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# Relationship between Household Air Pollution of Particulate Matter and Acute Respiratory Infections Among Young Children in Central Kenya

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***Abstract***

***Background***

*Household Air Pollution is perceived to increase the risk of developing acute respiratory infection (ARI) specifically among young children. A cohort study involving 430 children (228 exposed to Household Air Pollution and 202 non-exposed to indoor air pollution) was conducted to explore the association of Household Air Pollution upon respiratory infections of children aged less than five years, in Nyeri, central Kenya.*

***Methods***

*Quantitative data were collected monthly for twelve months from participants using a questionnaire. Meteorological data on temperature, relative humidity, and rainfall were also collected at the same interval and period. A randomized sample of 60 households was selected in which 24hour monitoring for particulate matter of less than 10 microns in diameter was carried out.*

***Results***

*The overall, mean particulate matter of less than 10 microns in diameter was 68.6 + 90 .1 µg/$m^3$ in the exposed households, the mean PM10 level was 100.8 + 95.3 µg/$m^3$ compared to mean of particulate matter of less than 10 microns in diameter level in the non-exposed household of 4.1 + 4.0 µg/$m^3$ (students t-test 4.88, $p<0.01$). The particulate matter of less than 10 microns in diameter from exposed households was 2 times (100.8g/m) higher than the 50g/m tolerable standards set by British Health Panel and adopted by European Commission, whereas those from non-exposed households were 12.2 times lower (4.1 µg/$m^3$). Children exposed to particulate matter of > 50 µg/$m^3$ were (10) at times at risk of developing more than five (5) episodes of acute respiratory infections within a year (RR= 10, 98% CI 3.3 to 30). There was also a positive linear correlation between a particulate matter of less than 10 microns in diameter and the development of acute respiratory infection ($R = 0.358$, $p<0.01$). The rates ratio for acute respiratory infections (ARI) was 1.57(95% CI, 1.19-2.07) and an attributable fraction of 68.6%. The risk of getting ARI during the cold season was 3.6 (RR=3.6, 95% CI 3.1-4.2) compare to the hot season. Exposure to HAP, stunting condition of the children were variables identified by the multivariate model as independently associated with ARI.*

***Conclusion***

*In conclusion, this study found that the incidence rate for acute respiratory infections was high in the study population and there was a statistically significant association between Household Air Pollution and acute respiratory infections (RR=1.57, 95% CI,1.19-2.07). the mean PM10 levels were higher than the standard set by the British Health Panel and European Commission. Besides exposure to indoor air pollution, stunting and time spent in the cooking place were risk factors for acute respiratory infections.*

***Recommendations***

*It is recommended that the government develop an air pollution policy aimed at reducing Household Air Pollution and also promote the adoption of superior fuel for cooking to reduce indoor air pollution. there is a need to conduct further studies on HAP monitoring and conduct comparative cost-effective studies between curative and preventive interventions for acute respiratory infections. Future similar studies should incorporate spirometry assessments. Finally, interventions on acute respiratory infections should not only involve reducing Household Air Pollution which are potential risk factors for acute respiratory infections.*

***Key Words:*** *Household Air Pollution, Acute Respiratory Infections, Cohort Study, Particulate Matter, Children Under Five Years, Metrological Parameters*

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## I. Background

Air pollution particularly pollution of indoor air, arising from biomass fuel which is used for cooking and heating has for a long time been recognized to have significant deleterious effects on human health. There is consistent evidence that Household Air Pollution ((IAP) increases the risk of childhood acute respiratory infections, the most important cause of death of children under five years in the least developed countries (McCracken *and Smith,* 1998). Although the risks remain poorly quantified, Household Air Pollution may be responsible for nearly two million excess deaths in the least developed countries, and around 4% of the global burden of disease (Smith et al., 2000). In developing countries and especially in rural Kenya, most of the cooking is done by women in poorly ventilated houses that hold heavy deposits of soot on the inside walls and roofs of the kitchens (Smith *et al*., 1994). In addition, sooty conditions are common in living houses where occupants spend several sleeping hours because of economic limits and culture.

Concerning morbidity from air pollution in developing countries, the acute respiratory tract infections due to indoor air pollution, as defined by World Health Organization, 1972 include any combinations of the following symptoms; cough with or without fever, blocked or runny nose, sore throat, and/or ear discharge. Other forms of acute respiratory infections including infection of the lungs, especially pneumonia have been reported. These severe infections are mostly caused by bacteria, although they may occasionally be caused by viruses (World Health Organization, 1972).

Air pollution is a result of discharge into the atmosphere of foreign gases and particulates, in excessive amounts to normal constituents (Union of International Associations, 2021). This air pollution falls into two main categories, namely particles and gases. The particles include ash, the un-burnt leftover of ordinary coal, soot, and incomplete burnt carbon lead, the unburnable additive in gasoline, and dust which may consist of wastes and by-products of technical processing, or airborne dust particles due to windstorms. Other toxic metals, asbestos, pesticides, microorganisms may be present (World Health Organization, 1972).

Air pollution as a health issue is old and of global concern. A review of several studies published both by several others and reports (*United* States National air pollution control administration 1969) provide strong evidence which suggests a constant association of air pollution and health impairment of varying degrees. Such associations are found between: -

i) Acute pollution exposure and morbidity and mortality
ii) Chronic low-level exposure and morbidity and mortality
iii) Exposure and impairment of functions and performance of vital organs
iv) Exposure and symptoms of sensory irritation and
v) Exposure and other effects on well-being.

Smoke and sulphur dioxide have been reported as the main pollutants measured in epidemiological studies on the assumption that these two substances set a pattern for other important pollutants.

In Kenya, the Household Air Pollution situation cannot be accurately described at the moment because accurate and reliable data have not yet been collected to determine the severity and magnitude of pollution to the health status of the people. However, observations made indicate that taking into consideration of pollution trends, known potential sources of pollutants, type of housing, and prevalence of diseases of the respiratory system, there could be a relationship between Household Air Pollution and the development of acute respiratory infections.

Children are particularly at higher risk of developing acute respiratory infections. They are affected more severely than adults because their airways are relatively narrower and more easily obstructed, and their oxygen demand relative to body weight is higher, resulting in relatively larger inhaled volumes. Asthmatic people are another group at special risk *(*World

Health Organization, 1972*).* A few studies that employed appropriate design to investigate the relationship between Household Air Pollution and the development of acute respiratory infections, such as intervention study designs have had smaller sample sizes to allow detection of any significant difference between two groups of individuals and thus making it difficult to extrapolate the results into a larger population. Therefore, this study, which used a follow-up design, attempted to bridge the gaps that many researchers had failed to accomplish and generate data and evidence that Household Air Pollution contributes to increased acute respiratory infections as well as identifying and quantifying their role in the incidence of acute respiratory infections.

## II. Objectives

The purpose of the study was to determine the contributions of Household Air Pollution on the development of acute respiratory tract infections in children aged under years.

Specifically, the study objective was to: -
1. Quantify the particulate matter levels in exposed and unexposed households
2. Compare the incidence of respiratory tract infections in households perceived to be exposed and those unexposed to Household Air Pollution and
3. Establish the existence of other factors that could increase the risk of acute respiratory infections other than Household Air Pollution

## III. Methodology

**Study Design and subjects**

This was a prospective cohort study. Children aged less than five years and living in the study area were recruited based on exposure status and followed up for twelve months and data was collected monthly. The subjects were enrolled based on written consent to participate by parents or guardians. Information on Household Air Pollution exposures, illness, and deaths was collected. To control for weather variations, meteorological parameters including temperature, rainfall, and humidity were collected daily from the Wambugu weather station and summarized on monthly basis. Before the commencement of the study, a household census was conducted to identify all houses with children aged less than 4 years (<48 months) and for the selection of exposed and unexposed households.

Children in the sampled households were categorized on exposure or non –exposure status based on the criteria below: -

- Type of stove used
- Type of housing in terms of ventilation
- Types of fuel used
- Time the child spent in the cooking place (kitchen)
- Evidence or lack of soot deposits

**Sampling technique**

The cluster-sampling technique was used to sample the villages where the cohorts of children were drawn. A total of 5 villages were selected (1 from Kiganjo location and 4 from Mukaro location). The sample size from each village was determined through probability proportion to size (PPS) criteria.

A total of sixty households were selected from the 430. 330 households were selected from 4 villages in Mukaro and 100 households from 1 village in Kiganjo. From the identified sixty households, forty were grouped in the exposed category and twenty un-exposed. These are the households where indoor air sampling for particulate matter pollutants on a 24-hourly monitoring period was conducted.

**Sample size determination**

The determination of both sample sizes for study children and houses to monitor particulate matter of less than 10 microns in diameter was based on the hypotheses of the study. For study children, the hypotheses had to do with the comparison of ARI incidence rates in exposed and unexposed, and thus the appropriate statistical formulae used were that dealing with comparison of two rates. On the other hand, for monitoring of indoor air pollution, the hypothesis had to do with the comparison of the mean PM10 in exposed and unexposed households thus appropriate statistical formulae of choice was that of comparing two means.

**The sample size for study children**

The minimum sample size for this study is 358 children which are 179 exposed groups and 179 unexposed groups. The sample size was determined using the below statistical formula for comparison of two rates (sample size of each group) as described by Kirkwood, 1998.

$n \geq (u+v)^2 (\mu_1+ \mu_2)/ (\mu_1- \mu_2)^2$

$n \geq (1.28+1.96)^2 (0.356+0.179)/(0.356-0.179)^2$

$n \geq 1.4976 \times 0.535/0.031329 \geq 179$ for one group thus $n \geq 358$ for the two groups.

n denotes sample size

u = one-sided percentage point of the normal distribution corresponding to 100% - the power. Since the power for this study was taken to be 90%, u then was 100% - 90% = 10% thus u = 1.28.

v = percentage of the normal distribution corresponding to the required (two-sided) significance level. For this study, the significance level was set at 5%, thus v = 1.96.

$\mu_1$ denotes the prevalence rate of ARI in an exposed population. For this study, the MOH Nyeri report for 2005 indicated that the prevalence of ARI in children aged under five years in Kieni East (data from a health center that serves the catchment area) where a majority of the households use biogas as the main fuel for cooking was 35.6%.

$\mu_2$ denotes the prevalence rate of ARI in the unexposed population. For this study, the same MOH Nyeri report for 2005 indicated that the prevalence of ARI in children aged under five years in Kiganjo Police Training College (data from Kiganjo dispensary), where the majority use electricity and gas for cooking was 17.9%

Therefore, the minimum sample size for each group (exposed and unexposed) was 179 thus making the required minimum sample size of 358 children. However, due to anticipated non-response by some participants (drop out) arising from attrition; deaths from disease not related to acute respiratory infections, migration outside the study area, and withdrawals from the study, the sample size was increased by 20% thus making the final sample size to be 430 (228, exposed, and 202 unexposed) in individuals in each household.

The number of households could have been less than the enrolled children in the study but it was decided that in households with more than one child including twins aged under four years, a simple random selection of which child to participate was done.

**2.3.2: Sample size for houses where indoor air monitoring was conducted**

The minimum sample size for houses considered for indoor air monitoring was 26 which were 13 exposed (houses using biogas) and 13 not exposed (houses not using biogas). The sample size was determined using the below statistical formula for comparison of two means (sample size of each group) as described by Kirkwood, 1998.

$n \geq (u+v)^2 (\sigma^2_1+ \sigma^2_2)/ (\mu_1- \mu_2)^2$

$n \geq (1.28+1.96)^2 (1327^2+258^2)/(1814-574)^2$

$n \geq 19184290.5168/1537600 = 12.5$

n denotes sample size

u = one-sided percentage point of the normal distribution corresponding to 100% - the power. Since the power for this study was taken to be 90%, u then was 100% - 90% = 10% thus u = 1.28.

v = percentage of the normal distribution corresponding to the required (two-sided) significance level. For this study, the significance level was set at 5%, thus v = 1.96.

$u_1$ denotes mean PM10 where no intervention is done to the living houses. For this study, Intermediate Technological Development Group (2002), findings of PM10 Kajiado houses which were using biomass during the pre-intervention period had a mean PM10 of 1814 $\mu g/m^3$.

$u_2$ denotes mean PM10 where intervention is done in living houses. For this study, Intermediate Technological Development Group, 2002, findings of PM10 Kajiado houses, which had smokeless stoves, installed, the mean PM10 during the post-intervention period was 574 $\mu g/m^3$.

$\sigma_1$ stands for a standard deviation for $u_1$, which for Intermediate Technological Development Group study in Kajiado was 1327 $\mu g/m^3$.

$\sigma_2$ stands for a standard deviation for $u_2$, which for the Intermediate Technological Development Group study in Kajiado was 258 $\mu g/m^3$.

Therefore, since it was a 2-arm study, then $n \geq 13$ x2 makes $n \geq 26$. However, the actual sample size was raised to 60 houses (40 exposed and 20 not exposed).

**2.4: Method for examination of respiratory infections**

A questionnaire was administered to screen for respiratory infections in children enrolled in the study. Questions about Household Air Pollution exposure and the development of any illness within the last 30 days at the time of interview were posed to the parents or guardians of the enrolled children by trained interviewers who acted as research assistants for the study. Cough, wheezing, sneezing, running nose, oozing of pus from the ear, and fever was used as signs and symptoms for acute respiratory tract infections. All interviewers were employees of the Ministry of Health and worked in the study area. The questionnaire was administered in either vernacular, Kiswahili, or English based on the preference of the respondents. The interviewers were conversant and fluent in the three languages. To build control for Intra and inter variations, the interviewers were exchanged between the areas and households, and responses were checked at the end of the day for correctness, accuracy, and completeness. The interviewers returned to the respondents to complete any unfilled items of the questionnaire.

**Clinical examination**

A clinical examination was conducted by a Clinician with the assistance of a Nurse by taking their medical history. Examination results were entered into the subject's health facility attendance notebook and this exercise was repeated in a subsequent visit by the study subject. The patients kept the notebooks for use in any other visits. Mothers were asked questions as to whether their children aged < 5 years had suffered a respiratory disease through the questionnaire and this was further verified by the use of the patient notebook. Home-based diagnosis form and the log kept by the mother on observed symptoms of ARI were also used.

**Laboratory Examination**

Before laboratory examination, the examination procedures were described and explained to mothers or guardians in detail. The testing of HIV for children was explained in detail and informed consent and assent for their children's participation were obtained. Mothers who gave written consent and assent had their children examined for the presence or absence of any illness of the respiratory system. The procedures were explained and mothers were informed that they could refuse HIV testing for their children without loss of any benefits or consequences, and could also withdraw from the study at any time.

**2.6: Data collection procedures**

**a) Acute respiratory infection data**

Observations and identification of ARI and treatments were performed by a clinician and their treatment cards were counter-checked. Inpatient notes for those hospitalized were reviewed to identify the exact type of acute respiratory infection.

**b) Metrological data**

Metrological data on humidity, temperature, and rainfall (both minimum and maximum), were collected daily from the Wambugu weather station, serving the study area. Both wet and dry bulb thermometers were used to measure humidity, while temperatures were measured by use of an ordinary thermometer and a rain gauge for measuring rainfall.

**2.7: Air Sampling and Analysis**

For assessment of pollution by particulates, 24 hours respirable particulates (approximate aerodynamic diameter 10 microns or less) were measured in houses using a Buck I.H pump sampling at 2.2 liters per minute, a Higgins – Dewell type cyclone, and 35 mm glass fiber filters (whatmann). This equipment conforms to BSEN – 481 for an inhalable thoracic response.

The pumps were calibrated using a Munro RM1069 rotometer before each sampling period. The cyclone was placed at 4 feet above the floor of the kitchen, and 4 feet horizontally from the hearth.

Due to the very moderate (not high) levels of pollution, filter cassettes were not changed after 12 hours because clogging was not anticipated. The equipment was designed such that if the sampling flow rate dropped by 5% or more, the Buck pump sampler suspends air sampling, and records the time and volume sampled before shutting down.

**Air pollution data collection through questionnaire**

Information on the household, house construction (windows, doors, and ventilation), stove type, and the fuel used for various purposes, during the air sampling period, was collected using a standard questionnaire developed for the purpose.

**Air pollution data collection through laboratory methods (glass fiber filters)**

Filters for gravimetric analyses were prepared using standard procedures, in the department of analytical chemistry, University of Nairobi. The filters were dried and then weighed on a 6-place balance, before insertion into the cyclone cassettes, labeled, and dispatched to the field for air sampling. On return from the field, the filters (still in cassettes) were dried, and then re-weighed. Regular checks were made on calibrations of the balance, and laboratory blank filters were used to check quality control.

**Field supervision and quality assurance during air sampling**

Two field officers had a supervisor who was responsible for the preparation of the questionnaires, filter cassettes, and air sampling equipment (charging and calibration) for each house visit. The supervisor directly observed 50 % of household assessments (air sampling and questionnaires). Ten percent of the questionnaires were repeated independently by the supervisor. The supervisor also repeated 10% of the households' air monitoring using the University of California, Berkery (UCB) particulate monitor and filter method. On return

from the field, the supervisor re-checked and recorded the stored pump data (time elapsed, volume sampled, flow rate). A proportion of filters prepared in the laboratory were used as blanks and returned from the field unused to act as controls.

**Data management and analysis**

All data were coded and double entered into computer entry files using (SPSS version 10). Data entry and cleaning were carried out continuously and the final analyses were performed after all the databases were ready. Analyses were carried out at primary and detailed levels of descriptive statistics including medians, means, with 95% confidence intervals, and frequency distributions. Correlation analyses to identify parameters associated with respiratory infections and multivariate analyses to determine which factors were independently associated with respiratory infections in children aged under five years were performed on the generated data. Comparative analyses between the study groups and adjusting for possible confounding variables were conducted.

## IV. Results

**Descriptive results**

Table 1 summarises the socio-demographic and economic characteristics of the 430 study participants

**Table 1: Distribution of characteristics in the study group (n = 430)**

| | | Frequency | Percent |
|---|---|---|---|
| | **Socio-demographic characteristics** | | |
| **Sex** | Male | 221 | 51.4 |
| | Female | 209 | 48.6 |
| | Total | 430 | 100.0 |
| **Age** | <=2 | 278 | 64.7 |
| | >2 | 152 | 35.3 |
| | Total | 430 | 100.0 |
| | Mean age | 20± 12.9 SD months | |
| **Marital status** | Married | 393 | 91.4 |
| | Not Married | 37 | 8.6 |
| | Total | 430 | 100.0 |
| **Religion** | Muslim | 46 | 10.7 |
| | Christian | 384 | 89.3 |
| | Total | 430 | 100.0 |
| **Education** | Low Education | 195 | 45.3 |
| | Higher Education | 235 | 54.7 |
| | Total | 430 | 100.0 |
| **Occupation** | Salaried/Business | 267 | 62.1% |
| | House Wife | 163 | 37.9 |
| | Total | 430 | 100.0 |
| **Housing and air pollution characteristics** | | | |
| **Exposure** | Unexposed | 202 | 47.0 |
| | Exposed | 228 | 53.0 |
| | Total | 430 | 100.0 |
| **PM10** | Mean in exposed HHs | 100.8±95.1 µg/$m^3$ | |
| | Mean unexposed HHs | 4.1± 4 µg/$m^3$ | |
| | Overall mean | 68.6±90.1 µg/$m^3$ | |
| **Smoking** | No | 316 | 73.5 |
| | Yes | 114 | 26.5 |
| | Total | 430 | 100.0 |
| **Time in cooking place** | < 1 Hr | 183 | 42.6 |
| | >=1 Hr | 247 | 57.4 |
| | Total | 430 | 100.0 |
| **Breastfeeding and anthropometric characteristics** | | | |
| **Breastfeeding** | No | 269 | 62.6 |
| | Yes | 161 | 37.4 |
| | Total | 430 | 100.0 |
| **Stunting** | Stunted | 88 | 27.6 |
| | Normal | 231 | 72.4 |

| | | | |
|---|---|---|---|
| | Total | 319 | 100.0 |
| **Underweight** | Underweight | 36 | 11.3 |
| | Normal | 283 | 88.7 |
| | Total | 319 | 100.0 |
| **Wasting** | Wasted | 40 | 12.5 |
| | Normal | 279 | 87.5 |
| | Total | 319 | 100.0 |
| | **Weather characteristics** | | |
| **Temperatures** | Mean | 18.6±1.7 $^0$C | |
| | Minimum | 12.2$^0$C | |
| | Maximum | 28.8$^0$C | |
| **Rainfall** | Annual total | 1109.3 mm | |
| | Total rainy days | 106 | |
| **Relative humidity** | Mean | 68.7%±6.7 | |
| | Minimum | 54% | |
| | Maximum | 78.5% | |
| **HIV/AIDS** | Number tested (n =430) | 12 (2.8%) | |
| | Positive | 1(8.3%) | |
| | Negative | 11(91.7%) | |

**Correlations and association of specific risk factors of acute respiratory infections and exposure status**

there existed a significant relationship (R = 0.110) was observed between marital status and the development of ARI. Children from unmarried families were more at risk of developing ARI compared to children from married families.

Parental education was highly correlated with ARI (R= 0.248), implying that children born to parents who were more educated recorded fewer cases of ARI.

Time the child spent in the cooking place was highly correlated with the development of ARI (R = 0.2620), implying that children who spent more time in the cooking place developed more ARI episodes (R = -0).

Parental smoking was highly correlated with the development of ARI, implying that children from households where parents were smokers developed more ARI cases (R = 0.149).

There was a strong correlation between the development of ARI and stunting (R = 0.141), implying that stunted children developed more cases of ARI. However, there was no statistical correlation between underweight, wasting, and development of ARI.

Table 3 details the correlation coefficients of different variables with the number of ARI episodes; sample sizes (N) and, coefficient correlation values. Details of the p-values are in appendix 8.

Exposure was highly correlated with the number of episodes a child suffered $p< 0.05$. An increase in exposure increased the number of episodes a child suffered.

Parental smoking was also highly correlated with the number of episodes a child suffered (R = 0.112). The stunting status of the children was also statistically correlated (R = 0.200) with the number of episodes a child suffered.

**Table 2: Correlation of ARI (binary) with variables**

| | ARI | Sex | Age | Marital | Exposure | Religion | Education | Occupation | Time | Breastfeeding | Smoking | Stunting | Underweight | Wasting |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| ARI | 1 | | | | | | | | | | | | | |
| Sex | 0.022 | 1 | | | | | | | | | | | | |
| Age | 0.085 | -0.03 | 1 | | | | | | | | | | | |
| Marital | 0.119(*) | 0.005 | 0.264(**) | 1 | | | | | | | | | | |
| Exposure | 0.323(**) | 0.072 | -0.006 | 0.127(*) | 1 | | | | | | | | | |
| Religion | -0.021 | -0.054 | 0.032 | -0.074 | -0.120(*) | 1 | | | | | | | | |
| Education | 0.248(**) | 0.086 | -0.041 | 0.115(*) | 0.344(**) | -0.149(**) | 1 | | | | | | | |
| Occupation | -0.066 | -0.108 | 0.019 | -0.250(**) | -0.261(**) | 0.062 | -0.246(**) | 1 | | | | | | |
| Time | 0.267(**) | -0.042 | -0.021 | -0.108 | -0.680(**) | 0.154(**) | -0.469(**) | 0.387(**) | 1 | | | | | |
| Breastfeeding | -0.014 | 0.04 | -0.515(**) | -0.091 | 0.045 | -0.053 | 0.001 | -0.134(*) | -0.066 | 1 | | | | |
| Smoking | 0.149(**) | 0.079 | 0.013 | -0.021 | 0.253(**) | -0.202(**) | 0.158(**) | -0.110(*) | -0.281(**) | -0.024 | 1 | | | |
| Stunting | 0.141(*) | 0.019 | 0.011 | 0.02 | 0.09 | 0.03 | -0.058 | -0.053 | -0.024 | 0.019 | -0.031 | 1 | | |
| Underweight | 0.009 | -0.02 | 0.051 | 0.124(*) | 0.016 | -0.041 | 0.005 | 0.008 | 0 | -0.033 | -0.031 | -0.017 | 1 | |
| Wasting | 0.016 | 0.002 | 0.019 | 0.132(*) | -0.032 | -0.009 | 0.044 | 0.006 | 0.05 | -0.003 | -0.037 | -0.083 | 0.587(**) | 1 |

** Denotes correlation is significant at the 0.01 level (2-tailed). *Denotes correlation is significant at the 0.05 level (2-tailed).

**Table 3: Correlation of ARI episodes (continuous) with variables**

| | ARI Episodes | Sex | Age | Marital | Exposure | Religion | Education | Occupation | Time | Breastfeeding | Smoking | Stunting | Underweight | Wasting |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| ARI Episodes | 1 | | | | | | | | | | | | | |
| Sex | 0.063 | 1 | | | | | | | | | | | | |
| Age | -0.009 | -0.03 | 1 | | | | | | | | | | | |
| Marital | -0.021 | 0.005 | 0.264(**) | 1 | | | | | | | | | | |
| Exposure | 0.275(**) | 0.072 | -0.006 | 0.127(*) | 1 | | | | | | | | | |
| Religion | -0.064 | -0.054 | 0.032 | -0.074 | -0.120(*) | 1 | | | | | | | | |
| Education | -0.016 | 0.086 | -0.041 | 0.115(*) | 0.344(**) | -0.149(**) | 1 | | | | | | | |
| Occupation | -0.066 | -0.108 | 0.019 | -0.250(**) | -0.261(**) | 0.062 | -0.246(**) | 1 | | | | | | |
| Time | -0.021 | -0.042 | -0.021 | -0.108 | -0.680(**) | 0.154(**) | -0.469(**) | 0.387(**) | 1 | | | | | |
| Breastfeeding | 0.046 | 0.04 | -0.515(**) | -0.091 | 0.045 | -0.053 | 0.001 | -0.134(*) | -0.066 | 1 | | | | |
| Smoking | 0.112(*) | 0.079 | 0.013 | -0.021 | 0.253(**) | -0.202(**) | 0.158(**) | -0.110(*) | -0.281(**) | -0.024 | 1 | | | |
| Stunting | 0.200(**) | 0.019 | 0.011 | 0.02 | 0.09 | 0.03 | -0.058 | -0.053 | -0.024 | 0.019 | -0.031 | 1 | | |
| Underweight | -0.047 | -0.02 | 0.051 | 0.124(*) | 0.016 | -0.041 | 0.005 | 0.008 | 0 | -0.033 | -0.031 | -0.017 | 1 | |
| Wasting | -0.059 | 0.002 | 0.019 | 0.132(*) | -0.032 | -0.009 | 0.044 | 0.006 | 0.05 | -0.003 | -0.037 | -0.083 | 0.587(**) | 1 |

** Denotes correlation is significant at the 0.01 level (2-tailed). *Denotes correlation is significant at the 0.05 level (2-tailed).

**Correlations between ARI and main risk factors**

Table 4 gives a summary of the correlations between risk factors and ARI. Exposure to indoor air pollution, cigarette smoking, and stunting were correlating for both binary and continuous ARI variables. Marital status, education, and time spent in the cooking place were correlates of binary ARI and not continuous ARI. Strong correlates for ARI were exposure to indoor air pollution, education, time spent in the cooking place, cigarette smoking, and stunting which were all significant at the 0.01 level (2-tailed).

**Table 4: The association of risk factors with the development of ARI**

| **Risk factor** | **n** | **R for binary ARI** | **R for continuous ARI Episodes** |
|---|---|---|---|
| Sex | 319 | 0.022 | 0.063 |
| Age | 319 | 0.085 | 0.009 |
| **Marital status** | 319 | 0.119* | 0.021 |
| **Exposure** | 319 | 0.322** | 0.275** |
| Religion | 319 | -0.021 | -0.064 |
| **Education** | 319 | 0.248** | 0.016 |
| Occupation | 319 | -0.066 | -0.066 |
| **Time** | 319 | 0.267** | 0.021 |
| B/feeding | 319 | -0.014 | 0.046 |
| **Smoking** | 319 | 0.149** | 0.112* |
| **Stunting** | 319 | 0.141* | 0.200** |
| Underweight | 319 | 0.009 | 0.047 |
| Wasting | 319 | 0.016 | 0.059 |

**Incidence rates of ARI by person –month at risk and exposure status**

Calculations generated from table 7 indicates the following:

- Incidence rate in exposed group (r1) = 186.9 per 1000 persons-months
- Incidence rate in unexposed group (r0) = 123.4 per 1000 person-months
- Overall incidence rate = 155.2 per 1000 person-months
- Rate difference = 63.5 per 1000 person –months
- Population attributable risk (PAR) = 63.5 per 1000 person –months
- Population Attributable fraction (PAF) 34%
- RR (Rates ratio) = Exposed/unexposed= incidence rate in exposed/incidence rate in unexposed = r1/r0 = 150x5163/87x5661 = 1.57(95% CI, 1.19-2.07)

**Table 5: Subjects follow up and ARI**

| **Exposed** | | | | **Unexposed** | | | | |
|---|---|---|---|---|---|---|---|---|
| **New cases** | **Number Followed** | **Months followed** | **IR/1000** | **New cases** | **Number followed** | **Months followed** | **IR/1000** | **Overall IR/1000** |
| 102 | 215 | 3 | 158.1 | 59 | 187 | 3 | 105.2 | 131.7 |
| 114 | 190 | 6 | 168.6 | 66 | 184 | 6 | 115.5 | 140.1 |
| 125 | 204 | 9 | 174.6 | 73 | 190 | 9 | 115.6 | 145.1 |
| 150 | 170 | 12 | 186.9 | 87 | 149 | 12 | 123.4 | 155.2 |

**Table 6: Exposure to IAP and ARI case by person – month at risk**

| | **Exposed to indoor air pollution** | |
|---|---|---|
| | Yes | No |
| ARI cases | 150 | 87 |
| Person – month at risk | 5661 | 5163 |

Relative risk (RR) = 1.57 (95% CI, 1.19-1.2.07)

**Incidence rate during months of follow up**

Figure 1 shows changes in incidence rates in the exposed and unexposed groups in selected points of follow-up. The incidence rate of ARI was higher in the exposed group (186.9 per 1000 person-months) than in the unexposed group (123.4 per 1000 person-months). Higher incidence rates were recorded during the first three months preceding the follow-up and progressively curved upward to the final months of follow-up. The chi-square value for the differences between exposed and unexposed incidence rates was 11.23, p-value 0.008 thus statistically significant.

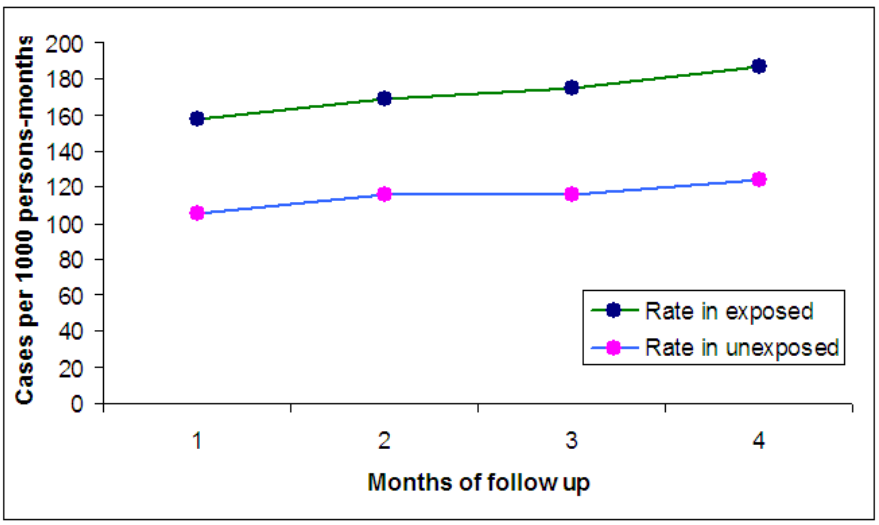


**Figure 1: Incidences rates**

X2 11.23, p-value, 0.008

**Comparison of particulate matter levels in exposed and unexposed households**

**Mean PM 10 level in exposed and unexposed households**

Table 7 details the mean values of PM10 in exposed versus non-exposed households. According to exposure classification, the overall average particulate matter of 10 microns in diameter was 68.6 ± 90.1μg/$m^3$. In the exposed households, the average PM10 level was 100.8 ± 95.3 μg/$m^3$ compared to PM10 level in non-exposed households of 4.1 ± 4.0 μg/$m^3$. The difference in the mean PM10 between exposed and non-exposed households was statistically significant by students' t-test, 4.88, p-value 0.000.

**Table 7: Mean PM 10 level in exposed and unexposed households**

| Exposure status | n | Mean | SD | 95% CI for Mean | |
|---|---|---|---|---|---|
| | | | | Lower Bound | Upper Bound |
| Unexposed | 20 | 4.10 | 4.012 | 2.22 | 5.98 |
| Exposed | 40 | 100.84 | 95.255 | 70.38 | 131.30 |
| Total | 60 | 68.59 | 90.099 | 45.32 | 91.87 |

**Association of Particulate matter of 50μg/$m^3$ cut off point and ARI episodes**

Table 8 shows the number of episodes that were recorded between the exposed and non-exposed groups. The mean overall number of ARI episodes a child suffered per year was 4.85(approximately 5). The relative risk for development of ARI by children in the exposed group was 10, 95% C. I 3.3-30.0. This indicated that children who were exposed to PM10 of >50 μg/$m^3$ were 10 times likely to develop more than 5 episodes of ARI within one year. The chi-square $\chi^2$ for the difference of the number of episodes between the two groups was $\chi^2$ = 34.3, p-value 0.000.

Figure 14 shows the relationship between the PM10 and the number of ARI episodes that the children developed in 12 months. As levels of particulate matter pollutants of less than 10 microns in diameter increased, the episodes of acute respiratory infections also increased thus giving a positive linear correlation (R = 0.358, p = 0.005).

**Table 8: Mean ARI episodes between exposed and non-exposed households**

| **PM10 levels** | **ARI Episodes** | | | | **Total** |
|---|---|---|---|---|---|
| | **> 5** | | **≤ 5** | | |
| | Freq | % | Freq | % | |
| **>50 μg/$m^3$** | 20 | 83.3 | 4 | 16.7% | 24 |
| **≤ 50 μg/$m^3$** | 3 | 8.3 | 33 | 91.7% | 36 |
| **Total** | 23 | 38.3 | 37 | 61.7% | 60 |

Mean ARI episodes = 4.85 (approximately = 5)

$\chi^2$, 34.266, p -value 0 .000

Relative risk = 10.0 (95% C.I 3.3 -30.0).

**Other factors that could increase the risk of acute respiratory infections other than indoor air pollution**

**3.4.1: Nutrition situation versus exposure status**

The difference in proportion between children not breasting and living in exposed and unexposed households was 8.4% compared to 4.2% who were breastfeeding. This difference was not statistically significant p-value 0.422. On the other hand, the difference in underweight and wasting and between exposure groups was also statistically insignificant, p-values 0.773 and 0.112 respectively. However, the proportions derived from stunting and exposure against that of not stunting and exposure were statistically significant, p-value 0.022 (see table 9).

**Table 9: Nutrition and exposure**

| | | | **Exposed** | | **Unexposed** | | **$X^2$** | **p** |
|---|---|---|---|---|---|---|---|---|
| | | **n** | **Freq** | **%** | **Freq** | **%** | **0.645** | **0.422** |
| | | **n** | **Freq** | **%** | **Freq** | **%** | | |
| Stunting | Not normal | 88 | 56 | 63.6 | 32 | 36.4 | **5.225** | **.022** |
| | Normal | 231 | 114 | 49.4 | 117 | 50.6 | | |
| **Total** | | **319** | **170** | **53.3%** | **149** | **46.7%** | | |

**Association of malnutrition and ARI**

Concerning malnutrition and development of ARI, table 10 below shows that stunted children were 1.4 times (RR= 1.38, 95% CI 1.16-1.64, p-value, 0.001) at risk of developing ARI than those who were not stunted.

**Table 10: Malnutrition and development of ARI**

| | **n** | **ARI** | | | | **$\chi^2$** | **p** | **RR** | **95% C.I for RR** | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | **Yes** | | **No** | | | | | | |
| | | **Freq** | **%** | **Freq** | **%** | **$\chi^2$** | **p** | **RR** | **95% C. I for RR** | |
| Stunted | 88 | 65 | 73.9 | 23 | 26.1 | 10.72 | **0.001** | 1.38 | 1.16 | 1.64 |
| Normal | 231 | 124 | 53.7 | 107 | 46.3 | | | | | |
| Total | 319 | 189 | 59.2 | 130 | 40.8 | | | | | |

**Association of seasons and number of ARI episodes the child developed**

According to the results in table 11, the cold season was a risk factor for developing more ARI episodes in children. During the cold months of June, Augusts November, December, and January, the children were 3.6 times at risk of developing more than 5 ARI episodes than during the hot season (RR = 3.6, 95% CI 3.1- 4.2, $\chi^2$ 494.98, p-value 0.00).

**Table 11: Seasons versus ARI episodes**

| **Season** | **ARI Episodes** | | **Total** |
|---|---|---|---|
| | > 5 | ≤ 5 | |
| Cold months (Jan, May, Jun, Aug, & Nov, | 467 | 36 | 503 |
| Hot months (Feb, Mar, Apr, Jul, Sep, Oct, Dec) | 122 | 348 | 470 |
| Total | 589 | 384 | 973 |

Relative risk = 3.6, 95% CI 3.1- 4.2

$\chi^2$ 494.98; P-value 0.00

**Association of relative humidity and acute respiratory infections**

**Logistic regression**

Using binary logistic regression for the dependent variable as ARI present or absent against binary risk variables (independent variables) which were significantly correlated with one or more variables from the sample of 319 (children who were fully followed up for 12 months) shown in table 12, it was found that three variables i.e. exposure (RR=2.6, 95% CI, 1.3-5.1, p-value, 0.0059), education (RR= 2.1, 95%CI 1.2-3.6, p-value 0.0109) and stunting (RR= 2.5, 95% CI, 1.4-4.5, p-value, 0.00320) were directly and independently associated with the development of ARIs as evidenced by values generated by odds ratios, 95% confident intervals and p-values.

In table13 binary logistic regression for the dependent variable as ARI episodes of ≤5 and >5 episodes were conducted from the small data which was generated from the households where PM 10 was monitored, it was found that PM10 of >50µg/$m^3$ was directly associated with the development of ARI episodes that were more than 5 in number as evidenced by RR of 1953.5, 95% CI, 9.2-416425.6, and p-value of 0.0056 and breastfeeding (RR = 0.0033, 95% CI 0.0000-0.7264, p-value, 0.0379) thus suggesting that breastfeeding was protective for ARI.

**Table 12: Binary Logistic regression with the dependent variable as ARI (n=319)**

| Variable | B | Wald | Sig | RR | 95% CI for RR | |
|---|---|---|---|---|---|---|
| | | | | | | |
| Sex | -0.3028 | 1.3546 | 0.2445 | 0.7387 | 0.4437 | 1.2301 |
| Age | 0.2823 | 0.7303 | 0.3928 | 1.3261 | 0.6941 | 2.5337 |
| Marital | 0.2786 | 0.3984 | 0.5279 | 1.3213 | 0.5562 | 3.1390 |
| Exposure | 0.9474 | 7.5733 | **0.0059** | 2.5791 | 1.3135 | 5.0644 |
| Religion | 0.3640 | 0.6304 | 0.4272 | 1.4390 | 0.5860 | 3.5339 |
| Education | 0.7220 | 6.4853 | **0.0109** | 2.0586 | 1.1810 | 3.5883 |
| Occupation | 0.4290 | 1.9782 | 0.1596 | 1.5357 | 0.8447 | 2.7919 |
| Time | 0.0336 | 0.0056 | 0.9403 | 1.0341 | 0.4293 | 2.4909 |
| Breastfeeding | 0.5114 | 1.9805 | 0.1593 | 1.6675 | 0.8181 | 3.3992 |
| Smoking | 0.0433 | 0.0136 | 0.9070 | 1.0442 | 0.5052 | 2.1585 |
| Stunting | 0.9052 | 8.6919 | **0.0032** | 2.4725 | 1.3545 | 4.5132 |
| Underweight | 0.4961 | 1.4057 | 0.2358 | 1.6423 | 0.7232 | 3.7292 |
| Wasting | 0.3808 | 0.8764 | 0.3492 | 1.4634 | 0.6594 | 3.2477 |
| Constant | -8.7048 | 18.0160 | 0.0000 | | | |

**Table 13: Binary Logistic regression with the dependent variable as ARI cut point of greater or less than 5 episodes a child suffered (n=60)**

| Variable | B | S.E. | Wald | Sig. | RR | 95.0% CI. for RR | |
|---|---|---|---|---|---|---|---|
| | | | | | | Lower | Upper |
| Sex | -3.0413 | 1.7603 | 2.9852 | 0.0840 | 0.0478 | 0.0015 | 1.5049 |
| Age | -4.8493 | 2.4868 | 3.8026 | 0.0512 | 0.0078 | 0.0001 | 1.0250 |
| Marital | -1.6485 | 2.0316 | 0.6584 | 0.4171 | 0.1923 | 0.0036 | 10.3131 |
| Religion | -3.1528 | 2.1759 | 2.0995 | 0.1474 | 0.0427 | 0.0006 | 3.0401 |
| Education | 2.8796 | 2.0058 | 2.0611 | 0.1511 | 17.8078 | 0.3494 | 907.7386 |
| Occupation | 0.4833 | 1.5557 | 0.0965 | 0.7560 | 10.6215 | 0.0769 | 34.2047 |
| Exposure | -0.8514 | 2.7092 | 0.0988 | 0.7533 | 0.4268 | 0.0021 | 86.3654 |
| Smoke | -0.8254 | 1.6216 | 0.2591 | 0.6107 | 0.4381 | 0.0182 | 10.5158 |
| PM10 | 7.5774 | 2.7358 | 7.6712 | **0.0056** | 1953.4778 | 9.1639 | 416425.55 |
| Time | 0.4265 | 1.7314 | 0.0607 | 0.8054 | 1.5319 | 0.0515 | 45.6030 |
| Breastfeeding | -5.7086 | 2.7495 | 4.3106 | **0.0379** | 0.0033 | 0.0000 | 0.7264 |
| Stunting | 1.0568 | 1.5791 | 0.4479 | 0.5033 | 2.8771 | 0.1303 | 63.5441 |
| Underweight | 0.5695 | 2.4460 | 0.0542 | 0.8159 | 1.7673 | 0.0146 | 213.4926 |
| Wasting | 2.5580 | 2.2474 | 1.2955 | 0.2550 | 12.9094 | 0.1577 | 1056.4751 |
| Constant | 2.0053 | 8.9657 | 0.0500 | 0.8230 | | | |

**Univariate analysis with dependent Variable as ARI present or absent against various independent**

On univariate analysis for all the variables that had shown significant correlations using the data for all the subjects that were fully followed for 12 months, exposure, education, and stunting were independently associated with ARI as evidenced by p-values of 0.002, 0.004, and 0.011 respectively as shown in table 14.

**Table14: Univariate analysis with the dependent variable as ARI present/absent (n=319)**

| | B | Sig. | 95% Confidence Interval for B | |
|---|---|---|---|---|
| **Parameter** | | | **Lower Bound** | **Upper Bound** |
| Intercept | 0.333 | 0.351 | -0.370 | 1.037 |
| Sex | -0.052 | 0.320 | -0.155 | 0.051 |
| Age | 0.003 | 0.164 | -0.001 | 0.008 |
| Marital | 0.099 | 0.243 | -0.068 | 0.267 |
| Exposure | 0.227 | **0.002** | 0.086 | 0.367 |
| Religion | 0.066 | 0.391 | -0.085 | 0.217 |
| Education | 0.068 | **0.004** | 0.021 | 0.114 |

| | | | | |
|---|---|---|---|---|
| Occupation | 0.019 | 0.194 | -0.010 | 0.048 |
| Time | -0.019 | 0.694 | -0.116 | 0.077 |
| Breastfeeding | 0.055 | 0.527 | -0.116 | 0.226 |
| Smoking | 0.103 | 0.140 | -0.034 | 0.239 |
| Stunting | 0.042 | **0.011** | 0.010 | 0.075 |
| Underweight | -0.010 | 0.733 | -0.066 | 0.046 |
| Wasting | 0.013 | 0.605 | -0.035 | 0.061 |

**Multivariate analysis**

The results as shown in table 15 revealed that exposure, time, and stunting were independently associated with the number of ARI episodes suffered by the children as evidenced by p-values of 0.000, 0.000, and 0.002 respectively. The rest of the variables were not significantly independently associated with ARI episodes. When the same analysis was performed on using the data for the subjects who were living in the households where PM10 monitoring was done, time spent in cooking place became the only variable that was independently associated with ARI episodes as shown in table 16

**Table 15: Multivariate analysis for Dependent Variable as number of ARI episodes against various independent variables (n=319)**

| | | **B** | **Sig.** | **95% Confidence Interval** | |
|---|---|---|---|---|---|
| **Dependent Variable** | **Parameter** | | | **Lower Bound** | **Upper Bound** |
| ARI Episodes | Intercept | -1.778 | .461 | -6.523 | 2.967 |
| | Sex | .174 | .621 | -.520 | .869 |
| | Age | .011 | .492 | -.021 | .043 |
| | Marital | -.636 | .269 | -1.766 | .494 |
| | Exposure | 3.091 | **.000** | 2.143 | 4.039 |
| | Religion | -.525 | .311 | -1.543 | .492 |
| | Education | -.101 | .524 | -.413 | .211 |
| | Occupation | -.120 | .226 | -.315 | .075 |
| | Time | 1.398 | **.000** | .747 | 2.048 |
| | Breastfeeding | .455 | .438 | -.698 | 1.607 |
| | Smoking | .648 | .167 | -.273 | 1.569 |
| | Stunting | .344 | **.002** | .123 | .565 |
| | Underweight | -.100 | .604 | -.480 | .279 |
| | Wasting | -.041 | .805 | -.365 | .283 |

**Table 16: Multivariate analysis for Dependent Variable as number of ARI episodes against various independent variables (n=60)**

| | | B | Sig. | 95% Confidence Interval for B | |
|---|---|---|---|---|---|
| **Dependent Variable** | Parameter | | | Lower Bound | Upper Bound |
| ARI Episodes | Intercept | 6.071 | .117 | -1.585 | 13.727 |
| | Sex | -.515 | .364 | -1.645 | .616 |
| | Age | .010 | .749 | -.052 | .072 |
| | Marital | -.451 | .633 | -2.337 | 1.436 |
| | Religion | .308 | .714 | -1.378 | 1.995 |
| | Education | -.356 | .303 | -1.044 | .333 |
| | Occupation | -.294 | .134 | -.682 | .094 |
| | Exposure | 1.121 | .228 | -.725 | 2.966 |
| | Smoke | -.629 | .358 | -1.990 | .733 |
| | P/matter | .002 | .545 | -.005 | .009 |
| | Time | .910 | **.036** | .060 | 1.760 |
| | Breastfeeding | -.032 | .967 | -1.568 | 1.505 |
| | Stunting | .109 | .546 | -.253 | .472 |
| | Underweight | -.049 | .871 | -.660 | .561 |
| | Wasting | .200 | .447 | -.325 | .724 |

## V. Discussion

Children who lived in households exposed to Household Air Pollution were 1.6 times at risk of developing acute respiratory infections compared to those who stayed in non-exposed households (RR 1.57, 95% C.I, 1.19-2.07). The study also found that the mean number of ARI episodes that a child suffered per year was 4.85 (approximately 5), whereas children who were exposed to PM10 of > 50 $\mu g/m^3$ were 10 times likely to develop more than 5 episodes of ARI within a year (RR = 10, 95 CI 3.3-30, p = 0.000), results that are similar to Wafula *et al.,* 1990, Pandey, 1984 and 1989 and Smith, 2000a)

Based on this study results, ARIs are not rare infections. The overall prevalence for acute respiratory infections ranged from 22.8% to 42.3%, which compared fairly well with outpatient morbidity presented in the Kenya health information management system report of 1996-99. The Kenya health information management system showed that acute respiratory infections were the leading cause of illness in children, with the annual outpatient treatment of 40%. Children who were living in households that were emitting PM10 of > 50 $ug/m^3$ were 10 times at risk of developing ARI than those who were staying in households emitting PM10 of ≤ 50 $\mu g/m^3$ (RR= 10, 95% CI 3.3-30, p = 0.00). There is further concurrence with these findings and in particular studies by Thomas and Zelikoff, (1999) who found that children living in homes with PM10 levels of 65 ug/m3 and above had an odds ratio that was 7 times higher than for children with levels below 65 ug/m3.

The levels of PM10 increased the number of episodes of acute respiratory infections also increased with a correlation coefficient of 0.358, and p-value of 0. 005. This positive linear correlation confirms similar observations made by Mazur, (1995), who reported higher morbidity from respiratory diseases in areas of high atmospheric pollution than observed otherwise in similar areas but of low atmospheric pollution. Our findings were further similar to those for Holgate *et al*., (1999) and Reynodes *et al*., 1998 who reported odds ratio for incidence in the range of 2-4 and Pope, (1989) who found that the closing and re-opening of a steel mill which was the primary source of PM10, was associated with elevated PM10 levels and hospital admissions for ARI and the 24 hr PM10 levels greater than 50$\mu g/m^3$, resulted to higher average admission of 2-3 times.

The incidence rate difference in this study was 63.5 per 1000 person-months and the proportion attributable fraction was 34%. Such results imply that Household Air Pollution alone was responsible for 63.5 cases of ARI in every 1000 children during the 12 months of exposure and that if Household Air Pollution was controlled through an intervention to tolerable levels such as PM10 ≤ 50 ug/m3, this would have resulted in reducing acute respiratory infections by 34% in the study area. These are very useful findings as they provide policymakers and implementers with an estimate of the impact of reducing Household Air Pollution to maximum tolerable levels.

There was no statistically significant correlation in age and development of ARI in our study but the coefficient was negative indicating that children who were two years and below were more at risk of developing ARI than those whose age was more than two years. Failure of our results to reach statistically significant levels slightly contradicts conclusions where inferences of age being a risk factor in a developing country were made (Thomas and Zekoff, 1999, and Stansfield and Shepard, 1993, and Berman, 1991).

In the 40 households that were selected for 24-hour monitoring for PM10, on the basis that they were classified as exposed to indoor air pollution, three fifths (60%) were emitting PM10 of >50 $\mu g/m^3$, whereas in all 20 (100%) households classified in the unexposed group emitted PM10 of ≤50$\mu g/m^3$. This gave a strong association between classification by housing type (ventilation and type of fuel used) and classification by PM10 of 50 $\mu g/m^3$ cut point (p=0.000). Overall, the average particulate matter of 10 microns in diameter was 68.6 ± 90.1$\mu g/m^3$. In the exposed households, the average PM10 level was 100.8 ± 95.3 $\mu g/m^3$ compared to 4.1 ± 4.0 $\mu g/m^3$ for non-exposed households. The difference in the mean PM10 between exposed and non-exposed households was statistically significant 100.8 versus 4.1(students t-test 4.88, p-value 0.000). These mean levels are much higher than the tolerable levels set by the British health panel and European commission (United states Environment protection Agency, 1997).

In general, although the levels of PM10 found in this study were not in compliance with standards for the British health panel and European commission, previous studies in Kenya recorded higher levels. Smith et al., (1994) found that PM10 was 1998 $\mu g/m^3$ in exposed households compared to 546 $\mu g/m^3$ in controls, p<0.01. Pope, et al., (1995a and b), found high concentrations of 3700-4800 ug/m3 in kitchens. The 24 hrs monitoring of Household Air Pollution for PM10 by Intermediate Technology Development Group 2002(unpublished)**,** in Kenya, found the mean levels for Kajiado district were 5526 $\mu g/m^3$, Mumia/Butere and Nyamira in western Kenya mean levels were 1713 $\mu g/m^3$. Wafula E M *et al.,* 1990 also found the mean levels for particulate matter of 10 microns in diameter in Maragwa households, which we're using 3 traditional stone stoves to be 4459 $\mu g/m^3$. Smith *et al.,* 1994 and McCracken *et al.,* 1998 found 24 hours mean particulate matter concentrations in houses in rural areas ranging between 300-3000 ug/m3 and up to 30,000ug/m3 during periods of cooking respectively.

The mean PM10 levels found in this study were lower than those reported in previous studies in Kajiado where the communities use semi-dry cow dung and Butere/Mumias and Nyamira probably because of

differences in the fuel type used. High PM10 concentration levels found in other studies such as those of Smith et al., (1994) could be attributable to the type of equipment used and the methodologies adopted. The issue of equipment for monitoring Household Air Pollution has been and remains a major challenging technology, which is still being nurtured. The findings that the PM10 levels were far above those tolerable standards set by the European Commission and British health panel are a confirmation of the poor and inappropriate interventions practiced in reducing IAP and ARI in Kenya. This is as evidenced by the stipulated strategies for air pollution control guide for the environment management and coordination act of 1999 (Wamukuya and Situma, 2000)

Further analyses carried out using PM10 cut point of 50 $\mu g/m^3$ and that of fuel and housing revealed a strong association between the two classifications for indoor air polluted households (p-value 0.000). This, therefore, confirmed the criteria used to assign the 430 households and, strongly agreed with the pollution criteria based on PM10 emission levels. In addition, the strong association also confirmed the possibility of having eliminated differential and non-differential bias among study subjects, and therefore the effect of exposure on the outcome might not have been overestimated or underestimated.

The strong association found between the type of fuel used and emission of PM10 of > $50\mu g/m^3$ (P = 0.000) suggests that in households that use firewood and charcoal (inferior fuel), the risk of ARI increased compared to households that used superior fuel (gas and electricity). In almost all the households which used other types of fuel other than firewood and charcoal, the levels of PM10 measured were less than $50\mu g/m^3$.

Malnutrition in this study was strongly associated with the development of ARI. Our data for, stunting and wasting suggest that malnutrition increases the risk of acute respiratory infections. Stunted children were 1.4 times more at risk of developing ARI than those who were not stunted (RR=1.4,95% CI 1.16-1.64). our findings confirm those of Terri *et al.,* (1995) who concluded that improved nutrition contributed to lowering ARIs in rural Kenyan children.

According to Graham, 1999, chilling weather is a risk factor for acute respiratory infections in children. Similarly, acute respiratory infection trends in this study appeared to increase with decreased temperatures and humidity and increase in rainfall. The cold season was a risk factor for developing more ARI episodes in children. During the cold months, the children were 3.6 times at risk of developing more than 5 ARI episodes than during the hot season. This could be postulated that during the cold season, the temperature inversion made pollution concentrations build up because of the absence of turbulence thus increasing the incidence of ARI as concluded by the World Health Organization, (1972).

Logistic regression was performed to identify any predictors of acute respiratory infections. All the variables that were significant at the correlation analysis were further fed into the logistic regression, univariate and multivariate models. The end product was that education, stunting, breastfeeding, exposure to indoor air pollution, PM10, and time spent in the cooking place, became statistically significant. It, therefore, implies that parental literacy, stunting, breastfeeding; PM10, Household Air Pollution, and time spent by a child in the cooking place were independently associated with the development of acute respiratory infections, although breastfeeding was protective in terms of its association with ARI.

Education becomes a risk factor for acute respiratory infection, especially where the parent or guardian of the child innocently is unaware of the cause-and-effect logic for acute respiratory infections. This is in particular when the mother had always to stay with the child in a smoky kitchen all the time she was cooking thus exposing the child to high levels of particulate pollutants.

Stunting should essentially be viewed as an intermediate variable in the causal pathway and through an independent effect. This variable contributes to increasing the risk of developing ARI by reducing the body's immunity, which is crucial in fighting infectious agents (McBride, 1999). The other two variables (exposure to Household Air Pollution and particulate matter of less than 10 microns in diameter are the same. They both increase the risk of ARI by reducing the functioning of specific and non-specific host defense systems. Breastfeeding is generally seen to be playing the role of improving the nutritional status of the child, which in essence increases the body immunity to prevent ARI.

## VI. Conclusions

Disease incidence due to Household Air Pollution was relatively high in the study population.
If interventions in all households were implemented to a state where they were able to emit PM10 of $<50\mu g/m^3$, the acute respiratory infections would have been reduced by 34%.

Children who were exposed to Household Air Pollution were 1.57 times more at risk of developing ARI than those in unexposed households (RR=1.57,95% CI 1.19-2.07)

The cold weather season was a risk factor for the development of more episodes of ARI. Children were 3.6 times at risk of developing more than 5 episodes of ARI during the cold season compared to episodes in the hot season.

The mean PM10 levels of 100.8 $\mu g/m^3$ in this study are above the recommended tolerable levels set by the British health panel and the European commission at $\leq 50$ $\mu g/m^3$) but are lower than those found in other parts of Kenya.

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The use of inferior fuel and especially firewood was associated with emission of higher PM10 levels (p=0.000).

Education level, stunting, breastfeeding, time spent in a cooking place, and exposure to Household Air Pollution (including PM10) were risk factors that were independently associated with the development of acute respiratory infections in children.

**RECOMMENDATIONS**

1. The government through the ministries of Finance and Energy should promote the adoption of superior fuel for cooking and warming to reduce indoor air pollution. This could be approached by reducing the cost of gas and electricity and ensuring that electricity is accessible to the rural population who commonly use biomass.
2. There is a need to conduct further studies on Household Air Pollution monitoring for PM 2.5 as the results of this study were purely on PM 10 for comparison purposes in particular the outcome on acute respiratory infections.

3. While designing a Household Air Pollution reduction project, there is a need to develop interventions, in areas of improving the educational level, nutritional status especially reduction of stunting, promoting exclusive breastfeeding (protective factor for ARI), and reducing exposure to IAP including PM10 because these were the factors which were independently associated with increased risk of developing ARIs.

## References


[1]. **Berman S, (1991).** Epidemiology of acute respiratory infections in children in developing countries. Journal of infectious diseases, 13 (supplement 6); 454-62.

[2]. **Graham N .M, (1990).** The epidemiology of acute respiratory infections in children and adults, a global perspective. Epidemiology review; 12: 149-178.

[3]. **Holtgrave D.R., Crossby R.A**, **(2000).** Socio-capital, poverty, and income inequality as predictors of gonorrhea, syphilis, chlamydia, and AIDS cases rates in the United States. Sex Transm Inf. 79: 62-64.

[4]. **McCracken J.P., Smith K.R, (1998).** Emissions and efficiency of improved wood-burning cookstoves in highland Guatemala. Environment international journal, 24; 739-747.

[5]. **Muzar B, (1995).** Peak expiratory flow values in children relative to the degree of atmospheric air pollution, Acta Paediatr 84 (2); 203-5.

[6]. **Pandey, M.R, (1984).** Domestic smoke pollution and chronic bronchitis in a rural community of the hill region of Nepal. Thorax; 39:337-39.

[7]. **Pandey M.R., Neupane R.P., Gautam A., Shrestha I.B, (1989).** Domestic smoke pollution and acute respiratory infections in rural communities of the region of Nepal. Environment international; 15:37-40.

[8]. **Reynolds H.Y., Elius J.A, (1998).** Pulmonary defense mechanisms against infections. In-Fisherman A, Fisherman's pulmonary diseases and disorders, 3rd edition. New York: McGraw-Hill.

[9]. **Smith K.R., Samet J.M., Romieu I, (1994).** Air pollution and the energy ladder in Asia cities. Energy, 19: 587-600.

[10]. **Smith K.R., Samet J.M., Romieu I., Bruce N, (2000).** Indoor air pollution in developing countries and acute lower respiratory infections in children. Thorax; 55:518-32.

[11]. **Stansfield S., Shepard D, (1993),** an acute respiratory infection. Disease control priorities in developing countries. Oxford: Oxford University Press; 67-90.

[12]. **Terri J., Ballard P H., Charlotte G N, (1995).** The effects of malnutrition, parental literacy, and household crowding on acute respiratory infections in young Kenyan children. Journal of tropical pediatrics, (41); 9-11.

[13]. **Thomas P.T., Zelokoff J.T, (1999).** Air pollutants: moderators of pulmonary host resistance against infection. In: Holgate ST, Samet JM, Koren HS, et al., 91998). Air pollution and health. San Diego: Academy Press, 357-9.

[14]. **The United States Environment Protection Agency, (1997).** National ambient air quality standards for particulate matter. Federal register. Volume 62, Number 138, page 38658.

[15]. **Thomas P.T., Zelokoff J.T, (1999).** Air pollutants: moderators of pulmonary host resistance against infection. In: Holgate ST, Samet JM, Koren HS, et al., 91998). Air pollution and health. San Diego: Academy Press, 357-9.

[16]. **United Nations National air pollution controls administration, (1969).** Air quality criteria for particulate matter, Washington DC. The United States of America public health services (publication No.AP-49).

[17]. **Wafula E.M., Onyngo F.E., Thairu H., Boleij J.S., Hoek F., Ruiewaard P., Kagwanja S., De koning H., P, Kimani E, (1990).** Indoor air pollution in a Kenyan village: East African Medical Journal 67(1), 24-32.

[18]. **Wamukoya G.M., Situma F.D.P, (2000).** Environment management in Kenya. A guide to the environment management and coordination Act, 1999. Nairobi.

[19]. **World Health Organization publication, (1972).** Health hazards of the human environment. Air pollution and health 22-23 &342-4.


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