevalence of symptoms, electrocardiogram (ECG), and lung function abnormalities in jeepney drivers in selected areas in Metro Manila.

Methods: Between December 2019 and March 2020, we studied the health status of jeepney drivers plying two routes with different levels of measured BC, using a modified version of the Burden of Obstructive Lung Disease (BOLD) questionnaire, ECG, and spirometry. The effects of BC severity and other respiratory risk factors, such as smoking, environmental exposures, and other occupational exposures, were also determined.

Results: One hundred three (103) jeepney drivers with a mean driving experience of 21.4 ± 11.3 years participated in the study, grouped according to the severity of BC levels in the route. Jeepney drivers on the route with higher BC severity reported five times more symptoms (p = 0.005) and showed three times more ECG abnormalities (p = 0.014) using univariate analysis. There was no substantial difference in lung function parameters. Logistic regression analysis showed that high BC levels accounted significantly for increased prevalence of symptoms (OR = 5.84 [95% CI: 1.60, 21.39], p < 0.01) and ECG abnormalities (OR = 3.2 [95% CI: 1.22, 8.37], p = 0.018) in the high BC group. No other respiratory risk factors were found to have significant effects.

Conclusion: The study shows the effect of BC levels on the prevalence of symptoms and ECG abnormalities in the highly exposed jeepney driver population. Further studies are required to determine the effect of BC exposure on lung function in this population.

Key words: *ambient black carbon, environmental epidemiology, cardiac conditions*

Corresponding Author:
Vincent M. Balanag, Jr., MD, MSc, FPCP, FPCCP

Institutional Affiliation:
Lung Center of the Philippines
Contact Number: 09175301331
E-mail: vmbalanag@gmail.com

Year Completed: 2021
Date Received: December 21, 2025
Date Accepted: January 5, 2026

Disclosure of Conflict of Interest:
The authors declare they have no real or perceived conflicts of interest. The use of trade names in this manuscript is for identification only and does not imply endorsement by the authors or their institutions.

Transport- or traffic-related air pollution has been shown to increase mortality from cardiopulmonary causes, increase the risk of respiratory symptoms and diseases not related to asthma or allergies, and cause impaired lung function and cardiovascular morbidity.[1-3]

Particulate matter measurements (like PM10 and PM2.5) have been correlated with levels of air pollution and adverse health effects in vulnerable populations such as bus drivers,4,5 taxi drivers,6–8 and traffic policemen.[9-11]

Black carbon (BC) is another component of particulate matter that is formed due to the incomplete combustion of fossil fuels, biofuels, and biomass, and is emitted directly into the atmosphere in the form of fine particles. Mortality and morbidity time are higher for BC than for PM10 or PM2.5 for each 1 µg/m3 increase in exposure.12–14 BC levels also varied more widely between weekends and weekdays (50 to 100%) compared to PM10 (5 to 15%).[15]

Traffic in the Metropolitan Manila area is said to be one of the worst in the world, with the iconic jeepney, a traditional mode of travel in the Philippines since the Second World War, contributing greatly to air pollution. With its pre-Euro4 diesel engine, the jeepney accounts for 94% of the total roadside emitted BC as soot, and up to 99% of the total measured PM2.5.[16]

It is therefore not surprising that jeepney drivers are also considered vulnerable to the effects of air pollution. Subida et al. reported a COPD prevalence of 32.5% among jeepney drivers, much higher than among commuters and bus drivers.[17]

The present study was conducted to determine the association of traffic-related air pollution measured in black carbon levels and adverse health effects in the vulnerable jeepney driver population.

This study aimed to compare the prevalence of respiratory symptoms, electrocardiogram, and lung function abnormalities among jeepney drivers operating in selected areas in Metro Manila with high and low levels of measured BC in the air.

METHODS

This was a cross-sectional study of jeepney drivers plying two selected routes in Metro Manila with different levels of measured black carbon (BC). The two routes were selected based on the observed volume of vehicular traffic and actual BC measurements. The high BC route, which passes through several congested roads, had an average BC level of 23.4 ± 14.0 µg/m^3, while the low BC route, which circumnavigates a tree-lined university campus, had an average ambient BC level of 6.2 ± 5.2 µg/m^3. Details of the BC levels are discussed elsewhere.[18] Criteria pollutants, such as gases (SO2, NO2, CO) with possible confounding effects, were not considered because data on these were not available. Survey data and health-related data of jeepney drivers plying the high BC route were categorically analyzed and labelled as "High Black Carbon" or HBC group, while those plying the low BC route were categorized as "Low Black Carbon" or LBC group.

Participants were licensed professional jeepney drivers who had regularly driven in the specified areas for at least five years and who were willing to provide personal information and undergo study procedures after signing an Informed Consent. Jeepney drivers with unstable co-morbid cardiac and pulmonary conditions, recent hospitalization for a medical illness, and ongoing treatment for active pulmonary tuberculosis were excluded. The study protocol was approved by the Technical Review Board and Ethics Review Board of the Lung Center of the Philippines.

Participants' vital signs, height, weight, and BMI, and medical history such as atopy, asthma, history of tobacco, alcohol, or drug intake, respiratory or any other previously diagnosed medical conditions were recorded. Participants' work characteristics and environment, such as educational background, mode of transport, years in the occupation, transit time (in hours) per day, and exposure to work-related indoor air pollutants, were also recorded. The study used the Filipino translation of the Burden of Obstructive Lung Disease (BOLD) Questionnaire with modifications (added items regarding solid fuel use and mode of transportation) to gather relevant information on environmental and occupational factors that may affect respiratory function. The Filipino translation has been validated and used in two national surveys.[19,20]

Pulmonary function was measured in forced vital capacity (FVC) and forced expiratory volume in 1 sec (FEV_1). Ratios of FEV_1 to FVC (FEV_1/FVC) were calculated. The measurements were performed according to methods recommended by the American Thoracic Society10 using the *EasyOne Air* device (Medizintechnik AG, Zurich, Switzerland). The highest of three acceptable measurements was taken as the actual value. The obtained data was compared with predicted values based on age, sex, height and ethnic group (non-smoking male adults) by Crapo et al.[21] Reduced values of FVC, FEV_1 and FEV_1/FVC were the primary criteria used to diagnose respiratory function impairment based on the following Global Initiative for Chronic Obstructive Lung Diseases (GOLD) criteria [21]: Normal: FEV_1 and FVC above 80% predicted; FEV_1/FVC ratio above 0.7; Obstructive: FEV_1 below 80% predicted; FVC can be normal or reduced, usually to a lesser degree than FEV_1; FEV_1/FVC ratio below 0.7; and Restrictive: FEV1 normal or mildly reduced; FVC below 80% predicted; FEV1/FVC ratio normal, above 0.7.

Cardiovascular status was evaluated through a review of cardiac symptoms and medical history, blood pressure measurements, physical examination, and official ECG reading by the in-house cardiologist.

Percentages were reported for categorical variables, while means ± standard deviation were obtained for quantitative variables. Differences in continuous variables (age, height, BMI, and duration on the job) between the two groups were computed using a t-test, and relationships between two categorical variables were tested using a chi-square test. Univariate analysis and logistic regression were performed to test the association between the presence of respiratory and other symptoms, ECG abnormalities, and spirometry abnormalities, and BC levels, as well as the interaction of risk factors like age, tobacco and biomass fuel use, and other environmental or occupational exposures. Odds ratios and the corresponding 95% confidence intervals were obtained. Statistical analysis was performed using Stata 15 software.

RESULTS

One hundred and three participants were recruited, with 49 jeepney drivers belonging to the HBC group and 54 to the LBC group. All of the participants were male. There were comparable distributions of age and comorbid conditions between the two groups. The LBC group had a slightly greater percentage of college-level education (Table 1).

Participants had been driving, on average, for 12 hours a day, five days a week, for more than 20 years, representing a significant exposure to air pollution. There were no significant differences between the two groups in terms of driving profile (hours per day, days per week, and total years of professional driving), smoking profile (years of smoking and total pack-years smoked), exposure to biomass fuel (charcoal and wood), and other environmental and occupational exposures (Table 1).

Most participants were smokers, with 38 (71%) in the LBC group and 33 (66%) in the HBC group reporting a history of smoking. Duration (in years) of smoking and estimated number of pack-years did not differ significantly between the two groups ($p = 0.781$) (Table 1).

Table 1. *Demographic and clinical profile of participants.*

Profile	LBC GROUP		HBC GROUP		
	NO.	%	NO.	%	*p*-value
Age (years)					
18–40	15	27.8	13	26.5	
41–50	16	29.6	17	34.7	0.239
51–60	13	24.1	16	32.7	
61 and above	10	18.5	3	6.1	
Gender					
Male	54	100	49	100	
Educational Attainment					
Elementary	13	24.1	10	20.4	
High School	25	46.3	30	61.2	0.277
College	16	29.6	9	18.4	
Co-Morbidity					
Allergy	8	14.8	8	16.3	0.523
Hypertension	7	13.0	7	14.3	0.535
Diabetes Mellitus	3	5.6	3	6.1	0.613
Asthma	5	9.3	3	6.1	0.414
Tuberculosis	2	3.7	6	12.2	0.106
Pneumonia	1	1.9	0	0.0	0.524
Fuel for Cooking					
Wood	9	16.7	7	14.3	0.477
Charcoal	14	25.9	20	40.8	0.081
LPG	53	98.2	48	98.0	0.728
Electric	9	16.7	11	22.5	0.311
Driving other vehicles	20	37.0	16	33.3	0.428
Burning trash	10	18.5	13	27.1	0.213
Farming	7	13.0	2	12.5	0.455
Roadside home	3	5.6	5	10.4	0.294
	Mean	SD	Mean	SD	*p*-value
Smoking Profile					
No. of years of smoking	19.1	16.1	17.3	14.6	0.557
Pack-years	14.6	17.7	10.4	12.7	0.175
Jeepney driving					
No. of hours per day	11.3	2.4	11.9	2.2	0.167
No. of days per week	4.8	0.8	4.5	0.9	0.087
Health profile on PE					
Height	1.6	0.1	1.6	0.1	0.112
Weight	69.9	10.3	66.5	10.0	0.094
BMI	25.8	3.4	25.2	3.6	0.359
SBP	122.9	19.2	124.6	13.7	0.602
DBP	83.7	12.2	83.4	9.0	0.875
HR	78.6	12.8	77.6	11.1	0.669
O2 Saturation	97.1	3.2	97.4	1.1	0.570

The HBC group reported more symptoms, with 15 (30.6%) reporting at least one respiratory or constitutional symptom compared with only 4 (7.4%) in the LBC group (p = 0.002). Shortness of breath was also more prevalent in the HBC group, with 12% compared with just 2% in the LBC group (p = 0.042). None of the participants reported any symptoms of malaise, anorexia, or hemoptysis. There were also more electrocardiographic abnormalities recorded in the HBC group (n = 19, 38.8%) compared to the LBC group (n = 9, 16.7%, p = 0.002). In particular, 13 jeepney drivers from the HBC group had signs of arrhythmia, significantly higher compared with six drivers from the LBC group (p = 0.039). Seven patients in the HBC group had ECG findings of ischemia compared with none in the LBC group (Table 2).

Table 2. *Comparison of symptoms and ECG findings of Low and High Black Carbon groups.*

	LBC GROUP		HBC GROUP		FISHER'S EXACT TEST
	NO.	%	NO.	%	*p*–value
Any symptom	4	7.4	15	30.6	0.002
Cough	2	3.7	5	10.2	0.180
Fever	0	0.0	1	2.0	0.476
Sputum	2	3.7	3	6.1	0.454
Weight loss	0	0.0	1	2.0	0.476
SOB	1	1.9	6	12.2	0.042
Night sweats	1	1.9	2	4.1	0.463
Chest/back pains	1	1.9	4	8.2	0.152
Any findings on ECG	9	16.67	19	38.78	0.011
Axis Deviation	2	3.70	2	4.08	0.654
Arrythmia	6	11.11	13	26.53	0.039
Atrial enlargement	1	1.85	4	8.16	0.152
Ischemia	0	0.00	7	14.29	0.004

**LBC: Low Black Carbon group; HBC: High Black Carbon group*

Univariate analysis showed that the presence of any symptom was significantly related to the level of BC exposure in their respective routes. Jeepney drivers from the HBC group were 5.5 times more likely to report any symptoms than the drivers from the LBC group (p = 0.005, 95% CI: 1.57, 19.7) (Table 3).

Table 3. *Univariate analysis of the association of the presence of symptoms with BC severity and other risk factors among jeepney drivers.*

Factor	*N*	With Symptoms (*n* %)	Odds Ratio	95% Confidence Interval of OR	*p*–value
Black carbon severity					
LBC group	54	4 (7.4)			
HBC group	49	15 (30.6)	5.51	1.68, 18.05	0.005
Use of charcoal for cooking					
No	69	9 (13.0)			
Yes	34	10 (29.4)	2.78	1.00, 7.68	0.049
Use of firewood for cooking					
No	87	15 (17.2)			
Yes	16	4 (25.0)	1.60	0.45, 5.65	0.465
Exposure to environmental pollutants					
No	42	5 (11.9)			
Yes	61	14 (23.0)	2.20	0.73, 6.68	0.162
No. of years of driving			0.98	0.94, 1.03	0.484
Pack-years of smoking			1.01	0.98, 1.04	0.641
Age			1.01	0.96, 1.05	0.741

On the other hand, driving-years, pack-years, use of biomass, and environmental exposure were not significant risk factors for the development of symptoms. Further logistic regression analysis showed that the BC severity was significantly associated with the presence of any symptom (Table 4).

Table 4. *Logistic regression analysis of the presence of symptoms with BC level and other risk factors among jeepney drivers*

Variable	Odds Ratio	Std. Err.	95% Interval	Conf.	*p*-value
Black carbon level	5.8401	3.8558	1.6011	21.301	0.008
Exposure to environmental pollutants	1.9544	1.2507	0.5575	6.9509	0.295
Use of firewood for cooking	1.1361	0.3354	0.2688	4.801	0.862
Use of charcoal for cooking	2.0415	1.1989	0.6457	6.4544	0.224
Age, years	1.0506	0.0446	0.9667	1.1418	0.244
Smoking, pack-years	1.0094	0.0197	0.9715	1.0488	0.631
Driving, years	0.9371	0.0393	0.8629	1.0715	0.122
Constant	0.0109	0.0186	0.0003	0.3065	<0.001

Log likelihood = -41.1166
p = 0.0229

Univariate analysis showed that the level of BC exposure was related to the prevalence of ECG abnormalities among the jeepney drivers (Table 5).

Table 5. *Univariate analysis of the association of abnormal ECG findings with BC severity and other risk factors among jeepney drivers.*

Factor	*N*	With ECG Findings (*n* %)	Odds Ratio	95% Confidence Interval of OR	*p*-value
Black carbon severity					
LBC group	54	9 (16.7)			
HBC group	49	19 (38.8)	3.17	1.26, 7.93	0.014
Use of charcoal for cooking					
No	69	17 (24.6)			
Yes	34	11 (32.4)	1.46	0.59, 3.61	0.409
Use of firewood for cooking					
No	87	23 (26.4)			
Yes	16	5 (31.3)	1.26	0.40, 4.03	0.691
Exposure to environmental pollutants					
No	42	9 (21.4)			
Yes	61	19 (31.2)	1.66	0.66, 4.14	0.278
No. of years of driving			0.99	0.95, 1.03	0.537
Pack-years of smoking			1.00	0.98, 1.03	0.794
Age			1.00	0.96, 1.04	0.922

Jeepney drivers from the HBC group were three times more likely to show abnormalities on ECG than drivers from the LBC group (p = 0.014). Logistic regression confirmed that High BC levels accounted for the increase in ECG abnormalities (Table 6)

Table 6. *Logistic regression analysis of the presence of ECG findings with BC level and other risk factors among jeepney drivers.*

Variable	Odds Ratio	Std. Err.	95% Interval	Conf.	*p*-value
Black carbon level	3.2013	1.5714	1.2231	8.3786	0.018
Exposure to environmental pollutants	1.5269	0.7761	0.5638	4.1349	0.405
Use of firewood for cooking	1.0559	0.6842	0.2965	0.2965	0.933
Use of charcoal for cooking	1.1145	0.565	0.4126	3.0103	0.831
Age, years	1.0295	0.0364	0.9605	1.1034	0.410
Smoking, pack-years	1.0045	0.0165	0.9727	1.0375	0.781
Driving, years	0.9646	0.0326	0.9026	1.0309	0.288
Constant	0.0710	0.0937	0.0053	0.9449	0.045

Log likelihood = -55.852
p = 0.2656

There were no significant differences in the lung function parameters (both pre- and post-bronchodilator) between the groups (Table 7).

Table 7. *Comparison of lung function values of Low and High BC groups.*

SPIROMETRY	LBC GROUP		HBC GROUP		T-TEST
	Mean	SD	Mean	SD	*p*-value
PRE-BD					
Pre-FEV Predicted	3.04	0.39	2.98	0.38	0.503
Pre-FEV Actual	2.64	0.52	2.61	0.54	0.732
Pre-FEV % Predicted	0.88	0.18	0.87	0.13	0.851
Pre-FVC Predicted	3.68	0.44	3.60	0.42	0.346
Pre-FVC Actual	3.31	0.50	3.26	0.53	0.628
Pre-FVC % Predicted	0.91	0.14	0.91	0.10	1.000
Pre-FEV/FVC Predicted	0.82	0.02	0.83	0.02	0.180
Pre-FEV/FVC Actual	0.78	0.09	0.80	0.08	0.463
Pre-FEV/FVC % Predicted	0.95	0.10	0.96	0.09	0.631
POST-BD					
Post-FEV Predicted	3.04	0.40	3.00	0.37	0.627
Post-FEV Actual	2.69	0.51	2.70	0.51	0.974
Post-FEV % Predicted	0.89	0.16	0.90	0.12	0.838
Post-FVC Predicted	3.68	0.44	3.61	0.42	0.431
Post-FVC Actual	3.33	0.51	3.27	0.51	0.551
Post-FVC % Predicted	0.91	0.15	0.91	0.10	0.767
Post-FEV/FVC Predicted	0.82	0.02	0.83	0.02	0.133
Post-FEV/FVC Actual	0.81	0.09	0.82	0.07	0.363
Post-FEV/FVC % Predicted	0.98	0.10	0.99	0.08	0.542

A total of twenty-eight or 27.2% of the whole study cohort had abnormal spirometry findings. Table 8 shows the univariate analysis of factors that may be related to findings of pulmonary function test (PFT) abnormalities. There were more PFT abnormalities seen in the LBC group (37.0%) compared with the HBC group (16.3%); thus, BC severity was paradoxically associated with a lower risk for PFT abnormalities (OR = 0.33; $p = 0.03$). The number of years of driving, the number of pack-years of smoking, and age showed a trend toward increased risk of PFT abnormalities.

Table 8. *Univariate analysis of the association of PFT abnormality with BC severity and other factors among jeepney drivers.*

Factor	*N*	With PFT Findings (*n* %)	Odds Ratio	95% Confidence Interval of OR	*p*-value
Black carbon severity					
LBC group	54	20 (37.0)			
HBC group	49	8 (16.3)	0.33	0.13, 0.85	0.021
Use of charcoal for cooking					
No	69	22 (31.9)			
Yes	34	6 (17.7)	0.46	0.17, 1.27	0.132
Use of firewood for cooking					
No	87	26 (29.9)			
Yes	16	2 (12.5)	0.34	0.07, 1.58	0.167
Exposure to environmental pollutants					
No	42	15 (35.7)			
Yes	61	13 (21.3)	0.49	0.20, 1.17	0.109
No. of years of driving			1.04	1.00, 1.08	0.061
Pack-years of smoking			1.03	1.00, 1.06	0.047
Age			1.03	0.99, 1.08	0.097

DISCUSSION

The present study measured the health effects of BC exposure on jeepney drivers in two categorized routes in Metro Manila, with an average of more than 20 years of driving their vehicles.

The results of the study show that exposure to higher levels of BC led to more reported respiratory or cardiac symptoms. Jeepney drivers in the HBC group were five times more likely to report any symptoms compared to those in the LBC group. It is widely recognized that acute or chronic exposure to air pollution is responsible for an increase in respiratory symptoms and diseases in communities or specific occupational groups.[22–26] There was also a higher prevalence of any electrocardiographic abnormality, ischemia, or arrhythmias seen in the high BC group (OR > 3). These abnormal ECG findings may indicate the presence of chronic cardiac conditions, but may also indicate the acute effects of recent exposure to air pollutants.

Exposure to particulate matter air pollution, whether acute or chronic, is associated with increased risk of death from cardiovascular diseases, including ischemic heart disease, heart failure, and ischemic/thrombotic stroke.[27] Levels of PM2.5 have consistently correlated with negative cardiovascular outcomes regardless of location.[28] Experimental studies have shown that exposure to increasing levels of ultrafine particulate matter in a controlled setting causes immediate electrocardiogram changes. Breitner et al.,[29] using four different study designs involving different study populations, exposure scenarios, and exposure levels, found evidence that recent exposures (within hours) to ultrafine particles and PM2.5 can induce acute pathophysiological responses that are apparent in ECG measurements. Another prospective panel study conducted in 56 males with ischemic heart disease showed the following ECG changes following 24 hours of exposure: a significant increase in QT duration in response to exposure to organic carbon; a significant decrease in T-wave amplitude with exposure to ultrafine, accumulation mode, and PM2.5 particles; and a corresponding significant increase in T-wave complexity in association with PM2.5 particles. Variability of T-wave complexity also significantly increased with exposure to components of the particulate matter (for the panel study mentioned, organic and elemental carbon) in the same time interval.[30]

We did not find significant differences in the FEV_1, FVC, and FEV_1/FVC ratio before and after inhalation of bronchodilators between the two groups. Univariate analysis showed that high BC exposure was not a

significant factor in the development of PFT abnormalities, with a higher percentage of PFT abnormalities seen with the LBC group. There was a trend toward a higher number of PFT abnormalities due to increasing age, pack-years of smoking, and number of driving years, but these were not statistically significant.

The effect of air pollution on the lung function of adults remains controversial. Few studies have assessed this association, but individual cohort studies from Europe,[31-33] the USA,[34] and Asia,[35] and a recent multi-cohort study of five European cohorts within the European Study of Cohorts for Air Pollution Effects (ESCAPE) project,[36] suggest adverse effects on adult lung function, despite having mixed results. The ventilatory effects are shown to be obstructive in some studies,[23,34,37] and restrictive in other studies.[29,31,32,35]

Several factors explain the lack of conclusive evidence of the adverse ventilatory effects of BC. The study by de Jong et al.[32] suggests that the association of PM10 and PM2.5 with impaired lung function levels in adults is stronger in subjects who are female, have higher BMI, and have underlying respiratory diseases such as asthma or COPD. Notably, our cohort consisted of all males, with borderline BMI, and low prevalences of asthma and COPD. A study in Parisian taxi drivers also did not find a relationship between BC levels and lung function,[38] but this was probably due to the very low BC levels of 2.2 to 3.9 ug/m3 measured inside the taxis compared to the 6.2 to 23.4 ug/m3 of BC measured in ambient air in our study. We also had a small sample size, making it the major limitation of this study. Our recruitment of study participants and data collection were cut short due to community lockdowns to prevent the spread of COVID-19. Future studies on the health impacts of the gaseous pollutants may also be considered once the data are made available. Certainly, more studies are needed to further define the associations of BC levels and other air quality criteria pollutants with lung function.

CONCLUSIONS

In conclusion, our study provides evidence of the categorized levels of chronic BC exposure that affect the local prevalence of symptoms and ECG abnormalities in the vulnerable jeepney driver population. There was no association between abnormalities in lung function parameters and high BC exposure in this cohort. However, further studies are required to determine the interactions between exposure to BC (and other risks) and lung function.

Author Contributions: Conceptualization: Tamara Schikowski, Luisito F. Idolor, Vincent M. Balanag, Mylene G. Cayetano; recruitment of study cohort: Mylene G. Cayetano, Sheryllyn M. Linda; Data curation: Luisito F. Idolor, Mylene G. Cayetano, Sheryllyn M. Linda; formal analysis: Vincent M. Balanag; funding acquisition: Tamara Schikowski; investigation: Luisito F. Idolor; methodology: Luisito M. Idolor, Vincent M.Balanag, Mylene G. Cayetano, Tamara Schikowski; Project administration: Luisito F. Idolor; Resources: Tamara Schikowski Qi Zhao, Luisito F. Idolor, Mylene G. Cayetano; Software: Sheryllyn M. Linda; Supervision: Luisito F. Idolor, Mylene G. Cayetano, Tamara Schikowski; Validation: Mylene G. Cayetano, Sheryllyn M. Linda; Writing – original draft: Vincent M. Balanag; Writing – review and editing: Vincent M. Balanag, Luisito F. Idolor, Tamara Schikowski, Mylene G. Cayetano, Sheryllyn M. Linda, Qi Zhao

FUNDING

This research was funded by the Federal Ministry of Education and Research (Bundesministerium für Bildung und Forschung, BMBF) No. URBAN_SEA2-034.

Institutional Review Board Statement: The study was conducted according to the guidelines of the Declaration of Helsinki, and approved by the Institutional Ethics Review Board of the Lung Center of the Philippines (protocol code LCP-CT-021-2019 on 28 November 2019).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study. All adult participants (Over 18 years old) provided written informed consent to participate in this study.

Data Availability Statement: Data supporting statements for high and low BC values will be provided once the doi is made available. Cayetano M.G., Linda S.M., Lamorena-Lim R., and Bagtasa G. (2021). *"Low and high emission zones at the pedestrian breathing level identified using multi-parameter air pollution measurements"*, Manuscript in preparation.

ACKNOWLEDGMENTS

Thank you to Clean Air Asia, Inc., for facilitating the BMBF funding. We also thank Prof. Peter G. J. Burney and his group at the BOLD Centre, UK, for permitting us to use the BOLD (Burden of Obstructive Lung Disease) questionnaire. Sincere gratitude to the jeepney operators and drivers' association and transport cooperative that participated in the study cohort, to our biostatistician Marianette T. Inobaya, PhD, for her invaluable help in assessing our data, to the Nurses and Radiology Technicians of Lung Center of the Philippines, and the researchers and staff of the Environmental Pollution Studies Laboratory of UPD-IESM (Enrique Mikhail Cosep, Raian Lapresca, Melody Batongbakal).

CONFLICTS OF INTEREST

The authors declare they have no real or perceived conflicts of interest. The use of trade names in this manuscript is for identification only and does not imply endorsement by the authors or their institutions.

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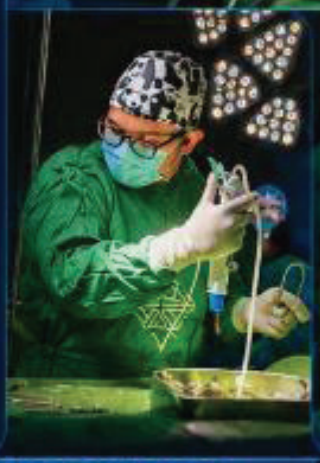

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ORIGINAL ARTICLE

RISK FACTORS FOR VENTILATOR–ASSOCIATED PNEUMONIA IN A TERTIARY HOSPITAL FROM JANUARY 2017 TO DECEMBER 2021

Dixie Mae R. Trinidad, MD, Mary Claire R. Orden, MD, FPCP, FPCCP** and Marie Charisma Laborte, MD, FPCP, FPCCP****

** Pulmonary Medicine Fellow–in–Training, Lung Center of the Philippines*
*** Critical Care Medicine Fellow–in–Training, Lung Center of the Philippines*
**** Pulmonary Medicine Consultant, Lung Center of the Philippines*

ABSTRACT

Background: Ventilator–Associated Pneumonia (VAP) is recognized as a significant nosocomial infection globally contributing to higher mortality. Currently, there are no local studies to document VAP prevalence and risk factors.

Objective: To determine the prevalence of VAP and the risk factors for VAP among intubated patients in the Lung Center of the Philippines (LCP) from 2017 to 2021

Methodology: This is a retrospective cohort study that reviewed the medical charts of adult intubated patients admitted to the critical care units of LCP from 2017–2021. The following data were extracted: the number of VAP cases and patient–, treatment–, and environment–related factors. Descriptive statistics were used to describe the baseline data. Binary Logistic Regression analysis was performed, reporting Odds Ratios (OR) with 95% Confidence Intervals. Multiple Regression analysis was utilized to assess predictors for continuous secondary outcomes, such as length of hospital stay. Chi–square test was used for determining associations between categorical variables where regression was not applicable. A p–value of < 0.05 was considered statistically significant. Data analysis was performed using Minitab statistical software.

Results and Discussion: Out of the 412 patients, 269 developed VAP (65.3%). The mean age was 61 years, with the majority being male (66%). Patient–related factors: sex, age, and smoking history were not associated with increased VAP risk, while Chronic Kidney Disease (CKD) was associated with lower risk of VAP (OR 0.11, $p = 0.03$). This inverse relationship was likely driven by the conservative fluid management and earlier extubation typical in renal patients. Admission diagnoses requiring intubation, specifically Community–Acquired Pneumonia High Risk (CAP–HR) (OR 2.67, $p = 0.01$) and Hospital–Acquired Pneumonia (HAP) (OR 3.84, $p = 0.04$), were associated with a higher risk of VAP, reflecting pre–existing lung inflammation and compromised local immunity. Treatment and environmental variables strongly influenced infection development; longer intubation days (OR 9.85, $p < 0.01$), tracheostomy (OR 6.86, $p < 0.01$), multiple antibiotic use (OR 1.13, $p < 0.01$), and a longer duration of hospital admission (OR 1.10, $p < 0.01$) were all linked to a higher risk of VAP, underscoring the hazards of prolonged device exposure and dysregulated microbial flora. Conversely, empiric antibiotic therapy (OR 0.37, $p < 0.01$) was associated with a lower risk, suggesting early suppression of initial colonizing pathogens. Regarding clinical outcomes, acute coronary syndrome, septic shock, and lung cancer were linked to higher mortality, whereas Chronic Obstructive Pulmonary Disease (COPD) was associated with lower mortality. Furthermore, HAP, cor pulmonale, late–onset VAP, multiple antibiotic use, dialysis, and tracheostomy significantly prolonged hospital stays. Notably, specific components of the VAP prevention bundle, including oral care and suctioning, correlated with increased mortality, a finding likely reflecting documentation bias among the most critically ill rather than direct causation.

Conclusions: The prevalence of VAP in the critical care units was high at 65.3%. Significant risk factors driving VAP development included admission for CAP–HR or HAP, prolonged intubation and hospitalization, tracheostomy, and multiple antibiotic use, while CKD and empiric antibiotic therapy were associated with a lower risk. We recommend standardizing VAP prevention bundles and strictly enforcing antimicrobial stewardship programs to mitigate these risks.

Keywords: *Ventilator–Associated Pneumonia (VAP), Nosocomial infections, Intubation, Risk factors, Critical care*

Corresponding author:
Dixie Mae R. Trinidad, MD

Institutional Affiliation:
Lung Center of the Philippines

Contact Number: 09192444037
E–mail: trinidaddixiemae@gmail.com
Year Completed: 2021
Date Received: October 20, 2025
Date Accepted: November 29, 2025

Disclosure of Conflict of Interest:
The investigators have no conflict of interest. This was presented as a poster at the American College of Chest Physicians (ACCP) Annual CHEST Convention in Boston, Massachusetts, on October 9, 2024, with support from a travel grant awarded by the Philippine College of Chest Physicians (PCCP).

Ventilator-Associated Pneumonia (VAP) is a form of pneumonia that develops more than 48 hours after mechanical ventilation and is a leading cause of hospital-acquired infections (HAI), contributing to high morbidity and mortality rates globally.[1] It affects approximately 5% to 40% of patients on a mechanical ventilator for over two days.[2] This variability may reflect differences in diagnostic criteria and hospital settings.[1] Understanding of VAP pathogenesis, risk factors, and prevention strategies has made notable strides. Identified risk factors include patient-specific variables like age and comorbidities, as well as treatment and procedural factors.[3]

However, challenges remain, such as inconsistent diagnosis and the rise of multidrug-resistant organisms (MDRO) due to improper antibiotic use. In regions like the WHO South-East Asia Region, VAP continues to be a major burden despite advancements in infection control practices.[4] Prevention efforts focus on bundled interventions, including head-of-bed elevation, sedation management, secretion drainage, oral care, and maintaining endotracheal cuff pressure.[5,6] While these measures have shown varying success, they remain promising in reducing VAP incidence.[7]

Thus, this study aimed to determine the VAP prevalence and the patient-related, treatment-related, and environment-related risk factors in adult intubated patients in the critical care units of Lung Center of the Philippines (LCP) from 2017 to 2021. Hospital-specific VAP incidences and risk factors are expected to inform tailored infection control strategies. Improved data collection and analysis can further refine prevention bundles and educational efforts, ultimately enhancing hospital policies and mitigating VAP's impact on patient outcomes.

METHODS

Study Design

This research utilized a retrospective cohort study design. Medical records of adult intubated patients admitted in critical care units of the LCP from 2017 to 2021 were reviewed to gather the target exposures, such as patient-related, treatment-related, and environment-related factors, and the outcome of interest, the occurrence of VAP, during their hospital stay.

Study Population and Duration

The target population included all admitted patients who were intubated (with a diagnosis of Acute Respiratory Failure [ARF] or Acute Respiratory Distress Syndrome [ARDS]) in LCP from January 1, 2017, to December 31, 2021. We included 1) intubated adult patients admitted to the Lung Center of the Philippines from January 1, 2017, to December 31, 2021, and 2) those who were admitted to a critical care unit or ward 3B. We excluded patients who 1) were admitted in the hospital for less than 48 hours, 2) signed limitation in medical care (with DNR or DNI directives), 3) were discharged against medical advice, 4) were transferred out to another facility, 5) were admitted as a transfer from another facility, or 6) had incomplete records or charts (unable to view Chest X-ray result and endotracheal aspirate culture result).

Sample Size and Sampling Strategy

Using G*Power 3.1.9.7, a minimum of 190 patients was required for this study based on an odds ratio of 1.9 for patients with COPD developing Ventilator-Associated Pneumonia.[8] This computation accounted for a 5% level of significance and 90% power. Total enumeration was used to achieve the desired sample size and maximize statistical power, with all charts screened and assessed for eligibility.

Study Procedure

A comprehensive roster of patients diagnosed with ARF and/or ARDS was retrieved from the medical records of the Lung Center of the Philippines. Each patient's chart underwent a detailed review by the principal investigator based on predefined inclusion and exclusion criteria. A final cohort was then identified, with each participant assigned a unique identifier code. Data were extracted by the principal investigator and a trained research assistant using a structured data collection tool from hospital charts and the WEB laboratory information system online database. VAP identification was based on the diagnosis of the attending physician and/or LCP-Infection Control Committee (ICC) records.

Data extracted included patient-related factors (age, sex, smoking history, comorbidities, reason for intubation, and etiologic agents), treatment-related factors (VAP bundle orders, duration of mechanical ventilation, antibiotic use history, and tracheostomy), and environment-related factors (ward of admission and duration of admission before VAP). Data were encoded in an MS Excel Worksheet by the trained research assistant. Data privacy and confidentiality were observed, and data protection measures (restricted access, de-identified data) were implemented. Regarding missing data, charts with incomplete critical records (missing Chest X-ray or endotracheal aspirate culture results) were excluded from the study (listwise deletion) to ensure data integrity.

Statistical Analysis

Descriptive statistics were used to summarize the data; categorical variables were reported as counts and percentages, while continuous variables were expressed as means and standard deviations. To determine the association between risk factors and dichotomous outcomes (such as VAP development and mortality), binary logistic regression analysis was performed, reporting Odds Ratios (OR) with 95% Confidence Intervals. Multiple regression analysis was utilized to assess predictors for continuous secondary outcomes, such as length of hospital stay. Chi-square test was used for determining associations between categorical variables where regression was not applicable. A p-value of < 0.05

was considered statistically significant. Data analysis was performed using Minitab statistical software.

Ethical considerations

This study was approved by and was under the oversight of the LCP-Institutional Ethics and Review Board (LCP-PF-009-2023) before implementation. A waiver of informed consent was secured from the LCP-IERB.

RESULTS

Recruitment

From January 2017 to December 2021, a total of 3,462 patients were identified with a diagnosis of ARF and/or ARDS (Figure 1). A total of 1,762 charts were successfully retrieved, and a total of 412 patients met the criteria for inclusion. A total of 1,350 patients were excluded for the following reasons: 535 were admitted for less than 48 hours, 367 had limitations of care such as do-not-intubate (DNI) and/or do-not-resuscitate (DNR) directives, 169 refused ICU admission, 117 were on non-invasive ventilation (NIV), 102 were intubated for less than 48 hours, 19 were transferred from another hospital, and 39 were discharged against medical advice. Of the 412 included patients, 269 developed VAP while 143 did not have VAP during admission.

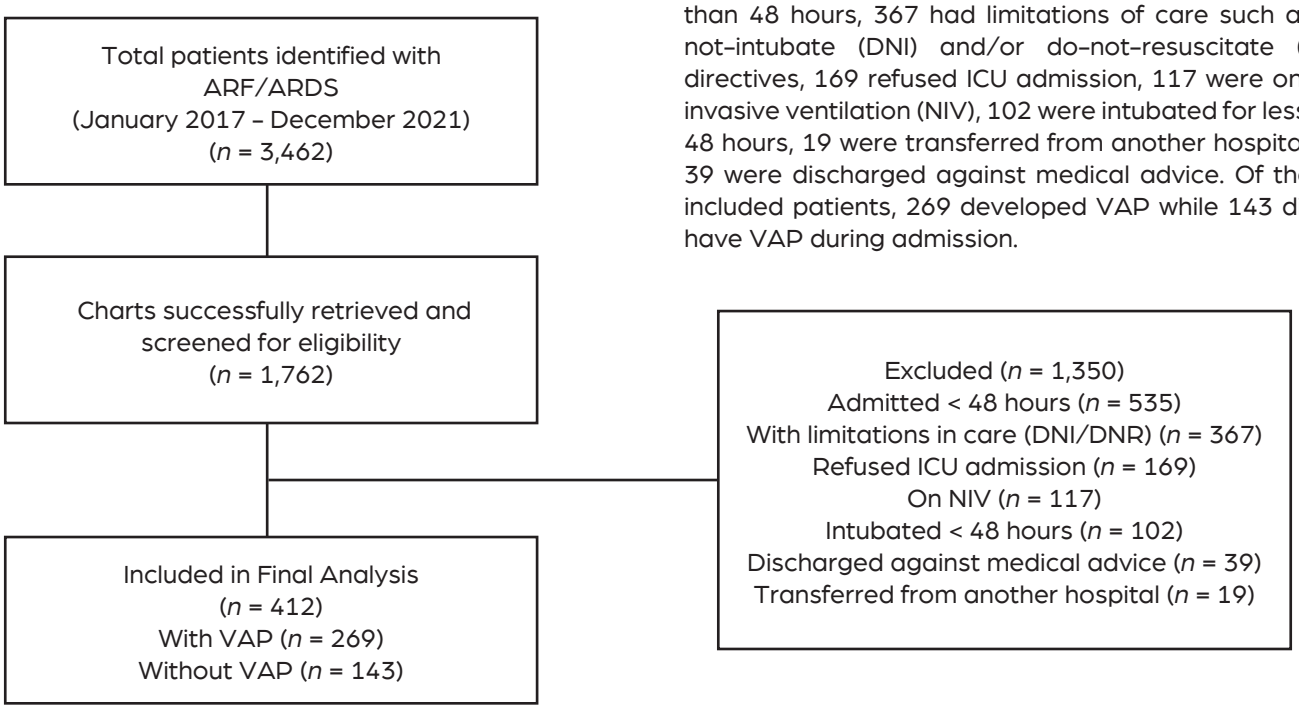
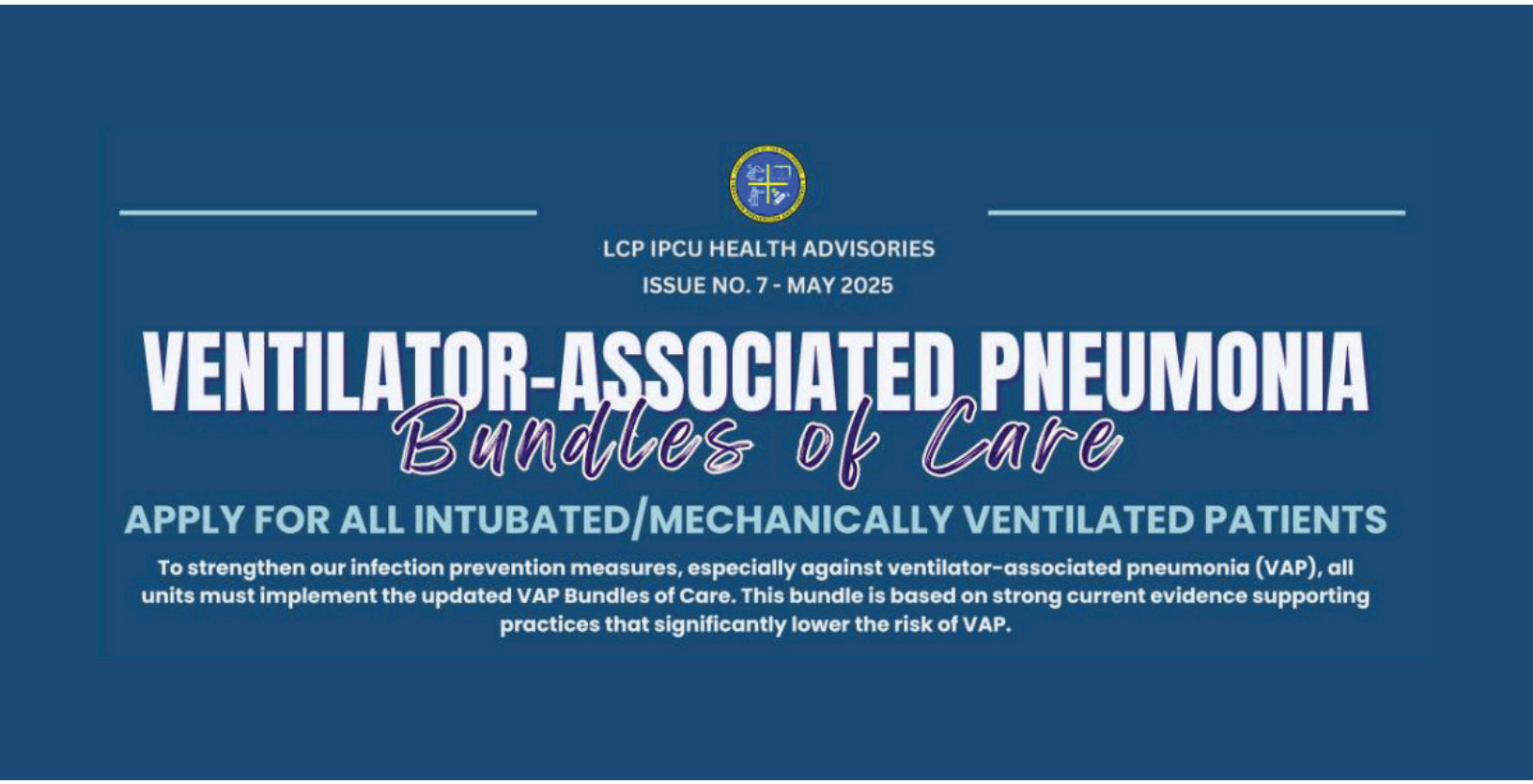


Figure 1. *Flow diagram showing patient selection, exclusion and final study population*



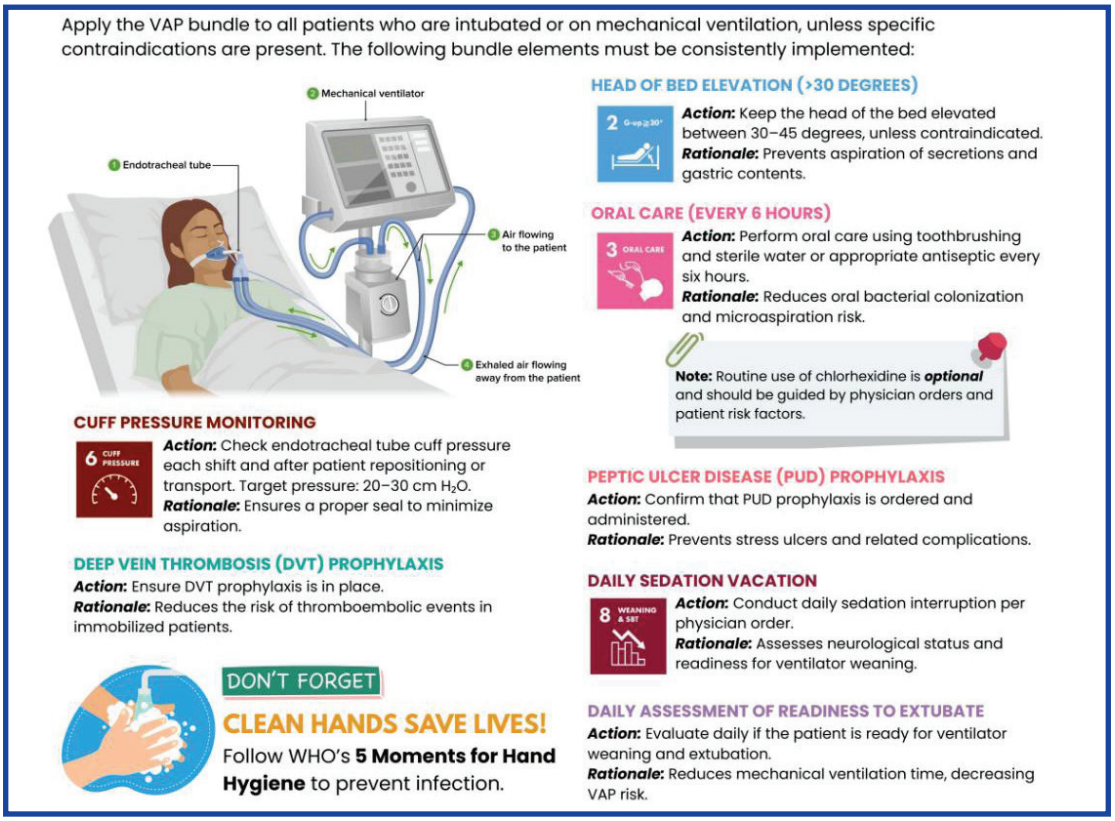
Demographic and Clinical Characteristics

A total of 412 intubated patients were included in the study (Table 1). The mean age was 61.21 years (SD 15.00), with a male predominance (66.01%). Hypertension (40.00%) was the most common comorbidity, followed by pulmonary tuberculosis (21.05%) and diabetes mellitus (20.53%). Demographic factors and comorbidities were generally similar between patients with and without VAP.

Table 1. *Baseline characteristics and presence of VAP*

	Ventilator–Associated Pneumonia		
	Total (*n* = 412)	With (*n* = 269)	Without (*n* = 143)
	Frequency (%); Mean ± SD		
Age, years	61.21 ± 15	61.52 ± 16.74	60 ± 14.57
Sex: Male	272 (66.01)	169 (62.82)	103 (72.02)
Female	140 (33.99)	100 (37.17)	40 (27.07)
Smoking history: Non–smoker	190 (46.11)	152 (56.50)	38 (26.57)
Smoker	170 (41.26)	115 (42.75)	55 (53.39)
Previous smoker	52 (12.62)	37 (13.75)	15 (10.48)
Comorbidity			
Hypertension	169 (40)	94 (35.83)	75 (46.38)
Pulmonary tuberculosis	88 (21.05)	82 (20)	6 (23.19)
Diabetes mellitus	82 (20.53)	54 (20.66)	28 (20.29)
COPD	81 (20.53)	45 (17.36)	36 (26.09)
Bronchial asthma	58 (14.21)	37 (14.05)	21 (14.49)
Post TB bronchiectasis	41 (10.53)	31 (12.4)	10 (7.25)
CVA/CVD	20 (4.74)	13 (5.79)	7 (2.9)
CKD	6 (2.63)	0	6 (7.25)
Lung Cancer	6 (2.63)	4 (3.31)	2 (1.45)
Malignancy other than lung	6 (2.63)	3 (2.48)	3 (2.9)
Other comorbidities	9 (4.74)	6 (4.96)	3 (4.35)

VAP = Ventilator–Associated Pneumonia, COPD = Chronic Obstructive Pulmonary Disease, CVA/CVD = Cerebrovascular Accident/Disease, CAD = Coronary Artery Disease, CKD = Chronic Kidney Disease, Other comorbidities = include Neuromuscular dystrophy, Cor pulmonale, Polycythemia, Kyphoscoliosis, Thymoma, Parkinson's Disease



Patient-Related Risk Factors for VAP

Among the comorbidities, CKD significantly reduced the odds of developing VAP by approximately 89% (Table 2). In contrast, specific reasons for intubation were strong predictors of VAP. Intubation due to CAP-HR more than doubled the risk of VAP, while HAP nearly quadrupled the odds compared to patients intubated for other reasons.

Table 2. *Patient-Related Factors and Risk of VAP Development*

	Coefficient	SE Coefficient	*P*-Value	Odds Ratio (OR)	95% CI for OR
Comorbidities					
Asthma	-0.26	0.37	0.48	0.77	(0.37, 1.59)
CAD	0.556	0.66	0.40	1.74	(0.47, 6.35)
CKD	-2.24	1.01	0.03*	0.11	(0.01, 0.77)
COPD	-0.10	0.41	0.81	0.90	(0.40, 2.03)
Cor Pulmonale	-15	325	0.96	0.00	(0.00, 6.01E+27)
CVA/CVD	0.28	0.64	0.66	1.32	(0.38, 4.67)
Diabetes Mellitus Type 2	0.30	0.35	0.39	1.35	(0.68, 2.70)
Hypertension	-0.01	0.29	0.98	0.99	(0.56, 1.74)
Lung Cancer	1.60	0.91	0.08	4.95	(0.83, 29.55)
Malignancy other than lung	-0.17	0.87	0.84	0.84	(0.15, 4.67)
Neuromuscular dystrophy	11.00	325	0.98	58384.71	(0.00, 1.55E+28)
Post TB bronchiectasis	0.34	0.67	0.62	1.40	(0.38, 5.25)
Others	1.64	1.19	0.17	5.18	(0.50, 53.72)
Reason for Intubation					
ACS/NSTEMI/STEMI	-0.19	0.39	0.64	0.83	(0.38, 1.80)
BAIAE	-0.33	1.57	0.83	0.72	(0.03, 15.66)
Central airway obstruction	12	325	0.971	118488.19	(0, 3.15E+28)
CAP HR	0.98	0.39	0.011 *	2.67	(1.25, 5.71)
COPDIAE	-0.04	0.44	0.93	0.96	(0.40, 2.29)
Cor Pulmonale	-1.04	0.69	0.13	0.35	(0.09, 1.36)
COVID 19	-0.22	0.66	0.74	0.81	(0.22, 2.93)
CVA/CVD	0	0.68	0.99	0.99	(0.26, 3.79)
Decompression sickness	12	325	0.97	143771.60	(0, 3.81E+28)
DRTB	1.25	1.16	0.28	3.50	(0.36, 33.82)
HAP	1.35	0.66	0.042*	3.84	(1.05, 14.05)
Lung Cancer	-0.59	0.65	0.36	0.55	(0.15, 1.98)
Malignancy other than lung	0.65	0.76	0.40	1.91	(0.43, 8.52)
Massive Hemoptysis	1.72	1.53	0.26	5.56	(0.28, 110.94)
Myasthenia gravis	-1	354	1	0.31	(0, 5.12E+30)
Pericardial effusion	-1.24	1.49	0.41	0.29	(0.02, 5.42)
Pleural Effusion	1.42	1.54	0.36	4.16	(0.20, 85.78)
Pneumohydrothorax	-12	325	0.97	0.00	(0, 1.66E+27)
Pneumothorax	0.57	1.63	0.73	1.78	(0.07, 43.74)
Post TB bronchiectasis IE	-0.61	0.58	0.30	0.54	(0.17, 1.71)
PTB	-0.91	0.72	0.21	0.40	(0.10, 1.66)
Pulmonary Congestion	0.06	1.42	0.97	1.06	(0.06, 17.26)
Pulmonary Embolism	-14.00	325	0.97	0	(0, 2.65E+27)
Sepsis/Septic Shock	0.36	0.39	0.36	1.43	(0.66, 3.06)

*Note: *Significant at p < 0.05 using Binary Logistic Regression Analysis; Deviance R-Sq = 14.59%; Deviance R-Sq(adj) = 2.23%*
VAP = Ventilator-Associated Pneumonia, CKD = Chronic Kidney Disease, CAP-HR = Community-Acquired Pneumonia High Risk, HAP = Hospital-Acquired Pneumonia, ACS = Acute Coronary Syndrome, COPDIAE = chronic obstructive pulmonary disease in acute exacerbation, NSTEMI = non-ST-segment elevation myocardial infarction, STEMI = ST-segment elevation myocardial infarction.

Microbiological Profile of Endotracheal Aspirates

Analysis of the 412 endotracheal aspirates revealed a predominantly gram-negative microbial landscape (Table 3). Acinetobacter baumannii was the most frequently isolated organism overall (12.63%), maintaining a consistent presence in both the VAP (12.40%) and non-VAP (13.04%) groups. This was followed by Candida species (11.05%) and Pseudomonas aeruginosa (10.53%). A distinct divergence in pathogen distribution was observed between the two cohorts. Specifically, Streptococcus pneumoniae was notably more prevalent among patients who did not develop VAP (10.14%) compared to those who did (2.48%). Conversely, opportunistic nosocomial pathogens, including Acinetobacter lwoffii (6.61%) and Enterococcus species (4.13%), were isolated exclusively in the VAP cohort and were absent in the non-VAP group.

Table 3. *Microbiological Profile of Endotracheal Aspirates*

	Total (*n* = 412)	With VAP (*n* = 269)	Without VAP (*n* = 143)
	Frequency (%); Mean + SD		
ETA organism isolated			
Acinetobacter baumannii	49 (12.63)	32 (12.4)	17 (13.04)
Candida sp	45 (11.05)	29 (11.57)	16 (10.14)
Pseudomonas aeruginosa	41 (10.53)	24 (9.92)	17 (11.59)
Klebsiella pneumoniae	24 (6.84)	13 (4.96)	11 (10.14)
Streptococcus viridans	24 (6.32)	15 (5.79)	9 (7.25)
Streptococcus pneumoniae	20 (5.26)	5 (2.48)	15 (10.14)
Acinetobacter lwoffii	16 (4.21)	16 (6.61)	0
Coagulase Negative Staphylococcus	16 (4.21)	10 (4.13)	6 (4.35)
Escherichia coli	12 (3.16)	10 (4.13)	2 (1.45)
Enterococcus	10 (2.63)	10 (4.13)	0
Enterobacter cloacae	8 (2.11)	5 (2.48)	3 (1.45)
Staphylococcus aureus	8 (2.11)	4 (1.65)	4 (2.9)
Stenotrophomonas maltophilia	8 (2.11)	5 (2.48)	3 (1.45)
Sphingomonas paucimobilis	8 (2.11)	4 (1.65)	4 (2.9)
Serratia marcescens	6 (1.58)	4 (1.65)	2 (1.45)
Burkholderia cepacia	6 (1.58)	4 (1.65)	2 (1.45)
Enterococcus faecalis	6 (1.58)	4 (1.65)	2 (1.45)
Klebsiella oxytoca	6 (1.58)	6 (2.48)	0
Elizabethkingia Meningoseptica	6 (1.05)	3 (0.83)	3 (1.45)
Staphylococcus warneri	6 (1.05)	3 (0.83)	3 (1.45)
Pseudomonas flourescens	6 (1.05)	3 (0.83)	3 (1.45)
Moraxella sp	6 (1.05)	6 (1.65)	0
Staphylococcus saprophyticus	2 (0.53)	2 (0.83)	0
Pseudomonas luteola	2 (0.53)	2 (0.83)	0
Streptococcus sanguinis	2 (0.53)	2 (0.83)	0
Pseudomonas stutzeri	2 (0.53)	2 (0.83)	0

Treatment and Environmental Risk Factors

Tracheostomy was the strongest independent risk factor, increasing VAP risk nearly sevenfold (Table 4). Similarly, prolonged mechanical ventilation (>4 days) increased the likelihood of VAP nearly tenfold. The use of multiple antibiotics during hospitalization quadrupled the odds of developing VAP, whereas the use of empiric antibiotic therapy was associated with a 63% reduction in risk compared to targeted therapy. Additionally, every day of hospital admission before intubation increased the odds of VAP by 10%. Conversely, neither the specific ward of admission nor individual VAP bundle components ordered at intubation significantly influenced VAP development.

Table 4. *Treatment and Environmental Factors and VAP Risk*

	Coefficient	SE Coefficient	*P*-Value	Odds Ratio (OR)	95% CI for OR
Treatment-Related Factors					
Use of Bundle of Care Head of Bed Elevation (30°–45°)	0.24	0.22	0.29	1.27	(0.82, 1.97)
Oral Care	0.18	0.22	0.42	1.20	(0.77, 1.85)
Proton Pump Inhibitor (PPI) Use	0.48	0.35	0.17	1.62	(0.81, 3.22)
Suctioning	0.56	0.3	0.07	1.75	(0.96, 3.17)
Duration of Mechanical Ventilator Use					
Intubation > 4 days	—	—	< 0.01*	9.85	—
MDRO Risk Factors					
Multiple Antibiotic Use	1.48	0.26	<0.001*	4.40	(2.64, 7.34)
Empiric Antibiotic Therapy	–0.98	0.21	<0.001*	0.37	(0.25, 0.57)
Chemotherapy/Radiotherapy	0.67	0.84	0.42	1.96	(0.38, 10.08)
Current Hospitalization > 5 days	0.43	0.4	0.28	1.54	(0.70, 3.40)
Dialysis Use	0.04	0.31	0.89	1.04	(0.57, 1.91)
Immunocompromised State	0.61	0.52	0.24	1.83	(0.66, 5.05)
Previous Antibiotic Use	0.03	0.32	0.92	1.03	(0.55, 1.94)
Tracheostomy	1.93	0.53	<0.001*	6.86	(2.41, 19.52)
Environment-Related Factors					
Ward of Admission					
MICU / RICU / SICU / 3B / STU	—	—	>0.05	NS	NS
Duration of Admission					
Duration before VAP	0.09	0.01	<0.001*	1.1	(1.07, 1.13)

**Significant at p < 0.05*

Factors Associated with Mortality

Among patient-related factors, the reason for intubation was a critical predictor for mortality (Table 5). Patients intubated due to Sepsis or Septic Shock had nearly a 12-fold increase in mortality odds, while those with Lung Cancer had a fivefold increase. Acute Coronary Syndrome (ACS) also significantly tripled the risk of death. Conversely, intubation due to COPD exacerbation was associated with an 80% reduction in mortality risk compared to other causes.

Regarding treatment-related factors, interventions intended for VAP prevention were associated with higher mortality in this cohort. Orders for suctioning and oral care were associated with a twofold increase in mortality risk. Additionally, chronic dialysis was a strong predictor, increasing mortality odds by nearly five times. Interestingly, a current hospitalization duration of more than five days before VAP onset was associated with a 68% reduction in mortality, possibly reflecting a survival bias where early mortality cases were excluded. Environmental factors, including the specific ward of admission, did not significantly impact mortality outcomes.

Table 5. *Factors Associated with Mortality*

	Coefficient	SE Coefficient	*P*-Value	Odds Ratio (OR)	95% CI for OR
I. Patient-Related Factors					
Demographics					
Age	0.01	0.01	0.37	1.01	(0.99, 1.03)
Sex (Female vs Male)	-0.47	0.39	0.23	0.63	(0.29, 1.34)
Smoking (Non-Smoker)	-0.08	0.43	0.85	0.92	(0.39, 2.16)
Comorbid Conditions					
COPD	-1.02	0.54	0.06	0.36	(0.13, 1.04)
Lung Cancer	-0.79	0.87	0.37	0.45	(0.08, 2.52)
Diabetes Mellitus	0.2	0.41	0.62	1.22	(0.55, 2.70)
Hypertension	-0.05	0.34	0.88	0.95	(0.48, 1.85)
Asthma	-0.45	0.47	0.34	0.64	(0.25, 1.61)
Reason for Intubation					
Sepsis / Septic Shock	2.47	0.54	<0.001*	11.77	(4.06, 34.11)
Lung Cancer	1.63	0.7	0.02*	5.12	(1.31, 19.99)
ACS / NSTEMI / STEMI	1.22	0.45	<0.01*	3.37	(1.41, 8.06)
COPD Exacerbation	-1.6	0.78	0.04*	0.2	(0.04, 0.93)
CVA / CVD	1.43	0.83	0.08	4.19	(0.83, 21.19)
COVID-19	0.66	0.78	0.4	1.93	(0.42, 8.83)
CAP-HR	-0.01	0.44	0.97	0.99	(0.42, 2.33)
HAP	-0.09	0.77	0.91	0.91	(0.20, 4.14)
II. Treatment-Related Factors					
VAP Bundle Orders					
Suctioning	0.78	0.35	0.03*	2.18	(1.10, 4.34)
Oral Care	0.66	0.23	<0.01*	1.93	(1.23, 3.02)
PPI Use	-0.25	0.37	0.49	0.78	(0.38, 1.59)
Head of Bed Elevation	-0.32	0.22	0.16	0.73	(0.47, 1.13)
Interventions & MDRO Risks					
Dialysis Use	1.57	0.3	<0.001*	4.78	(2.63, 8.68)
Current Hosp > 5 Days	-1.14	0.43	<0.01*	0.32	(0.14, 0.74)
Immunocompromised State	0.86	0.46	0.06	2.37	(0.96, 5.86)
Immunosuppressive Drug	0.5	0.29	0.09	1.65	(0.93, 2.92)
Multiple Antibiotic Use	0.13	0.28	0.66	1.13	(0.65, 1.98)
Empiric Antibiotics	0.01	0.2	0.97	1.01	(0.68, 1.50)
Intubation > 4 Days	-0.29	0.26	0.27	0.75	(0.45, 1.26)
III. Environment-Related Factors					
Ward of Admission					
MICU (Reference)	—	—	—	1	—
RICU	-0.68	1.12	0.54	0.51	(0.06, 4.54)
SICU	0.45	0.89	0.61	1.57	(0.27, 8.99)
Ward 3B	-12.5	450	0.98	0	(0.00, >100)
STU (Special Treatment Unit)	13.5	215	0.95	>100	(0.00, >100)

**Significant at p < 0.05*
Note: Coefficients, Standard Errors (SE), and Odds Ratios (OR) are rounded to 2 decimal places.

Factors Associated with Length of Hospital Stay

Among the reasons for intubation, Hospital-Acquired Pneumonia (HAP) and Cor Pulmonale were significant predictors of prolonged stay, showing substantial positive coefficients. Environmental factors also played a role; notably, late-onset VAP and a longer duration of admission before VAP diagnosis were strongly associated with extended hospital stays. Treatment-related factors

such as tracheostomy and multiple antibiotic use were also linked to longer admissions. Conversely, intubation due to lung cancer or CKD did not significantly extend the length of stay in this cohort.

Table 6. *Factors Associated with Duration of Hospitalization*

	Coefficient	SE Coefficient	*P*-Value
I. Patient-Related Factors			
Demographics			
Age	0.03	0.07	0.63
Sex (Female)	2.23	2.5	0.37
Smoking (Non-Smoker)	-1.03	2.82	0.72
Previous Smoker	-1.72	2.65	0.52
Comorbid Conditions			
Asthma	4.18	2.9	0.15
CAD	-2.93	5.12	0.57
CKD	-6.26	7.46	0.4
COPD	-1.37	3.3	0.68
Cor Pulmonale	-7.2	19.7	0.72
CVA/CVD	-3.38	4.87	0.49
Diabetes Mellitus	-0.1	2.68	0.97
Hypertension	1.04	2.24	0.64
Lung Cancer	11.43	6.59	0.08
Reason for Intubation			
HAP	13.17	5.07	0.01*
Cor Pulmonale	12.45	5.47	0.02*
Sepsis/Septic Shock	5.16	3.03	0.09
Lung Cancer	-7.72	5.23	0.14
COVID-19	-5.99	5.15	0.25
CAO (Central Airway Obstruction)	-21.3	19.2	0.27
Pulmonary Congestion	-12	11	0.28
CAD	6.22	6.05	0.31
Pulmonary Embolism	-18.5	19.5	0.34
Pericardial Effusion	-10.8	11.9	0.37
Massive Hemoptysis	6.74	8.65	0.44
Obstructive Pneumonia	4.82	7.62	0.53
COPD Exacerbation	-1.6	3.49	0.65
Pneumothorax	6	13.3	0.65
ACS/NSTEMI/STEMI	-0.81	3.07	0.79
CAP-HR	0.18	2.8	0.95
II. Treatment-Related Factors			
Interventions & MDRO Risks			
Tracheostomy	–	–	<0.001*
Multiple Antibiotic Use	–	–	<0.001*
Dialysis Use	–	–	<0.001*
Late-Onset VAP	–	–	<0.001*
Duration of Admission	1.1	–	<0.001*
III. Environmental-Related Factors			
Ward of Admission (Ref: MICU)			
RICU	–	–	>0.05
SICU	–	–	>0.05
Ward 3B	–	–	>0.05
STU (Special Treatment Unit)	–	–	>0.05

**Significant at p < 0.05*

DISCUSSION

This study at our institution identifies key risk factors for the development of VAP in critical care units. In this study, baseline demographic characteristics such as age, sex, smoking history, and general systemic comorbidities were not significantly associated with the development of VAP. While ARF and ARDS were highly prevalent among elderly males with comorbid conditions, these clinical profiles were equally distributed between the VAP and non-VAP groups and did not correlate with increased mortality or prolonged hospital length of stay. While these findings challenge several historically established cohorts that identified advanced age and systemic comorbidities as primary drivers of VAP,[9,10] they strongly align with a growing body of contemporary literature. Recent investigations have similarly demonstrated that baseline demographics are frequently overshadowed by device-related variables and the acute severity of the underlying illness. This paradigm shift suggests that in modern critical care settings, a patient's susceptibility to VAP is driven less by their intrinsic baseline characteristics and more by the profound environmental disruption and duration of invasive mechanical ventilation itself.

Notably, CKD was associated with a reduced incidence of VAP. This inverse relationship may reflect clinical management differences and selection bias rather than true physiological protection. CKD patients often require aggressive fluid restriction and closer hemodynamic monitoring; literature confirms that such conservative fluid strategies promote negative fluid balance, shorter ventilator days, and earlier extubation, thereby narrowing the risk window for VAP.[11] However, when renal impairment progressed to require dialysis, this intervention was associated with both VAP development and mortality, likely reflecting the profound vulnerability, immune dysregulation, and overall severity of illness in patients requiring invasive organ support. Furthermore, this apparent protection may be compounded by selection bias, such as the earlier transfer of these complex patients, and the routine administration of broad-spectrum, renal-adjusted antibiotic regimens that incidentally suppress early airway colonization.

The microbiological profile of endotracheal aspirates underscores a distinct ecological shift in the airways of intubated patients. The dominance of gram-negative organisms, specifically Acinetobacter baumannii, Pseudomonas aeruginosa, and Klebsiella pneumoniae, aligns with the known pathogenesis of VAP. These resilient pathogens rapidly form protective biofilms on endotracheal tubes, creating a persistent reservoir for micro-aspiration and subsequent infection.[12] Comparing the VAP and non-VAP cohorts clarifies the crucial distinction between airway colonization and nosocomial superinfection. The higher prevalence of Streptococcus pneumoniae in the non-VAP group likely reflects residual colonization from their initial community-acquired pneumonia (CAP) after targeted therapy. Conversely, the exclusive isolation of Acinetobacter lwoffii and Enterococcus species in the VAP cohort highlights their role as true opportunistic, device-associated pathogens that emerge under the selective pressure of broad-spectrum empiric antibiotics.[13] Finally, the prominent isolation of Candida species (11.05% overall) and its independent association with mortality warrants attention. Often dismissed as benign airway colonization, its heavy presence is clinically significant. Extensive Candida colonization serves as a surrogate marker for profound host immunosuppression, prolonged ICU stays, and fungal overgrowth following heavy antibiotic exposure.[14] Furthermore, dense Candida colonization can promote the growth and virulence of co-existing bacteria like Pseudomonas aeruginosa, complicating clinical recovery and driving up mortality.

Conversely, admission diagnoses of high-risk community-acquired pneumonia and hospital-acquired pneumonia significantly increased the odds of developing ventilator-associated pneumonia. This predisposition is likely driven by a combination of pre-existing lung inflammation, prior heavy antibiotic exposure, and the subsequent disruption of normal respiratory flora. The initial pneumonic insult causes diffuse epithelial damage and impairs ciliary clearance.[15] When combined with an endotracheal tube, this compromised microenvironment facilitates a clear pathogenic sequence. Specifically, the pre-existing pneumonia necessitates prolonged mechanical ventilation, which in turn provides an extended opportunity for bacterial colonization and biofilm formation, ultimately resulting in the development of ventilator-associated pneumonia.[12]

Therapeutic interventions and device utilization heavily influenced both the development of ventilator-associated pneumonia and patient mortality. The duration of mechanical ventilation directly correlated with disease incidence and extended hospital stays, reinforcing the fundamental critical care principle that limiting intubation time is paramount. Similarly, tracheostomy was significantly associated with both the development of the infection and mortality. However, our retrospective analysis did not stratify tracheostomy by timing, such as early versus late intervention. This is a critical distinction, as literature demonstrates that delayed tracheostomy prolongs endotracheal intubation and increases colonization risk. Early tracheostomy, typically performed within the first seven to ten days, has been shown to reduce overall disease incidence and facilitate faster weaning.[16,17]

Antimicrobial strategies also yielded complex outcomes. While multiple antibiotic use was strongly tied to the development of ventilator-associated pneumonia and mortality, an observed inverse association between empiric antibiotic therapy and disease development requires careful contextualization. This apparent protective effect may stem from early broad-spectrum

coverage effectively suppressing early-onset pathogens. Alternatively, it may reflect confounding by indication, wherein patients presenting with profound sepsis receive immediate empiric therapy, and their clinical outcomes, including mortality, are determined rapidly by their primary illness before the pneumonia has the opportunity to develop.[18] Consequently, critical care practitioners must continuously balance the immediate life-saving benefits of early empiric therapy against the vital principles of antimicrobial stewardship to prevent the subsequent selection of multidrug-resistant organisms.

Analysis of prevention bundles revealed inconsistent implementation across the institution. Individual components, such as proton pump inhibitors, head of bed elevation, and venous thromboembolism prophylaxis, did not significantly alter the risk of developing ventilator-associated pneumonia. Strikingly, orders for oral care and secretion suctioning were associated with increased mortality. This association likely reflects documentation and selection bias; these specific orders are frequently documented more rigorously in the most critically ill patients who inherently carry a higher mortality risk, rather than suggesting a direct causal harm from oral hygiene. Environment-related factors, specifically the specific ward or intensive care unit of admission, showed no significant association with VAP development. This indicates a uniform standard of critical care delivery and baseline infection control practices for mechanically ventilated patients across the hospital's various departments.

LIMITATIONS

Several limitations must be considered when interpreting these findings. First, the study is constrained by its retrospective design, which is inherently vulnerable to missing documentation, unstratified variables (such as the exact timing of tracheostomies), and difficulties in verifying non-standardized bundle adherence. Second, there is a potential for misclassification of VAP due to diagnostic heterogeneity, as clinical and radiographic criteria frequently overlap with other underlying pulmonary pathologies. Finally, the single-center design limits the broad generalizability of our results, as local microbial ecologies and institutional protocols heavily dictate VAP dynamics.

RECOMMENDATIONS

Given that baseline diagnoses of CAP-HR and HAP directly increased the odds of developing VAP, we recommend enhanced surveillance and targeted prevention strategies for these specific patient populations upon admission. Additionally, future prospective studies should investigate the potentially protective patient-level clinical mechanisms, such as conservative fluid management, observed in those with CKD. Regarding treatment-related factors, the significant associations of prolonged mechanical ventilation, tracheostomy, and multiple antibiotic exposure with both VAP development and subsequent mortality highlight the need for the institutional implementation of protocolized weaning strategies, early tracheostomy evaluations, and stringent antimicrobial stewardship programs. Furthermore, because our institution lacked a formalized VAP bundle checklist during the study period, relying instead on individual orders, and because specific interventions like oral care were unexpectedly associated with higher mortality, we recommend that healthcare facilities transition to unified, protocolized VAP prevention bundles. Regular audits of these formalized bundles are highly encouraged to evaluate true bedside implementation fidelity, while future research should explore optimal antibiotic regimens and durations to minimize multidrug-resistant organism development. Finally, to address environmental and institutional variables, healthcare facilities should consider standardizing VAP diagnostic criteria across all critical care units to ensure consistent reporting and infection control. To comprehensively advance this field, future multi-center, prospective studies with larger sample sizes and longer observational periods are essential to mitigate the inherent biases of this single-center retrospective design and validate these findings across diverse clinical settings.

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LUNG CENTER OF THE PHILIPPINES
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ORIGINAL ARTICLE

DIAGNOSTIC PERFORMANCE AND OPTIMAL CUT-OFF VALUES OF PLEURAL FLUID ADENOSINE DEAMINASE IN DIAGNOSING TB PLEURAL EFFUSION AMONG ADULT PATIENTS AT THE LUNG CENTER OF THE PHILIPPINES: A CROSS-SECTIONAL ANALYTICAL STUDY

*Jellicoe Maine Asuncion-Lugtu, MD**
*Bonn Ma, MD **
*Racquel Ibanez, MD, FPCP, FPCCP***

**Adult Pulmonary Medicine Fellow-in-Training, Pulmonary, Critical Care, and Sleep Medicine Department*
***Medical Specialist, Pulmonary, Critical Care, and Sleep Medicine Department*
Lung Center of the Philippines

ABSTRACT

Background: Tuberculosis (TB) remains a major global health problem, with 10.6 million cases and 1.3 million deaths reported by the World Health Organization in 2023. In the Philippines, 591,000 cases and 31,000 deaths were recorded in 2020. Tuberculous pleural effusion (TPE) is common but diagnostically challenging due to the low sensitivity of conventional tests.
Pleural fluid adenosine deaminase (pfADA) is a simple, rapid, and cost-effective biomarker for TPE. Its performance may vary by population. While international studies support a 40 U/L cut-off, data suggest lower thresholds for settings with a high TB burden, such as the Philippines. This study aims to evaluate the diagnostic accuracy of pfADA and determine optimal cut-off values in adult Filipino patients.

Objectives: The general objective is to appraise pfADA's diagnostic effectiveness in TB pleural effusion compared to other standard references. Specific goals included determining pfADA's accuracy, sensitivity, specificity, positive and negative predictive values, and discriminative ability through ROC curves. Through subgroup analyses, we sought optimal cut-off points for differentiating TB from non-TB effusions in patients with TB, age >45, and a history of TB treatment at least two years prior.

Methods: This retrospective, analytical, cross-sectional study involved adults with exudative, lymphocytic-predominant pleural effusion at the Lung Center of the Philippines (April-November 2023). We used ROC curves and Youden's J index to ascertain pfADA's optimal cut-off, and compared pfADA against GeneXpert TB/MTB and TB culture.

Results: Out of 660 cases, we extensively reviewed 146. The mean age was 45 years, with pfADA exhibiting a notable difference in various diagnostic categories. sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) stood at 82.08%, 92.40%, 90.16%, and 85.66%, respectively. The optimal cut-off value was 47.2 U/L. Stratifying by age, the study affirmed that patients aged 45 and older require a lower cut-off value at 25.2 U/L (sensitivity 72.34% and specificity 80.95%, respectively). The study also showed that retreatment cases of tuberculosis had a higher cut-off value at 94 U/L (sensitivity 88.89 and 48.08, respectively).

Conclusion: The optimum cut-off value for pfADA among the Filipino population is 47.2U/L. The study justifies pfADA's diagnostic performance in TB pleural effusion. The findings align with existing literature, highlighting pfADA's potential as a dependable TPE marker. However, given the study's single-center nature and limited sample size, broader multi-center validations are advocated for enhanced generalizability.

Keywords: Tuberculosis, pleural effusion, adenosine deaminase, diagnostic performance, cut-off point

Corresponding Author:
Bonn Ma, MD, FPCP, FPCCP

Institutional Affiliation:
Lung Center of the Philippines

Contact Number: 09267461917
E-mail: bonnyongit@gmail.com
Year Completed: 2025
Date Received: October 30, 2025
Date Accepted: November 5, 2025

Disclosure of conflict of Interest:
The investigators have no conflict of interest (COI) This study received no grant from any funding agency.

Tuberculosis is still a global health problem, especially in low-income countries. In 2023, WHO reported that TB afflicted an estimated 10.6 million worldwide and caused 1.3 million deaths, making it the 13th leading cause of mortality from a single infection. The global incidence of people newly diagnosed with TB was 7.5 million, the highest number since WHO began global TB monitoring in 1995.[1] In 2020, 591,000 people in the Philippines developed TB, while 31,000 people died.[2] Extrapulmonary disease is the initial presentation in about 25% of patients, involving lymph nodes and pleura.[3] TB is currently one of the most frequent causes of exudative pleural effusion worldwide.[4]

Tuberculous pleural effusion (TPE) is a paucibacillary manifestation of tuberculosis, diagnosed through examination of the pleural fluid, including bacteriological tests and biomarker detection.[5] Despite TPE being a common clinical entity, it can be difficult to diagnose. Conventional smear microscopy of pleural fluid has low yield rates of less than 10%.6 Even rapid molecular tests such as Xpert Mycobacterium tuberculosis/ Rifampicin resistance (Xpert MTB/RIF) report a wide range of sensitivity, from 28% to 81% in pleural fluid and 90% in pleural tissue.[5] While medical thoracoscopy has a sensitivity of up to 100% for TPE and can improve culture and Xpert MTB/RIF yield rates, it is invasive and not always available in resource-limited settings. Pleural fluid adenosine deaminase (pfADA) is a simple, rapid, inexpensive surrogate marker for tuberculous pleural effusion (TPE) that is used particularly to rule out the disease. A cut-off value of 40 U/L is currently being used based on overseas data. However, the level of pleural pfADA is influenced by several factors, which include geography (high TB burden vs low TB burden countries), age, and comorbidities (malignancy, previous history of TB treatment, etc.). The use of pleural fluid ADA has been limited in our locality, as only a few centers can perform such tests. Furthermore, only one local study has evaluated its diagnostic value and importance.

To bridge these gaps, this study aimed to evaluate the diagnostic performance of pleural fluid ADA compared with the composite standard of care references in a Filipino population and determine the different cut-off values to diagnose TB effusion appropriately for patients with different age groups and comorbidities.

MATERIALS AND METHODS

Study Design

This was a retrospective, analytical, cross-sectional study of medical records of adult patients who presented with exudative, lymphocytic-predominant pleural effusion.

Study Setting

This was a single-center study of patients' medical records managed at the Lung Center of the Philippines from April 2023 to November 2023.

Study Population

The study included adult patients with pleural effusion (e.g., malignancy, pneumonia, heart failure, and chronic kidney disease) identified through non-probability sampling, who underwent pleural fluid drainage via thoracentesis, video-assisted thoracoscopic surgery, thoracoscopy, chest tube thoracostomy, pigtail catheter insertion, or pleural catheter placement. Eligible participants were Filipino patients aged ≥19 years with imaging-confirmed pleural effusion (chest radiograph, computed tomography, or ultrasound) and exudative, lymphocyte-predominant pleural fluid (lymphocytes >50%).

Only cases with pleural fluid adenosine deaminase (pfADA) measured at the Lung Center of the Philippines and a complete composite reference standard work-up were included. The composite reference standard consisted of sputum and pleural fluid Gram stain and culture and sensitivity, GeneXpert, acid-fast bacilli smear, and tuberculosis culture; pleural fluid glucose, protein, and lactate dehydrogenase; corresponding serum studies; and pleural fluid cytology.

Patients were excluded if pfADA testing was performed in external laboratories, pfADA results were unavailable, the composite reference standard was incomplete, or duplicate specimens were identified. For patients undergoing multiple thoracenteses, only the first pleural fluid sample was included in the statistical analysis, although all samples were reviewed to establish the reference standard diagnosis. A positive culture, GeneXpert, or acid-fast bacilli smear in any specimen was considered diagnostic.

Among the 146 lymphocytic predominant pleural effusion cases, 61 were Bacteriologically confirmed tuberculosis, 39 were clinically diagnosed tuberculosis, 41 were Not Tuberculosis without malignancy, and 5 were not TB with malignancy.

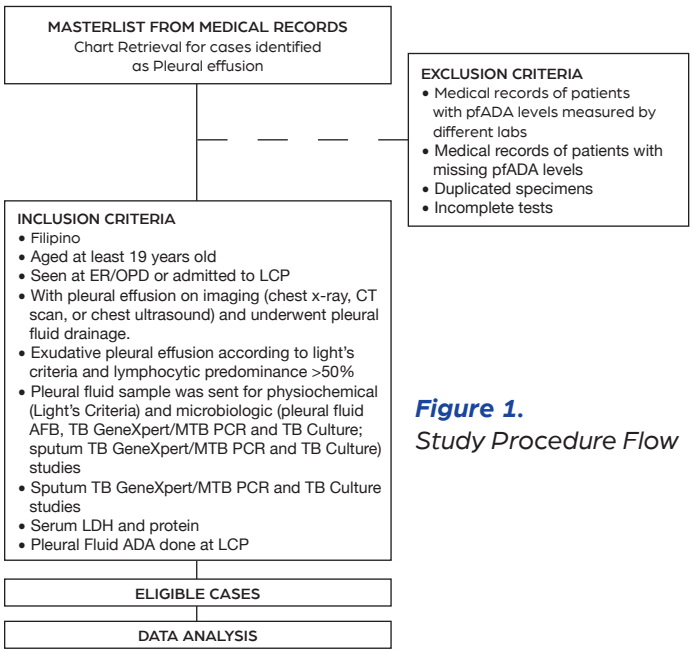

Figure 1.
Study Procedure Flow

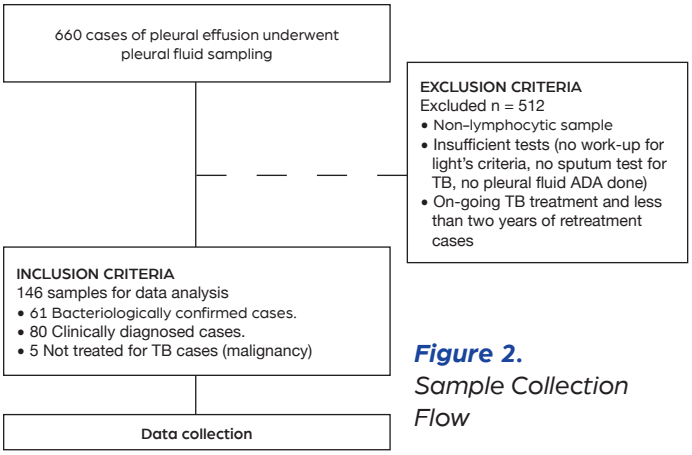

Figure 2. *Sample Collection Flow*

Study Instrument/Intervention

Descriptive statistics were used to summarize participant characteristics, using frequency and proportion for categorical variables, mean/standard deviation for normally distributed interval/ratio variables, while median/interquartile range was used for non-normally distributed ones. The Shapiro-Wilk test was used to assess data normality. For determining the optimal cut-off of pleural fluid adenosine deaminase (pfADA) in tuberculosis pleural effusion detection, a receiver operating characteristic (ROC) curve was constructed. The AUC-ROC indicated accuracy, categorized as failing, poor, exemplary, or excellent. Youden's J index was used to calculate overall diagnostic effectiveness, with the maximum value determining the optimal cut-off. PfADA's diagnostic performance-using sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), positive likelihood ratio (LR+), negative likelihood ratio (LR-), and accuracy-was compared against a composite reference standard comprising GeneXpert MTB/Rlf, TB Lamp, AFB smear, cytology, and TB culture, serving as the gold standard.

Study Procedure

The study was conducted from April to November 2023. Approval was obtained from the Institutional Review Board of the Lung Center of the Philippines before study initiation. To ensure confidentiality, all records were anonymized using coded identifiers.

Data were extracted from the medical charts, including baseline characteristics (age, sex, smoking history, comorbidities, tuberculosis exposure, and clinical features), imaging findings (chest radiograph, computed tomography, and ultrasound), and procedural details. Pleural fluid analyses collected included clinical chemistry (pleural fluid ADA) and microbiologic tests (pleural fluid TB culture, GeneXpert/PCR, AFB smear; sputum TB culture, GeneXpert/PCR, and AFB smear).

Outcome Measures

The diagnostic performance of pleural fluid adenosine deaminase (ADA) for tuberculous pleural effusion was analyzed for sensitivity, specificity, positive and negative predictive values, likelihood ratios, and receiver operating characteristic (ROC) curve analysis, with the optimal cut-off defined as the value maximizing sensitivity and specificity.

Primary outcomes included the accuracy and diagnostic indices of pleural fluid ADA compared with a composite reference standard across bacteriologically confirmed tuberculosis, clinically diagnosed tuberculosis, and malignant cases. The study also aimed to determine the optimal ADA cut-off values for differentiating tuberculosis from non-tuberculous exudative pleural effusions among patients aged ≥45 years and those with ≥2 years of tuberculosis treatment.

Pleural fluid ADA was the primary predictor. Potential confounders included age and prior tuberculosis treatment; effect modification was assessed by categorizing cases according to diagnostic category, age group, and treatment history.

Table 1. *Case classification among the study population*

DIAGNOSIS	NO OF PATIENTS (%)
Bacteriologically Confirmed	61 (42.1)
Clinically Diagnosed TB	39 (26.9)
Not Clinically Diagnosed TB Without Malignancy	41 (28.3)
Not Clinically Diagnosed TB With Malignancy	5 (3.4)
TOTAL	146

TB = Tuberculosis

Diagnostic Criteria

The ADA test is preferred over the IFN-gamma test in countries with a high prevalence of TB because of its long history of success, simplicity, and cost-effectiveness.[7] ADA was made available in the St. Luke's Medical Center in May 2019 (Danganan MD, Tan W. Diagnostic value of adenosine deaminase in tuberculous pleural effusion among adult patients in St. Luke's Medical Center Quezon City. Unpublished data, 2021) The normal value of ADA in pleural fluid at SLMC-QC is 0-30 U/L, and as a highly sensitive test, a result of <30 U/L can exclude the diagnosis of tuberculous pleural effusion.

Tuberculous pleural effusion was diagnosed microbiologically when at least one pleural fluid microbiologic test (GeneXpert, TB culture, or AFB smear) yielded a positive result. Pleural fluid cytology

demonstrating granulomatous inflammation suggestive of tuberculosis was also considered diagnostic. Cases with positive direct microbiologic tests (pleural fluid GeneXpert, TB culture, or AFB smear) were classified as bacteriologically confirmed tuberculous pleural effusion. Clinically diagnosed tuberculous pleural effusion included cases with pleural fluid cytology showing chronic inflammatory patterns consistent with tuberculosis in the absence of microbiologic confirmation.

In this study, cases were classified based on the attending physician's diagnosis.

Statistical Analysis

For this study, prevalence referred to the number of cases of a given kind of pleural effusion divided by the total number of pleural effusions studied. Non-normal distributions were identified from their skew and kurtosis. The Student's T-test was used to estimate the statistical difference between means of normally distributed variables, and the Wilcoxon's T-test was used for non-normally distributed variables. The ADA levels were evaluated in terms of sensitivity, specificity, positive predictive value (PPV); and negative predictive value (NPV). Positive likelihood ratio (PLR), accuracy, and precision (Table 2) were computed as well.

The area under the ROC curve (AUROC) was used to measure the discriminative ability of pleural fluid ADA in different clinical situations, particularly in differentiating tuberculosis from non-TB, identifying TB in patients 45 years old and older, and identifying TB in retreatment cases. The Youden Index was used to summarize the receiver operating curve and select the optimal cut-off point.

Ethical Considerations

The study was approved by the Lung Center of the Philippines Ethics Research Committee and the Lung Center of the Philippines Institutional Review Board before data collection commenced. We also secured permission to review charts. All data were kept confidential.

RESULTS

Study Participants

Out of 660 pleural effusion cases from April 2023 to November 2023, 146 cases with complete work-up were extensively reviewed, achieving a confidence interval of 95% (level of confidence of 5%).

Baseline Characteristics

The population's demographic and clinical profiles are summarized in Table 3, delineating individuals based on varying Adenosine Deaminase (ADA) levels using a 30U/L cut-off, with associated p-values signifying the statistical significance of inter-group disparities.

Table 2. *Summary of Formula*

PPV	{PPV = (prevalence x sensitivity)/[(prevalence x sensitivity) + (1 – prevalence) (1 – specificity)]}
NPV	{(1 –prevalence) x specificity]/[(1 – prevalence) x specificity + prevalence x (1 – sensitivity)]}.
PLR	[sensitivity / (1 – specificity)], negative likelihood ratio [(1 – sensitivity) / specificity]
Accuracy	[true positive + true negative/(TP + FP + TN + FN)]
Precision	[true positive/(true positive + false positive)

Table 3. *Demographic and clinical profiles of the respondents according to pleural fluid adenosine deaminase (ADA)*

DEMOGRAPHIC & CLINICAL PROFILES	TOTAL (*n* = 146)	ADA ≤ 30U/L (*n* = 62)	ADA > 30 U/L (*n* = 84)	*p*-VALUE
	Frequency (%); Mean ± SD; Median (IQR)			
Age, years	45 (IQR 35–55)	42 (IQR 33–50)	48 (IQR 39–58)	<0.001
Sex				
MALE	51 (35%)	21 (34%)	30 (36%)	0.701
FEMALE	95 (65%)	41 (66%)	54 (64%)	
Comorbidities				
Hypertension	77 (53%)	27 (44%)	50 (60%)	0.052
Diabetes mellitus	50 (34%)	19 (31%)	31 (37%)	0.338
History of PTB	34 (23%)	14 (23%)	20 (24%)	0.838
COPD	29 (20%)	11 (18%)	18 (21%)	0.529
Pneumonia	10 (7%)	4 (6%)	6 (7%)	0.763
Heart Failure	15 (10%)	6 (10%)	9 (11%)	0.791
Cancer	14 (10%)	5 (8%)	9 (11%)	0.398
Others	5 (3%)	1 (2%)	4 (5%)	0.348
None	32 (22%)	15 (24%)	17 (20%)	0.515
Sign and Symptoms				
Cough	72 (49%)	30 (48%)	42 (50%)	0.819
Dyspnea	89 (61%)	38 (61%)	51 (61%)	0.989
Fever	65 (45%)	28 (45%)	37 (44%)	0.903
Orthopnea	23 (16%)	10 (16%)	13 (16%)	0.975
Chest pain	31 (21%)	12 (19%)	19 (23%)	0.575
Weight loss	54 (37%)	21 (34%)	33 (39%)	0.408

Main Results

The population's mean age was 45 years (IQR 35–55), with those having ADA ≤30U/L and ADA >30U/L demonstrating mean ages of 42 years (IQR 33–50) and 48 years (IQR 39–58), respectively. The age distribution differed significantly between individuals with ADA levels ≤30U/L and those with ADA levels > 30U/L (p-value < 0.001). The population was 35% male and 65% female, with comparable proportions observed in both ADA groups (p = 0.701). There was a higher prevalence of hypertension and diabetes among patients with elevated ADA, though these differences did not reach statistical significance (p = 0.052 and p = 0.338, respectively).

No significant differences were observed in the history of previous tuberculosis (p = 0.838), chronic obstructive pulmonary disease (p = 0.529), pneumonia (p = 0.763), heart failure (p = 0.791), or cancer (p = 0.398) between the ADA groups. There was likewise no difference in the prevalence of signs and symptoms (including cough, dyspnea, fever, orthopnea, chest pain, and weight loss).

Among the 61 cases with bacteriologically confirmed Tuberculosis (TB), 55 (90.16%) exhibited ADA levels > 30 U/L, representing true positives (TP). However, 12 cases with ADA levels > 30 U/L did not have bacteriologically confirmed TB, constituting false positives (FP). Conversely, among the 85 cases without bacteriologically confirmed TB, 73 with ADA levels ≤ 30 U/L were correctly identified as true negatives (TN), while 6 cases with ADA levels ≤ 30 U/L had bacteriologically confirmed TB, constituting false negatives (FN) (Table 4).

Sensitivity and specificity were calculated using a 2x2 table (Table 4). The sensitivity was 82.08%, indicating the proportion of true positives among bacteriologically confirmed TB cases. The specificity was 92.40%, signifying the accuracy of identifying cases without bacteriologically confirmed TB. The positive predictive value was at 90.16%, while the negative predictive value was at 85.66%, demonstrating the pfADA's ability to discriminate true positive and true negative results.

Likelihood ratios were computed to discriminate the likelihood of pfADA among patients with TB effusion compared to the likelihood that the same result would be expected in a patient without TB effusion. Likelihood ratios of 1 indicate that the test is uninformative, whereas the likelihood ratios of more than 1 or close to 0 indicate that the test is informative. Positive and negative likelihood ratios were at 10.18 and 0.09, respectively. Thus, a positive pfADA would be 10.18 times more likely among patients with TB effusion than those without the disease, with a 0.09 likelihood to miss out on the disease in those with negative pfADA. The accuracy rate was computed at 87%, and the precision rate was at 90.16%. These results highlight the diagnostic performance of ADA levels.

Table 4. *Sensitivity and Specificity*

	TB	NON-TB	TOTAL
ADA >30	A 55	B 6	61
ADA <30	C 12	D 73	85
TOTAL	67	71	146

The boxplot analysis indicates that the distribution of values for each diagnostic category is positively skewed, as evidenced by the elongated right tails in the plots (Figure 2). For the Bacteriologically Confirmed diagnosis, the median (Q2) is approximately 53.1, with the majority of the data falling between 43.1 (Q1) and 66.8 (Q3), and some higher values extending up to 144.6. Similarly, the Clinically Diagnosed TB category displays positive skewness, with a median (Q2) of around 18.6. Most data range from 10.5 (Q1) to 46.4 (Q3), with outliers reaching 92.5. The Not Clinically Diagnosed TB Without Malignancy category also exhibits positive skewness, with a median (Q2) of approximately 13.2. Most of the data range from 8.6 (Q1) to 41.5 (Q3), with some higher values extending up to 136.7. Lastly, the Not Clinically Diagnosed TB With Malignancy data is positively skewed, with a median (Q2) of around 20.2. Most of the data range from 5.08 (Q1) to 42.4 (Q3), with a few outliers reaching up to 48.

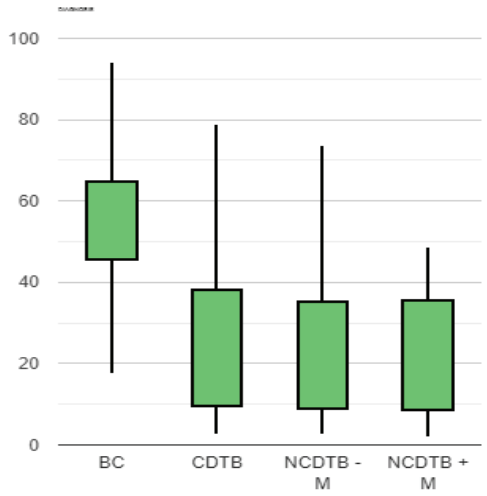

Figure 2. *Boxplot analysis: Distribution of values for each diagnosis*

The Receiver Operating Characteristic (ROC) curve (Figure 3) is a graphical representation of the performance of a binary classifier system. It plots the true positive rate (sensitivity) against the false positive rate (1 – specificity) for different threshold values. The area under the ROC curve (AUROC) quantifies the overall performance of the test. An AUROC closer to 1 indicates better discrimination ability, while 47.2 suggests the test performs no better than chance.

The AUROC was 0.867 (CI = 0.786 to 0.926, standard error = 0.039, p-value < 0.001), reaching statistical significance. This suggests that the performance of pfADA in diagnosing tuberculosis is significantly better than chance (Figure 3, Table 5).

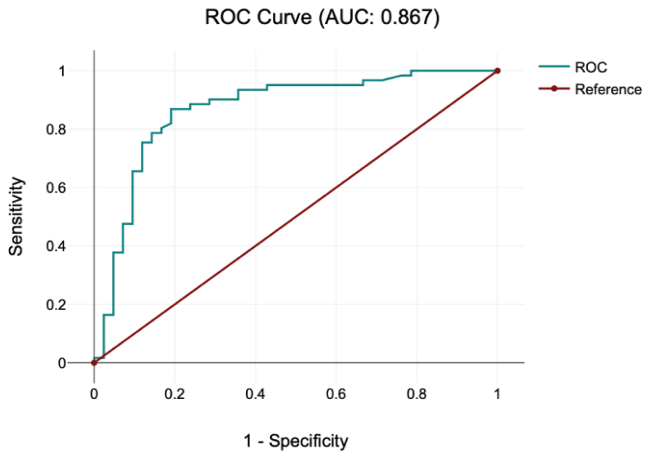

Figure 3. *The Receiver Operating Characteristic (ROC) Curve of Pleural Fluid ADA in Diagnosing Tuberculosis*

Table 5. *The Area Under the ROC Curve (AUROC) of Pleural Fluid ADA in Diagnosing Tuberculosis*

Test Result Variable	Area	Std. Error	P-Value	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
Pleural Fluid ADA (pfADA)	0.867	0.039	<0.001 *	0.786	0.926

*Note: * Significant at 0.05; Null hypothesis: true area = 0.50*

The AUROC for patients 45 years old and older was 0.782 (CI = 0.665 to 0.873, standard error = 0.0618, p-value < 0.001), suggesting that the performance of pfADA in diagnosing tuberculosis in these patients was significantly better than chance (AUROC = 25.2) (Figure 4, Table 6).

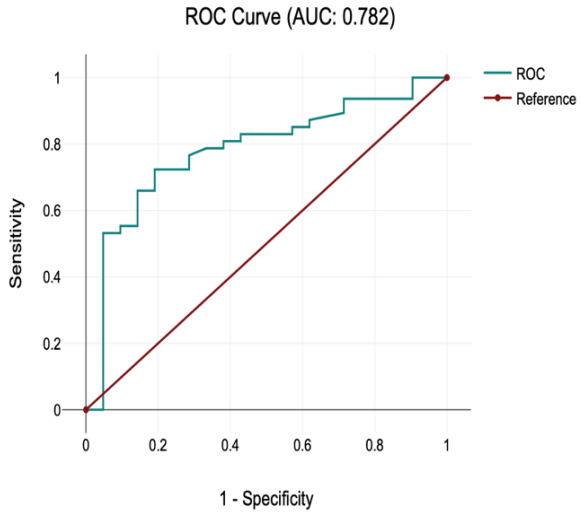

Figure 4. *The Area Under the ROC Curve (AUROC) of Pleural Fluid ADA in Diagnosing Tuberculosis based on Age >45*

Table 6. *The Area Under the ROC Curve (AUROC) of Pleural Fluid ADA in Diagnosing Tuberculosis based on Age > 45*

Test Result Variable	Area	Std. Error	P-Value	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
Pleural Fluid ADA (pfADA)	0.782	0.0618	<0.001 *	0.665	0.873

*Note: * Significant at 0.05; Null hypothesis: true area = 0.50*

The AUROC for retreatment cases was 0.641 (CI = 0.508 to 0.760, standard error = 0.108, p-value = 0.1906) and did not reach statistical significance (Figure 5, Table 7).

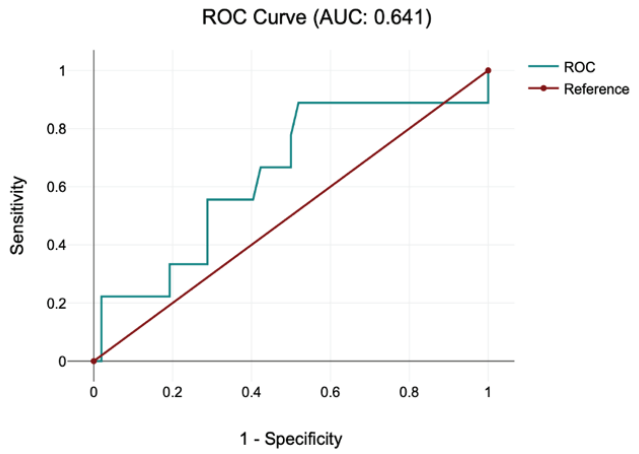

Figure 5. *The Receiver Operating Characteristic (ROC) Curve of Pleural Fluid ADA in Diagnosing Tuberculosis based on Previous TB therapy, with at least 2 years of treatment*

Table 7. *The Area Under the ROC Curve (AUROC) of Pleural Fluid ADA in Diagnosing Tuberculosis based on retreatment.*

Test Result Variable	Area	Std. Error	P-Value	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
Pleural Fluid ADA (pfADA)	0.641	0.108	0.1906	0.508	0.760

*Note: * Significant at 0.05; Null hypothesis: true area = 0.50*

DISCUSSION

PfADA demonstrated good diagnostic accuracy, with a sensitivity of 82.08% and specificity of 92.40%.

A cut-off value of 47.2 U/L provided better diagnostic performance compared with the conventional 30 U/L threshold, supporting the use of higher cut-offs in settings with a high tuberculosis burden. Among patients aged ≥45 years, a lower cut-off (>25.2 U/L) was observed, consistent with prior studies, although the association between age and ADA was not statistically significant within the TPE subgroup-likely due to limited sample size. No significant difference in ADA levels was observed among retreatment cases based on AUROC analysis, although this finding may also be limited by small sample size.

These findings highlight the importance of population-specific cut-offs and reinforce the role of pfADA as a practical, rapid, and cost-effective diagnostic tool in exudative, lymphocytic pleural effusion. Subgroup analyses further support its utility across different clinical scenarios.

However, the study has limitations, including its single-center design, limited sample size, and potential selection and misclassification biases. Some cases of TPE may have been misclassified due to spontaneous resolution or uncertain etiology. In addition, the lack of physician blinding to ADA results may have introduced diagnostic bias.

CONCLUSION

In conclusion, pfADA is a reliable diagnostic marker for TPE. While the conventional cut-off of 30 U/L performs well, a higher threshold (~47.2 U/L) may be more appropriate in the Filipino population, with lower thresholds considered for older patients. Further multicenter studies are recommended to validate these findings and refine diagnostic thresholds.

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ACKNOWLEDGEMENT

The authors thank Dr. Ma. Lalaine Gumiran Cheng for her guidance in the statistical analysis and interpretation of the study. We also acknowledge the clinical departments and medical staff of the Lung Center of the Philippines for their support during data collection. Lastly, we extend our gratitude to the patients whose participation contributed to knowledge that may improve the care of future patients.

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THE PHILIPPINES' FIRST SINGLE-CELL GENOMICS CORE FACILITY: ADVANCING TRANSLATIONAL RESEARCH AT THE LUNG CENTER OF THE PHILIPPINES

*By Heidie F. Cabanos, MSc, PhD**

The Lung Center of the Philippines (LCP) has reached a major milestone in biomedical research with the establishment of the Philippines' First Single-Cell Genomics Core Facility within its Clinical Research Division (CRD). The facility was established through the Department of Science and Technology – Philippine Council for Health Research and Development (DOST-PCHRD) Balik Scientist Program (BSP), with major funding support from a Department of Science and Technology – Grant-in-Aid (DOST-GIA) research project led by the Lung Center of the Philippines and institutional infrastructure support from LCP. Together, these initiatives established the country's first dedicated platform for single-cell genomics. More than a new laboratory, the facility provides the foundation for advanced genomics and clinical and translational research, bringing these technologies closer to Filipino researchers, clinicians, and ultimately, patients.

Single-cell genomics combines high-throughput single-cell or single-nucleus RNA sequencing (sc/snRNA-seq) with computational analysis to characterize the transcriptomic profiles of thousands of individual cells simultaneously (Figure 1). Unlike conventional bulk RNA sequencing, which measures average gene expression across heterogeneous tissues, single-cell approaches resolve cellular heterogeneity, identify rare cell populations, define cellular states, and reveal disease-associated molecular pathways. These capabilities have advanced biomarker discovery and precision medicine by enabling researchers to study disease at cellular resolution.

The establishment of the facility began through the DOST-PCHRD Balik Scientist Program, which introduced advanced single-cell genomics technologies to the Philippines while strengthening local genomics research capacity. Beyond establishing the country's first Single-Cell Genomics Core Facility, the initiative focused on creating a sustainable research platform through standardized workflows for clinical sample processing, biobanking and biorepository management, DNA/RNA extraction, nucleic acid quality assessment, next-generation sequencing (NGS) library preparation, REDCap-based clinical and biospecimen data management, quality management systems, and specialized technical training (Photos 1–4).

A major milestone in this effort was the Philippines' First Single-Cell Genomics Workshop, held on November 11–12, 2024, at the Lung Center of the Philippines. The workshop brought together clinicians, scientists, pathologists, medical technologists, and laboratory personnel for the country's first dedicated training activity in single-cell genomics, featuring lectures, demonstrations, and discussions on the principles and applications of single-cell transcriptomics (Photos 5–7). On November 13, 2024, the facility was publicly introduced during the National Reference Laboratory Building event at the Lung Center of the Philippines, providing an early institutional showcase to the wider research and clinical community (Photo 8).

The facility is central to the Clinical Research Division's vision of transitioning into a Clinical and Translational Research Division (CTRD), bridging laboratory discovery and clinical practice. While its primary focus is pulmonary medicine, it is envisioned as a national genomics resource supporting multidisciplinary research on diseases of major public health importance and accelerating the translation of scientific discoveries into improved patient care.

The facility currently supports two flagship tuberculosis (TB) research programs addressing one of the Philippines' most important public health challenges. The first investigates post-tuberculosis lung disease, particularly TB-associated bronchiectasis, using single-cell RNA sequencing and complementary molecular approaches to characterize immune cell populations and molecular pathways associated with persistent inflammation, immune dysregulation, and lung injury. A complementary program focuses on TB-HIV co-infection, integrating longitudinal clinical data, biobanking, transcriptomic profiling, and advanced genomic analyses to identify molecular signatures associated with disease susceptibility, treatment response, and long-term outcomes. Together, these studies seek to improve diagnosis, guide therapeutic strategies, and advance the understanding of tuberculosis biology. Beyond tuberculosis, the facility also supports collaborative research in lung cancer, chronic respiratory diseases, infectious diseases, cardiovascular diseases, autoimmune disorders, metabolic diseases, and other conditions affecting the Filipino population.

The facility's early translational research initiatives were also presented at the 2026 Human Cell Atlas General Meeting in Boston, Massachusetts, USA, where two research posters highlighted the establishment of the

Philippines' First Single-Cell Genomics Core Facility and the Lung Center's emerging genomics research program. The meeting also provided opportunities to establish new collaborations and exchange scientific expertise (Photos 9–11).

Looking ahead, the Lung Center of the Philippines aims to further strengthen its research capabilities through expanded in-house next-generation sequencing (NGS), advanced bioinformatics and multi-omics analysis, the establishment of a Flow Cytometry Core Facility, and a cell culture laboratory capable of developing patient-derived cell lines and disease models for mechanistic studies and functional validation. These complementary platforms will expand the Lung Center's capabilities from clinical sample processing and molecular profiling to functional validation and translational research.

The establishment of the Philippines' First Single-Cell Genomics Core Facility marks an important milestone in the Lung Center of the Philippines' continuing commitment to research and innovation. By integrating advanced genomics with clinical research, the facility provides a strong foundation for generating locally relevant evidence that can improve disease prevention, diagnosis, and treatment. As the country's apex referral and premier specialty hospital for pulmonary diseases, the Lung Center of the Philippines is well positioned to lead genomics-driven clinical and translational research while fostering collaboration across institutions in the Philippines and abroad.

** About the Author. Dr. Heidie F. Cabanos is a Balik Scientist under DOST-PCHRD and Scientific Consultant to the Clinical Research Division, LCP. Through the DOST-PCHRD Balik Scientist Program, she led the establishment of the Philippines' First Single-Cell Genomics Core Facility and continues to support genomics and clinical and translational research initiatives at the Lung Center of the Philippines.*

Figure 1A

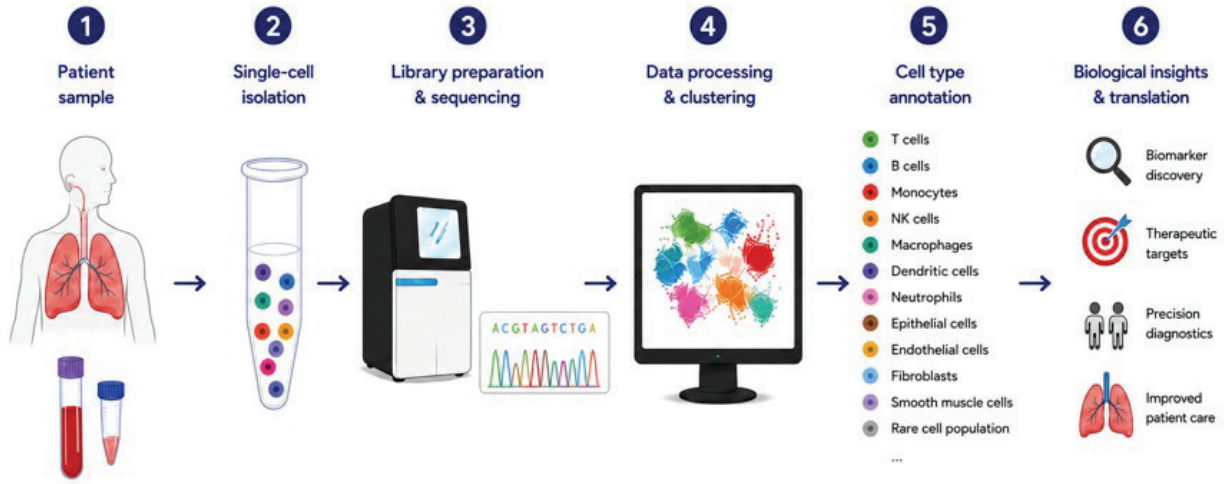


Figure 1B

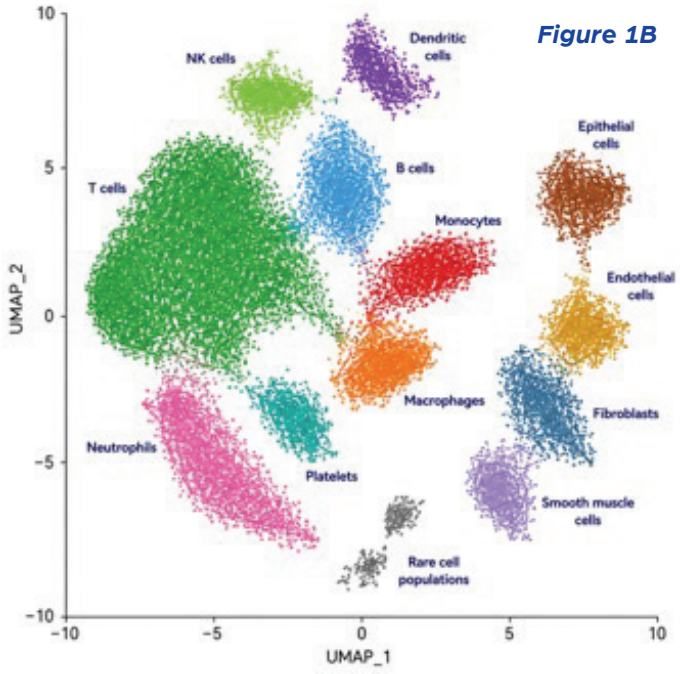
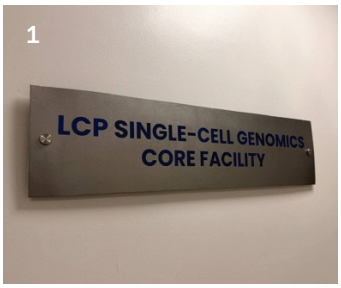
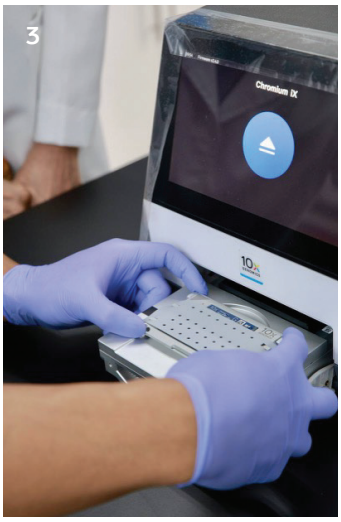


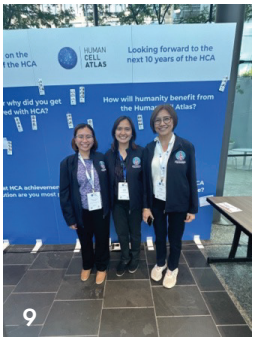
Figure 1. Single-cell genomics workflow and representative single-cell transcriptomic analysis from the Lung Center of the Philippines Tuberculosis (TB) Research Program. ***(A)*** Patient-derived specimens undergo single-cell RNA sequencing (scRNA-seq), followed by bioinformatic processing, dimensionality reduction, clustering, and cell type annotation to identify distinct cell populations and disease-associated molecular signatures. ***(B)*** Representative Uniform Manifold Approximation and Projection (UMAP) illustrating the cellular heterogeneity resolved by single-cell transcriptomics. The UMAP shown is an artist's rendition based on the Lung Center of the Philippines TB Single-Cell Project and was modified for illustrative purposes to depict representative immune and non-immune cell populations while preserving the overall structure of the original dataset.





Photos 1–4. The Philippines' First Single-Cell Genomics Core Facility, Clinical Research Division, Lung Center of the Philippines, Quezon Avenue, Quezon City, Metro Manila, Philippines. The facility supports clinical sample processing, biobanking and biorepository operations, DNA/RNA extraction, nucleic acid quantification and quality assessment, cell counting and viability analysis, single-cell library preparation, and molecular genomics workflows for single-cell RNA sequencing (scRNA-seq) and other next-generation sequencing (NGS) applications. (1) Entrance signage of the facility. (2) Overview of the single-cell genomics laboratory. (3) Single-cell library preparation using the 10x Genomics Chromium platform. (4) Lab tour and visit by Lung Center of the Philippines Clinical Research Division leadership to discuss the Division's translational research roadmap and future development of the Single-Cell Genomics Core Facility. Shown are CRD officials Dr. Virginia Delos Reyes, Dr. Raquel Ibanez, and Dr. Bernadette Asuncion, together with the Single-Cell Genomics Core Facility team led by Dr. Sullian S. Naval, with Dr. Jared L. dela Rosa, Angelo Arabit, Zion S. Bañes, and Jaz Amper.

Photos 5–8. Activities during the Philippines' First Single-Cell Genomics Workshop and the public introduction of the Philippines' First Single-Cell Genomics Core Facility. (5–7) The workshop, held on November 11–12, 2024, at the Lung Center of the Philippines, brought together clinicians, scientists, pathologists, medical technologists, and researchers for lectures, hands-on demonstrations, and discussions on the principles and applications of single-cell transcriptomics. (8) On November 13, 2024, the Single-Cell Genomics Core Facility was publicly introduced during the National Reference Laboratory (NRL) Building event at the Lung Center of the Philippines, providing the institution's first public showcase of the facility ahead of its planned official launch. The event was attended by representatives from the Department of Science and Technology – Philippine Council for Health Research and Development (DOST-PCHRD), including Executive Director Dr. Jaime C. Montoya, together with Lung Center of the Philippines officials Dr. Vincent M. Balanag Jr., Dr. Sullian S. Naval, Dr. Norberto Francisco, and Dr. Treah S. Sayo.



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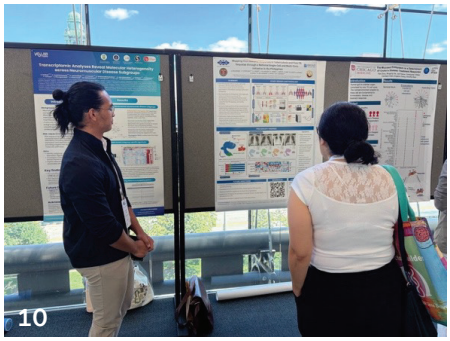
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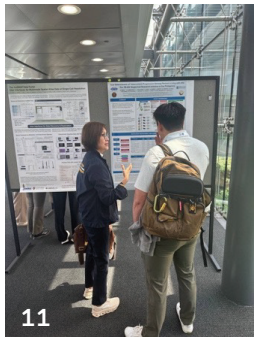
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Photos 9–11. Presentation of research posters highlighting the Philippines' First Single-Cell Genomics Core Facility and the Lung Center of the Philippines' early clinical and translational research initiatives at the 2026 Human Cell Atlas General Meeting, held on June 16–18, 2026, in Boston, Massachusetts, USA. The meeting provided an international platform to showcase the Philippines' growing capabilities in single-cell genomics, share early research initiatives, engage with the global scientific community, and establish new research collaborations. Representatives from the Lung Center of the Philippines included Dr. Heidie F. Cabanos, Dr. Sullian S. Naval, Dr. Fresthel M. Climacosa, and Dr. Jared L. dela Rosa, who were among the scientists involved in establishing the country's first Single-Cell Genomics Core Facility.

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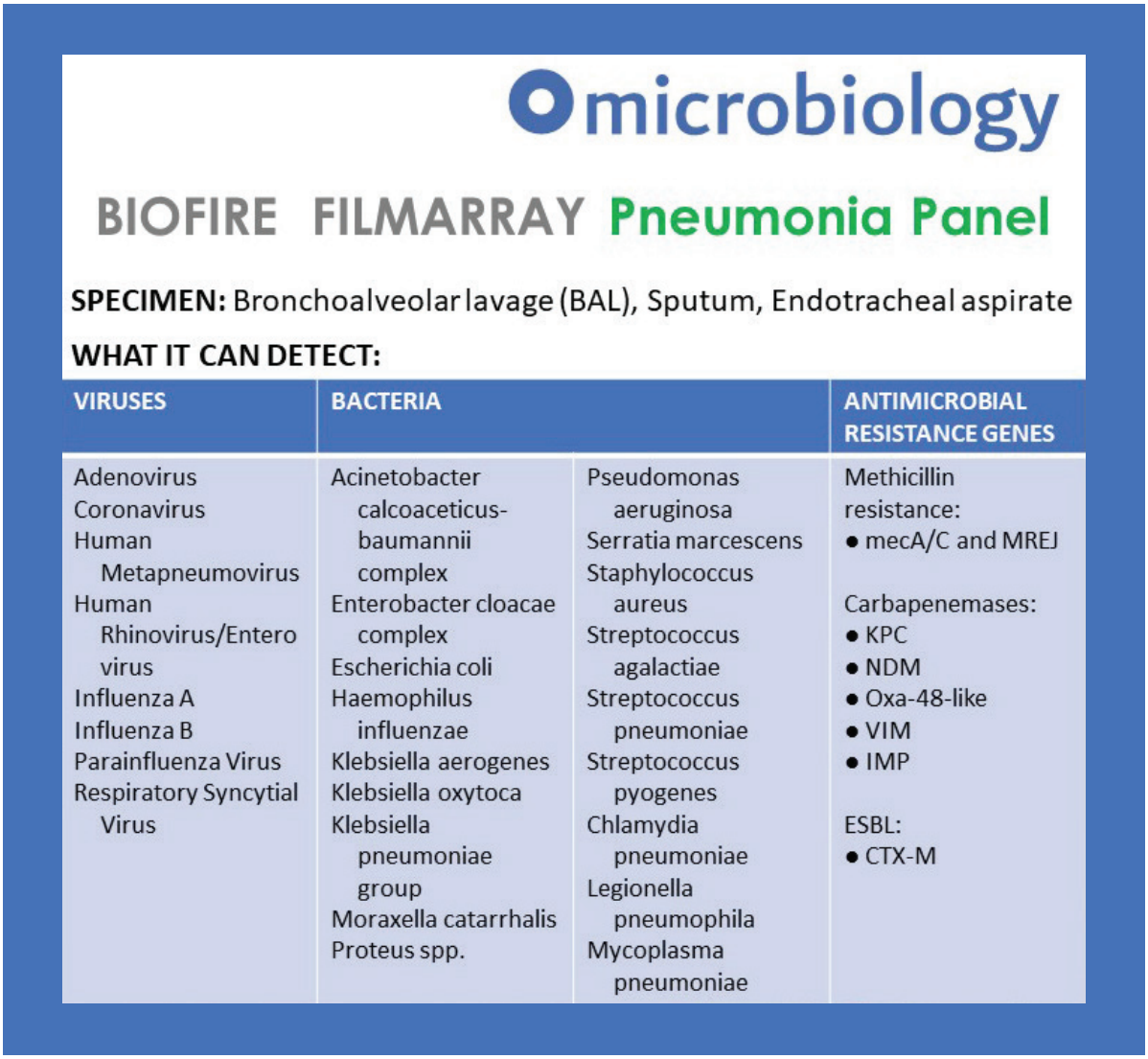

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  Libshitz HI, Mckenna RJ, Haynie TP, et al. Mediastinal evaluation in lung cancer. Radiology 1984; 151:295-99.

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  Meltzer PS, Kallioniemi A, Trent JM, Chromosome alterations in human solid tumors. In: Vogelstein B, Kinzler KW, editors. The genetic basis of human cancer. New York: McGraw-Hill; 2002. p. 93-113.

  Book

  Murray, PR, Rosenthal KS, Kobayashi GS, Pfaller MA. Medical microbiology. 4th ed. St. Louis: Mosby; 2002.

  Gilstrap LC 3rd, Cunningham FG, VanDorsten JP, editors. Operative obstetrics. 2nd ed. New York: McGrawHill; 2002.

  Website

  World Health Organization. Hospital infection control guidelines for severe acute respiratory syndrome. April 16, 2003: http://who.int/csr/sars/infectioncontrol/en (accessed April 24, 2003).

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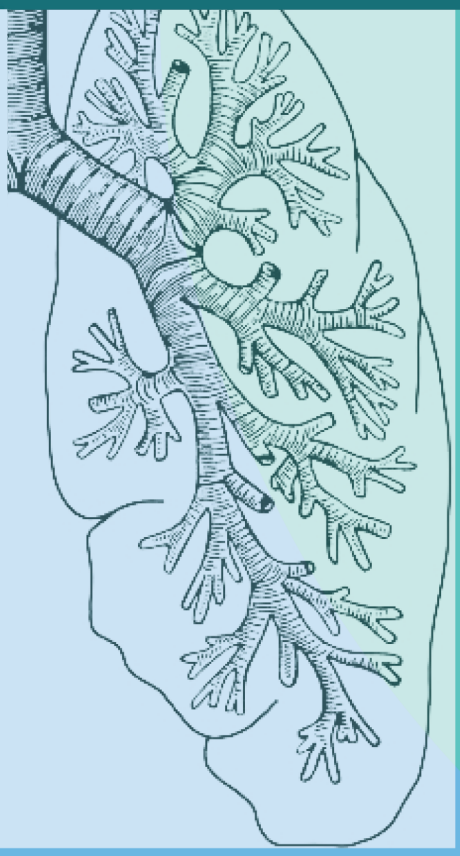

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