EXPOSURE TO INDOOR AIR POLLUTION FROM HOUSEHOLD FUELS AND EFFECT ON TREATMENT RESPONSE AMONGST CHILDREN AGED BETWEEN 2 AND 59 MONTHS ADMITTED WITH PNEUMONIA AT THE KENYATTA NATIONAL HOSPITAL, NAIROBI, KENYA

BY

Dr. Francis Ng'ang'a Gicheru

H58/11104/2018

A DISSERTATION SUBMITTED IN PARTIAL FULFILLMENT FOR THE AWARD OF THE DEGREE OF MASTERS OF MEDICINE IN PAEDIATRICS AND CHILD HEALTH OF THE UNIVERSITY OF NAIROBI.

NOVEMBER 2021

Declaration

Student's Declaration

This dissertation is my original work and has not been presented for a degree or any other award in any University.

Francis Ng'ang'a Gicheru (MB ChB)

Department of Paediatrics, University of Nairobi

Signature……  ……. Date 11/11/2021

Supervisors' Approval

This dissertation has been presented with our full approval as supervisors.

Dr. Lawrence Owino. (MB ChB, M. Med, FRheum.)

Lecturer and Paediatric Rheumatologist,

Department of Paediatrics and Child Health, University of Nairobi

Signed Date 08/11/2021

Prof. Elizabeth Obimbo. (MB ChB, M. Med, MPH, FPulm.)

Professor and Paediatric Pulmonologist,

Department of Paediatrics and Child Health, University of Nairobi

Signed.....Date.....8th Nov 2021

Collaborating Institutions

1. The University of Nairobi

2. Kenyatta National Hospital

Funding

This research project was fully self-funded.

Dedication

This dissertation is dedicated to lovely wife, Nancy and my precious daughters, Khoi, Kenya and Wambui whose love and patience have been a great source of inspiration.

Thank you all for your unending support, prayers, words of encouragement and constant support throughout this journey.

Acknowledgements

I would like to thank God for seeing me through this journey.

I wish to acknowledge the invaluable support and encouragement I received from my supervisors' Prof. Elizabeth Obimbo and Dr. Lawrence Owino of the University of Nairobi.

I am grateful to the faculty members of Department of Paediatrics and Child Health, University of Nairobi for their contribution in making this study a success.

I appreciate the research assistants and statisticians for their hard work and their co-operation at the various stages of this research.

I am thankful to the Kenyatta National hospital management, the Paediatric in-patient unit staff and all the children and their guardians who agreed to participate in this study.

My sincere gratitude to my wife, daughters and parents for their understanding and prayers.

Table of Contents

Declaration……...……ii

Supervisors' Approval……...…….iii

Dedication……..v

Acknowledgements…….....…vi

Table of Contents……..……..…vii

List of Tables……..….x

List of Figure…….…….....…..xi

Abbreviation and Acronyms ……..……...…..….....xii

Definition of Operational Terms …….……xiii

Abstract….……...……..…..xiv

Chapter 1:Introduction.……...….....…….……...……1

Chapter 2: Literature Review……...…….…….……...……..2

2.1 Exposure to Indoor Air Pollution…….……....2

2.2 Pathophysiologic Effects of Indoor Air Pollutants on the Human Respiratory Tract. .5

2.3 Management of Pneumonia…….……7

2.4 Effect of household fuel exposure on pneumonia treatment outcome in children…..11

2.5 Measurement of household Indoor Air Pollution……...…....14

2.6 Household Indoor Air Pollution and risk of Pneumonia in children……..…16

2.7 Study Justification…….…..….17

2.8 Study Utility…….…….……...17

Chapter 3: Research Questions and Study Objectives ……....18

3.1 Research Questions……..…….……..18

3.2 Research Objectives…….…….……..…..18

Chapter 4: Methodology…….……19

4.1 Study Design……...…….……19

4.2 Study Location……...…….……19

4.3 Study Population……..……..……..19

4.4 Case definitions…….……20

4.5 Sample Size Determination……..…22

4.6 Sampling Method…….…….……23

4.7 Study Tools…….…….……..…23

4.8 Study Procedures……..…….24

4.9 Data Management……....25

4.10 Ethical Consideration……...…26

4.11 Dissemination of Study Findings ……..…….26

CHAPTER 5: RESULTS…….……...27

5.1 Participants' screening, enrolment and follow-up ……..27

5.2 Socio-demographic characteristics of the study respondents……..28

5.3 Environmental characteristics of the study respondents……..29

5.4 Age, gender and physical findings of the study respondents at admission……...30

5.5 Clinical status of the study respondents at admission ……..…...31

5.6 Follow up clinical status of the study respondents …….....…33

5.7 Oxygen saturation at admission and 48-hours…….33

5.8 Treatment outcome Analysis……..34

5.9 Five-day outcome of children admitted with pneumonia……...36

5.10 Survival Analysis……..38

CHAPTER 6: DISCUSSION……..39

6.1 Study Strengths and Limitations……..…….44

6.2 Conclusions…….……..…….……..…44

6.3 Recommendations……..……..……..44

7.0 References…….…….…...…46

Appendix A Checklist - to estimate level exposure of IAP from household fuels……50

Appendix B Questionnaire……..…..……..……..…….....51

Appendix C Parents/Guardian Consent Form…… …….…….…59

Appendix D Fomu ya Kupata Idhini la Wazazi/Walezi…….....62

List of Tables

Table 2.1: Percentage of the population using solid fuels as their main cooking fuel per region of the world and the death rate from IAP per 100,000 individuals……3

Table 2.2: Energy efficiency, average 24 hour $PM_{2.5}$ concentration and level of emissions above the WHO air quality guideline recommendation…….……5

Table 2.3: Studies on prevalence of pneumonia treatment failure in children hospitalized with pneumonia in different settings…….…….……..…12

Table 5.1: Socio-Demographic characteristics of the study respondents……..……..28

Table 5.2 Household fuel type exposure…….……29

Table 5.3: Home environment characteristics of the study respondents……..30

Table 5.4: Age, gender and physical findings at admission …….....31

Table 5.5: Clinical features of children with pneumonia at admission ……32

Table 5.6: Follow up clinical status of the study respondents ……33

Table 5.7: Oxygen saturation at admission…….……....34

Table 5.8: Outcomes of study subjects at 48 hours – survival and oxygenation response to treatment ……..….34

Table 5.9: 48-hour Pneumonia treatment outcome Analysis ……...….35

Table 5.10: Five-day outcome of children admitted with pneumonia……36

Table 5.11: Five-day pneumonia treatment response analysis of the study respondents.37

List of Figures

Figure 2.1: Total Global Exposure to Particulate Matter Pollution……...……....…2

Figure 2.2: Major categories of variables likely to influence to household air pollutant exposure at individual, household or community level……..…….4

Figure 2.3: Pathologic effects of air pollutants on the respiratory tract…….……..7

Figure 2.4: Classification of Pneumonia WHO (2014) and Kenya Paediatric Protocol..…8

Figure 5.1: study participants screening and enrolment……28

Figure 5.2: Survival Curve…….....28

Acronyms and Abbreviations

ALRI:	Acute Lower Respiratory Infections
ARI:	Acute Respiratory Infections
CDC:	Centre for Disease Control
IAP:	Indoor Air Pollution
IMCI:	Integrated Management of Childhood Illnesses
KNBS:	Kenya National Bureau of Statistics
KNH:	Kenyatta National Hospital
KNH-UoN ERC:	Kenyatta National Hospital-University of Nairobi Ethical Review Committee.
LMICs:	Lower and middle Income Countries
LPG:	Liquefied Petroleum Gas
PM:	Particulate matter
PCP:	Pneumocystis Jirovecii Ppneumonia
RR:	Relative Risk
SPSS:	Statistical Package for Social Sciences
UNDP:	United Nations Development Program
WHO:	World Health Organisation

Operational Definition of Terms

Particulate matter: Particle generated from burning fuels and classified according to their aerodynamic properties.

Pneumonia : Will be defined using the WHO classification of severe pneumonia which is fast breathing and/or chest indrawing (pneumonia) plus general danger signs (inability to drink, convulsions, lethargic or unconscious stridor in a calm child, persistent vomiting or severe malnutrition).

Poor response to treatment: Defined as presence oxygen saturation (SPO_2) same as or decreased when compared to the baseline saturation at admission or death. Any decline in oxygen saturation (SPO_2) from above 95% to less than 95%. Clinical persistence of disease at day five of treatment.

Good response to treatment: Any increase in oxygen saturation (SPO_2) from the baseline, maintenance of oxygen saturation (SPO_2) above 95% or been discharged from hospital at or before 48 hours of treatment. Discharge before or at day 5 of treatment.

ABSTRACT

Background On average, 50% the world's population relies on high polluting fuels for their domestic energy requirements. The primary target of air pollutants emitted by these fuels is the respiratory system and can result in pneumonia which at 16% is the second leading cause of death of children less than 5 years of age in Kenya. Approximately 20% of children admitted to hospital in Kenya with pneumonia and receive treatment as per the WHO recommended guidelines experience a poor response to treatment at 48 hours.

Objectives The main aim of the study was to determine the prevalence of exposure to IAPs from household fuels and its effect on treatment outcomes amongst children aged between 2 and 59 months admitted with pneumonia at the Kenyatta National Hospital (KNH) in Nairobi, Kenya.

Methodology A prospective cohort study undertaken over a three month period of time in the general pediatric wards of KNH. The study population was of children aged between 2 and 59 months admitted with WHO-defined severe pneumonia and started on the WHO recommended treatment regimen.

Data Management and Analysis A standardized questionnaire was used to collect data. Data entry was through the Epi-info computer package and was then exported to the statistical package for social sciences (SPSS) for analysis. Association is presented using odds ratio with a 95% confidence interval to determine whether there is an association between household fuel exposure and response to treatment after 48 hours in children aged between 2 and 59 months admitted with clinical features of pneumonia KNH. Survival analysis was done to assess the effects of IAP exposure from household fuels on 5-day outcome using variable of time from admission to outcome death versus time to survival/recovery as the outcome in the model.

Results A total of 127 respondents were recruited for the study. Exposure to high levels of IAP from household fuels was found to be significantly associated with a poor response to pneumonia treatment with an odds ratio of 1.49, a 95% confidence interval of 1.02 to 2.21 and a P value of 0.0406 . There was also a significant association between

exposure to IAP and response to pneumonia treatment at five days with an odds ratio of 2.04, a 95% confidence interval of 1.07 to 3.90 and a P-value of <.03.

Conclusion Exposure to high polluting household indoor fuels is high among children admitted with severe pneumonia and is associated with a poor response to pneumonia treatment both at 48 hours and five days of treatment.

Recommendations Use of low polluting household fuels should be promoted and facilitated both at a household and at a national level to reduce associated poor response to pneumonia treatment.

.

1.0 INTRODUCTION

Pneumonia is an acute respiratory infection that affects the lung's alveoli. Pus and fluid accumulate in the lungs which limits gaseous exchange thus making breathing difficult. It is the leading cause of death globally in children less than 5 years of age. It causes approximately 12.8% annual deaths beyond the neonatal period (1) and 21% of all deaths in the developing countries (2). In Kenya, pneumonia causes 16% of all deaths of children aged less than five years making it the second leading killer in this age group (3).

Worldwide, approximately 3.9% of all deaths can be attributed to indoor air pollutant exposure in LMICs (4). Over 250,000 deaths of children less than 5 years are attributed to exposure to IAP with the highest percentage of these deaths occur in Africa especially in Sub-Saharan Africa (5).

IAP refers to contamination of air by physical, chemical or biological elements within homesteads. In LMICs, the main source of IAP is from the use of household fuels which contain carbon monoxide, sulphur dioxide, particulate matter, nitrogen dioxide, formaldehyde and polycyclic aromatic hydrocarbons. Approximately 50% of the world's population depends on solid fuels for their domestic fuel needs (6). The Kenya Demographic Health Survey (KDHS) of 2014 determined that 74.6% of households in Kenya use solid fuels for cooking while 11.9% use paraffin (7). Environmental factors increase a child's susceptibility to acute lower respiratory infections especially pneumonia (8).

The highest risk of exposure to indoor air pollutants is amongst women and young children of the developing world. The risk of developing pneumonia increases by 80% following solid fuel smoke exposure and can be a cause of poor response to treatment (9). In Kenya, indoor air pollution contributes to 8-10% of early deaths (10).

2.0 LITERATURE REVIEW

2.1 Exposure to Indoor Air Pollution

Household fuels can be classified into two major groups' namely high polluting and clean/low polluting household fuels. The high polluting fuels are kerosene/paraffin and solid fuels which include dung, crop waste, wood and charcoal. The low-polluting household fuels are electricity and liquefied petroleum gas (LPG). The highest exposure to air pollutants occurs indoors in the developing countries at 76%. In these countries, the burden of disease from indoor air pollutants is approximately five times that of outdoor air pollution (11) (figure 2.1).

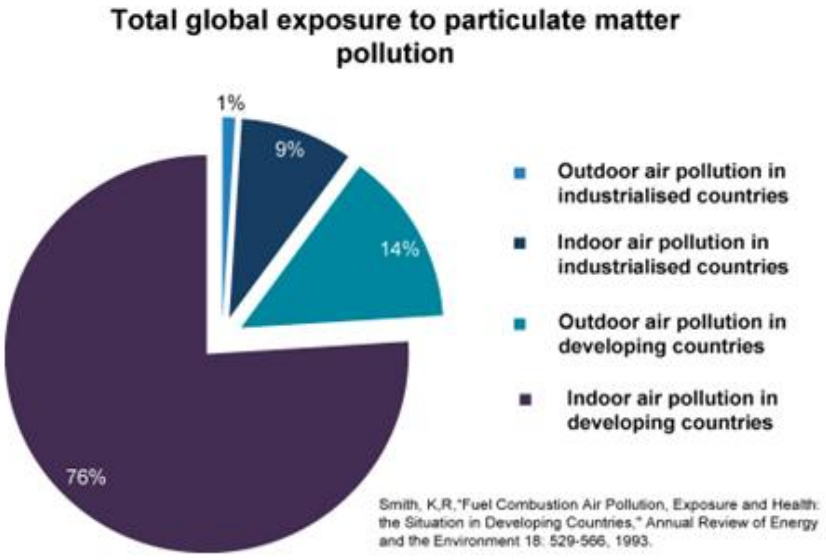

Figure 2.1: Total global exposure to particulate matter pollution (Smith KR, 1993).

At 77%, Africa had the greatest percentage of its population using solid fuels as their primary source of domestic cooking fuel in 2016 while South East Asia followed by Europe had the least users of domestic solid fuels at 6% and 7% respectively as shown in table 2.1 (5).

Africa also had the greatest death rate from IAP per 100,000 individuals ranging from 90-150 while Europe, South East Asia and America had less than 10 deaths per 100,000 individuals dying from IAP as shown in figure 2.1. It is estimated that more than 250,000 children aged less than 5 years die due to IAP exposure annually (5).

Table 2.1: Population (in percentage) who use solid fuels as their primary cooking fuel per region of the world and the death rate from IAP (5).

Region	Households in percent using solid fuels as their primary source of cooking energy	IAP Death rate per 100,000 Deaths of individuals
Africa	77%	90-150
South East Asia	6%	<10
Western Pacific	46%	60-90
Eastern Mediterranean	35%	10-30
America	14%	<10
Europe	7%	<10

The most important sources of domestic energy in Kenya are solid fuels and paraffin. They meet over 85% of the total primary energy consumption needs due to their cheap prices and availability. However, they contribute the most to indoor air pollution that adversely affects the health status of household members. Electricity and LPG are used by 0.4% and 11.5% of Kenyans respectively. In the urban areas of Kenya, solid fuel use stands at 45.6%, kerosene use at 26.6%, LPG use at 24.5% and electricity use at 0.9%. In the rural areas, solid fuel use is at 95.5%, kerosene use at 1.3%, LPG use at 2% and electricity use at 0.1% (7).

Stacking/ simultaneous use of charcoal and kerosene fuels is widely practiced in Kenya (12). In LMICs, households tend to use a combination of fuels rather than change completely to an alternative or modern energy sources. This may involve combining modern fuels with solid fuels as a source of energy. This implies that rather than move up the ladder gradually as income increase, these households prefer to use a variety of fuels simultaneously. Most choose a combination of low and high-cost fuels, which depends on their budgets, needs and preferences (13). This gives rise to the concept of multiple/combination fuel use or fuel stacking rather than an energy ladder (14).

Air pollution varies over time and space. Levels of air pollution in the environment where people spend most of their time determine their exposure levels. The primary source of indoor air pollutant exposure in developing countries is from the solid fuels as the of cooking fuel. Pollution is enhanced by use of stoves that are inefficient and are used in poorly ventilated areas. The major categories of variables likely to influence to household air pollutant exposure at individual, household or community levels are displayed in Figure 2.2.

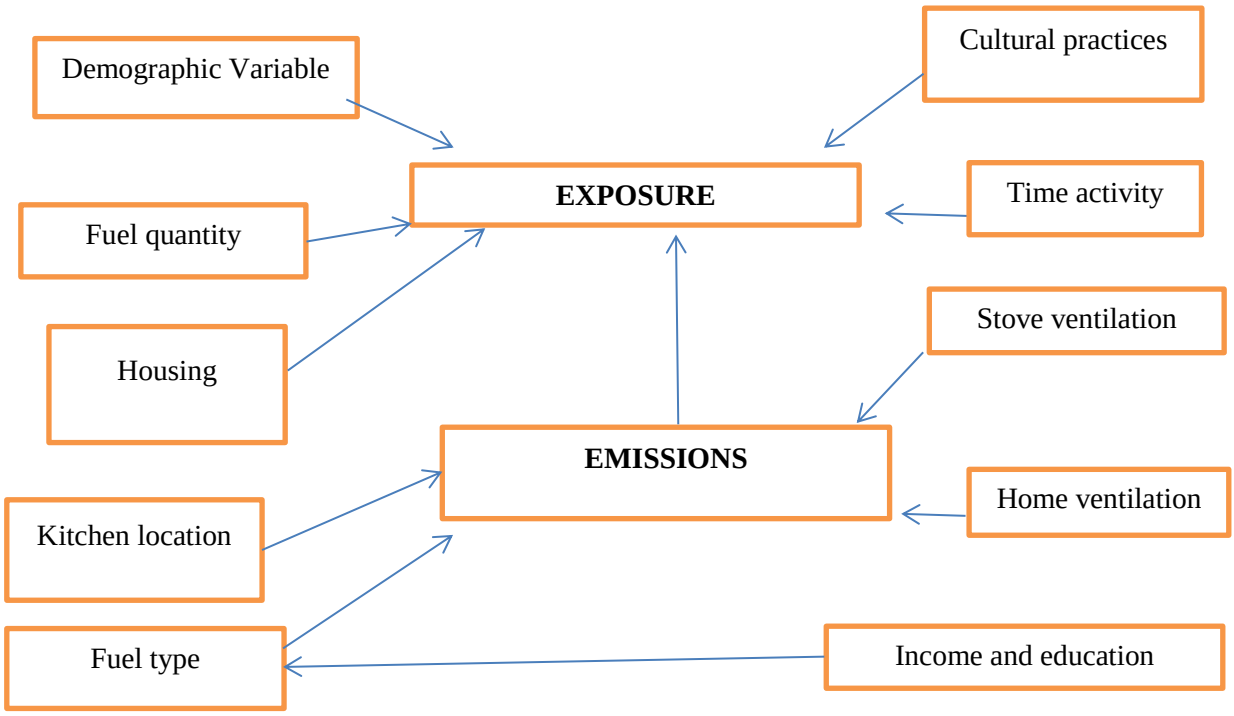

Figure 2.2: Major factors likely to influence to household air pollutant exposure at individual, household or community levels (Kurmi et al 2004).

The composition of the emissions varies widely and some are more toxic than others. In most instances, these emissions exceed national and international recommended levels (15).

The particulate matter (PM) are particles generated from burning of fuels their classification is based on their aerodynamic properties. The PM influences how particles are transported and removed from the air, how particles are deposited in the respiratory system and their

chemical components (16). PM_{10} are particles that measure up to 10 micrometers in diameter. $PM_{2.5}$ are particles in air which are less than or equal to 2.5 micrometers in diameter. Compared to $PM_{10,}$ $PM_{2.5}$ is a better predictor of health effects of particulate matter.. The WHO global ambient air quality require that the 24 hour PM_{10} concentration should not be above 50 $\mu g \cdot m^{-3}$ and the annual mean not above 20 $\mu g \cdot m^{-3}$. The 24 hour $PM_{2.5}$ should not be above 25 $\mu g \cdot m^{-3}$ while the annual should not be above 10 $\mu g \cdot m^{-3}$ (17). Where solid fuels are used, the 24-hour kitchen concentrations for $PM_{2.5}$ and PM_{10} are usually beyond their respective WHO recommended levels (Table 2.2). The efficiency of cooking fuels range from 84% for electricity to between 12% -25% with the use of wood/charcoal (18).

TABLE 2.2: Energy efficiency, average 24 hour $PM_{2.5}$ concentration and level of emissions above the WHO air quality guideline recommendations (17).

Type of fuel	**Energy efficiency of the fuel (%)**	**Average 24 hour kitchen $PM_{2.5}$ concentration (ug/m^3)**	**Emissiom level above or below WHO air quality guideline $PM_{2.5}$ ug/m^3 mean 24 hours**
Charcoal/wood	10-22%	249	Very high
kerosene	55%	172	Moderately high
LPG	60%	66	Slightly high
Electricity	84%	66	Slightly high

2.2 Pathophysiologic Effects of Indoor Air Pollutants on the Human Respiratory Tract

The pathophysiologic effects of many indoor pollutants act together through a series of inter-connected biological mechanisms. Air pollutants exert their effects by influencing the host defense mechanism activity against micro-organisms that invade the respiratory tract. Some of the health-damaging pollutants emitted by solid fuels include carbon monoxide, respirable particulates, benzene, nitrogen oxides, formaldehyde, 1,3 butadiene, and polyaromatic

compounds. Use of these fuels in poorly ventilated homesteads, in open fires or in inefficient stoves, a common occurrence in LMICs, results in IAP levels well above the recommended exposures levels. This causes a large numbers of people to be at a higher risk of contracting acute lower respiratory illnesses (ALRI), tuberculosis, chronic obstructive pulmonary disease (COPD), asthma and lung cancer (19).

The pollutants cause a defect in the host immune response mechanism against micro-organisms in the respiratory tract. The alveolae are lined by epithelial cells which secrete cytokine and radical elements in response to foreign invading bodies (20). Normally**,** the cytokines released mediate the recruitment and activation of inflammatory cells including phagocytes and macrophages to the site of invasion. The pathogens are then engulfed and digested by these immune cells. Where high levels of indoor air pollutants exist, these host filtration and sterilization immune components of the respiratory tract are compromised. This in turn increases the risk of the development of ALRIs. The pathophysiologic mechanisms by the pollutants include triggering oxidative stress and the induction of local and systemic inflammation. They may also reduce the clearance of microorganisms by the mucociliary system, they can cause an enhanced reactivity of the respiratory epithelial cells, the macrophage response to microorganisms may be reduced, epithelial permeability may be increased and they may cause an increased adhesion of microorganisms to epithelial cells or bronchial irritation. These mechanisms of disease causation differ with the extent of exposure and by the type of polluting agent (21). Children are at a higher risk of contracting ALRIs because their airways are narrower, their resting metabolism is usually increased (22) and their aerobic metabolism is higher when compared to their size and adults (23). The most polluting agents responsible for a lot of health risks due to the use of solid fuels are PM and carbon monoxide. $PM_{2.5}$ whose concentration markedly increase inside kitchens during cooking to milligrams per cubic metre way above the WHO recommended guidelines is minimally filtered by the naso-oropharynx and can as a consequence enter the bronchi and alveoli causing respiratory tract irritation (17). The particles also result in defective humoral and cellular immune mediated pathways making infectious micro-organisms easily access the respiratory system. Black carbon, a component of particulate matter promotes the micro-organisms ability to tolerate multiple antibiotics. It also enables the spread of bacteria to the lungs which enhances disease occurrence. The mucociliary clearance system and cellular

mediated immune defences are significantly reduced by nitrogen dioxide thus causing defective micro-organism clearance (22). These pathophysiologic mechanisms are as summarized in figure 2.3.

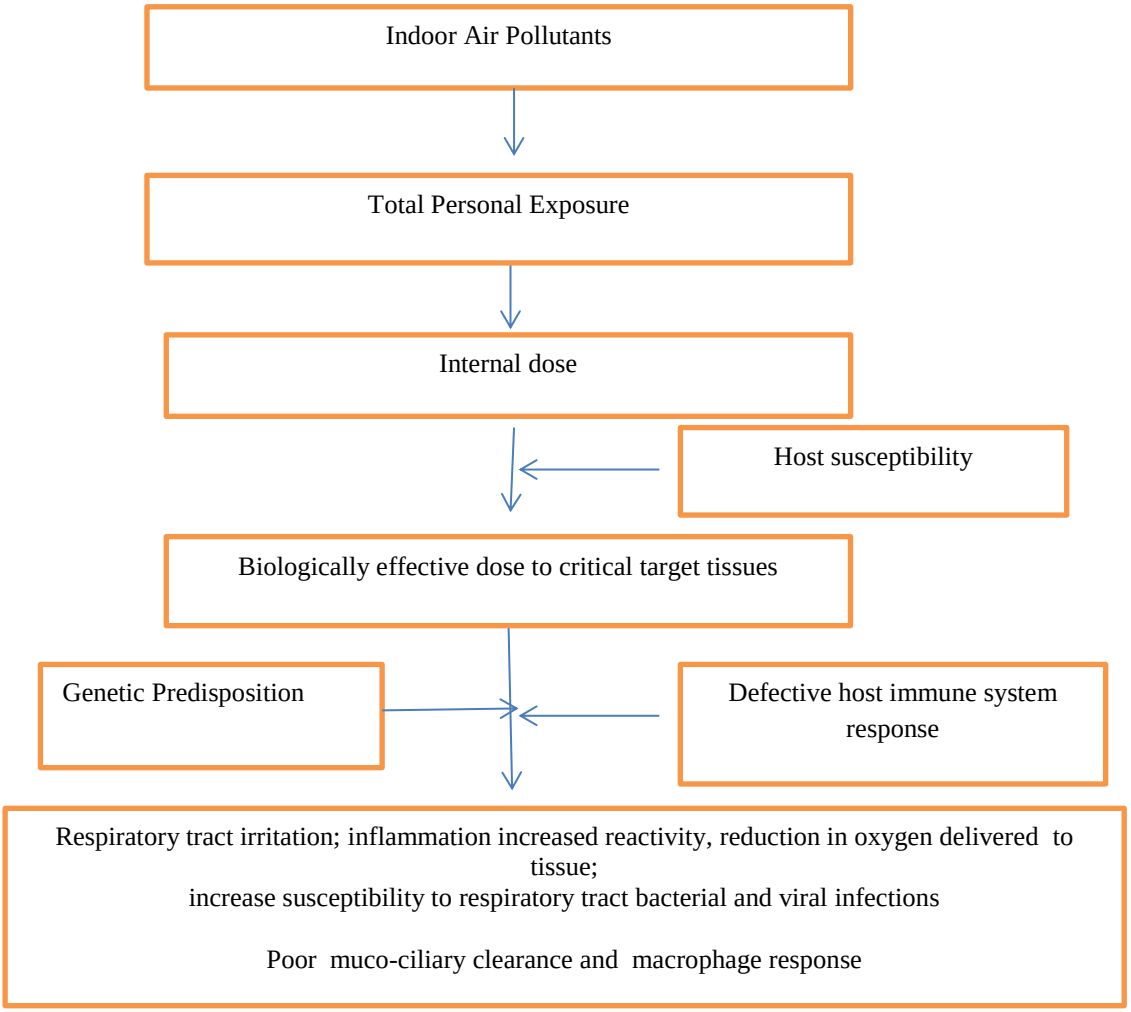

Figure 2.3: Pathologic effects of air pollutants on the respiratory tract (Smith KR 2000).

2.3 Management of pneumonia

Pneumonia in Kenya is diagnosed and treated clinically according to the WHO guidelines of case management of pneumonia. Use of physical examination findings helps to identify three levelsof acute respiratory illnesses based on the severity of their clinical presentation. These are no pneumonia which is predominantly cough/cold (an upper respiratory infection), pneumonia and severe pneumonia (24). Up to 13% of cases of pneumonia cases require

hospitalization due to their severity. Children who develop severe pneumonia are at an increased risk of long-term respiratory disease burden and morbidity (25).

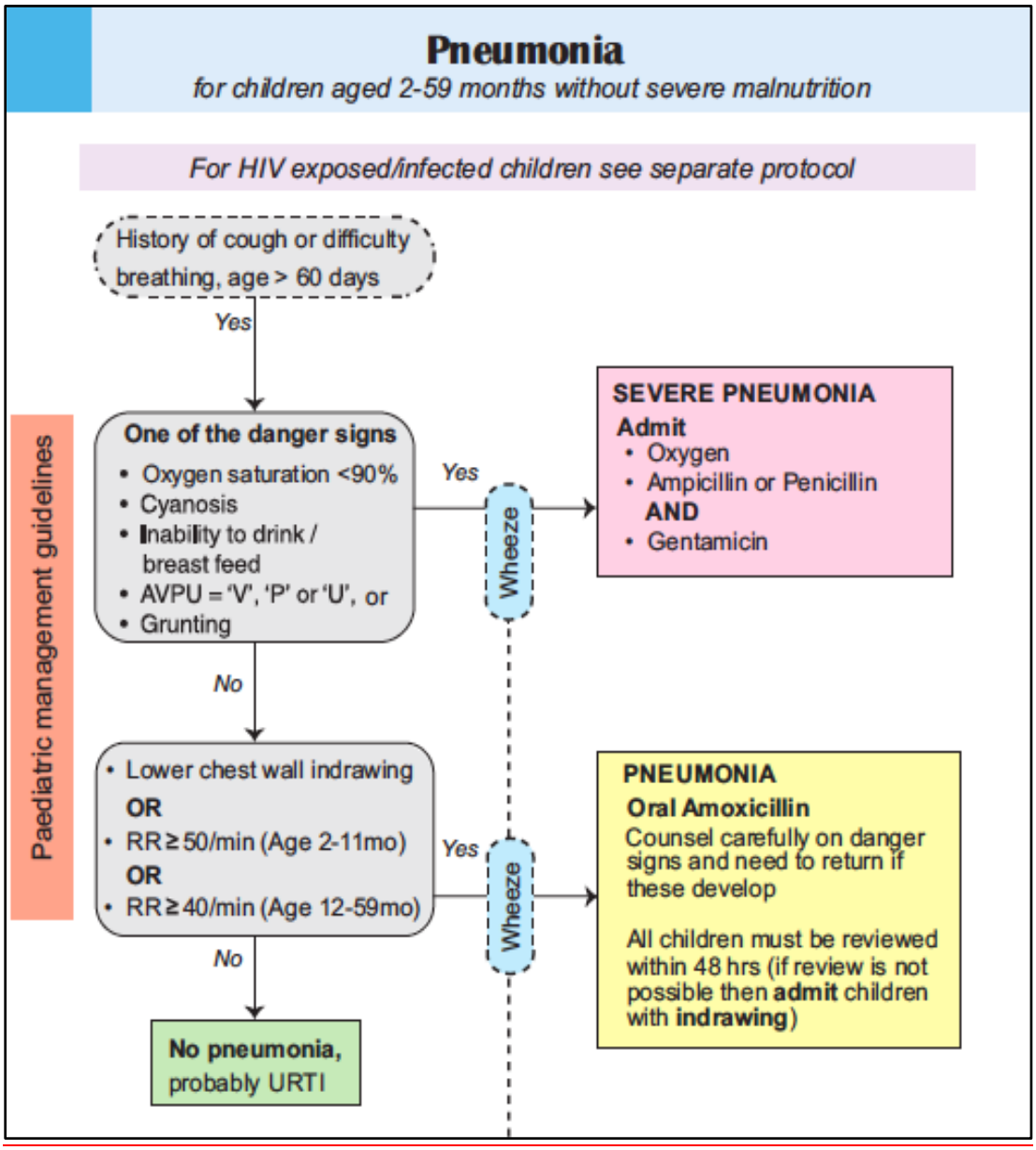

Figure 2.4: Classification of Pneumonia in children aged 2-59 months (WHO 2014) and Kenya Paediatric Protocol (2016).

Case management is a key component of pneumonia control strategies (2). This involves the classification of the severity of illness using the WHO clinical criteria then applying the appropriate treatment. The WHO recommendations for the treatment of pneumonia which is used in Kenya's public and private facilities are (24):

Recommendation 1: In children with fast breathing pneumonia but without chest in-drawing or general danger sign, oral amoxicillin should be used.

Recommendation 2: High dose oral amoxicillin should be used in children age 2–59 months with chest in-drawing pneumonia.

Recommendation 3: Parenteral penicillin and gentamicin are used as first-line treatment in children aged 2–59 months with severe pneumonia. In children with severe pneumonia who fail the first-line treatment, ceftriaxone should be used as a second-line treatment.

Recommendation 4: The first-line antibiotic regimen for HIV-infected and exposed infants and for children under 5 years of age with chest in-drawing pneumonia or severe pneumonia the recommended treatment is either penicillin and gentamicin or ceftriaxone

Recommendation 5: For HIV-exposed and HIV infected infants of 2 months to 1 year of age with severe or very severe pneumonia empiric cotrimoxazole treatment for suspected *Pneumocystis jirovecii* pneumonia (PCP) is recommended as an additional treatment but it is not recommended for HIV exposed and HIV infected children over 1 year of age with chest in-drawing or severe pneumonia.

Up to 20% of children admitted in Kenya with pneumonia or severe pneumonia and treated according to the WHO guidelines experience treatment failure. The two key elements that are of importance in the definition of treatment failure are the time before the clinical re-assessment and the parameters used to define failure.

The parameters that may be applied for poor response to treatment definition include a general lack of improvement including in oxygen saturation, an increase in heart rate, respiratory rate or temperature. They may also include an increase in oxygen requirement, the appearance of a new danger sign, care giver withdraw of the consent, a child leaving the hospital against medical advice, a change of antimicrobial for positive culture, a new comorbidity or death (25). Poor response to treatment occurs more in children whose families use of wood fuel as their primary source of fuel energy (8). The normal value of oxygen

saturation at the altitude of Nairobi at 1670 metres is should be above 95% as determined by Duke et al in Papua New Guinea at 1600 metres which showed oxygen saturation of children between 1-60 months was expected to be about 95.7% (SD=2.7%). Oxygen saturation measurement was found to be an objective way to determine response to pneumonia treatment (25).

Response to treatment of pneumonia is re-evaluated clinically after 48 hours, 60 hours, and one week or at any time after initiation of treatment (2) and classified as either a good response to treatment or a poor response to treatment according to the WHO (24).

Addo Yobo *et al*. in 2004 undertook a multi-centre, randomized study at tertiary-care centres in eight developing countries in Africa, South America and Asia. Their objective was to determine whether parenteral penicillin and oral amoxicillin were equivalent in their treatment of severe pneumonia in children aged 3–59 months admitted with severe pneumonia. The children were first admitted and randomly allocated to either arm of treatment. They were then assessed after 48 hours where treatment failure which was the primary outcome was ascertained. A poor response to treatment was defined as a low oxygen saturation, the appearance of a new danger sign, a change in antibiotics, a new comorbidity, persistence of lower chest in-drawing or death at 48 h. Prevalence of poor response to treatment was found to be 19% at 48 hours in either arms and the main risk factors of a poor response were infancy (age 3–11 months; odds ratio 2·72, 95% CI 1·95 to 3·79), and hypoxia (1·95, 1·34 to 2·82) and fast breathing (1·94, 1·42 to 2·65) (27).

McNally *et al*. in 2007, South Africa, did a prospective descriptive study where they investigated children with WHO defined severe or very severe pneumonia and who were commenced on standard antimicrobial treatment of benzylpenicillin and gentamicin. Infants also received a high dose trimethoprim-sulfamethoxazole. The primary outcome was to determine predictors of poor response to treatment and to determine the cause of non-responsiveness pneumonia in these children at 48 hours. Poor response to treatment was defined as a lack of improvement or an increase in temperature, the respiratory rate or heart rate. It also included an increase in oxygen requirement, the appearance of a new danger sign, care giver withdraw of the consent or the child left against medical advice. A change of antimicrobial for positive culture, a new comorbidity or death was also included in the treatment failure definition. They determined that a poor response at 48 h was predicted by

disease severity-very severe disease (2·47, 1·17–5·24, p=0·0181), age of less than one year (adjusted odds ratio 6·38, 95% CI 2·72–14·91, p<0·0001), polymicrobial disease (one organism 2·06, 1·05–4·05; two organisms 10·75, 4·38–26·36; p<0·0001) and HIV status (HIV infected 10·3, 3·26–32·51; HIV exposed, uninfected 6·02, 1·55–23·38; p=0·0003), (28).

Webb *et al* in 2008 undertook a cohort study in Kenya whose main aim was to determine the frequency of poor response to treatment among children aged 2-59 months admitted to hospital with severe pneumonia. The two key elements in the definition of poor response to treatment were the time before re-assessment and the criteria for failure. They defined poor response to treatment at 48 hours as the worsening or a lack of improvement in any of the following clinical features- conscious level, oxygen saturation less than 90%, an increase in respiratory rate by at least five breaths per minute or a temperature more than 37.5°C. A poor response also included a new finding of bacterial meningitis, signs of shock, empyema, renal impairment or death. They determined that a poor response to treatment at 48 hours was 20%, 95% CI 17-23%. Poor response was mainly associated with HIV infection status and severe malnutrition (26).

Maria Alkinson *et al.* in 2007 in the United Kingdom, where domestic use of polluting fuels is minimal or non-existent, conducted a multicentre randomized but non-blinded trial comparing intravenous and oral treatment for pneumonia in previously well infants and children. A total of 252 children were randomized. The primary outcome of interest was the time from randomization until oxygen was no longer required and the temperature was less than 38°C. Those who failed treatment in the intravenous arm were approximately 7%. 13% of pneumonia was caused by viruses. They also determined that it took approximately 1.3 days for the temperature to settle and 1.2 days for the oxygen requirement to cease for the two groups (29).

Agweyu *et al* in 2015 determined that the average duration of hospital stay during severe pneumonia was 4 days. The study was undertaken in Kenya at the Kenyatta National Hospital (30). Webb *et al* in their study in Kilifi, Kenya established that in children with severe pneumonia but without HIV or severe malnutrition, 4.3% had a poor response to treatment at day 5 and 0.3% died by day 5 of treatment. Among children with severe

pneumonia complicated by HIV or severe malnutrition 22% failed treatment at day 5 day and 16% died by day 5 of treatment (26).

2.4 Effect of household fuel exposure on pneumonia treatment outcome in children

A prospective cohort study to determine whether household use of wood as a cooking fuel was associated with a worse pneumonia treatment outcome 48-hours amongst children less than two years of age admitted with a diagnosis of pneumonia in Botswana was conducted by Kelly *et al*. in 2015. The study was at a tertiary hospital in Gaborone the capital city of Botswana. . The use of wood as a household fuel was determined during a face-to-face questionnaire with caregivers. They established that household use of solid fuels as a cooking fuel was associated with an enhanced risk of a poor response to treatment by 35% in children aged 1 to 23 months after 48 hours of treatment (RR 1.44, 95% CI 1.09–1.92, $P = 0.01$) (8).

Table 2.3 below summarises the various studies on prevalence of pneumonia treatment failure in children hospitalized with pneumonia in different settings.

Table 2.3: Studies on prevalence of pneumonia treatment failure in children hospitalized with pneumonia in different settings

Country, Author	**Study design and sample size**	**Study population**	**Title**	**Clinical definition of treatment failure**	**Result**
Botswana, Kelly *et al* The International Journal of Tuberculosis and Lung Disease, 2015 (4).	Cohort study N=284	1 to 23 months of age with pneumonia	The effect of exposure to wood smoke on outcomes of childhood pneumonia	Persistent lower chest wall in-drawing, development of new WHO danger signs	Poor response-35%, (RR: 1.44; 95% CI: 1.09-1.92; *P*=0.01). Wood smoke exposure was associated with a 1.44 increased risk of poor

					pneumonia outcome at 48 hours.
Multi Centre-Colombia, Ghana, India, Mexico, Pakistan, South Africa, Vietnam and Zambia. Addo-Yobo *et al*. The Lancet, 2004 (34)	Randomised control study N=1702	3–59 month; International inpatient study population with severe pneumonia	Oral amoxicillin versus injectable penicillin for severe pneumonia in children aged 3 to 59 months	At 48 hours, any 1 of the danger signs, low oxygen saturation, persistent in-drawing, a new antibiotic/ comorbidity or death	Poor response to treatment was at 19% at 48 hours. Factors associated with poor response to treatment were infancy (age 3–11 months) hypoxia fast breathing
South Africa, McNally *et al*. The Lancet, 2007(35)	Prospective descriptive study N=358	1-59 months; South Africa; Inpatient; Severe and very severe pneumonia	Effect of age, polymicrobial disease, and maternal HIV status on treatment response and cause of severe pneumonia in South African children	At 48 hours, persistence or worsening of any 1 of: heart rate, respiratory rate, temperature, inability to drink, increased oxygen requirements; New danger signs; Absconded; Change of antibiotic for new disease or blood culture result; Death	Poor response to treatment was at 35% after 48 hours. They determined that a poor response to treatment at 48 h was predicted by age of less than one year, HIV status, disease severity and polymicrobial disease

Kenya, Webb *et al* The Pediatric infectious disease journal, 2012 (16).	Cohort Study N=710	Children aged 2-59 months with severe pneumonia	Treatment failure among Kenyan children with severe pneumonia.	No improvement or worsening of SaO_2, conscious level, temperature or respiratory rate; no improvement or a new finding of empyema, bacterial meningitis, renal impairment or signs of shock	Poor response to treatment in children was 20% after 48 hours of treatment (95% CI 17-23%) Poor response to treatment was associated with HIV infection status and severe malnutrition
United Kingdom, Atkinson M *et al*, Thorax, 2007 (36).	Multicentre randomized trial N=246	Children more than 6 months of age	Comparison of oral amoxicillin and intravenous benzyl penicillin for community acquired pneumonia in children (PIVOT trial):	A continued oxygen requirement; temperature more than 37.5°C.	Poor response to treatment was 7% 1.3 days needed for the temperature to settle; 1.2 days needed for the oxygen requirement to cease

2.5 Measurement of household Indoor Air Pollution

The WHO in its population levels of household air pollution and exposures Review 5, 2015, developed a checklist (Appendix A) on key variables that can be used to collect detailed data on IAP from household fuel exposure. Three variables best predict exposure to household indoor pollution and these include fuel type, kitchen type and the ventilation (31).

Fuel type is the best predictor of concentration of pollutants to household fuel exposure. Use of solid fuels is associated with more exposure to IAP when compared to use of other sources of household energy. Kitchen type can be categorized into either an enclosed kitchen with a partition, without a partition, a separate enclosed kitchen outside the house or an outdoor kitchen. The kitchen with the partition is associated with a greatest exposure to pollution while outdoor kitchens are associated with the least exposure to IAP. A well ventilated house is associated with less exposure to IAP when compared to a poorly ventilated house. The time-activity is used to determine the approximate time a family member is exposed to IAP from household fuels. It is based on a 24-hour recall that details the type of activities undertaken in the house with an emphasis on cooking.

Other factors that determine exposure include the cooking stoves characteristics which influence the emissions and thus exposure levels to indoor air pollution, household characteristics and the number of household members (31).

The extent to which a child is exposed to household IAP can be determined indirectly as demonstrated by Dherani *et al*. This involves determining the type of household fuel used, the time a child spends near the household fuel, whether the child is carried on mothers back during cooking, whether cooking occurs inside or outside the house. Exposure can also be determined directly when actual measurements of IAP are available (9).

Kurmi *et al* in their study on indoor air pollution and the lung in low-and medium-income countries determined that the choice of fuel is determined by availability of both modern fuels and of local biomass fuels and affordability, which is influenced by household income and the policy options existing such as taxes, prices and subsidies as shown in figure 2.4 (32).

Both structured questionnaires and biomedical measurement may be used to identify the levels, extent and the nature of exposures. They can also be used to understand the contributions of individual determinants. Data on exposure include the type of domestic fuel used, the location and type and of the kitchen and the type of stove used for cooking. Household sample surveys of fuel use, household characteristics which may include type of building material in use, type of stove, number of rooms including room ventilation may also be used to ascertain exposure.

Biomedical measurement of pollution exposure includes stationary air sampling devices that can be placed in the home for a timed period such as 24 hours or several days to measure amount of pollution within the home over the period. Alternatively worn devices, biological fluid or tissue biomarkers can also be used to measure personal exposures to pollution. These devices are expensive and home placement requires higher finances, transport and related resources. For this reason, few studies in LMIC have been able to employ this approach to verification of fuel exposure. Thus, most research studies in LMICs mostly use questionnaires to collect data on exposure due to the lower cost of this approach and limited funding for research in their setting. The questionnaires are usually self-reporting to facilitate the determination of the level of exposure related to IAP and the various health outcomes (33).

2.6 Household Indoor Air Pollution and risk of Pneumonia in children

The level of exposure determines the risk of ill health and (34) a dose–response relationship exists between the particulate matter and the increased risk of acute respiratory infection. A higher exposure to particulate matter in children increases the likelihood of developing pneumonia compared to children with minimal particulate matter exposure (35).

Nandesena *et al* in their meta-analysis of 24 studies, determined that the overall odds ratio of developing pneumonia was 1.78 (95% CI: 1.45-2.18) among children of the less than five years of age exposed to IAPs from solid fuels when compared to children who were not exposed.

They also established that a 75 min daily wood fuel exposure for 30-45 days is associated with severe lower respiratory tract infections (19).

There is a two fold increase in incidence of lower respiratory tract infections in children spending more than two hours near the cooking fire each day and when solid fuel is used while the child is on the mother's back (36). The average for 24-hr exposures for children is approximately 199μg/m3 for PM_{10} while it is 219μg/m3 for $PM_{2.5}$ (11).

In a case control study by karki *et al*. (2014) in Nepal, it was established that in houses where food preparation was done indoors, the risk of developing pneumonia increased by four times (OR 3.76, 1.20–11.82) (37). Smith *et al*. in Guatemala determined that the lack of or poor

ventilation in homes using biomass fuel and use of inefficient stoves which lack fireplaces to take the smoke out of the inhabited area often enhance the adverse effects of IAP on health (38).

2.7 Study Justification

The WHO estimates that 3.9% of all deaths in LMICs are attributable to IAP. 8-10% of early deaths are attributable to indoor air pollution in Kenya (14). The primary target body system of air pollutants is the respiratory system with the risk of pneumonia enhanced by 80% as a result of indoor air pollution (9).

Children are amongst the most vulnerable groups for adverse effects of IAP. However, indoor air pollution from household indoor exposure is a modifiable risk factor for pneumonia outcome to treatment with the potential of a variety of interventions to mitigate its effects. The improved knowledge on exposure is now a useful tool for developing effective intervention options.

Our aim was to determine if IAP following household fuel exposure impacts pneumonia treatment outcomes among children hospitalized in a tertiary hospital setting which provides insights to guide whether interventions are needed for these children.

2.8 Study Utility

Indoor air pollution is a modifiable determinant of pneumonia with the potential of a variety of interventions to mitigate its effects. The study documents pneumonia treatment response after 48 hours and 5 days of treatment following exposure to different types of household fuels. The improved knowledge on exposure will become a useful tool for developing effective intervention options.

3.0: RESEARCH QUESTIONS AND STUDY OBJECTIVES

3.1 Research Question

What is the effect of exposure to indoor air pollution from household fuels on the 48 hour response to pneumonia treatment amongst children aged between 2 and 59 months admitted with pneumonia at the Kenyatta National Hospital, Nairobi?

3.2 Research Objectives

3.2.1 Overall Objective

To evaluate the prevalence of exposure to indoor air pollution from household fuels and its effect on treatment responses amongst children aged 2 and 59 months admitted with pneumonia at the Kenyatta National Hospital, Nairobi.

3.2.2 Specific Objectives

The primary objectives of the study were:

1. To determine the prevalence of exposure to indoor air pollution from household fuel among children aged 2-59 months hospitalised with pneumonia at Kenyatta National Hospital.
2. To determine the effect of exposure to indoor air pollution from household fuels on 48 hour pneumonia treatment response amongst children aged between 2 and 59 months hospitalised at Kenyatta National Hospital, Nairobi.

The secondary objective of the study was:

To determine the effect of exposure to indoor air pollution from household fuels on 5 day of pneumonia treatment response amongst children aged between 2 and 59 months hospitalised at Kenyatta National Hospital, Nairobi.

4.0 METHODOLOGY

4.1 Study Design

This was a hospital based prospective cohort study.

4.2 Study Location

The study was conducted in the general pediatric wards of Kenyatta National Hospital which is the largest referral hospital in East and Central Africa. The hospital also serves as a teaching hospital of the University of Nairobi. It is located in the city of Nairobi, the capital city of Kenya with a population of about 4.3 million people according to the Kenya National Bureau of statistics, 2019 (38). It has a total bed capacity of approximately 2000 with 50 inpatient wards and 70,000 admissions yearly. The paediatric medical department has four in-patient general wards. Each ward has a bed capacity of 30 patients thus a total bed capacity of 120 but is usually overstretched with more than 200% bed occupancy at any given time. There are approximately 10,000 paediatric admissions annually. Most of the patients are admitted with acute childhood illnesses. The majority are referred from primary care facilities at 65%. Direct self-referrals are 20% while 15% are referred from private hospitals or public facilities. Approximately one third of all general paediatric admissions are due to clinically diagnosed pneumonia (40).

4.3 Study Population

The study population included children aged between 2 and 59 months admitted at Kenyatta National Hospital, Nairobi general paediatric wards with WHO-defined pneumonia and started on the WHO recommended treatment regimen.

The study population was divided into two main categories. Category one was of a population exposed to low-polluting household fuels which were electricity and LPG as their main source of household fuel. The second category was a population who used high-polluting household fuels as their main source of household fuel which included solid fuels - dung, crop waste, wood, charcoal and kerosene, a petroleum product.

Inclusion Criteria

- Children aged between 2 and 59 months hospitalised at Kenyatta National Hospital.

- Presence of clinical diagnosis of severe pneumonia per WHO pneumonia diagnostic criteria as detailed in the case definition.
- The children are started on antimicrobial treatment as per the WHO guidelines.
- Informed written consent from the parent.

Exclusion Criteria

- Children hospitalized in the prior 14 days to decrease the possibility of a hospital acquired pneumonia or an incompletely treated pneumonia.
- Children with co-morbid major organ conditions that might significantly impacted the outcome of interest including children with an underlying debilitating condition such as a heart disease or a chronic lung disease including chronic pulmonary infections except asthma.
- Children referred from an inpatient hospital in which pneumonia treatment has been initiated.
- A child whose initial antibiotic treatment given within KNH was not in line with WHO recommended treatment.

4.4 Case Definitions

Pneumonia case definition

Pneumonia in children aged between 2 and 59 months is categorised by the WHO into either severe pneumonia or pneumonia. Only children with severe pneumonia as defined by the WHO were included in the study.

Severe pneumonia was defined as a child with clinical features of pneumonia and one of the danger signs which can either be an oxygen saturationof less than 90%, been cyanosed, been unable to breastfeed or drink , AVPU at either 'V', 'P', or 'U', or a child who is grunting.

Pneumonia in a child aged between 2 and 59 months was diagnosed in a child presenting with a history of cough or difficulty in breathing with lower chest in-drawing or fast breathing (respiratory rate >50/minute in 2-11 month old children and a respiratory rate of >40 breaths per minute in 12-59 month old children).

WHO Recommended Pneumonia Treatment

Only those who were started on the WHO treatment recommendations were recruited to the study. The WHO recommendations for the treatment of pneumonia as used in Kenya's public facilities are (14):

- *Severe Pneumonia treatment:* In HIV negative, parenteral penicillin and gentamicin are used as first-line treatment Children aged 2–59 months with severe pneumonia. Ceftriaxone should be used as a second-line treatment in children with severe pneumonia who fail the first-line treatment.

- *For HIV-infected or exposed* : For HIV-exposed and HIV infected infants of 2 months to 1 year of age with severe or very severe pneumonia empiric cotrimoxazole treatment for suspected *Pneumocystis jirovecii* pneumonia (PCP) is recommended as an additional treatment but it is not recommended for HIV exposed and HIV infected children over 1 year of age with chest in-drawing or severe pneumonia.

- *Pneumonia with no chest indrawing:* oral amoxicillin should be used in children with fast breathing pneumonia with no chest in-drawing or general danger sign.
- *Pneumonia with chest in-drawing*: a high dose oral amoxicillin should be used in children age 2–59 months.

Response to Pneumonia treatment

The primary outcome, response to pneumonia treatment was assessed after 48 hours of treatment. The definition was drawn from a study conducted by Clare Webb and James Berkley et al in Kilifi (25).

A poor response to treatment at 48 hours was defined as:

-Death before 48 hours.

-By arterial oxygen saturation, a child was classified as having a poor response to treatment if:

Oxygen saturation (SPO_2) remained the same as baseline or declined from the baseline.

Oxygen saturation (SPO_2) declined from above 95% to below 95%.

A good response to treatment at 48 hours was defined as:

-Any increase in oxygen saturation (SPO_2) from the baseline

-Maintenance of oxygen saturation (SPO_2) above 95%

-Discharge from hospital before completing of 48 hours.

A poor response to treatment at five days was defined as:

-Death before 5 days of treatment.

-Clinical persistence of disease.

A good response to treatment at five days was defined as clinical resolution of disease resulting in discharge from hospital.

4.5 Sample Size Determination

Objective 1: Prevalence of IAP exposure among children hospitalized with pneumonia.

The sample size determination for prevalence was calculated by Fischers' formula as indicated below.

$$n = \frac{z^2 p(1-p)}{d^2}$$

n = Estimated minimum sample size.

Z^2_{α} ²= the square of the standard normal deviation corresponding to a confidence interval of 95%.

p = 0.721 (Prevalence of urban households exposed to indoor air pollutants (KDHS, 2014) (7).

d = level of precision (set at 10%)

$$n = \frac{1.96^2 \times 0.721(1 - 0.721)}{0.1^2}$$

$$n = 77$$

In order to cater for any loss to follow-up, or children missing data on key outcomes, we the size was increased by 10% to give a sample size of 85 for this study.

Objective 2: Effect of IAP exposure on 48 hour pneumonia outcome

The minimum required sample size to determine the effects of IAP on 48 hour pneumonia treatment outcome was computed using the Epi info, Version 3 (41), open source calculator-SS cohort, Fleiss formula for sample size estimation (42). The following assumptions and estimates are made:

-Two sided significance level (1-alpha) is 95%.

-Power (1-beta) is set at 80%.

-Estimated ratio unexposed/exposed is 1:1.

-Estimated percent of unexposed with outcome is 10%.

With the above assumptions, a sample size of 124 participants gives the power to detect a relative risk of three or higher of poor outcome in the exposed compared to the unexposed.

In order to cater for any loss to follow-up, or children missing data on key outcomes, this was increased by 10% to give a sample size of 137 for this study.

The higher of the two sample sizes obtained for the two objectives was used as the study's sample size thus a total of 137 study participants were to be recruited to the study.

4.6 Sampling Method

Consecutive sampling was used to recruit the study participants. All children aged between 2 and 59 months who met the inclusion criteria and were admitted during the duration of the study with clinical features of pneumonia or severe pneumonia as defined by the WHO and started on the WHO recommended treatment were consecutively recruited to the study until the sample size was reached.

4.7 Study Tools

Data was obtained using a structured researcher-assisted questionnaire (Appendix B). The case record form captured information on socio-demographic characteristics, home indoor fuel use and relevant clinical information at enrolment, 48 hours and 5 days.

4.8 Study Procedures

Data was be collected over a three month period at the Kenyatta National Hospital general paediatric wards by research assistants and the principal investigator on children aged 2-59 months admitted with WHO clinical features of pneumonia or severe pneumonia and started on the WHO recommended treatment. Data was collected within eight hours of admission in the general wards, then at 48 hours and at five days after initiation of treatment. Children admitted during the weekends were also included in the study. Screening for eligibility was done once the admitting doctor made the diagnosis pneumonia according to the WHO guidelines. Consent was obtained once the patient had been stabilised. The research person introduced him/herself to the guardian and then proceeded to explain the purpose, usefulness and voluntary nature of the study. Eligible patients were then selected. Consent and assent were both administered in written forms. This was done in either English (Appendix C) or Kiswahili (Appendix D) languages according to the guardian's preference.The interviewer then proceeded to fill the questionnaire both at admission and two days after initiation of treatment and at both instances he will thank the caregiver for the information provided.

At enrolment, information obtained through the interview was data on the socio-demographic data, home fuel use and relevant clinical history for pneumonia diagnosis and severity, and co-morbid chronic disease.

The physical exam was then undertaken to determine to determine relevant respiratory signs such as chest indrawing, cyanosis, pallor, respiratory rate and temperature and chest examination. Pulse oximetry to determine the oxygen saturation was also be recorded. Anthropometric measurements were collected by the researchers at their first contact with the study participant. A weighing machine was used to determine the weight of the patient while a standiometer or an infantometer was used to determine the height of the patient. A mid upper arm circumference tape was used to determine the child's mid upper arm circumference.

The details of antibiotic and oxygen treatment initiated were extracted from the child's medical record treatment sheet and administration of oxygen treatment verified at the bedside.

Evaluation after 48 hours and five days of treatment included a determination of key clinical features that define pneumonia response to treatment and specifically the oxygen saturation (SPO_2) whether it had remained the same as or decreased as compared to the baseline saturation at admission or death. Any decline in oxygen saturation (SPO_2) from above 95% to less than 95% was also determined. Participants were also reviewed at day 5 of admission to determine whether they had recovered or had a persistent clinical illness or mortality and their oxygen saturation (SPO_2) levels determined.

4.9 Data Management

Data was organized, screened, and checked for completeness. The data was then coded, inputed into a computer and cross-checked against the original data set for accuracy. Data entry was via a computer using Epi-info version 3.5.1./IBM SPSS v20 and was then converted to CSV format for analysis. The first objective, to determine the prevalence of exposure to indoor air pollution from household fuel among children aged 2-59 months hospitalised with pneumonia at Kenyatta National Hospital was analysed and presented using percentages and frequencies. The numerator included children from household using the type of fuel while the denominator included all children enrolled in the study.

The second objective, to determine the effect of exposure to indoor air pollution from household fuels on 48 hour treatment outcome (treatment failure and/or mortality), amongst children aged between 2 and 59 months hospitalised at Kenyatta National Hospital, Nairobi, was analysed using odds ratios for a cohort study and calculated as follows:
Odds ratio (OR) calculation= risk of outcome occurrence in exposed / risk of outcome in unexposed.

OR> 1 suggests high polluting household fuels predisposes to poor response to pneumonia treatment

OR<1 suggests high polluting household fuels predisposes to good response to pneumonia treatment

OR=1 was to be null and would indicate that there is no association between indoor fuel exposure response to pneumonia treatment.

Chi square for grouped or nominal variables and fishers exact correction are applied where a variable has a frequency of <5. Chi square for linear trend was used to assess incremental polluting fuel exposure against 48 hour and 5 day outcomes. Significant association is defined where the 95% confidence interval for odds ratio was 1.0 and a P-value <0.05.
For the secondary objective, survival analysis was done to assess the effects of IAP exposure on the 5-day outcome using variable of time from admission to outcome death versus time to survival/recovery as the outcome in the model. Cox regression hazards for poor outcome will be performed and Kaplan Meir survival curves developed.

4.10 Ethical Considerations

Ethical approval for the study was obtained from Kenyatta National Hospital-University of Nairobi Ethical Review Committee (KNH-UoN ERC). Permission to access caregivers and wards was obtained from the Kenyatta National Hospital administration. Both written and verbal consents were obtained from the study participants after a careful explanation. The voluntary aspect of the study was also explained.

4.11 Dissemination of the Study Findings

The results of the study was presented to the UoN paediatric registrars and the KNH/UoN paediatric consultants during poster presentation and a copy will be provided to the paediatric Kenyatta National Hospital unit. The manuscript after completion will be sent to an academic journal for approval of publication in order to reach a wider public. Arrangements will be made as well to send abstract to upcoming medical conferences for further dissemination discussion and policy interventions.

CHAPTER 5: RESULTS

5.1 Participants' screening, enrolment and follow-up.

A total of 169 respondents were eligible for the study during screening. Of those who were eligible, 42 were not included in the study. This was due to the fact that twenty declined to give consent, nine had the treatment initiated at admission not in line with WHO's recommended severe pneumonia treatment, six had been admitted in the preceding 14 days prior to the current admission while ten of the eligible participants had chronic medical conditions.One hundred and twenty seven (127) respondents consented to the study and were thus enrolled. On follow up, at 48 hours, 10 respondents had died while the remaining 117 respondents were still admitted in hospital. At 5 days, 76 respondents had been discharged, 17 had died while 34 were still admitted in hospital as shown in Figure 5.1.

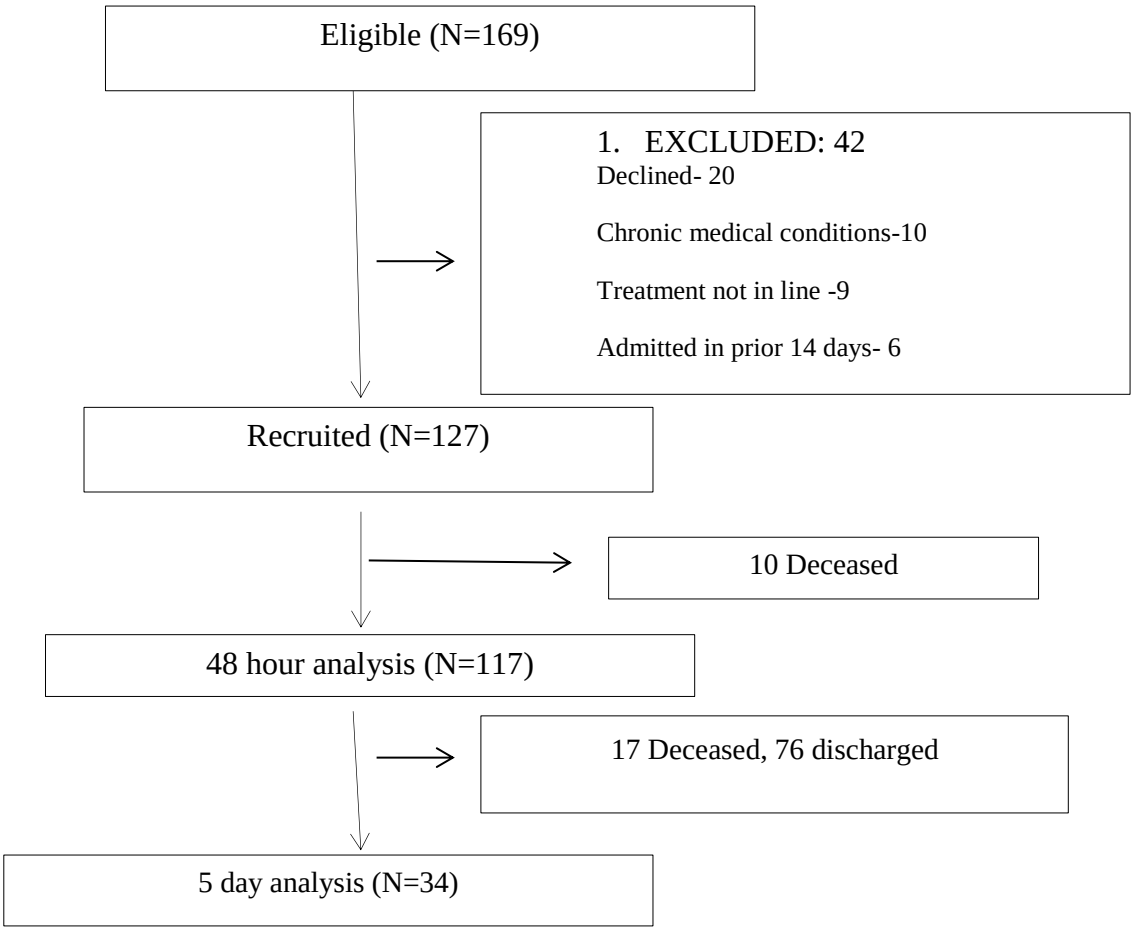

Figure 5.1: study participants screening, enrolment and follow-up.

5.2 Socio-demographic characteristics of the study respondents

Most of the participants parents were married at 101 (79.5%), were housewives at 63 (49.6%), had a secondary level of education at 83 (65.3%), their spouses were self-employed 44 (43.5%), their spouses also had a secondary level of education 58 (57.4%) and most resided in an urban are at 105 (82.6%) as shown in Table 5.1.

Table 5.1: Socio-Demographic characteristics of the study respondents

Characteristic of parent/guardian		**Frequency (N=127)**	**Percent**
marital status	Single	13	10.2
	Married	101	79.5
	Divorced/ separated	13	10.3
Level of education	No formal	4	3
	Primary	19	15
	Secondary	83	65.3
	Tertiary	21	16.5
Occupation	Employed	8	6.2
	Self employed	31	24.4
	Unemployed	25	19.6
	housewife	63	49.6
Relationship to the child	Mother	124	97.6
	Father	2	1.5
	Grandmother	1	0.7
Spouse occupation	Employed	14	13.8
	Self employed	44	43.5
	unemployed	12	11.8
Spouse level of education	Primary	14	13.8
	Secondary	58	57.4
	tertiary	8	7.9
Residence	urban	105	82.6
	rural	22	17.4
Mothers average age	29 years		
Fathers average age	34.2 years		

5.3 Environmental characteristics of the study respondents

5.3.1 Household fuel exposure

92 (72.5%) and 35 (27.5%) of the respondents were exposed to high and clean/low levels of indoor air pollutants from household fuels respectively. Amongst the participants 'exposed to high levels of indoor air pollutants from household fuels, 28 (22 %) used kerosene as their preferred source of household fuel. This was followed by use of charcoal at 21 (16.5%) and wood only 7(5.5%). 37 (28.3%) mixed their fuel use and were classified as exposed to high level of indoor air pollution. Amongst the participants' exposed to low levels of indoor air pollutants, all of them used LPG as their preferred source of cooking fuel (Table 5.2).

Table 5.2 Household fuel type exposure

Household fuel use	Frequency (N=127)	Percent (%)
Polluting Fuels:	**92**	**72.5**
Kerosene	28	22
Charcoal	21	16.5
wood	7	5.5
Mixed/Stacking	37	28.3
Clean fuels:	**0**	**27.5**
LPG	35	27.5
electricity	0	0.0

5.3.2 Home environment characteristics of the study respondents

Most households had an indoor kitchen with a partition at 84 (66.1%). In a majority of households, cooking took less than one hour at 118 (93%), was done in the same room the

child was located at 90 (70.8%) but was not done while the child was been carried on the parents/guardians back at 106 (83.5%). Most households prepared 2-3 meals per day at 105 (90.5%) and most had between 2-3 rooms at 58 (45.6%). Most kitchens had at least one window at 122 (96%) as shown in Table 5.3.

Table 5.3: Home environment characteristics of the study respondents

	Frequency (N=127)	**Percent (%)**
Kitchen characteristics		
Indoor kitchen with partition	84	66.1
Indoor kitchen without partition	20	15.7
Kitchen outside the house	23	18.1
Average duration of cooking		
Less than 1 hour	118	93
More than one hour	9	7
Childs location during cooking		
In the same room	90	70.8
Different room	31	24.4
Sometimes same/ different	6	4.7
Child carried on mothers back during food preparation	21	16.5
Number of designated kitchen windows		
0	5	4
1	122	96
Number of rooms in the house		
1	15	25.8
2-3	58	45.6
>3	54	42.5
Meals cooked per day		
1	7	5.5
2	69	54.3
>3	49	38.5
Smoking within the house	3	2.3

5.4 Age, gender and physical findings of the study respondents at admission

The majority of participants were female at 50 (55.6%) and most were aged between 2-12 months at 106 (83%).

The mean weight of the participants IAP was 7.4 kgs (SD=2.29), height was 66.1 (SD=13.5), temperature was 38.1°C, respiratory rate per minute 53 (SD=16.9), heart rate per minute137 (SD=25.9). Most of the respondents were well nourished at 123 (96%) as shown in Table 5.4.

Table 5.4: Age, gender and physical findings at admission

Age:	
2-12 months (%)	106 (83.4)
13-60 months (%)	21 (16.6)
Sex	
Male	70 (55.1)
Female	57 (16.6)
Physical findings at admission	
weight kilograms	7.4 (2.29)
Temperature	38.1ºC (SD=1.3)
Respiratory rate (SD)	53 (SD=6.9)
Heart rate (SD)	137 BPM (SD=25.9).
Nutrition status	Malnutrition 4 (W/H Zscore 2 SD)
	Normal 123 (W/H Zscore 2 SD)

5.5 Clinical status of the study respondents at admission

All of the study participants recruited to the study had a cough as their main presenting symptom. Most also had difficulty in breathing at 120 (94.4%) and hotness of the body 117 (92.1%). Most the participants at 110 (86.7%) did not have any significant medical/ family/ social history at admission. Most were referrals from other health facilities at 67 (52.7%). Most the participants at 121 (95.2%) had their immunization status up to date and most were breastfeeding appropriately at 103 (81.1%). 119 (93.7%) were not exposed to HIV/AIDS as shown in Table 5.5.

All of the study participants recruited to the study had fast breathing for their age. Most had lower chest wall indrawing at 121 (95.2%) and grunting 69 (54.3%). 102 (80.3) were alert at admission and only 7 (5.5%) had a stridor at admission (Table 5.5).

Table 5.5: Clinical features of children with pneumonia at admission

Admission Symptoms of the study participants		**Frequency (N=127)**	**Percent (%)**
Difficulty in breathing		120	94.4
Hotness of the body		117	92.1
Abnormally sleepy		10	7.8
Relevant medical/family/social history	yes	17	13.3
	no	110	86.7
Referral status: From another facility	yes	67	52.7
Referring facility	Dispensary/health centre	30	23.6
	Level four/five health facility	37	29.1
	Self-referral	60	47.2
Immunisation status	Up to date	121	95.2
Breastfeeding status	Appropriate	103	81.1
HIV status	Sero-exposed	8	6.3
	Not exposed	119	93.7
Confirmed HIV positive	Yes	2	1.6
Grunting		69	54.3
Inability to drink/breastfeed		61	48
AVPU	A V P U	102 7 17 1	80.3 5.5 13.3 0.7
Severe respiratory distress		32	25.1
Lower chest wall indrawing		121	95.2
Stridor		7	5.5

5.6 Follow up clinical status of the study respondents

After 48 hours of treatment, 117 (92.2%) of the respondents were still admitted in hospital and were still on treatment. 10 (7.8%) had died while no patient was discharged within the 48 hours of admission. After 5 days of treatment, 34 (26.7%) respondents were still admitted, 17 (13.3%) had died while 76 (59.8%) had been discharged from hospital. The average duration of hospital stay for those discharged was 3.9 days (Table 5.6).

Table 5.6: Follow up clinical status of the study respondents

Follow-up		**Number**	**Percent (%)**
48 hour	Dead	10	7.8
	Alive still on Rx	117	92.2
	Discharged	0	0.0
Day 5	Dead	17	13.4
	Alive still on Rx	34	26.8
	Discharged	76	59.8
Duration in ward	Median days	3.9	IQR 2 days
Average Hours to discharge (days)	High polluting household fuels	98 (4)	
	Low polluting household fuels	89 (3.7)	

5.7 Oxygen saturation at admission and 48-hours

A majority of the participants had oxygen saturation between 90-94% at 74 (58.2%)) at admission. 29 (22.8%) had oxygen saturations at between 85-89%, 12 (9.4%) had saturation of less than 85% and another 12 had oxygen saturation above 95%.

After 48 hours of treatment, the majority at 58 (49.5%) had their oxygen saturation at between 90-94%, 30 (25.6%) at above 95%, 25 (21.3%) at between 85-89% while 4 (3.4%) had their oxygen saturation below 85%.

Table 5.7: Oxygen saturation at admission

Oxygen saturation levels	**At admission, percent (%)**	**After 48 hours, percent (%)**
<85%	9.4	3.4
85-89%	22.8	21.3
90-94%	58.2	49.5
>95%	94	25.6

5.8 Treatment outcome Analysis

Treatment responsiveness was determined by comparing pulse oximetry recordings at both admission and at 48 hours. Poor response to treatment was said to occur if the oxygen saturation reading at 48 hours was the same or less than the recording at admission or the death of a patient.

A total of 10 (8%) of the study respondents lost their lives within 48 hours of admission.

Among the 117 respondents who were alive at 48 hours, 69 (58.9%) experienced a poor response to treatment while 48 (41.1%) had a good response to treatment.

79 (62.2%) of the total respondents experienced a poor response to treatment while 48 (38%) respondents had a good response to treatment.

Table 5.8: Outcomes of study subjects at 48 hours – survival and oxygenation response to treatment

Characteristic		**Number of children**	**Percentage (%)**
Vital status	Dead	10	7.8
	Alive	117	92.8
Oxygenation response	SPO2 decline or remained the same as admission	69	54.3

	Improved	48	37.8
Overall outcome at 48 hours	Poor response to treatment	79	62.2
	Good response to treatment	48	37.8

92 participants were exposed to high polluting household fuels amongst them, 63 (68%) had a poor response to treatment and 29 (32%) had a good response to treatment at 48 hours.
35 participants were exposed to clean household fuels amongst them 16 (46%) had a poor response to treatment and 19 (54%) had a good response to treatment at 48 hours (Figure 5.9).

Table 5.9: 48-hour Pneumonia treatment outcome Analysis

		Primary treatment outcome		**total**	**RR[95% CI]**	**P-value**
		Poor response	**Good response**			
IAP Exposure	Polluting fuels	68%	32%	100%	1.49[1.0,2.2]	0.04
	Clean fuels	46%	54%	100%	Ref	
Total				100%		

$\boldsymbol{f}$: Proportion of poor response to treatment in the group exposed to polluting household fuels=63/92 = 0.68

$\boldsymbol{g}$: Proportion of poor response to treatment in the group exposed to polluting household fuels= = 16/35 = 0.46

Risk Ratio calculation = $\frac{f}{g} = \frac{0.685}{0.457}$ = 1.498

$\boldsymbol{RR}[\mathbf{95}\%\boldsymbol{CI}] = 1.49[1.02,2.21]$ **(p-value = 0.041)**

Children who were exposed to indoor air pollution were 1.49 times more likely to have an unfavorable 48 hour response to pneumonia treatment in comparison to those not exposed to

indoor air pollution from household fuels (OR 1.49, 95% CI 1.02, 2.21, P=0.041) as shown in table 5.9.

5.9 Five-day outcome of children admitted with pneumonia

Most of the study respondents at 76 (60%) had recovered and 34 (26%) were still admitted in hospital at day five of treatment while 17 (13.3%) died within five days of admission. Of those who died within five days of admission, 13 (76%) were exposed to high levels of indoor air pollutants from household fuels while 4 (24%) were exposed to low levels of indoor air pollutants from household fuels. There was also no significant association between the death/ recovery and exposure to household fuels with P values above .05.

The average number of days of hospital stay for those discharged was 3.9 days. The average number of days of hospital stay for those exposed to high levels of indoor air pollutants was 4.12 days while the average number of days of hospital stay for those not exposed to high levels of indoor air pollutant was 3.7 days. Of those exposed to high levels of IAPs, 52% had been discharged at five days while those not exposed to high levels of IAPs, 87% were discharged within 5 days of admission.

There was a significant association between time to discharge from hospital and exposure to household fuels with a P-value < .00. T-test (95% CI) = 6.07 (6.04 – 11.95). Thus, those exposed to high polluting household fuels were likely to be discharged later compared to those who were exposed to clean household fuels.

Table 5.10: Five-day outcome of children admitted with pneumonia

Characteristic		**Number of children**	**Percentage**
Vital status	Dead	17	13.3

	Alive	110	86.7
Discharged before day 5	Still on treatment in the ward	34	26.7
	Discharged	76	59.8
Overall outcome at day 5	Poor response to treatment	51	40.1
	Good response to treatment	76	59.8

51 (35%) of the total respondents experienced a poor response to pneumonia treatment while 76 (59%) respondents responded well at day 5 of treatment shown in table 5.10.

Of those exposed to indoor air pollutants from household fuels, 43 (46%) experienced a poor response to treatment while 8 (22%) of participants exposed to clean household fuels experienced a poor treatment response based on their clinical status at 5-days.

The odds ratio of a poor response to treatment for those exposed to high indoor pollutants compared to those who were exposed to low levels of indoor air pollutants from household fuels was 2.04 with a 95% confidence interval of 1.07 to 3.90 and a P-value of ,<.03. This indicates that there was a significant association between exposure to indoor household fuels and response to pneumonia treatment at five days (Table 5.11).

Table 5.11: Five-day pneumonia treatment response analysis of the study respondents

		Primary treatment outcome (n=127)		**Total**	**Odd ratio**	**P-value**
		Poor response	Good response			
Household fuel Exposure	polluting	47.7%	53%	100 %	2.045 [1.07 – 3.90]	.03
	clean	22%	78%	100 %		
Total				100 %		

Proportion of poor response in the group exposed to indoor fuel pollutants= 0.47

Proportion of poor response in the exposed to clean household fuels group = 0.0.23

$\boldsymbol{OR[95\%CI]}$ $= 2.04[1.07 - 3.90]$, **P-value 0.03**

5.10 Survival Analysis

We documented the time from admission to death, or time from admission to poor outcome at 5 days for each child who was still sick on treatment at day 5 (poor outcome group). Among children who survived and were discharged before the 5^{th} day time was censored at date of discharge (good outcome group). The survival curve indicates that been exposed to low levels of indoor air pollutants from household fuels was associated with a times 3.3 better survival probability when compared to exposure to high polluting indoor fuels. Exposure to low levels of indoor air pollution increases the survival rate by 16%, P<0.46.

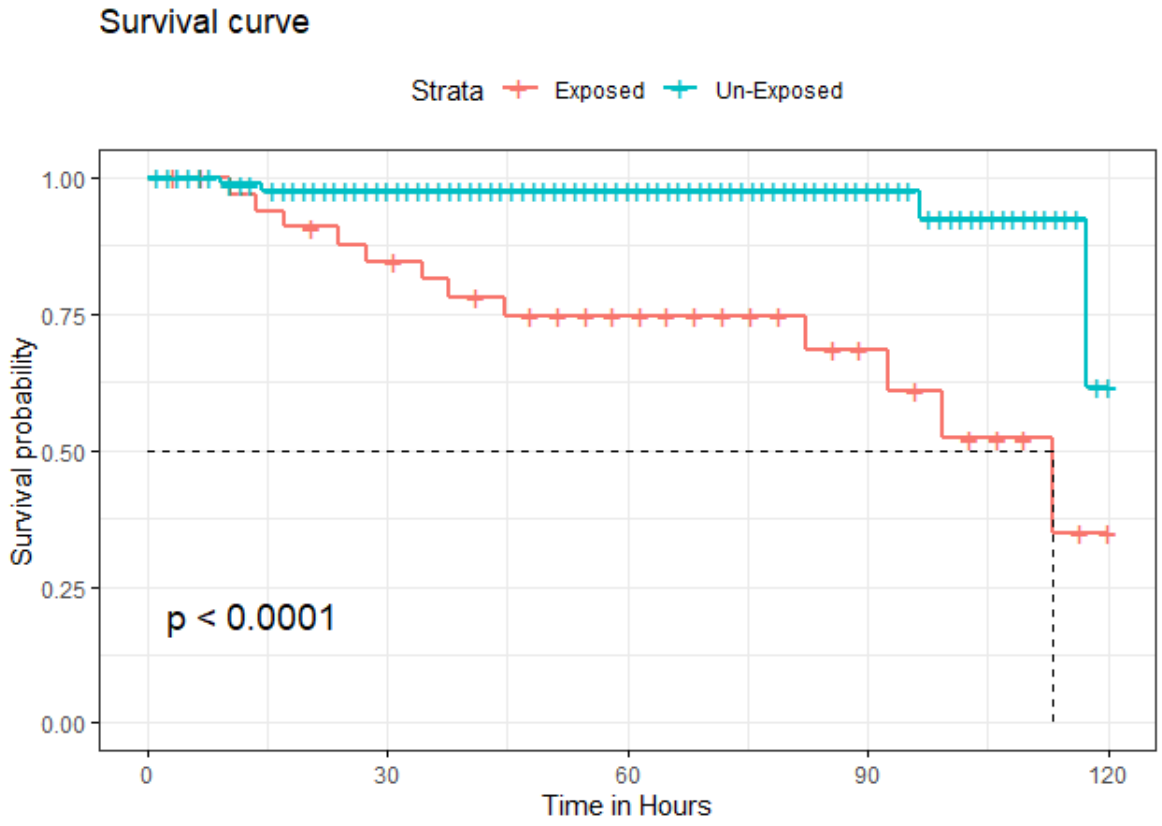

Figure 5.2: A Survival Curve showing association between indoor air pollution exposure and 5-day pneumonia treatment outcome among children at KNH.

CHAPTER 6.0: DISCUSSION

This study aimed at determining the prevalence of exposure to indoor air pollution from household fuels and its effect on treatment outcomes amongst children aged between 2 and 59 months admitted with pneumonia at the Kenyatta National Hospital (KNH) in Nairobi, Kenya.

The study showed an almost equal distribution of females and males at 55.1% and 44.9% respectively amongst the participants. The majority of the respondents were aged less than one year at 83% indicating that infants were more vulnerable to severe pneumonia a finding similar to a study carried out by Mugane *et al* (43) in KNH 2010 which also determined that infants were the most at risk to acquire severe pneumonia.

The effect of exposure to household fuels on treatment response tended to be more pronounced among children less than one year of age. Although this did not reach statistical significance, this finding is consistent with prior studies suggesting that the impact of exposure to household fuels differs by age in children. Gurley *et al*. (44) found that high levels of indoor particulate matter increased the incidence of ALRI in children aged 0–11 months, but not in older children. Participants did not differ by age, gender, their parents/guardians ages, marital status, or parents/guardians occupation.

The majority of study respondents at 72.5% were exposed to high levels of indoor air pollutants while 27.5% were exposed to low levels of indoor air pollutants from household fuels. The study findings are similar to the Kenya National Bureau of Statistics,2015 (KNBS) findings which determined that in the urban areas of Kenya, exposure to high polluting household fuels was 70% with low level polluting fuels been used by 25% of the urban population (7).

However, the study indicates that urban areas use cleaner fuels when compared to rural areas where 55% of the population uses wood, 14% charcoal and 14% kerosene as their sources of

household fuels. LPG is used by only 13% of the rural population. This can be attributed to higher incomes amongst urban dwellers compared to rural dwellers (7).

Amongst the study participants 'exposed to high levels of indoor air pollutants from household fuels, 22 % used kerosene as their preferred source of household fuel. This was followed by use of charcoal at 16.5% and wood only at 5.5%. 28.3% mixed/stacked the fuels used in their households. This is because kerosene is the cheapest cooking fuel in urban Kenya. Charcoal is the most costly cooking fuel but it can be bought in small amounts thus increasing its prevalence. Stacking was also found to be prevalent amongst the respondents at 28.3% which was in keeping with the Kenya National Bureau of Statistics integrated household budget survey which also determined that stacking/ simultaneous use of fuels is widely practiced in Kenya (12) which depends on their budgets, needs and preferences (13).
27.5% the study population used a clean source of household fuel with all of them using LPG. No one in the study population used electricity as a household fuel source. Similarly, a report by KNBS of 2017 found that clean modern cooking fuels are available in Kenya.
However, they are yet unaffordable, not easily accessible with limited consumer awareness to significantly lower the use of traditional fuels.
In urban Kenya, consumers are aware of LPG and there has been an increased investment towards its provision. However, it is unlikely to become the main fuel source due to its limited availability and exorbitant cost. It is also perceived as unsafe as a result of poor safety practices of illegal LPG traders which is estimated at 30-50% of the market (12).
The report also showed that electricity for cooking is not viable today in Kenya and has minimal penetration ar <2% in urban Kenya due to the high costs of efficient electric cookstoves and service charges.
The study determined that at admission, 91% of the study respondents had oxygen saturations below 95% with a majority at 58% having oxygen saturations of between 90-94%.The study determined that 32% of the respondents were admitted with oxygen saturation levels below 90% which is lower than that of a study conducted at KNH by Mugane *et al* (43) which determined prevalence of hypoxemia below 90% at 50.7% and a similar study finding in Papua Guinea showed a prevalence of 54.2% (25).

58% of the total study respondents had oxygen saturations of between 90-94% at admission which was still below the expected normal values at the altitude of Nairobi at 1670 metres with expected oxygen saturation levels of approximately 95.7% (SD=2.7%) as determined by Duke et al in Papua New Guinea (25).

Treatment regimens instituted at admission between the two groups were similar. All the children admitted had crystalline penicillin and gentamicin prescribed at admission.

Amongst the exposed to high polluting household fuels, after 48 hours of treatment, 68% of the respondents were determined to have a poor response to treatment based on an their oxygen saturation level of below 95%. 29.1% had an oxygen saturation of below 90%, 59.5% between 90-94% while 10.7% had saturations above 95%.Amongst those exposed to low levels of IAP from household fuels, after 48 hours of treatment, 45% of the respondents had a poor response to treatment based on their oxygen saturation levels of below 95%. 6% had an oxygen saturation of below 90%, 51.5% between 90-94% while 27.2% had saturations above 95%.

Been exposed to high levels of indoor air pollution was significantly associated with a poor response to pneumonia treatment $RR[95\%CI] = 1.49[1.02,2.21]$ (p-value = 0.0406) in comparison to exposure to low levels of indoor air pollutants. This findings are consistent with T Kelly *et al*. in 2015 findings at a tertiary hospital in Gaborone the capital city of Botswana where they established that household use of high polluting indoor cooking fuels was associated with an enhanced risk of treatment failure or a poor response to treatment by 35% in children aged 1 to 23 months after 48 hours of treatment (RR 1.44, 95% CI 1.09–1.92, $P = 0.01$) (8).

The study poor response to treatment at 48 hours is higher when also compared to the study conducted in Botswana by Kelly *et al* that determined that homesteads whose cooking fuel was wood as had a nearly 50% increase in the risk of treatment failure at 48 hours among children aged two years and below admitted with pneumonia. However, in this study by Kelly, the primary outcome, treatment failure, was assessed at 48 hours. They defined treatment failure the as emergence of new WHO danger signs, having oxygen saturation at less than 80% for a patient on room air, the need for continuous positive airway pressure

(CPAP) or mechanical ventilation, a continued lower chest wall indrawing, or death which could explain their low failure outcome based on the oxygen (8).

Similarly, Webb *et al* in 2008 determined that poor response at 48 hours was 20%, 95% CI 17-23% when basing their oxygen saturation at less than 90% (26).

A higher poor response rate occurred in children aged one year and below in this study which is similar to a study by McNally *et al*. in 2007, South Africa, who determined that a poor response to treatment at 48 h was predicted by age of less than one year (adjusted odds ratio 6·38, 95% CI 2·72–14·91, p<0·0001) (28).

The mortality rate at 48 hours was 7.8% and it increased with decreasing age and lower oxygen saturation levels. Our study determined that 58% of all the deaths occurred in the first 48 hours of admission which is slightly lower than that of the study undertaken at KNH by Maina et al which showed a 48-hour mortality rate of 63.6%.Mortality was more in the group exposed to high indoor air pollution at 8.6% compared to exposure to low levels of indoor air pollutants at 5.7% (45). The findings are similar to that of the study conducted Mugane et al at KNH which showed mortality increased by 3.3 for children admitted with oxygen saturation of less than 90% (43). A study by Onyango et al also determined that children with low oxygen saturations were 4.3 times more likely to die of pneumonia compared to those who normal oxygen saturations (3). The 8.6% death rate amongst the respondents exposed to high polluting fuels is slightly higher when compared to the Kelly et al study determined the death rate at 48 hours was 6% children for children exposed to high polluting cooking fuels (8). However from the study, there was no significant association between the type household fuel exposure and mortality.

At five days, 35% of the total respondents experienced a poor response to pneumonia treatment while 59% respondents responded well to treatment based on their oxygen saturation levels. Of the exposed, 46% participants experienced a poor response to treatment while 22% of participants of the unexposed experienced a poor treatment response which was defined as oxygen saturation same as baseline or decreased as measured by an oximeter or death.

The odds ratio of a poor response to treatment for those exposed to high indoor pollutants compared to those who were exposed to low levels of indoor air pollutants from household

fuels was 2.04 with a 95% confidence interval of 1.07 to 3.90 and a P-value of ,<.03. This indicates that there was a significant association between exposure to indoor household fuels and response to pneumonia treatment at five days.

For those discharged, 3.9 days was the average number of hospital stay days. For those exposed to high levels of indoor air pollutants was 4.12 days was their mean duration of hospital stay while the average duration of hospital stay for those not exposed to high levels of indoor air pollutant was 3.7 days. There were no disharges within 48 hours of admission. Similarly, Agweyu *et al* in 2015 determined that the average duration of hospital stay during severe pneumonia was 4 days (30) but a study by Maina *et al* at KNH which determined the average duration of hospital stay at for children admitted with severe pneumonia was 5.2 days (44). There was no significant association between time to discharge and exposure to polluting household fuels with P values >.05.

13% of the respondents died within five days of admission a figure which is comparable to study undertaken by Maina *et al* at KNH which determined that 13.1% of pneumonia cases die within a week of admission (43). The survival curve develop in this study indicated that been exposed to low levels of indoor air pollutants from household fuels was associated with a better survival probability when compared to exposure to high polluting indoor fuels.

6.1 Study Strengths and Limitations

6.1.1 Strengths

The cohort prospective study design of the study offered clarity of temporal sequence between exposure to indoor air pollution from household fuels, pneumonia and response to pneumonia treatment. This design also enabled an accurate determination of in-hospital outcome of pneumonia treatment.

6.1.2 Limitations

Indoor air pollution exposure was determined indirectly through a self-report thus the researcher will not be able to directly quantify the extent of the exposure at the household level. This is due to logistical and funding challenges.

Pneumonia diagnosis and treatment was limited to the clinical diagnosis using the WHO criteria for case management of pneumonia but the radiological and microbiologic evidence were not available or feasible including the covid-19 for all the enrolled children.

Findings of the study are limited to users of a tertiary public hospital and may not represent outcomes from those using lower level or private health facilities.

Normal oxygen saturation decreases with altitude which could have resulted in an over-estimation of poor response to treatment in the two cohorts. We used pulse oximetry/oxygen sat to determine treatment response whereas in clinical practice, clinical signs are more routinely used thus reducing clinical utility.

6.2 Conclusion

1. Exposure to high polluting household indoor fuels was high among children admitted with severe pneumonia.
2. Exposure to high polluting household indoor was associated with poor response to pneumonia treatment both at 48 hours and five days of treatment.
3. Exposure to indoor solid fuels was associated with longer duration of hospital stay amongst children admitted with severe pneumonia at KNH.

6.3 Recommendations

Assessment of children admitted with severe pneumonia for exposure to polluting household indoor fuels should be done, and they may require closer monitoring for deterioration within their first days of treatment.

Use of clean household fuels should be promoted and facilitated both at a household and at a national level.

Further research is required to better understand the pathophysiologic contributors to poor treatment response among children exposed to high indoor air polluting fuels who develop severe pneumonia.

7.0 REFERENCES

1. O'Brien KL, Baggett HC, Brooks WA, Feikin DR, Hammitt LL, Higdon MM, Howie SR, Knoll MD, Kotloff KL, Levine OS, Madhi SA. Causes of severe pneumonia requiring hospital admission in children without HIV infection from Africa and Asia: the PERCH multi-country case-control study. The Lancet. 2019 Aug 31;394(10200):757-79

2. World Health Organization. Ending preventable child deaths from pneumonia and diarrhoea by 2025: the integrated Global Action Plan for Pneumonia and Diarrhoea (GAPPD) 2013.

3. Onyango D, Kikuvi G, Amukoye E, Omolo J. Risk factors of severe pneumonia among children aged 2-59 months in western Kenya: a case control study. Pan African Medical Journal. 2012;13(1).

4. Mathers C, Stevens G, Mascarenhas M. Global health risks: mortality and burden of disease attributable to selected major risks. World Health Organization; 2009: 9-25.

5. Roser M, Ritchie H. (2016) Burden of disease. Published online at Our World In Data..org. Retrieved from https://ourworldindata.org/burden-of-disease' {Online Resource} 2016 Jan 25

6. Rehfuess EA, Bruce NG, Smith KR. "Solid fuel use: health effect." In encyclopedia of Enviromental Health, vol 5 ed.J.O. Nriagu,156-159. Burlinghton, MA:Elsevier, 2011

7. Kenya National Bureau of Statistics (KNBS) and ICF Macro 2015. Kenya Demographic Health Surveyv (KDHS), 2014. Calverton, Maryland: KNBS and ICF Macro.

8. Kelly MS, Wirth KE, Madrigano J, Feemster KA, Cunningham CK, Arscott-Mills T, Boiditswe S, Shah SS, Finalle R, Steenhoff AP. The effect of exposure to wood smoke on outcomes of childhood pneumonia in Botswana. The International Journal of Tuberculosis and Lung Disease. 2015 Mar 1;19(3):349-55.

9. Dherani M, Pope D, Mascarenhas M, Smith KR, Weber M, Bruce N. Indoor air pollution from unprocessed solid fuel use and pneumonia risk in children aged under five years: a systematic review and meta-analysis. Bulletin of the World Health Organization. 2008;86:390-8C.

10. International Climate Initiative (IKI) : Scaling up clean cooking in urban Kenya with LPG & Bio-ethanol *A market and policy analysis*. Dalberg. June 2018.

11. 17. Bruce N, Pope D, Rehfuess E, Balakrishnan K, Adair-Rohani H, Dora C. WHO indoor air quality guidelines on household fuel combustion: Strategy implications of new

evidence on interventions and exposure–risk functions. Atmospheric Environment. 2015 Apr 1;106:451-7.

12. Kenya National Bureau of Statistics. (2018). *2015/16 Kenya Integrated Household Budget Survey (KIHBS): Labor Force Basic Report*. Nairobi: Kenya National Bureau of Statistics.

13. Heltberg R. Household fuel and energy use in developing countries: A multi-country study. The World Bank. 2003 May 14:1-87.

14. Heltberg R. Factors determining household fuel choice in Guatemala. Environment and development economics. 2005 Jun;10(3):337-61

15. 18. Tasić M, Rajšić S, Novaković V, Mijić Z. Atmospheric aerosols and their influence on air quality in urban areas. Facta universitatis-series: Physics, Chemistry and Technology. 2006;4(1):83-91.

16. Thurston GD, Ito K, Mar T, Christensen WF, Eatough DJ, Henry RC, Kim E, Laden F, Lall R, Larson TV, Liu H. Workgroup report: workshop on source apportionment of particulate matter health effects—intercomparison of results and implications. Environmental health perspectives. 2005 Dec;113(12):1768-74.

17. World Health Organization. WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide: global update 2005: summary of risk assessment. Geneva: World Health Organization; 2006:31-54

18. O'Sullivan K, Barnes DF. Energy policies and multitopic household surveys: guidelines for questionnaire design in living standards measurement studies. The World Bank; 2006 Nov 16. Working Paper No. 90:27-40.

19. Nandasena S, Wickremasinghe AR, Sathiakumar N. Indoor air pollution and respiratory health of children in the developing world. World journal of clinical pediatrics. 2013 May 8;2(2):6-15.

20. Hussey SJ, Purves J, Allcock N, Fernandes VE, Monks PS, Ketley JM, Andrew PW, Morrissey JA. Air pollution alters Staphylococcus aureus and Streptococcus pneumoniae biofilms, antibiotic tolerance and colonisation. Environmental microbiology. 2017 May;19(5):1868-80.

21. Smith KR, Samet JM, Romieu I, Bruce N. Indoor air pollution in developing countries and acute lower respiratory infections in children. Thorax. 2000 Jun 1;55(6):518-32.

22. Bruce NG, Dherani MK, Das JK, Balakrishnan K, Adair-Rohani H, Bhutta ZA, Pope D. Control of household air pollution for child survival: estimates for intervention impacts *BMC Public Health* 13, S8 (2013). https://doi.org/10.1186/1471-2458-13-S3-S8.

23. Ad Hoc Statement Committee, American Thoracic Society. Mechanisms and limits of induced postnatal lung growth. American Journal of Respiratory and Critical Care Medicine. 2004 Aug 1;170(3):319-43.

24. World Health Organization. Revised WHO classification and treatment of pneumonia in children at health facilities: evidence summaries, 2014.

25. Duke T, Mgone J, Frank D Hypoxaemia in children with severe pneumonia in Papua New Guinea. .Int J Tuberc Lung Dis. 2001 Jun;5(6):511-9.PMID: 1140957

26. Webb C, Ngama M, Ngatia A, Shebbe M, Morpeth S, Mwarumba S, Bett A, Nokes DJ, Seale AC, Kazungu S, Munywoki P. Treatment failure among Kenyan children with severe pneumonia–a cohort study. The Pediatric infectious disease journal. 2012 Sep;31(9):e152

27. Addo-Yobo E, Chisaka N, Hassan M, Hibberd P, Lozano JM, Jeena P, MacLeod WB, Maulen I, Patel A, Qazi S, Thea DM. Oral amoxicillin versus injectable penicillin for severe pneumonia in children aged 3 to 59 months: a randomised multicentre equivalency study. The Lancet. 2004 Sep 25;364(9440):1141-8.

28. McNally LM, Jeena PM, Gajee K, Thula SA, Sturm AW, Cassol S, Tomkins AM, Coovadia HM, Goldblatt D. Effect of age, polymicrobial disease, and maternal HIV status on treatment response and cause of severe pneumonia in South African children: a prospective descriptive study. The Lancet. 2007 Apr 28;369(9571):1440-51

29. Atkinson M, Lakhanpaul M, Smyth A, Vyas H, Weston V, Sithole J, Owen V, Halliday K, Sammons H, Crane J, Guntupalli N. Comparison of oral amoxicillin and intravenous benzyl penicillin for community acquired pneumonia in children (PIVOT trial): a multicentre pragmatic randomised controlled equivalence trial. Thorax. 2007 Dec 1;62(12):1102-6

30. Agweyu A, Kibore M, Digolo L, Kosgei C, Maina V, Mugane S, Muma S, Wachira J, Waiyego M, Maleche-Obimbo E. Prevalence and correlates of treatment failure among Kenyan children hospitalised with severe community-acquired pneumonia: a prospective study of the clinical effectiveness of WHO pneumonia case management guidelines. Tropical Medicine & International Health. 2014 Nov;19(11):1310-20.

31. World Health Organization. WHO Indoor Air Quality Guidelines: Household Fuel Combustion: Population levels of household air pollution and exposures Review 5, 2015.

32. Kurmi OP, Lam KB, Ayres JG. Indoor air pollution and the lung in low-and medium-income countries. European Respiratory Journal 2012 40: 239-254.

33. PrayGod G, Mukerebe C, Magawa R, Jeremiah K, Török ME. Indoor air pollution and delayed measles vaccination increase the risk of severe pneumonia in children: results from a case-control study in Mwanza, Tanzania. PloS one. 2016;11(8).

34. Barnes DF, Krutilla K, Hyde W. The urban household energy transition The Urban Household: Energy Transition Energy, Poverty, and the Environment in the Developing World. World Bank, 2004.

35. Dherani M, Pope D, Mascarenhas M, Smith KR, Weber M, Bruce N. Indoor air pollution from unprocessed solid fuel use and pneumonia risk in children aged under five years: a systematic review and meta-analysis. Bulletin of the World Health Organization. 2008;86:390-8C.

36. Ezzati M, Kammen DM. Quantifying the effects of exposure to indoor air pollution from biomass combustion on acute respiratory infections in developing countries. Environmental health perspectives. 2001 May;109(5):481-8.

37. Karki S, Fitzpatrick AL, Shrestha S. Risk factors for pneumonia in children under 5 years in a teaching hospital in Nepal. Kathmandu University Medical Journal. 2014;12(4):247-52.

38. Smith KR, McCracken JP, Weber MW, Hubbard A, Jenny A, Thompson LM, Balmes J, Diaz A, Arana B, Bruce N. Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial. The Lancet. 2011 Nov 12;378(9804):1717-26

39. Kenya National Bureau of Statistics 2019. Kenya Population and Housing census. Population by County and Sub-County. Nairobi, Kenya National Bureau of Statistics, Volume 1.

40. Kenyatta National Hospital. 2018. Kenyatta National Hospital Strategic plan 2019-2023, Nairobi. Kenya National Hospital.

41. Nieves JE, Jones J. Epi Info™: now an open-source application that continues a long and productive "life" through CDC support and funding. The Pan African Medical Journal. 2009 Apr 30: 2:6

42. Kelsey JL, Whittemore AS, Evans AS, Thompson WD. Methods in observational epidemiology. Monographs in Epidemiology and Biostatistics; 2nd ed. New York: Oxford University Press; 1996. Chapter 4, Cohort Studies: Statistical Analysis 1; p130-66.
1996al Hospital.

43. Mugane S, Musoke R, Obimbo E,Irimu G. Hypoexemia among Children with severe pneumonia or very severe pneumonia at Kenyatta National Hospital, 2010. University of Nairobi. Nairobi, Kenya.

APPENDIX A: CHECKLIST - TO ESTIMATE LEVEL EXPOSURE OF IAP FROM HOUSEHOLD FUELS (WHO 2015)

Type of cooking fuel

Household characteristics

Rooms in the household

Number of windows/major openongs

Main source of lighting for the household

Kitchen characteristics

Type of kitchen- Indoor kitchen with partition, Indoor kitchen without partition, Separate indoor kitchen, outside house, open-air kitchen

Presence of a chimney

Time-activity data Mean duration (hours) spent by household subgroups in the kitchen while cooking; what type of fuel was used over the last 24 hours; Time and duration of cooking activities and number of people being cooked for.Who cooked, Number of people Duration/ being cooked for Morning/ Afternoon/ Evening

Total time that the fire was on (hours)

Cigarettes smoked indoors

Additional information on a child's exposure to IAP from household fuels:

Time child spends near the household fuel

Child is carried on mothers back during cooking

Appendix B:

QUESTIONNAIRE

INTERVIEWER NAME……
QUESTIONNAIRE NO……
DATE AND TIME……

Social demographic data

2. Client's age in months……3. Date of birth *(Day/month/year)*……..

4. Sex 1 =male 2=female ☐

5. Informant *(relation to child)* mother=1 father=2☐ Other *(specify)*……..

6. Mothers/fathers marital status

Married☐ Single ☐ Separated ☐ Divorced ☐

Widowed ☐ Remarried☐

7. Mothers age *(years*☐ Fathers age *(years)*☐

8. Birth weight *(grams)*……8. Gestation *(weeks)*……..

9. Number of siblings……

Others *(specify)*…….

11. Residence……..…..

12. Guardian Occupation

Employ☐ Self employe☐ Unemployed☐

☐

Housewife Others *(specify)*……..…

If married, spouse's occupation

Employed Self employed Unemployed

Housewife O *(specify)*……

13. Guardian Education status

a. None Primary

Secondary Tertiary

If married, spouse's level of education

a. No Primary

Seconda Tertiary

Medical/family/social history

14. Any known chronic illness/ allergies Yes=1, No=2

If Yes, *(specify)*……

15. Referral from another facility yes=1, no=2 if yes,

16. Referring facility……Dispensary Health Centre Level four facility

Level 5 facility self-referral

If referred, was referral from an inpatient setting yes=1, no=2

17. Reason for referral *(specify)*……

18. Prior hospital admission in the last 14 days with the same complaints Yes=1, No=2

19. Immunization status for age *(tick appropriately)* - up to date not up to date

20. Breastfeeding status – breastfeeding *(tick appropriately)*-Yes No

Breatfeeding status *(tick appropriately)* EBF complementary feeds

Not breastfeeding *(tick appropriately)* yes no

Reason *(specify)*……..

21. Symptoms 1=present, 2=absent, duration in days (D.I.D.)……

Cough/c☐ *duration* ☐ Difficulty in breathing☐ *duration* ☐

Hotness of the body ☐ *duration*☐ Vomiting ☐ *duration* ☐

Abnormally slee☐ *duration* ☐ Convulsions ☐ *duration* ☐

22. Physical exam (at admission)

Weight…… (kgs) height/length (cms)……Temp (°C)…….MUAC (cms)…….

Respiratory rate per minute ……Heart rate per minute……Oxygen saturation (%)…….

Signs, at admission Yes=1, No=2

Central Cyanosi☐ grunting ☐ audible wheeze ☐

Severe respiratory distress (e.g head nodding) ☐ Lower chest in-drawing☐

Inability to breast feed or drink ☐ Fast breathing ☐

2 months up to 12 months: 50 breaths per minute or more

12 months up to 5 years: 40 breaths per minute or more.

Stri☐

AVPU Sca☐ 1= A, 2=V, 3=P, 4=U

Severity of Pneumonia ☐

1=severe pneumonia, 2=pneumonia, 3=no pneumonia

Initial antibiotics at admission, correct dose 1=Yes; 2=No; Correct frequency 1=yes; 2=no

First line: Crystalline penicillin….. *dose* ☐ *frequency* ☐

Gentamicin *dose*☐ *frequency* ☐

Other treatment *(specify)*……

***23.* Symptoms *(After 48 hours)* 1=present, 2=absent**

Cough/cold☐ Difficulty in breathing ☐ Fever ☐

Convulsion☐ Vomiting ☐ Abnormally sleepy ☐

24. Physical exam (after 48 hours)

Weight……(kgs) Height/length (cms)…… Temp (°C)…….MUAC (cms)…….

Respiratory breaths per minute…… Heart beat per minute……Oxygen saturation (%)……

Oxygen saturation (%)……same as admission/decline from baseli☐

Oxygen saturation (%)……reduced from above 95%☐

Increased when compared to baselin☐ still above 95☐

25. Signs, after 48 hours

Cyanosi☐ Gruntin☐ Audible wheeze ☐

☐ ☐

☐ ☐

Severe respiratory distress (e.g head nodding), Lower chest indrawing,

Inability to breastfeed or drink Stridor

Fast breathing

2 months up to 12 months: 50 breaths per minute or more

12 months up to 5 years: 40 breaths per minute or more.

AVPU Sca 1= A, 2=V, 3=P, 4=U

Severity of Pneumonia

1=severe pneumonia, 2=pneumonia, 3=no pneumonia

26. Progression of infection to other systems YE Y NO N

System affected……..Effect……

26. Treatment at 48 hours (tick as appropriate)

Same as admissi… new/changed

Oxygen prescribed (*tick as appropriate)* – yes no

Mode of oxygen delivery *(tick as appropriate)…*

nasal prongs nasal catheter face mask with reservoir

Others *(specify)……*

Severity of Pneumonia

1=severe pneumonia, 2=pneumonia, 3=no pneumonia

31. HIV status

Sero-exposed(tick as appropriate) yes no

If yes, prophylaxis *(tick as appropriate)* yes no

If PCR positive or confirmed HIV positive, on ART yes no

Five-day outcome Assessment, state YES or NO

Full recovery ☐ Discharged ☐ If recovered, time to recovery in hours ☐

Persistent clinical illn ☐ oxygen saturation lev ☐ oxygen prescribed ☐

Mode of oxygen delivery if prescribed ☐ Mortality/death ☐

32. FUELS

a. Household fuel used (Last two months) wood ☐ charcoal ☐

kerosene ☐ LPG ☐ electricity ☐

Stacking *(specify)* y ☐ ……. no ☐

Others (specify)…….

b. What type of fuel did you use over the last 24 hours

Woo ☐ charco ☐

kerosene ☐ LPG ☐ electricity ☐

Stacking *(specify)* y ☐ ……. no ☐

Others (specify)…….

c. Cooking stove: traditional charcoal stove ☐ improved ☐

d. Household fuel acquisition -purchased/paid for ☐ not purchas ☐

e. Household source of lighting (tick appropriately) electricity ☐ kerosene ☐

others(specify)

……..

f. Who (mostly) prepares/cooks the food……relationship to the child…….

g. Approximate number of meals prepared in a day ☐

h. Number of rooms in the house *(tick appropriately)* 1 1-3 > 3

i. Type of kitchen

Indoor kitchen with partition ☐ In-door kitchen without partition ☐

Separate indoor kitch ☐ Kitchen outside hous ☐ Open-air kitch ☐

j.Number of windows / openings in kitchen

k. Fireplace/chimney *(tick appropriately)* prese ☐ abse ☐

l. In the last 24 hours

Who cooked…….relationship to the child…….

Number of people cooked f ☐

Duration of cooking (hours)

Morning…Less than 1 hour ☐ 1-2 hours ☐ more than 2 hours ☐

Afternoon….Less than 1 hou ☐ 1-2 hou ☐ more than 2 hours ☐

Evening…Less than 1 hour ☐ 1-2 hours ☐ more than 2 hours ☐

Other meals, specify……duration of cooking…….

Total time that the fire was on (hours)……

1 hour ☐ 1-2 hours ☐ more than 2 hours ☐

m. Child location/placement during food preparation *(tick appropriately*)

Same room ☐ Different location ☐

n. The proximity of children to stoves while fuel is burned *(approximate in metres)*

o. Child carried on mothers back while food is been prepared *tick appropriately*)

Y☐ N☐

q. Number of meals prepared per day while child is on mothers back *(tick appropriately*)

r. ☐ 2 ☐ 3 ☐ >3 ☐

s. Number of household occupants *(tick appropriately*)

2☐ 3☐ 4 ☐ 5 ☐ 6 ☐ ☐

t. Smoking within the house *(tick appropriately*) yes ☐ no ☐

u. Number of windows in the house

(tick appropriately-non☐ 1 ☐ 2☐ 3 ☐ 4 ☐ ☐

v. Number of windows in the kitchen *(tick appropriately*)

No☐ 1☐ 2☐ >2 ☐

Appendix C: Parents/Guardian Consent Form (English)

Patient's Study Number: _____

Date: _____

Study Title: EXPOSURE TO INDOOR AIR POLLUTION FROM HOUSEHOLD FUELS AND EFFECT ON TREATMENT OUTCOMES AMONGST CHILDREN AGED BETWEEN 2 AND 59 MONTHS ADMITTED WITH PNEUMONIA AT THE KENYATTA NATIONAL HOSPITAL IN NAIROBI, KENYA

Investigator: **Dr. Francis Ng'ang'a (MBChB)**
Paediatric Resident,
University of Nairobi
Tel Number: - 0720- 829362

Supervisors:

Dr. Lawrence Owino (MB ChB, M.Med, FRheum)
Senior Lecturer in Paediatrics and Child Health,
Department of Paediatrics, University of Nairobi.

Prof. Elizabeth Obimbo (MB ChB, M. Med, MPH, FPulm)
Professor Department of Paediatrics and Child Health,
University of Nairobi.

Investigator's Statement:

We are kindly requesting for your child to kindly participate in this research study. The purpose of this consent form is to provide you with the information you will need to help you decide whether to participate in the study or not. This process is called 'Informed Consent'.

Please read this consent information carefully and ask any questions or seek clarification on any matter concerning the study.

Introduction:

Indoor air pollution is a major contributor to acute respiratory infections. Understanding pneumonia response to treatment following exposure to household fuels will help in the development of interventions and policies that can help mitigate against such exposure.

Benefits:

The results of the study will be shared with the ministry of health and other relevant authorities for interventions that may be instituted to assist children in future. The results will as well help other children facing similar challenges. The results will be shared to all the guardians who participated in the study.

Risks:

There will be no risks to you or your child during the study. There will be no invasive procedures carried out in the study. Refusal to participate will in no way jeopardize the treatment of your child in any way.

Voluntariness:

The study will be fully voluntary. There will be no financial rewards to you for participating in the study. One is free to participate or withdraw from the study at any point. Refusal to participate will not compromise your child's care in any way.

Confidentiality:

The information obtained about you, your child and your family will be kept in strict confidence. No specific information regarding you, your child or your family will be released to any person without your written permission. We will, however, discuss general overall findings regarding all children assessed but nothing specific will be discussed regarding you or your child. We will also, not reveal the identity of you or your child in these discussions.

Problems or Questions:

For any question about the study or about the use of the results you can contact the principal investigator, Dr. Francis Ng'ang'a by calling 0720-829 362.

If you have any questions on your rights as a research participant you can contact the Kenyatta National Hospital Ethics and Research Committee (KNH- ESRC) by calling 2726300 Ext. 44355.

Consent Form: Participant's Statement:

I _____ having received adequate information regarding the study research, risks, benefits hereby AGREE / DISAGREE (Cross out as appropriate) to participate in the study with my child. I understand that our participation is fully voluntary and that I am free to withdraw at any time. I have been given adequate opportunity to ask questions and seek clarification on the study and these have been addressed satisfactorily.

Parents Signature: _____ Date _____

I _____ declare that I have adequately explained to the above participant, the study procedure, risks, and benefits and given him /her time to ask questions and seek clarification regarding the study. I have answered all the questions raised to the best of my ability.

Interviewers Signature _____ Date _____

Appendix D: Fomu ya Kupata Idhini la Wazazi/Walezi

Nambari la UsajiliyaUtafiti: _____

Tarehe ; _____

Utafiti: Athari ya Uchafuzi wa Hewa Manyumbani kwa matibabu ya Nimonia kwa Watoto kati ya Umri wa Miezi 2-59 Waliolazwa katika Hospitali Kuu ya Kenyatta, Kenya

NAIROBI

Mpelelezi: **Dr. Francis Ng'ang'a (MB ChB)**
Mwanafunzi katika Chuo Kikuu cha Nairobi,
Tel Number:- 0720- 829 362

Wasimamizi:

Dr. Lawrence Owino (MB ChB, M.Med,FRheum.)
Mwadhiri Mwandamizi , Idara ya Uzima watoto
Chuo Kikuu cha Nairobi

P**rof. Elizabeth Obimbo (MB ChB, M. Med, MPH, FPulm.)**
Profesa, Idara ya Uzima Watoto
Chuo Kikuu cha Nairobi.

Semi la Wachunguzi

Tafadhali tunaomba kwa mtoto wako kushiriki katika utafiti huu. Madhumuni ya fomu hii idhini Ni kukupa taarifa unahitaji kukusaidia kuamua kushiriki katika utafiti au la. Mchakato huu inaitwa 'Idhini. Sababu ya idhini hii ni kukupa mawaidha ya kukusaidia kuamua kama utajihusisha na utafiti huu. Tafadhali soma maelezo kwa makini, na ukiwa na swali sikia huru kuuliza.

Kianzishi

Kuelewa vile machafuzi ya hewa huadhiri matibabu ya nimonia kwa watoto kutachangia kubuni sera zinazoweza kuzuia huu ugonjwa siku za baabaye.

Faida:

Majibu ya utafiti yatatumiwa na wahudumu waafya kusaidia vijana hawa.

Hatari:

Hamtakuwa na hatari lolote litakalo mkabili yeyote katika utafiti.

Kujitolea:

Hamtakuwa na faida lolote la kifedha. Hakuna atakaye lazimishwa kushiriki katika utafiti. Kukataa kushiriki ni sawa na halitaleta kutolewa huduma ya mtoto wako kwa njia yoyote .

Usiri:

Maswali yote utakayo jibu kuhusu wewe, mtoto au familia yatakuwa kwa siri.

Maswali au shida?

Ukiwa na swali au tatizo lolote kuhusu utafiti huu, kuwa huru kuwasiliana na msimamizi wa utafiti huu **Daktari Francis Ng'ang'a** kwa kupiga simu nambari **0720-829 362.**

Ukiwa na swali kuhusu kujiunga na utafiti huu wasilianana Kenyatta **National Hospital Ethics and Research Committee (KNH- ESRC)** kwa kupiga simu nambari **2726300 Ext. 44355.**

Fomu Idhini la Mlezi

Mimi_____nimepewa mawaidha ya kutosha kuhusu utafiti huu na nina KUBALI / KATAA (Futa kama inavyofaa) kujihusisha na utafiti huu.

Idhini ya mlezi: _____ Tarehe_____

Mimi_____natangaza ya kwamba nimemshauri mshiriki wa utafiti yote kuhusiana na utafiti huu na kujibu maswali yote aliyouliza.

Sahihi la mchunguzi _____Tarehe_____