ENERGY TECHNOLOGY, INDOOR AIR POLLUTION, AND RESPIRATORY INFECTIONS IN DEVELOPING COUNTRIES

A FIELD STUDY FROM CENTRAL KENYA

Majid Ezzati

A DISSERTATION PRESENTED TO

THE FACULTY OF PRINCETON UNIVERSITY

IN CANDIDACY FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

RECOMMENDED FOR ACCEPTANCE

BY THE WOODROW WILSON SCHOOL OF

PUBLIC AND INTERNATIONAL AFFAIRS

November 2000

ENERGY TECHNOLOGY, INDOOR AIR POLLUTION, AND RESPIRATORY INFECTIONS IN DEVELOPING COUNTRIES

A FIELD STUDY FROM CENTRAL KENYA

Abstract

Globally, more than two billion people rely on biofuels as the primary source of domestic energy. Exposure to indoor air pollution, especially to particulate matter, from biomass combustion, is a causal agent of respiratory and eye diseases. Acute respiratory infections (ARI) and chronic respiratory diseases lead the causes of disease and mortality worldwide, and account for more than 10% of the global burden of disease, mostly in developing countries.

In this dissertation, I consider the linkages among household energy technology, indoor environment, and health. I provide quantitative analysis of (1) patterns of human exposure to indoor air pollution, (2) the exposure-response relationship for particulate matter and ARI, and (3) the pollution reducing performance of an array of stove-fuel combinations. Data are from three years (1996 – 1999) of field research in Central Kenya. I also briefly discuss the important issues in successful dissemination of household level technologies.

I construct *Profiles of exposure* using continuous real-time monitoring of pollution concentration and the location and activities of household members, supplemented by

data on the spatial dispersion of pollution and interviews. Exposure during brief high-intensity emission episodes accounts for 31% - 61% of the total exposure of household members who participate in cooking and 0% - 11% for those who do not. Simple models that neglect the spatial distribution of pollution within the home, intense emission episodes, and activity patterns underestimate exposure by 3% - 71% for different demographic sub-groups, resulting in inaccurate and biased estimations.

ARI and acute lower respiratory infections (ALRI) are increasing, concave functions of average daily exposure to PM_{10}. The rate of increase declines for exposures above approximately 2000 $\mu g.m^{-3}$. Consequently, programs aiming to reduce the adverse health impacts of indoor air pollution in developing countries should focus on measures that result in larger reductions in pollution, especially those that bring average exposure below 2000 $\mu g.m^{-3}$.

Improved wood stoves provide an overall reduction in the emission concentration compared to 3-stone fire. The largest reduction of emission concentrations and human exposure, however, is achieved through a transition from wood to charcoal. I discuss the implications for public health and technology transfer.

Table of Contents

List of Figures

List of Tables

Preface

In the course of my field research in Kenya, several events were reminders of how health and cooking, the focus of my research, pose difficult tradeoffs in day-to-day life.

In my first few weeks in Kenya, I entered a Turkana family's smoke-filled hut at Mpala Ranch. Within a few minutes, with cough and burning eyes, I understood the reality of all the numbers that I had read about indoor air pollution in developing countries. On another occasion, we arrived in a village to measure pollution only to find out that the month's food ration had not yet arrived and people had nothing to cook. One family kindly offered to make some tea, so that we could measure the smoke from their stove.

On numerous occasions during the early months, we came across sick people who could neither afford nor find transport to the hospital in Nanyuki. Although the basic medical services provided by our project nurse eventually addressed many of these cases, those referred to hospitals still had to deal with transportation and medical costs. During my last few weeks in Kenya, when our field project at Mpala had ended, I once again went to villages where sick people had little choice but to wait.

Finally, three children died at Mpala during the three years of my field research, two from pneumonia and another from an intestinal disease. All three deaths could have been prevented by simple means. I hope my thesis is a step in that direction.

Acknowledgments

"*I am grateful to those who guided me to think, not to what good thoughts are*" anonymous

I have been fortunate to have had tremendous intellectual and personal support from numerous people throughout my years at Princeton. I am especially grateful to my advisors and mentors, Dan Kammen, Burt Singer, Noreen Goldman, and Dan Rubenstein for their invaluable wisdom and encouragement. Dan Kammen, my primary advisor, far surpassed his initial commitment of providing me with the best possible educational opportunities. Burt Singer has been the ideal intellectual mentor to whom I could turn when I needed a boost of ideas. My interactions with him have profoundly influenced my thinking about, and approach to, research on public health, the environment, and social development. Noreen Goldman provided generous help and impeccable guidance in analyzing the mass of data that I collected in a rigorous manner, and organizing the results into a coherent dissertation. Dan Rubenstein offered kind support throughout my years at Princeton, especially in making my field work in Kenya smooth and productive. I benefited from the expertise, experience, and support of Clint Andrews, David Bradford, Angus Deaton, Jeff Herbst, Emmanuel Kreike, Denise Mauzerall, and Frank von Hippel in various stages of my Ph.D. work. Kirk Smith, of the University of California at Berkeley, kindly shared his wealth of knowledge on indoor air pollution and health. George Grinnell, in his course at McMaster University, raised the questions on science, technology, and human well-being that eventually led to my choice of Ph.D. program.

My friends and colleagues in the STEP program, Shardul Agrawala, Hrijoy Bhattacharjee, Dan Klooster, Mahesh Phandnis, Rachel Massey, David Romo Murillo, Alex Mutebi, Yesim Tozan, and Xiaoping Wang provided an enjoyable and stimulating environment. I should single out from this group Richard Duke, David Hassenzahl and Robert Margolis, with whom I entered graduate school. I have learned a great deal from them and I will miss their kind and intelligent presence.

My field research in Kenya has been the cornerstone of my dissertation. I would simply have been unable to conduct this work without the generous and kind support of a large number of people. Dan Kammen introduced me to research in Kenya, reminding me to observe carefully and to have fun during field work. Jackie Schatz provided willing help whenever a problem required a push from Princeton. Yousof Kaka and his sons drove me to hospital and looked after me when I had a road accident. Joseph Kithome assisted with setting up the research project and introduced me to Kenyan roads and villages. Bernard Mbinda led the project when I was away from Kenya, and contributed a great deal, intellectually and logistically. Bernard, David Kinyua, and Bell Okello became my close colleagues and friends at Mpala; I hope I will see them again.

My field research assistants, Mark Egelian, Peter Ekuam, Mary Lokeny, Grace Lokeny, and Jackson Ngisirkale not only worked long hours to collect the enormous amount of data that we needed, but also made my time at Mpala enjoyable. I hope I did as much for them as they did for me. Simon Munyi and Jolly Murithi worked under difficult conditions to collect an incredibly complete health data set. The administration of

Nanyuki District Hospital, especially Matron Wachanga, kindly provided two of their best nurses to assist us with collection of health data. Dr. A.W. Muriithi from the Kenyatta National Hospital and National ARI Programme provided valuable help in design and execution of the health monitoring system. I am also thankful to the staff and administration of Mpala Ranch and Mpala Research Centre, especially Joseph Leting, Nick Tomlinson, and Cyprian Gatua who made things work, even when others said it was not possible. I am forever indebted to the kind hospitality of the residents of Mpala Ranch and Mpala Research Centre. They not only agreed to all our data collection activities, but also generously shared their lives with us, by offering us tea; by pushing our truck when it broke down or was stuck in mud; by patiently showing me how to cut firewood, fetch water, cook ugali; and by teaching me (with mixed success) Turkana dances and songs.

African Academy of Sciences (Dr. Kone, Professor Odhiambo, Professor Okello, Mrs. Oriero, and Mr. Wafula), ASAL Development Programme, Laikipia District (Mrs. Phoebe Kipng'ok and Mr. Theo Hendriksen), Laikipia Research Programme (Mr. B. Kiteme and Mr. J. Mathuva), Mpala Research Centre (Dr. Nick Georgiadis), and numerous other Kenyan organizations provided institutional support. Ekero Jiko Sales and Mr. Mohammed Olunga conducted the cookstove workshops at Mpala and taught me about Kenyan stoves.

Financial support for my research was provided by the Summit and Compton Foundations, the Social Science Research Council International Predissertation

Fellowship Program, Center of International Studies (through a grant from MacArthur Foundation), Council on Regional Studies, the Woodrow Wilson Foundation, and ASAL Development Programme, Laikipia District.

This dissertation is the last stage of my lengthy formal education. My parents, Zari Ghasemian and Mohammad Ezzati, were always ready to give up everything for the sake of our education, and eventually moved to a far land to provide this opportunity. I could always count on my brother Saied, especially when things seemed uncertain or overwhelming. My wife, Riki Eggert, deserves my deepest gratitude. Her understanding, intelligence, and wonderful sense of humor have made my time in Princeton a very pleasant experience. Looking back, the best thing about coming to Princeton has been our relationship.

Chapter 1 Introduction [1]

Acute respiratory infections (ARI) and chronic respiratory diseases lead the causes of global disease, and together account for more than 10% of global burden of disease and mortality, mostly in developing countries (1, 2, 3). In 1997 and 1998, the leading cause of mortality from all infectious diseases was acute lower respiratory infections (ALRI) with an estimated 3.7 and 3.5 million deaths worldwide for the two years respectively, mostly among infants and children (3, 4).

Exposure to indoor air pollution, especially to particulate matter, from the combustion of biofuels (wood, charcoal, agricultural residues, and dung) has been implicated as a causal agent of respiratory and eye diseases (including cataracts, blindness, and possibly conjunctivitis) in developing countries (5, 6, 7, 8, 9, 10, 11). This association, coupled with the fact that globally more than two billion people rely on biomass as the primary source of domestic energy, has put preventive measures to reduce exposure to indoor air pollution high on the agenda of development and public health organizations (1, 12, 13, 14).

For efficient and successful design and dissemination of preventive measures, the following fundamental questions must be answered:

---

[1] This research was approved by The Institutional Review Panel for Human Subjects of the University Research Board, Princeton University (Case #1890) and by the Government of Kenya, under the Office of the President Research Permit No. OP/13/001/25C 167.

1. What are the factors that determine human exposure and what are the relative contributions of each factor to personal exposure? These factors include energy technology (stove-fuel combination), housing characteristics such as the size and material of the house and the number of windows, and behavioral factors such as the amount of time spent indoors or near the cooking area.
2. What is the quantitative relationship between exposure to indoor air pollution and the incidence of disease (i.e. the exposure-response relationship)?[2]
3. Which of the determinants of human exposure will be influenced, and to what extent, through any given intervention strategy?
4. What are the costs and benefits as well as the institutional requirements – at the national, local, and household level – for the implementation of each intervention?

Epidemiological and physiological studies over the past two decades in urban areas of industrialized countries have resulted in significant progress in identifying and quantifying the health impacts of outdoor particulates (15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25). These results, however, are applicable to a small range of exposures, generally below 200 $\mu g.m^{-3}$, which are primarily of concern in industrialized countries (13).[3] There is little information on the shape of the exposure-response relationship at concentrations of hundreds to thousands of $\mu g.m^{-3}$ which are commonly observed in indoor environments of developing countries (14). This is a critical gap in our understanding of

[2] Note that quantifying the exposure-response relationship for indoor suspended particulate matter itself requires accurate measurement or estimation of personal exposure.

[3] The most recent US-EPA National Ambient Air Quality Standards, for example, required PM_{10} concentration (particles below the diameter of 10 μm) to have a 24-hour average below 150 $\mu g.m^{-3}$.

the role of exposure to particulate matter as a causal agent of ARI, and thus as a contributor to the global burden of disease, since approximately 80% of total global exposure to this pollutant occurs indoors in developing nations (26, 27).

Research on the health impacts of indoor air pollution in developing countries has been hindered by a lack of detailed data on both exposure and illness outcomes. In these settings, many epidemiological studies have used indirect and often inaccurate measures, such as fuel or housing type, as proxies for personal exposure in cross-sectional studies (see for example 28, 29, 30, 31, 32, 33) (for a discussion of this issue see 34). Given the nearly universal use of biomass fuels in rural areas, this indirect approach to exposure estimation clusters numerous people into a single exposure category. Recent findings on large variations in emissions from individual stove types (14, 35) and in exposure profiles within individual households (36, 37, 38), however, demonstrate that aggregate analysis and grouping of individuals artificially reduces the variability of the explanatory variable in the exposure-response relationship, and therefore the reliability of the estimation of its parameters. From a public health policy perspective, ignoring the variability of individual technologies and intra-household variation in exposure may dramatically change the relative importance of various strategies for reducing exposure to indoor air pollution.

Initial works on the benefits of improved stoves, as a means for reducing exposure to indoor air pollution, were also marked by a lack of detailed data on stove performance. Efficiencies and emissions, for example, were often measured in controlled environments

as the stoves were used by technical experts under conditions very dissimilar to those of end-users (39, 40). More recently, the attention of the research community has shifted from such ideal operating conditions to monitoring stove performance under actual conditions of use, taking into account the various social and physical factors that would limit the use of these stoves all together or result in "sub-optimal" use (41, 42). As a result of these studies the initially-perceived high level of benefits from improved stoves has been called into question (35, 43).

In this dissertation, I provide full analysis of the first three of the questions posed above using original field data from Central Kenya. I also briefly discuss the important issues in successful transfer of household level technology. Over 3 years of field research (1996 – 1999) at Mpala Ranch and Mpala Research Centre in Central Kenya, I have developed a unique data set in which we simultaneously monitored both exposure to indoor air pollution and the health status of all the individuals in the study group.

I first integrate quantitative and qualitative data on individual time-activity budgets, household demographic characteristics, and continuous real-time monitoring of indoor air pollution to construct personal *profiles of exposure* to suspended particulate matter resulting from biofuel combustion. Further, the measurement of both exposure and health outcome at the level of individuals allows quantifying the exposure-response relationship for indoor particulate matter along a continuum of exposure levels.

In the analysis of interventions, I focus on an array of stove-fuel combinations used extensively by Kenyan households and analyze their performance under the actual conditions of use. With continuous data on instantaneous pollution levels, I go beyond the single measure of average daily pollution and develop exposure profiles using other descriptive statistics of emission data which better characterize human exposure. Finally, I discuss the question of technology transfer at the household level using current literature and qualitative observations from my field research.

The organization of this dissertation is as follows: In Chapter 2, I briefly discuss household energy use in developing countries, and the physiology and epidemiology of respiratory infections. Chapter 3 provides historical, geographical, social, and economic information about Kenya and Laikipia District, where my field research took place, with emphasis on public health characteristics. In Chapter 4, I describe Mpala Ranch, the site of this research, focusing on daily activities of residents. Chapter 5 explains the type of data collected and data collection strategies and protocols over the period of field research. Chapters 6, 7, and 8 provide details of data analysis and results. Chapter 6 focuses on the construction of personal exposure from pollution and time-activity budget data. In Chapter 7, I derive the exposure-response relationship for indoor particulate matter from exposure and health data. Chapter 8 compares the performance of an array of stove-fuel combinations in reducing exposure to indoor air pollution. Finally, in Chapter 9, I discuss the important issues in assessing household level technologies, such as improved cookstoves. Chapter 10 presents the conclusions, policy implications, and directions for future research.

Chapter 2 Household Energy, Indoor Air Pollution, and Acute Respiratory Infections: Global Picture and Current Research

2.1 Household Energy in Developing Countries

Globally, more than two billion people, almost all in developing countries, rely on biomass – wood, crop residues, dung, and charcoal – as their primary source of domestic energy (Figure 2.1) (44, 45, 46).

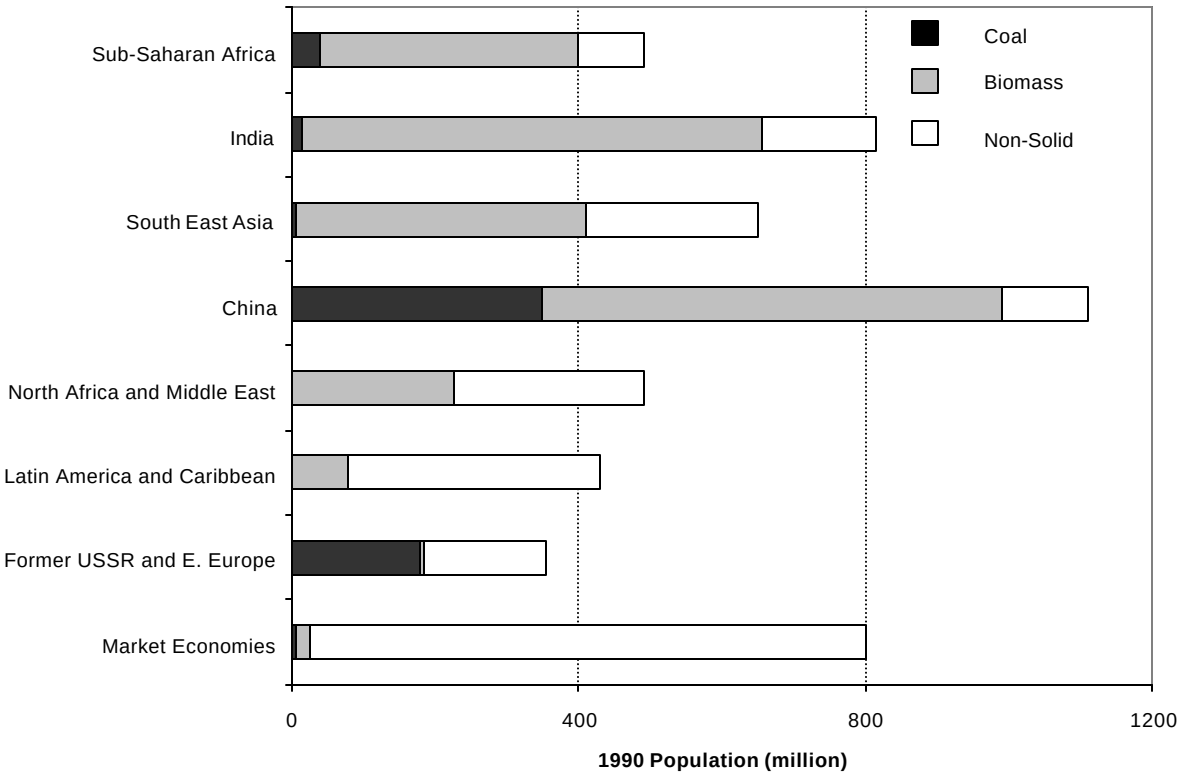


Figure 2.1: Sources of domestic energy in different geographical regions (source: 13)

Biomass accounts for more than one half of total national energy consumption and as much as 95% of household energy in some developing countries, especially in poorer regions of Sub-Saharan Africa and Asia (44, 46, 47, 48).

2.2 Biomass Combustion and Indoor Air Pollution

Combustion of biomass (and also coal which is common in China and the former Soviet Union and Eastern Europe) results in high concentrations of particulate matter and other pollutants. The average concentration of PM_{10} emissions (particles below 10 microns in diameter) from a wood-burning stove, for example, is normally on the order of thousands of $\mu g.m^{-3}$. For comparison, the most recent US-EPA standard for PM_{10} was 150 $\mu g.m^{-3}$.[4]

Since much of cooking in developing countries takes place indoors with limited ventilation (49), household members who cook or are present during cooking, are exposed to a large fraction of emissions from biomass stoves. Smith (27) estimates that the fraction of emissions from an indoor biomass stove that is inhaled is approximately 10,000 times that of a coal power plant in an industrialized country. As a result of such patterns of exposure, indoor air pollution from biomass consumption in developing countries is by far the most significant source of exposure to particulate matter in the world, as shown in the break-down of global exposure in Figure 2.2.

[4] In 1997 the US-EPA standards were redesigned in terms of the concentration of $PM_{2.5}$ (particles below 2.5 microns in diameter) due to increasing physiological evidence on the role of the smaller particles in health risks.

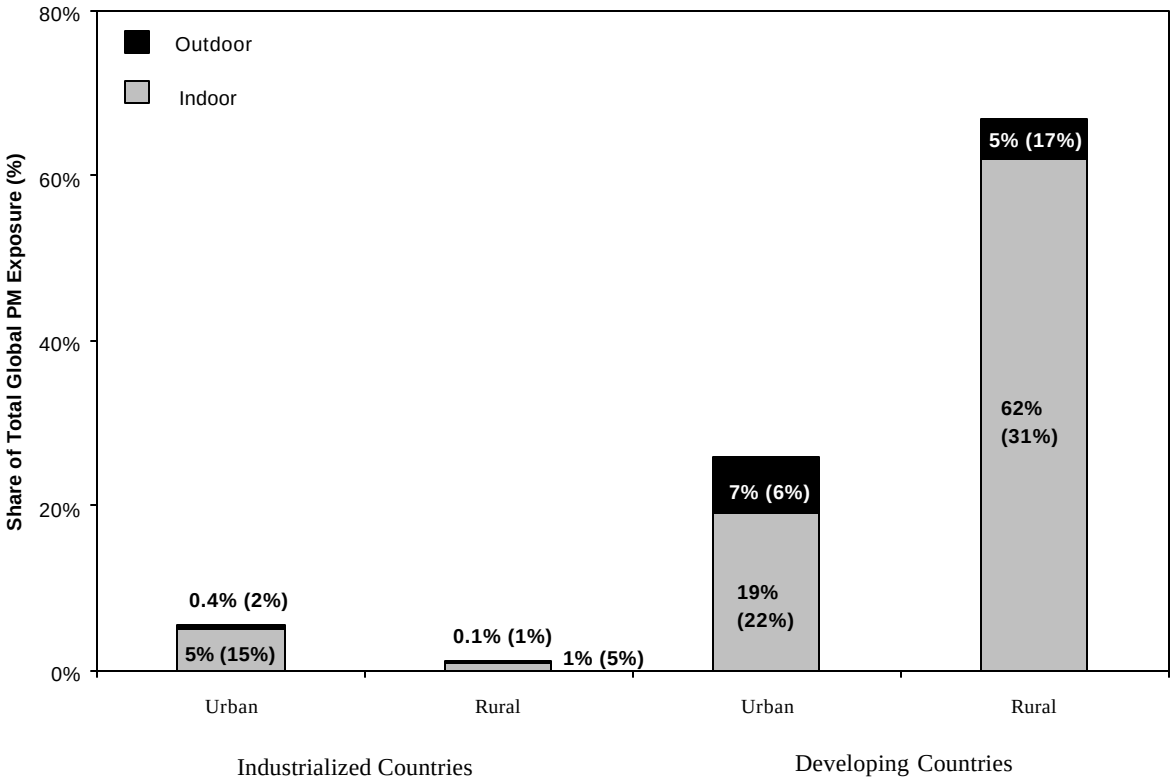


Figure 2.2: Break-down of global exposure to particulate matter by type of environment (also expressed by the first number reported for each group) (source: 26, 27, 50). Numbers in brackets indicate the share of global person-hours in each environment. The ratio of the share of global exposure to global person-hours for each environment is a relative index of pollution level (*population exposure = pollution* $\times$ *time* $\times$ *population*).

2.3 The Health Impacts of Exposure to Indoor Air Pollution

2.3.1 Physiology of Impacts on the Respiratory System

Much of the research on the non-carcinogenic impacts of air pollution has focused on particulate matter, which I discuss briefly in this section. Advances in measurement and monitoring technology in the past decade have especially led to rapid advances in research on the physiological processes underlying the health impacts of exposure to airborne particulates.

Particles deposit in the airways through three physical mechanisms: inertial impaction, gravitational sedimentation, and Brownian diffusion with larger particles removed in the upper airways (51). Total and regional dispersion and deposition in the specific regions of the airways are influenced by changes in the respiratory flow rates, respiratory frequency, and tidal volume (51). Until recently, particles between 0.1 and 10 microns in diameter were the subject of research on health impacts and regulation. In the past decade however, as a result of mathematical modeling and animal and human studies, researchers have found that smaller particles – those below 2.5 microns in diameter denotes as $PM_{2.5}$ – can travel farther in the airways and have more severe health impacts (51, 52).

It is not fully known whether the impact of particles is due to the total number of particles deposited in the airways or their total mass[5] but new advances in technology for particle count over a range of particle sizes may result in rapid advances in answering this question. Further, except for specific toxants, the role played by the chemical composition of particles versus their mere physical deposition is not fully understood.[6] Overall a combination of the following mechanisms are believed to be the cause of the health risks associated with suspended particulate matter: increased airways permeability,

[5] In a small range of particle sizes, particle mass is approximately proportional to particle number. Therefore, the two are interchangeable with close approximation.

[6] Unlike coal and other commercial fuels, biofuels contain fewer intrinsic contaminants such as sulfur and trace metals. Further, over time many societies are likely to have developed preferences for wood species that minimize pollution (53). Therefore, in biomass burning homes, particulate matter is a dominant source of health risks.

impaired host defenses, alveolar inflammation, exacerbation of chronic lung disease, and specific toxicities (51).

Acute respiratory infections (ARI), the central focus of this research, are the most common response from deposition of particles in the airways. Acute respiratory infections are divided into acute lower respiratory infections (ALRI) and acute upper respiratory infections (AURI). In its purest form, the division is based on the location of infection in the respiratory tract with the lower infections affecting the lungs, the bronchi, the trachea, and the larynx and the upper infections affecting the pharynx, the tonsilar glands, the eustachian tube, the nasal cavities, and the sinuses (see Figure 2.3) (54). Many infections however affect multiple parts of the respiratory tract especially where the affected areas are smaller. Further, infections of the bronchi (bronchitis) and of the lungs (pneumonia) are often considerably more severe than those in other parts of the respiratory tract and have more specific symptoms. For this reason, many public health and medical protocols use ALRI to refer to bronchitis, pneumonia, and broncho-pneumonia and combine the infections of the sections above and including the trachea into the category of AURI (55). I use this latter classification throughout this work.

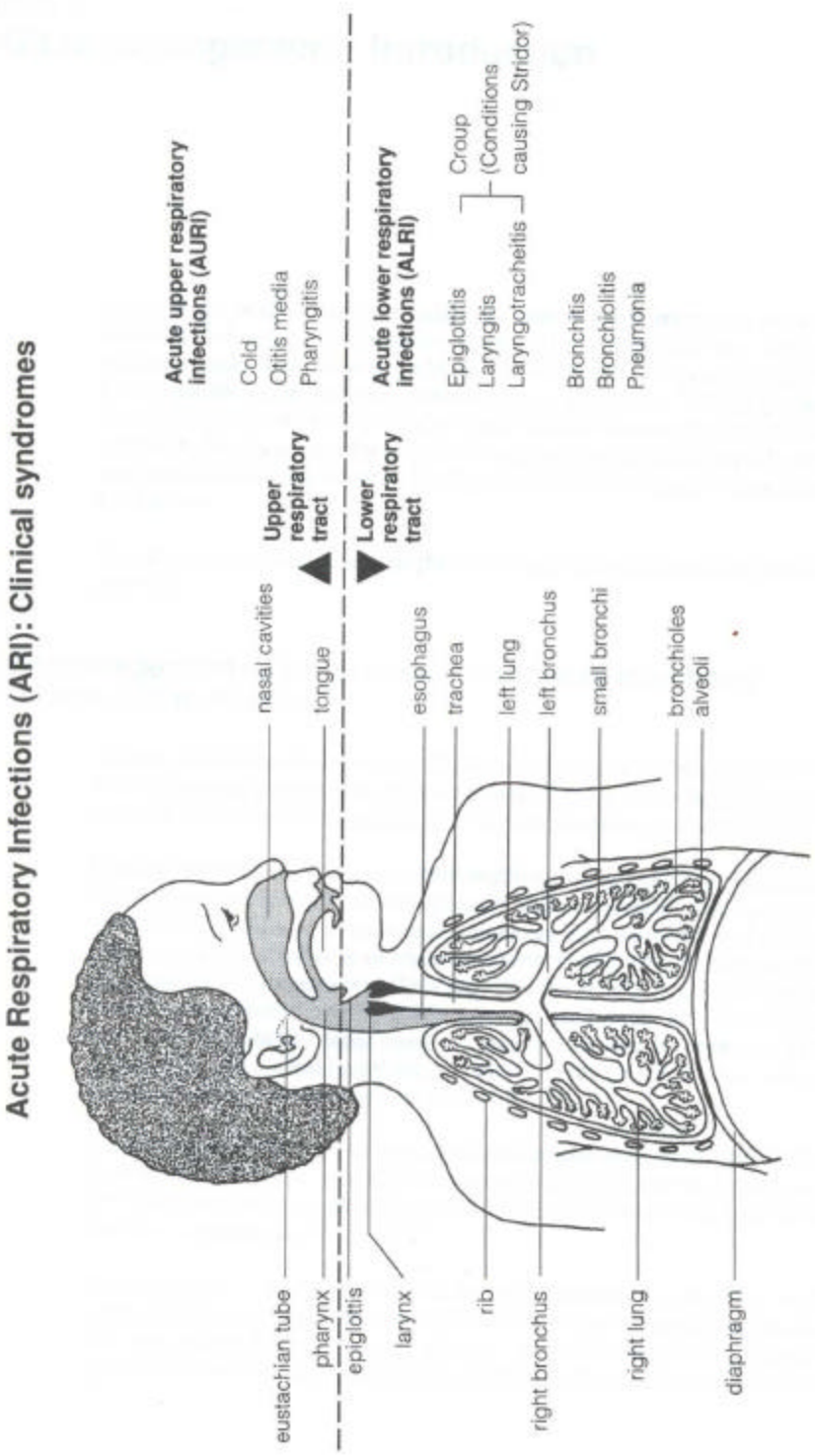


Figure 2.3: The respiratory tract. In ARI classification the infections of middle section of the respiratory tract are grouped together with the acute upper respiratory infections (AURI). ALRI refers to the infections of the bronchi and lungs (source: 54).

2.3.2 Research on Respiratory Diseases in Developing Countries: History

Respiratory diseases, and in particular pneumonia have consistently been among the most prevalent diseases of developing countries (see Chapter 3 for examples from Kenya). At the same time, with the exception of tuberculosis, they have received mixed attention in this setting. On the one hand, as early as the turn of the century, detailed research on the prevalence, causation, and management of pneumonia and other respiratory diseases was conducted in developing countries (see for example 56, 57). On the other, in many studies of health and development or tropical health, respiratory diseases were hardly mentioned or were not discussed in length (see for example 58, 59, 60, 61, 62, 63, 64, 65, 66).

Manderson also notices this systematic lack of attention for respiratory and diarrheal diseases in colonial Malay which she attributes to the "metaphoric weight" of other diseases, especially those of an epidemic nature (67). An equally important factor for this lack of attention may be traced to the evolution of medical sciences in the late part of the 19th century and much of the 20th century. The search for disease vectors and parasites, and for curative approaches that would eliminate them, dominated biomedical sciences in this period. This rise of "germ theory" in medicine, and in particular tropical medicine – which took place in a geographical context that was perceived to be ecologically and socially suitable for the spread of germs – shifted the attention of health authorities to those diseases that could be dealt with using modern biomedical tools. This dominance was intensified by the fact that colonial tropical medicine had a strong presence and contribution from military doctors whose biomedical approaches had achieved a great

deal of success in combating disease among European troops overseas in the 19th and 20th centuries (68, 69). In this intellectual and professional context of tropical health, "neither tuberculosis nor pneumonia appeared to 'yield' to [the dominant] methods of control …" (67).

This trend in coupling disease with germs, especially in tropical settings, was also the likely reason that even when respiratory infections received attention in the medical community, no reference to the role of air pollution in their incidence was made. In colonial Malay, where the medical services were "rather more successful for curative than preventive purposes," it was believed that "there was likely to be little change [in tuberculosis or pneumonia] under existing social and economic circumstances (67). But in Malay, as in other places, this was almost exclusively associated with overcrowding of houses and other factors that would facilitate the transmission of germs, rather than with air pollution. In the chapter of his book *Africa Emergent*, titled "*The Roots of Backwardness*", W. M. Macmillan cites the 1928 *Annual Medical Report* of Kenya:

> Pneumonia, broncho-pneumonia, and tuberculosis take a large toll of life. The circumstances of the people are such that they live under conditions which are admirably suitable for the existence and spread of the causal agents of disease or of their animal hosts. Even where huts and villages are not overcrowded with humans, they are always overcrowded with the causative organisms of disease or the carriers of these organisms, so that escape from infection is for the great majority of people impossible (70).

Other accounts of the "native huts" by health personnel also included indications of its crowdedness with people or objects, and its various smells (71) but not of its smokiness. Similarly, *crowded* urban conditions were blamed as "breeding grounds for respiratory and enteric diseases" (72). Even when the relationship between air pollution and

respiratory health was discussed in the context of occupational health (73), it was ignored in residential settings and cooking activities, a trend that continued until recent decades. In fact, as recently as the 1980's and 1990's, epidemiological studies, health care manuals, and health reports focused on the biological mechanisms of infection and biomedical management of respiratory infections, with some consideration of the role of temperature and crowding but little mention of the role of indoor air pollution (see for example 54, 74, 75, 76, 77, 78, 79, 80, 81).

2.3.3 Research on Respiratory Diseases: Current Research

In industrialized countries, epidemiological studies using both time-series and cross-sectional data, have also found evidence of increased incidence of acute and chronic respiratory diseases, asthma, and heart disease associated with air pollution, especially particulate matter pollution (16, 17, 19, 20, 21, 22, 23, 82) (for a summary of research on particle air pollution and health see 24). This research has further quantified the relationship between exposure to particulate matter and incidence of respiratory and cardiovascular symptom, but in the range of concentrations observed in this setting, below 200 $\mu g.m^{-3}$.

Research on the relationship between indoor air pollution and respiratory infections in developing countries began in the 1960's and 1970's in India, Nigeria, and Papua New Guinea (83, 84, 85, 86, 87, 88). In the 1980's, many research projects studied the association (mostly in cross-sectional studies) and by the early 1990's, the topic was on the agenda of research and policy communities (1, 12, 27, 49, 50, 89).

In developing countries, numerous epidemiological studies have established a causal link between exposure to indoor air pollution and a set of illnesses including acute respiratory infection (ARI), chronic respiratory ailments and in particular chronic obstructive pulmonary disease (including chronic bronchitis), lung cancer, eye diseases (including cataracts, blindness, and possibly conjunctivitis), and perinatal conditions (in particular low birth weight) (2, 5, 6, 7, 9, 10, 12, 27, 31, 90, 91, 92, 93, 94, 95, 96) (for a comprehensive review see 11). But all of these studies have used indirect measures of exposure – such as fuel type, housing characteristics, or time spent near fire – in case-control cross-sectional comparisons and little is known about the details of the quantitative relationship between exposure and health risks. [7]

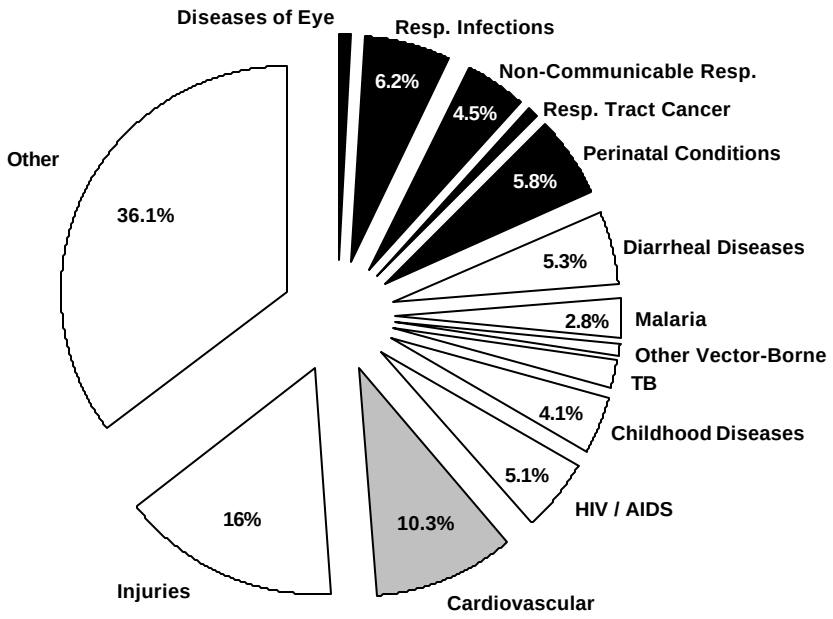
2.3.4 Contributions to the Global Burden of Disease

Figure 2.4a and Figure 2.4b show a break-down of the global burden of disease as measured by mortality and disability-adjusted-life-years (DALY)[8]. As seen in these figures, 15% of the global disease and mortality (without including cardiovascular diseases which have been related to air pollution in industrialized country studies only) are diseases whose incidence has been *partially* attributed to the exposure to indoor air

[7] Some studies have found no or inconclusive evidence of a relationship between sources of exposure to indoor air pollution and respiratory infections (see for example 28, 30, 32, 33, 87). As I will discuss in detail in Chapter 6, this is because indirect measures of human exposure such as fuel or housing can bias the results of risk assessment.

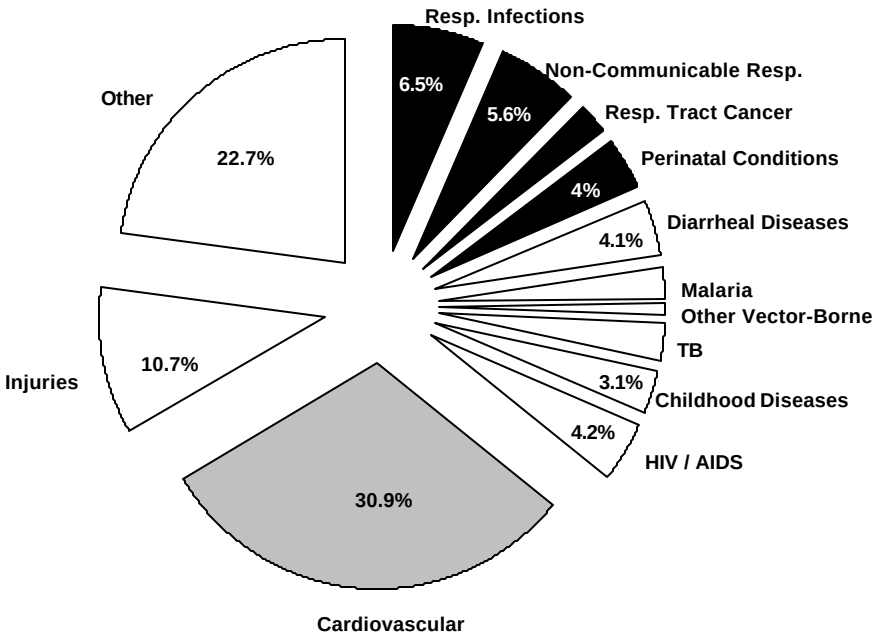
[8] Disability-adjusted-life-year (DALY) is a method for quantifying disease that in addition to incidence, considers the severity of disease (including fatality), its duration, and the age of the person at the time of disease (97).

pollution from biofuel combustion.[9] With the disease classification of the World Health Organization, one group of these diseases, acute lower respiratory infections (ALRI), is the leading cause of burden of disease in the world with 6% of total DALYs lost in 1998 (followed by perinatal conditions at 5.8% and diarrheal diseases at 5.3%) (3). Further in 1997 and 1998, the leading cause of worldwide mortality from infectious diseases was acute lower respiratory infections (ALRI) with an estimated 3.7 and 3.5 million deaths respectively, mostly among infants and children (3, 4).



(a)

[9] This does not mean that this share of global disease is "attributable" to exposure to indoor air pollution. A complete discussion of attributable risk is provided in (98). For indoor air pollution, in the specific case of India, (2) estimates that 410,000 – 570,000 of 9.5 million annual national deaths are caused prematurely because of exposure to indoor air pollution, mostly among infants and children.



(b)

Figure 2.4: The share of global disease partially associated with exposure to indoor air pollution in 1998 (source: 3). (a) Global burden of disease in units of disability-adjusted life years (DALY). Total number of DALYs lost globally in 1998 were 1.38 billion. (b) Mortality. Total number of global deaths in 1998 were 53.9 million. Non-communicable respiratory diseases also include chronic obstructive pulmonary disease (COPD) and asthma. Cardiovascular diseases have been linked to air pollution only in industrialized country studies and the mechanism of impact may be through modified lung functions.

Chapter 3 Kenya and Laikipia

Kenya is located on the east coast of Africa (between 34° and 41° east), on the equator (between 3° north and 5° south), and is a part of the Greater Horn of Africa (Figure 3.1). Kenya covers an area of 582,650 km^2 with a diverse landscape including deserts and plains, the Rift Valley, the central highlands and Mount Kenya, and tropical forests. Its climate varies from tropical along the coast to tropical highland or semi-arid and arid in the interior.

Figure 3.1: Kenya and the surrounding region (source: 99)

3.1 A Brief History of Colonial and Post-Independence Kenya

As a part of the "scramble for Africa" following the Berlin Conference of 1884-85, the formal colonial period in Kenya began with the granting of a royal charter to the Imperial British East Africa Company (IBEAC) in 1888 and the formation of the British East

Africa Protectorate in 1895. Kenya, along with Southern Rhodesia (Zimbabwe) and South Africa, had the largest concentration of European settlers in colonial Africa. As a result, changing land tenure and resettlement schemes, designed to provide land and labor for settlers, were a dominant feature of colonial Kenya. The effects of forced migration and resettlement continue to affect the politics and economics of Kenya well after independence, with particular impacts on Laikipia, the site of this research (100, 101, 102, 103, 104, 105, 106).

Following years of struggle and resistance aimed at gaining access to land and political participation for the African majority, on June 1, 1963 Jomo Kenyatta became the first African prime minister of Kenya. Kenya became an independent state on December 12, 1963. A year later Kenya became a republic with increased concentration of political power in the central government of President Kenyatta. Daniel Arap Moi became the second president of Kenya after President Kenyatta's death in 1978 and was re-elected in the country's first and second multiparty elections in 1992 and 1997.

Kenya has enjoyed political and civil stability of a degree rare in Sub-Saharan Africa. It has nonetheless faced extensive political debate and at times turmoil with divisions along ideological, geographical, ethnic, and economic lines, motivated by access to resources, in particular land, as well as political power. These divisions which began in President Kenyatta's era have continued to be a dominant feature of President Moi's government (105).

3.2 The Economy of Kenya

In the first two decades after independence, Kenya was regarded as one of the few countries in Sub-Saharan Africa headed for economic success including sustained growth. Kenya was praised for policies that reduced price distortion especially for agricultural commodities. National infrastructure was extensive and expanding, and growing tourism and commodity export promised economic growth which could ultimately benefit even the poorest sectors of the society. Underestimated in this picture was the fact that reduced price distortion was not a central policy of the national government, but a side-effect of an alignment of interests between the political elite in President Kenyatta's government and powerful (European and Kikuyu) agricultural groups (107, 108, 109). Like many other African countries, the first setback to Kenya's economy was from the oil shocks on the 1970's and fluctuations in international coffee prices.

Since the election of President Daniel Arap Moi in 1978, new economic and political alignments have intensified political and social tension in Kenya. Increased uncertainty and government corruption, coupled with attenuated civil strife in other parts of Africa, have directed investment and tourism away from Kenya. As a result, today the fundamental barriers to development in Kenya include not only a poorly maintained national infrastructure, increased demand for land, and a fragile ecology, but also the enormous concentration of political power and economic resources in the central government and away from other important social and economic institutions (110). Table 3.1 provides some of the current economic indicators for Kenya.

Table 3.1: Basic Economic Indicators for Kenya (source: 111, 112, 113, 114, 115). The data are from the early to the late 1990's.

GDP	$ 9.5 Billion
GNP per capita (1998) [a]	$330 – $350
GDP per capita (purchasing power parity)	$1,550
Annual GDP growth rate (1988 – 1998)	2.3%
GNP per capita growth rate (1988 – 1998) [a]	-0.2%
Inflation (consumer prices, 1998)	10.7%
% of population below national poverty line	42%
Household income/consumption of the lowest 10% group	1.2%
Household income/consumption of the highest 10% group	47.7%
Structure of economy (% of GDP) (1998)	Agriculture: 26.1%; industry: 16.2%; services: 57.7%
Structure of economy (% of labor force)	Agriculture: 75% – 80%; non-agriculture: 20% – 25%
Unemployment rate	50%

[a] For comparison the GNP per capita of Sub-Saharan Africa as a whole is $480; the rate of growth of GNP per capita of Sub-Saharan Africa as a whole in 1998 was 0.2%.

3.3 The Population of Kenya

Kenya has an estimated population of 29 million with demographic, social, and health characteristics provided by Table 3.2 and Table 3.3. The indicators in these tables show that:

- Child malnutrition and infant mortality in Kenya remain relatively high, although lower than the (45% and 90 per 1000 respectively) average for low-human-development-index nations, which include most countries in Sub-Saharan Africa. Since Kenya has a high population growth rate, even compared to other African countries, and a young population (approximately 50% below the age of 15), child health will remain an important public health issue in Kenya.

Table 3.2: Demographic Statistics of Kenya (source: 111, 112, 113, 114, 115, 116). The data are from the early to the late 1990's.

Rural population (% total)	70%
Population growth rate	2.5% – 2.8%
Total fertility rate	4.4 – 4.9
Age Structure (*% distribution*)	
Below 5 years	16%
5 – 14 years	28%
15 – 64 years	54%
65 years and over	3%
Sex Ratio	
At birth	1.03 males / female
Under 15 years	1.02 males / female
15 – 64 years	1.00 male / female
65 years and over	0.77 males / female
Ethnic Groups (*% distribution*)	
Kikuyu	22%
Lyhya	14%
Luo	13%
Kalenjin	12%
Kamba	11%
Other African	27%
Non-African (Asian, European, and Arab)	1%

- A large proportion of Kenya's population lives in rural areas and, as a result, has limited access to the scarce health facilities and resources.
- With a large share of household energy from biomass, especially in rural areas where biofuels are the exclusive source of energy, indoor air pollution is an important risk factor.

Table 3.3: Basic Social and Health Indicators for Kenya (source: 48, 111, 112, 113, 114, 115, 116, 117). The data are from the early to the late 1990's.

Health	
Life expectancy at birth (total population)	54 – 57
Infant mortality rate	61 – 75 deaths / 1000 live births [a]
Under 5 mortality rate	90 – 120 deaths / 1000 live births
Maternal mortality rate	590 – 650 deaths / 100,000 live births
Infants with low birth weight	16%
Child malnutrition (% children under 5) [b]	23%
Access to safe drinking water (% population)	Total: 44% – 53%; urban: 67% – 87%; rural: 30% – 49%
Access to adequate sanitation (% population)	Total: 77% – 85%; urban: 69% – 96%; rural: 81%
Physicians per 100,000 people	15
Nurses per 100,000 people	23
Total national health expenditure (% GNP)	5%
Total government health expenditure (% GNP)	1.5% – 2.7%
% national health expenditure devoted to local health care	21%
Education	
Adult (15+) literacy rate	Total: 78%; male: 86%; female: 69%
Energy	
Electricity consumption per capita (kw-h) (1995)	109 (world average: 1566)
Biomass fuel consumption (% total) (1995)	77% (world average: 6.8%)
% household energy from fuelwood and charcoal (1990)	79%

[a] Where ranges are given, they reflect differences in the estimates from different sources of data.

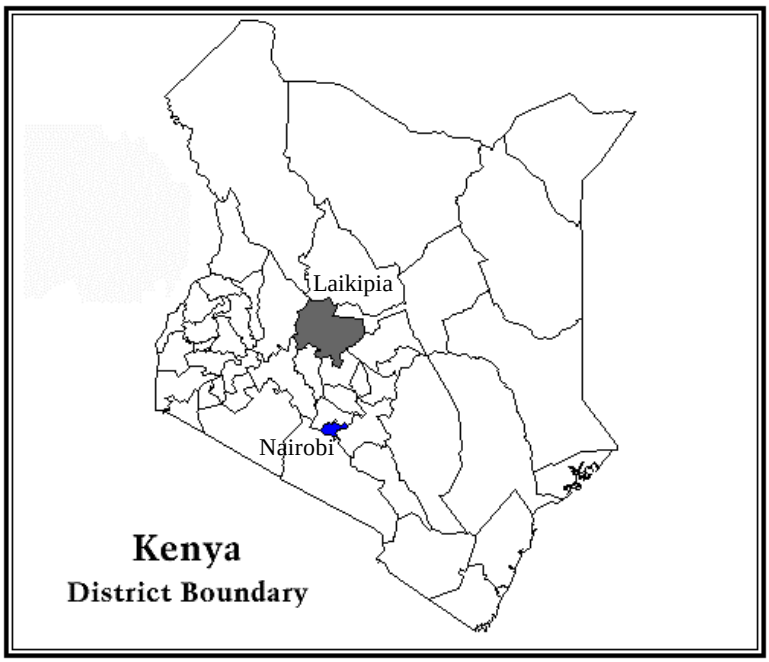
[b] Child malnutrition shows the percentage of children under five whose weight for age is more than two standard deviations below the median for the international reference population aged 0 – 59 months. The reference population, adopted by the WHO in 1983, is based on children from the United States, who are assumed to be well nourished (118).

3.4 Laikipia

3.4.1 Geography and climate

Laikipia is one of the 14 districts in the Rift Valley Province of Kenya, covering an area of 9,700 km^2 (Figure 3.2a) and comprising five divisions: Central, Lamuria, Mukogodo, Ngarua Rumuruti. Laikipia consists mainly of a level plateau bounded by the Rift Valley to the west and the Aberdare Mountains and Mount Kenya to the south. The altitude of

the district varies between 1800 meters in the north and 2100 meters in the south. Economic activities and human settlement in Laikipia are shaped by an interaction of geographical attributes, including topography, rainfall, and access to water, and historical events.



(a)

Figure 3.2: (a) Laikipia District (source: 99). (b) Land-use and population density (source: 119). (c) Precipitation and agro-ecological zones (source: 120).

The climate of the Laikipia is mainly shaped by the monsoons but is also affected by the rain shadow of Mount Kenya. Because of its elevation, the ecosystem is characterized by a mixture of cool and dry conditions. Rainfall is erratic, often obscuring the signature of the two typical monsoon generated rainy seasons. The dry season which begins in December and runs through February, is characterized by hot dry winds brought from Arabia by the north-east monsoon.

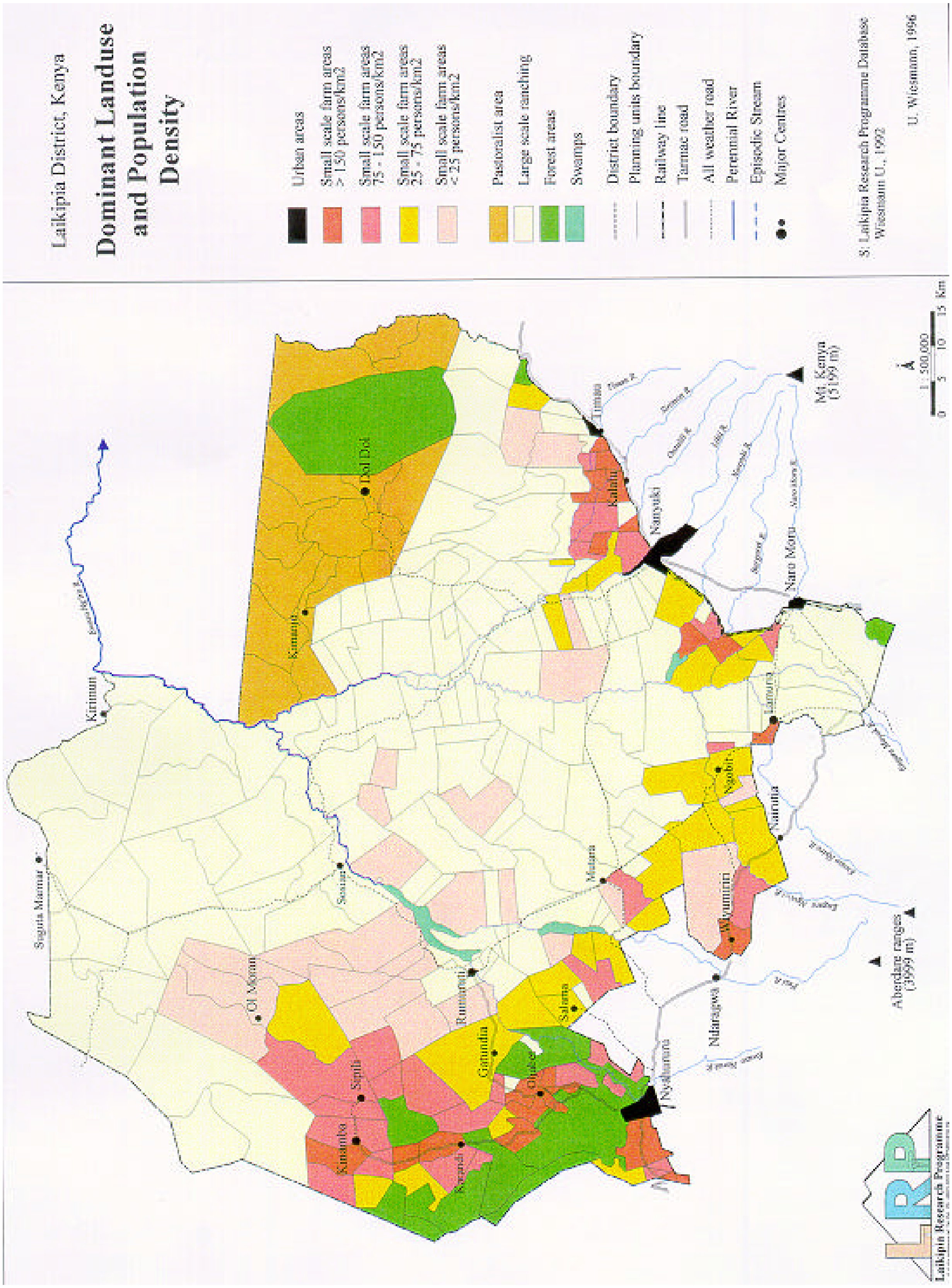

Laikipia District, Kenya
Dominant Landuse and Population Density
Urban areas
Small scale farm areas > 150 persons/km2
Small scale farm areas 75 - 150 persons/km2
Small scale farm areas 25 - 75 persons/km2
Small scale farm areas < 25 persons/km2
Pastoralist area
Large scale ranching
Forest areas
Swamps
District boundary
Planning units boundary
Railway line
Tarmac road
All weather road
Perennial River
Episodic Stream
Major Centres
S: Laikipia Research Programme Database Wiesmann U., 1992
U. Wiesmann, 1996
Mt. Kenya (5199 m)
Aberdare ranges (3999 m)
1 : 500,000
Laikipia Research Programme

Figure 3.2 (b)

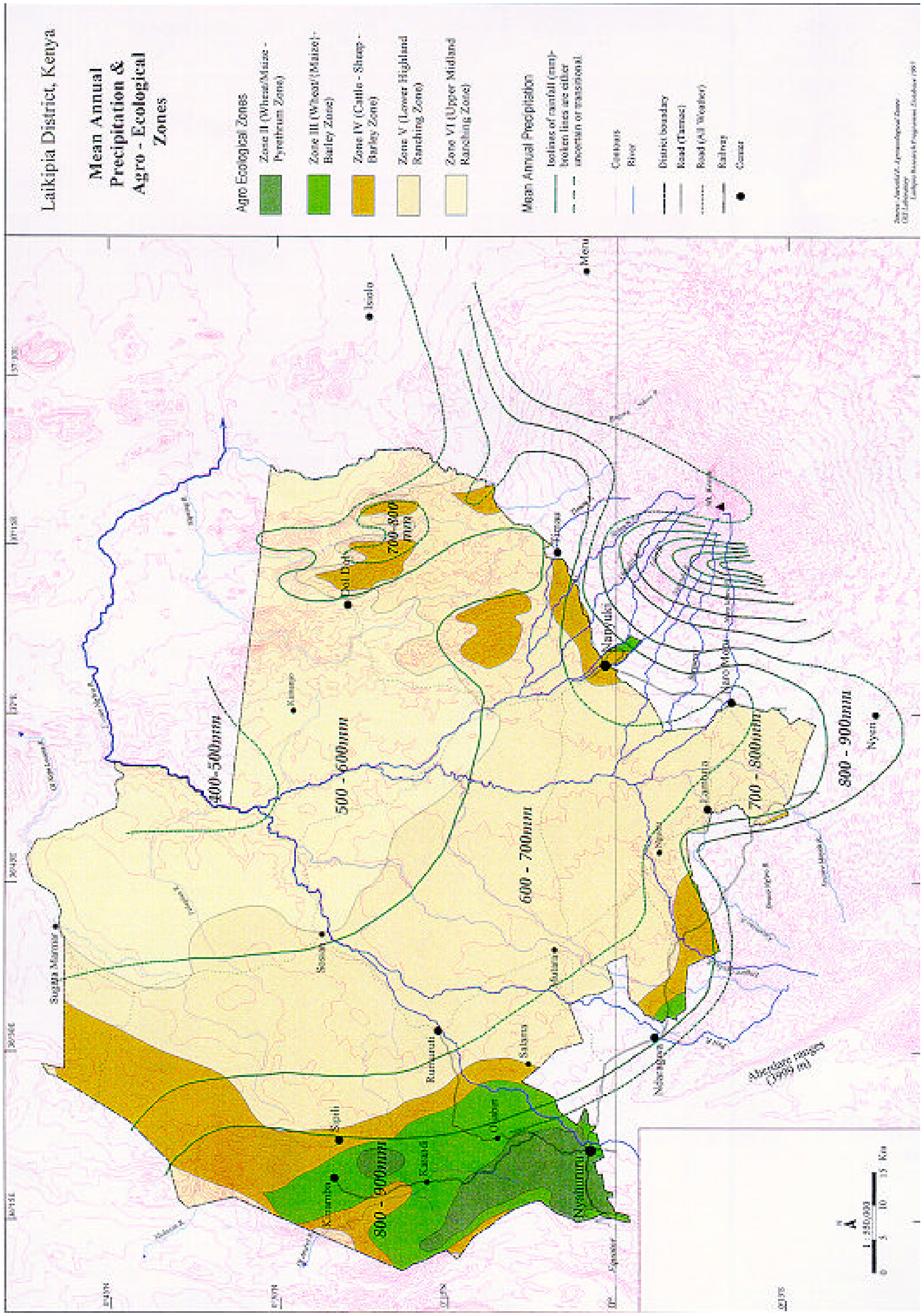

Laikipia District, Kenya
Mean Annual Precipitation & Agro - Ecological Zones
Agro Ecological Zones
Zone II (Wheat/Maize - Pyrethrum Zone)
Zone III (Wheat/(Maize) - Barley Zone)
Zone IV (Cattle - Sheep - Barley Zone)
Zone V (Lower Highland Ranching Zone)
Zone VI (Upper Midland Ranching Zone)
Mean Annual Precipitation
District boundary
Road (Tarmac)
Road (All Weather)
Railway
400-500mm
500 - 600mm
600 - 700mm
700 - 800mm
800 - 900mm
Isiolo
Meru
Dol Dol
Nanyuki
Nyeri
Sosian
Rumuruti
Nyahururu
Kinamba
Aberdare ranges (3999 m)

Figure 3.2 (c)

In March the monsoon winds shift, bringing moisture from the Indian Ocean. At this time cooling occurs, but whether these first, or "long", rains actually fall by May on the central Laikipia plateau depends upon the strength of convective cloud formation which can be extremely localized. Dry continental winds from the west take over in June and dominate the weather until September, although sporadic "continental" rains can occur. From October to December the winds again shift bringing coastal moisture from the east which typically produce a second, although shorter, rainy season (Figure 3.3).

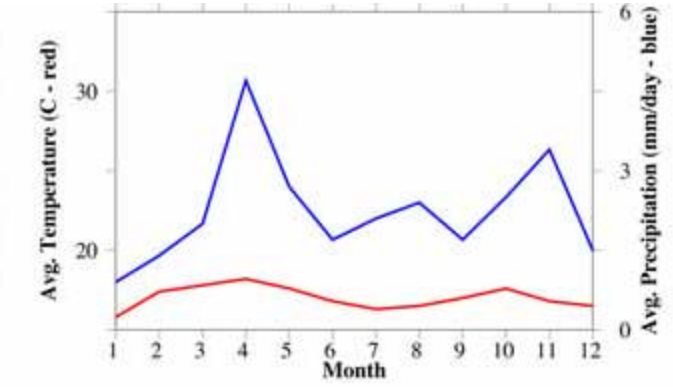


Figure 3.3: Seasonal distribution of rainfall and temperature in Laikipia District.

The slopes of Mount Kenya and the Aberdare Mountains, especially the southwestern part of the district, where total annual rainfall is approximately 900 mm and average monthly temperatures vary between 14.2°C and 17.3°C, are suitable for forestry and crop farming. The warmer and drier southeastern part and the level plateau between Mount Kenya and the Rift Valley, with approximate annual rainfall of 600 – 750 mm, are more suitable for livestock ranching, with fewer cropping activities.[10] Finally, the northeastern

[10] The level plateau of the district is drained by the tributaries of Ewaso Ngiro River which have their catchments in the slopes of the Aberdares and Mount Kenya. The rivers also determine human settlement as they are sources of water, both for human and livestock consumption and possible irrigation activities.

region of the district (Mukogodo Division) is dry and has the lowest rainfall and highest temperature (400 – 500 mm annual rainfall and 17.1°C – 23.2°C average monthly temperature). The high temperatures and dry conditions in this region prevent agricultural activities other than (nomadic) pastoralism (120, 121, 122) (Figure 3.2b and Figure 3.2c).

3.4.2 Population distribution and economy

The different agro-climatic regions of Laikipia are also marked by clear ethnic and economic divisions, owing to the colonial and post-independence land tenure policies. In 1904 much of Laikipia was designated as Maasai reserves as a part of schemes to provide land for white settlers and to reorganize the Maasai people from their traditional nomadic practices to more settled communities which could be controlled more readily. Between 1910 and 1913 most of Laikipia Reserve (North Reserve) was re-designated as "white highlands" and divided into large-scale ranches or farms owned by individuals, groups, or companies, forcing the Maasai to the northeastern and least hospitable part of the district (103, 106, 123).

After independence, as a part of land redistribution schemes, some of the foreign-owned farms, especially in the western, southern, and southeastern parts of Laikipia, were purchased by local people from neighboring districts, mainly Nyeri, Muranga and Eeru. Cooperatives and land buying companies have since subdivided many of the purchased farms and settled members on their plots (124). Currently, private ranches occupy almost 55% of the Laikipia District and the group ranches of Mukogodo Maasai cover 7%, with

the remainder of the district used as small scale farms, forest reserves, government land, and urban centers (125) (Figure 3.2b).

Today land tenure dynamics in Laikipia operate in a setting far from an economic market. European settlers consider Laikipia one of their strongholds and attempt to block purchase of land by Africans. This pattern is augmented by the fact that Laikipia, because of its privately owned game reserves, has one of the highest wildlife densities in Kenya. Blocking land division to preserve wildlife habitat blurs the distinction between conservation and ethnic segregation.

Finally, land redistribution in the western and southern parts of the district has created permanent and temporary immigration into these parts of the district by the new settlers, both with and without families, consisting mostly of the Kikuyu and Meru people of neighboring districts. This migration has brought many aspects of Kenya's national political tensions, which include divisions along ethnic lines, with specific emphasis on Kikuyu land-holding, into Laikipia. In this manner, Laikipia contains in its small area many of the social, economic, and ecological tensions of colonial and post-independence Kenya.

The population of Laikipia District was 65,500 in 1969 and 134,500 in 1979, representing an annual growth rate of 7.3%. The annual increase later fell to 4.5%, still considerably higher than the national average due to the migration of new land owners. In 1993 the total population of Laikipia was estimated at 253,700 (122). Rumuruti Division has the

largest population and Ngarua Division, with its high agricultural potential and small size of land-holding, the highest population density. Mukogodo and Central Divisions have the lowest density due to unfavorable climatic conditions and the presence of large ranches.

3.5 Public Health and Respiratory Infections in Kenya and Laikipia

I described the role of acute respiratory infections in the global burden of disease in the previous chapter. In Africa, acute lower respiratory infections account for 8.2% of mortality (HIV/AIDS 19%, malaria 10.7%, and diarrheal diseases 7.6%) and 7% of lost DALY's (HIV/AIDS 16.6%, malaria 10.6%, and diarrheal diseases 7.5%) (3).

Country level data on the causes of morbidity and mortality are often rare and unreliable due to uncertainty in recording and reporting protocols. But all existing evidence indicates that respiratory infections are an important source of disease in Kenya and Laikipia, and have been so during its recent history. Figure 3.4 shows the prevalence of some of the most common diseases in the last two decades of colonial Kenya as recorded by hospital records, illustrating the consistently important role of respiratory infections in colonial Kenya.[11]

[11] One may expect that in days when hospitals were less accessible, especially to the African population, only a fraction – and the most severe cases – of respiratory infections were reported, compared to infectious and parasitic diseases which are generally more severe and likely to have had higher relative reporting rates. In that case, there may be a downward bias in the estimates of the share of respiratory diseases.

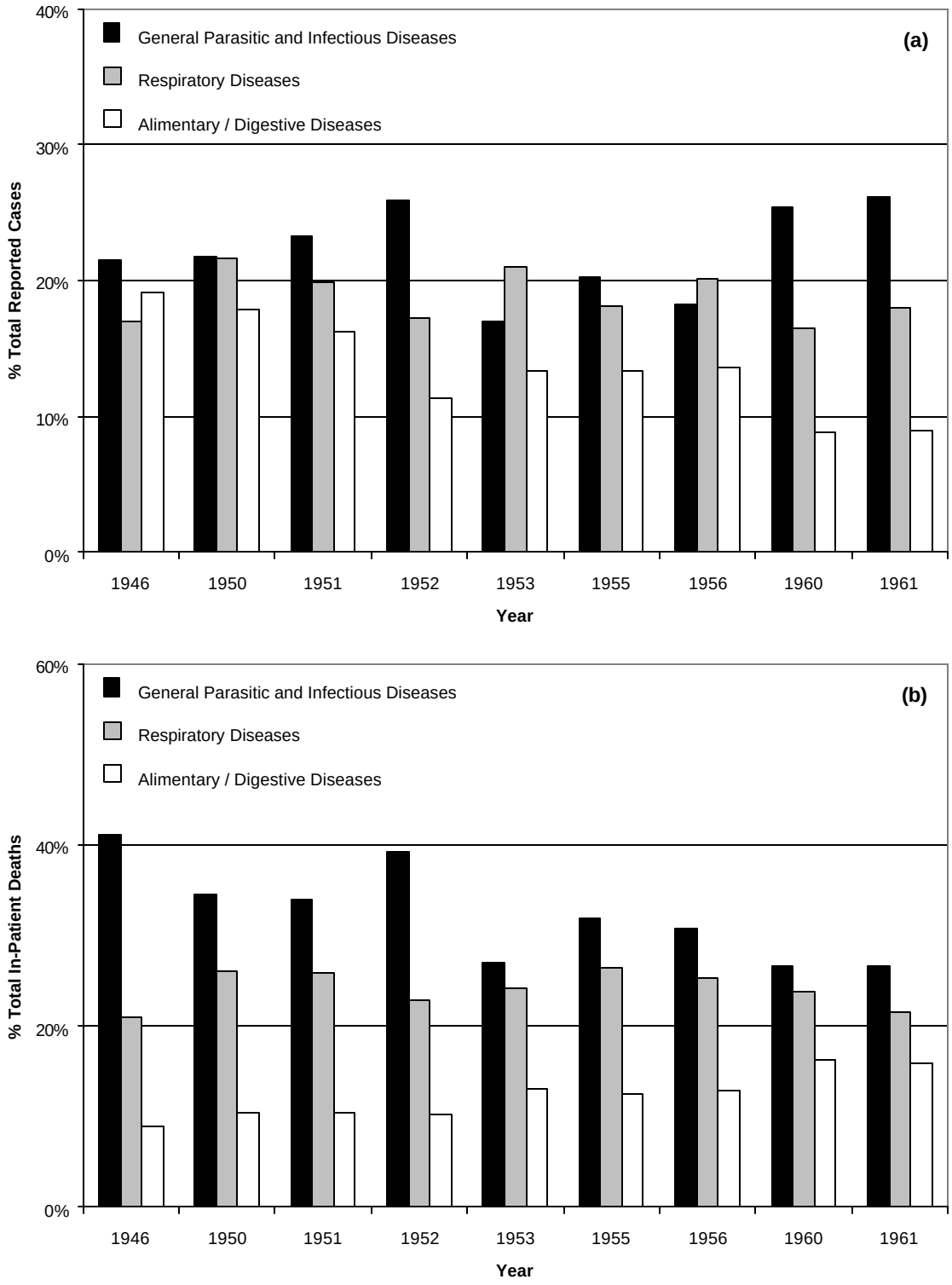


Figure 3.4: Common diseases of colonial Kenya. (a) Share of total number of cases treated in hospitals. The data are the share of total in-patient and out-patient cases in hospitals except 1960 and 1961 when data were available only on in-patient cases. (b) Share of in-patient mortality. Other common diseases of these years were skin diseases and injuries which in some years had more cases than alimentary / digestive diseases (source: 126).

Respiratory infections remained an important disease in Kenya over time. In 1968, hospital records of the causes of death for the estimated 9 million out-patients and 320,000 in-patients, show the following distribution for the five most common causes of death: diseases of respiratory system (30%), infectious and parasitic diseases (26%), diseases of the digestive system (14%), blood diseases (9%), accidents, poisoning, and violence (5%) (74).[12] In addition to being the leading national cause of mortality, respiratory diseases were also the first or second leading cause of mortality in all provinces. The contribution of respiratory diseases to morbidity in 1968 was similar. In out-patient attendances they ranked first with 25% of all cases, followed by infectious and parasitic diseases (21%), diseases of the digestive system (16%), and accidents, poisoning, and violence (9%). In out-patient admissions respiratory diseases ranked second with 17% of all cases, following infectious and parasitic diseases (25%), and followed by delivery, pregnancy, and puerperium (16%), accidents, poisoning, and violence (10%), and diseases of the digestive system (8%). Ranking of respiratory diseases in hospital admissions in different provinces was consistent with the national ranking (74).

A similar pattern existed in 1980. Acute respiratory infections and malaria led the number of cases treated in Kenyan hospitals with a share of 21% and 18% respectively.

[12] In non-hospital notification of death records the distribution of causes is as follows: diseases of respiratory system (20%), infectious and parasitic diseases (23%), diseases of the digestive system (9%), blood diseases (6%), accidents, poisoning, and violence (8%). The authors of this study also suspect a systematic under-reporting of deaths from diseases of respiratory system and other diseases that are more common in poorer households (74).

Infectious and parasitic diseases (20%) and respiratory diseases (18%) were the leading causes of death (66).

The role of respiratory infection in the burden of disease in Kenya is also confirmed in studies of specific regions or age groups. In a study of infant and child health in the Machakos District between 1975 and 1978 pneumonia and gastroenteritis were the leading causes of infant and child mortality each accounting for 20% of deaths (75). Respiratory diseases along with malaria and diarrhea led the out-patient morbidity in the health facilities of Turkana District (northern Kenya) in 1980-1981, the causes of morbidity in Samia (western Kenya) in 1984, and the cases reported to health facilities in Kibwezi Division (near Nairobi) in 1991 (66, 127, 128). Approximately 20% of children in households surveyed in 1993 and 1998 had had symptoms of respiratory infections in the two weeks preceding the survey (111, 112).

Finally respiratory diseases were consistently the leading disease among the cases reported to Laikipia District hospitals, clinics, and dispensaries between 1990 and 1999 (129).

Chapter 4 Research Location and Study Group

4.1 Mpala Ranch

My field research took place at Mpala Ranch, one of the private ranches (and game reserves) in the Central Division of Laikipia. Mpala Ranch (36° 50' E, 0° 20' N) is a 22,000-hectare (55,000-acre) privately owned ranch in the Central Division of Laikipia District located approximately 50 km northwest of the town of Nanyuki (Figure 3.2). Mpala is bounded on the east and north sides by the Ewaso Ngiro and Ewaso Narok rivers.

The northern two-thirds of Mpala Ranch consists of a dissected Archeas terrain covered with a thin layer of sandy red soil. The southwestern section of the ranch is characterized by a phonolite lava flow which is 100 – 200 m high. It is covered with a black clay vertisol with limited drainage and a brown calcareous soil (chestnut soil) on the higher elevations and steeper slopes. There are granitic inselbergs (called *kopjies*) scattered throughout the ranch (130).

The vegetation of Mpala Ranch is "characteristic of semi-arid African savannas, predominantly grassy savanna bushland, with patches of woodland and open grassland" with an estimated 800 plant species (130). The most common trees are species in the genera *Acacia* (*Mimosaceae*), *Euphorbia* (*Euphorbiaceae*), *Balanites* (*Balanitaceae*), and *Boscia* (*Capparaceae*). There are four broadly defined ecological zones in the Mpala

area: small acacia bushes dominate on the plateau, mixed vegetation and larger acacia trees below the plateau, an area of *euphorbia* in the northern part of the ranch, and riparian vegetation near the larger rivers, especially the Ewaso Ngiro (130, 131, 132).

There are more than 250 km of internal roads on Mpala Ranch and fifteen dams or water catchments to provide water for the more than 2,000 heads of cattle, camels, and sheep that are ranched on Mpala. Mpala is also a part of the larger Laikipia wildlife sanctuary system which contains an "intact" savanna mammal community, including Kenya's second largest elephant population (130).

4.2 Living and Working on Mpala Ranch

Most of the residents of Mpala are from Turkana and Samburu[13] ethnic groups. Cattle herding and domestic labor are the primary occupations of most of the 80 – 100 households residing on the ranch, with the remaining households employed as maintenance staff (such as tractor drivers, masons, mechanics, clerks, etc.).

Livestock is herded using traditional pastoral practices by Samburu and Turkana herdsmen (133, 134). In a herding village at Mpala, called a *boma* and shown in Figure 4.1, houses of the herders and guards surround a central enclosure where cattle are kept at night in protection from carnivores and cattle raiding. *Bomas* move their location

[13] Due to the various colonial resettlement schemes and mixing of various ethnic groups, there is a continuum of language and traditions between the ethnic groups of Samburu, Dorobo (or Laikipia Maasai), and Maasai (133).

regularly, in intervals that last from 2 months to more than 1 year. The timing and location of each move is decided by the ranch administration based on the criteria of availability of grass for grazing (itself influenced by climate), accessibility especially during the rainy season, and security and protection from cattle raiding. The number of households and the composition of the *bomas* also change based on the same criteria. A *boma* usually houses 4 – 10 households. Two other villages house the maintenance staff of Mpala ranch and the affiliated Mpala Research Centre.

Figure 4.1: A cattle-herding village or *boma* at Mpala Ranch. The central enclosure is made from thorny *Acacia* branches to protect the cattle at night. Herders and their families live in the surrounding houses.

Many of the households own small pieces of land in the reserve areas of northern Laikipia or the northern districts, in particular Turkana, Samburu, Marsabit, and Isiolo Districts. Some family members reside on the family land while others stay and work at Mpala. At Mpala, like many private ranches and plantations in Africa, the management exerts a great deal of power and control over employment conditions and lack of resources often prevents dismissed workers from seeking legal assistance.

Salaries of the employees vary by occupation and in general most unskilled staff receive a daily payment of approximately $US 1.5 - 2 per day in addition to some food (in particular milk) and uniform. Some households participate in income generating activities including making mats from ropes made from boiled tree barks, harvesting honey in traditional beehives (135), making traditional brew, and selling food (in particulate tea, sugar, maize flour and other dry foods), soap, and *mirraa*[14] purchased at wholesale prices from Nanyuki town. Both individuals and cooperative groups of women take part in the economic activities, with the latter especially involved in trade of food from town.[15] Distance and lack of ready transportation to the town of Nanyuki affect the number and shape of such activities. In particular, mats and honey are almost exclusively purchased by a monopsony of the relatives of the manager of Mpala Ranch who have access to transportation and marketing.

There was limited access to medical services at Mpala Ranch prior to the beginning of our research project in 1996. The district hospital, mission hospital, and private clinics in Nanyuki are the most accessible health facilities. But with a distance of nearly 50 kilometers and limited transportation, only the most serious cases would be referred to these facilities, when affordable. Traditional medicine and limited drugs administered by the ranch manager were the local sources of health care. A mobile mission clinic visited Mpala once per month for immunization and a family planning clinic from the Nanyuki

[14] *Mirraa* is a plant which is harvested on the Eastern slopes of Mount Kenya and whose leaf is chewed as a stimulant.

[15] Our project team was involved in setting up of the cooperatives of local women in early months of our research. Prior to that, the activities were mostly on an individual basis.

Cottage Hospital made occasional visits. Throughout this project (1996 – 1999), in an arrangement with Nanyuki District Hospital and Kenyatta National Hospital in Nairobi, two community nurses from the former facility provided basic medical services to the residents for two days each week while collecting health data.

A nursery school, taught by one of the residents, was the only form education available at Mpala until 1997 when a two room school was built by the visiting units of the British Army and staffed by a trained teacher. Since then classes covering first and second grades are also offered to the children of residents. But the expansion of education to higher grades was still under discussion in 1999 when field data collection for this project was completed. As a result, those residents who can afford the fees, send some or all of their children to school in areas where their relatives live or to mission-run boarding schools.

The day in a *boma* begins at or before 6:00 a.m. The fire is lit early in the morning for warmth and making tea. At this time, adult household members milk and bring the cattle out of the central enclosure of the *boma*. The cattle are counted and by 7:30 leave the *boma* for grazing accompanied by men and boys. After breakfast (sweet tea with milk and porridge or food left over from the previous day) is eaten, women and girls wash dishes from the previous night, store milk in gourds and make butter, clean the house and the compound. Water and firewood are collected in the morning or in the afternoon. Children, who wake up slightly later, play in the *boma* compound or in the house throughout the day or in the afternoon if they attend school (136).

In most households, the main meal of the day is cooked around noon. Those who go out with livestock eat their lunch / early dinner when they return in the late afternoon or take turns returning for lunch during the day if this can be done without being noticed by the foreman or manager. Tea is made 2 – 4 times during the day and water is heated for cleaning utensils a few times. The afternoons are usually spent resting outside, making mats, or collecting water and firewood. The cattle return to the *boma* and are counted by 17:30 and are sometimes also milked in the afternoon. After dusk, which begins at Mpala at 19:00 throughout the year, most people are inside. A small dinner is sometimes cooked in the evening but often food remaining from lunch is eaten (136). At each *boma*, a watchman is responsible for guarding the cattle overnight. The guard stays outside near a fire which is kept burning throughout the night and walks around the cattle enclosure once every 1 – 2 hours.

In maintenance villages the daily pattern of work (with the exception of cattle-related activities) is the same. The work day for men ends at 14:00 after which they eat lunch and gather in groups around the village to talk or play. Evening cooking is more common in these villages.

4.3 Housing

The houses in both cattle-herding and maintenance villages are cylindrical with conic straw roofs (Figure 4.2). Table 4.1 provides details of housing characteristics in the two

villages. Larger households, especially those with older children, are often allowed to build a second (often slightly smaller) hut.

(a)

(b)

Figure 4.2: Houses in *bomas* of Mpala Ranch. (a) A house under construction. (b) A completed house. See Table 4.1 for physical details.

Table 4.1: Housing characteristics in the cattle-herding and maintenance villages of Mpala Ranch.

	Cattle-herding villages (*Bomas*)	**Maintenance villages**
Diameter	3 m	4 – 5 m
Material of the walls	Mud, dung, and wood	Stones and mud
Height of the walls	1.5 m	2 m
Material of the roof	Wood and grass	Wood and grass
Height of the roof	1.5 m	2 m
Internal divisions	Yes (mud, dung, and wood)	Yes (plastic or brick)
Windows	No	Yes (2)

4.4 Food and Diet

The diet of Turkana, and herding societies in general, is often categorized as high-protein and low-energy (137). The diet of the residents of Mpala, described broadly in Table 4.2, is influenced and shaped by Turkana and Samburu traditions, modern staples of Kenya, and locally available resources. In particular, maize meal is now commonly available and consumed in Kenya and distributed at Mpala monthly by the ranch administration as payment-in-kind. Therefore, the general calorie intake at Mpala is likely to be higher than those observed among Turkana communities in their homeland.

Table 4.2: Common food items among the residents of Mpala Ranch.

Broad food category	**Cattle-herding villages (*Bomas*)**	**Maintenance villages**
Protein	Milk; occasionally blood, meat, eggs, and beans	Milk, beans; occasionally meat and eggs
Caloric	Maize flour (porridge or *ugali*) and sugar; occasionally sorghum flour, maize and beans (*githeri*)	Maize flour (porridge or *ugali*), maize and beans (*githeri*), and sugar; occasionally sorghum flour
Vitamins	Wild herbs and vegetables; occasionally purchased vegetables and fruits	Purchased vegetables, wild herbs and vegetables; occasionally fruits

Milk, both fresh and sour, is drunk regularly especially by children.[16] In general people in *bomas* have more access to milk than those in maintenance villages who have more access to foods purchased from town. Butter or cream is made from milk by sharply shaking a gourd full of milk suspended from the wooden poles in the walls or roof of the house as seen in Figure 4.3 (134, 136).

Figure 4.3: Turkana woman making butter/cream from milk by shaking the gourd sharply. The task is commonly done by women and girls in the cooking area.

Maize flour (maize meal) is consumed as porridge as well as *ugali*. *Ugali* is a "cake" made from maize flour (Figure 4.4). After adding flour to boiling water, the cook continuously stirs the mixture with a wooden spoon. As water evaporates and the mixture hardens, stirring becomes increasingly vigorous and then turns into folding the now-hardened layers of "dough". Finally the "cake" is turned over initially in the

[16] Milk is introduced into children's diet from the first few weeks/months after birth. In general, the nutrition of children receives a great deal of attention in Turkana and Samburu cultures (133, 134, 136,

cooking pot and then into a large dish for serving. Throughout the process, heat is controlled by increasing the burning rate or putting the fire into a smoldering (and hence very smoky) phase as stirring continues. After water has come to boil and flour is added, the process takes 15 to 40 minutes during most of which the cook is very close to the fire, actively controlling the heat or mixing the flour and stirring (136, 138).

(a)

---

137).

(b)

Figure 4.4: Cooking *ugali*. (a) Maize flour is added to boiling water. (b) After a long period of stirring and folding the mixture to ensure uniform consistency, ugali is ready in the shape of a cake.

A mixture of cooked maize and beans and some fried vegetables (including onions, potatoes, or tomatoes) is called *githeri*. Although there is widespread discussion in Kenya about methods for cooking *githeri* that consume less time and energy (in particular soaking the maize and beans the previous night), at Mpala all the work is done on the day of cooking. Cooking *githeri* from dry maize and beans often takes between 3 and 5 hours (136).

4.5 Energy Technology

With the exception of 4 or 5 households who occasionally use paraffin, firewood and charcoal are the exclusive sources of domestic energy at Mpala. Firewood is collected by

women and girls except in households in which a male migrant worker lives alone or with his small children.

Due to the small number of households living in a *boma* and regular movement of *bomas*, wood is readily accessible in most parts of Mpala and collection often takes less than 1 hour (53). Wood is cut with a large machete (called a *panga*) which is sharpened on a rock before collection (Figure 4.5a). There are clear preferences for particular species of trees as well as preferences for thicker and drier branches (53). The bark and small branches are cut from each piece, apparently because the thicker and more solid pieces burn longer and better, and with less smoke. After cutting, the wood is neatly arranged in bundles whose size is determined by the person's (perceived) carrying ability, tied with a thick, flat rope and carried on the back or on the head (Figure 4.5b, Figure 4.5c, and Figure 4.5d). We measured wood bundles as heavy as 35 kg but most bundles carried by adult women weigh between 15 and 25 kg. Wood is stored inside the house, especially during the rainy season, where it dries before use (Figure 4.5e) (136). The threat of wild animals, and in particular elephants, is identified by people as the main danger faced when collecting firewood.

(a)

(b)

(c)

(d)

(e)

Figure 4.5: Wood collection at Mpala. (a) Wood is cut by hitting it repeatedly at the same spot with a *panga.* (b), (c), (d) Wood is arranged and tied into bundles which are carried on the head or on the back. (e) Firewood is stored in the house to dry. *Panga* can be seen next to the stored wood.

Access to charcoal is more difficult and costly. The residents of Mpala are forbidden from making charcoal by the owner and management of the ranch. At least in one case, the residents believe that an employee was dismissed as a result of reports that he had made charcoal for his own use. Households who do want charcoal, sometimes bury the burning wood from their 3-stone fire and use the resulting charcoal, a method that has low conversion efficiency.

Charcoal is made in the community of *Naibo*, located on a neighboring group ranch, for sale. *Naibo* is 10 km from Mpala and transporting charcoal, especially when elephants

are in the area, is difficult and dangerous. Traders from *Naibo* at times visit Mpala Ranch and carry charcoal for sale, mostly on bicycles. For an extended period, the visits were prohibited by the ranch manager but this was not enforced consistently. Due to both Mpala Ranch restrictions and the difficulty of transporting charcoal the trader visits are of an irregular nature. A bag of charcoal, which could last a family of 4 – 6 for up to two weeks costs approximately $3 - $5, depending on the season and the size of the purchase.

Wood is burned in the 3-stone (open) fire as well as in ceramic wood stoves seen in Figure 4.6 and described in Table 4.3.[17] The ceramic stoves have an inner liner made from fired clay ceramic and a metal body. The body and the liner are connected with a mixture of cement and vermiculite which provides additional insulation. Charcoal is used in the older *Metal Jiko*[18] as well as the newer models of *Kenya Ceramic Jiko* (*KCJ*) and *Loketto* (Figure 4.7).

Table 4.3: Stoves used by the residents of Mpala Ranch.

Stove Name	**Material**		**Fuel**	**Price (US $ Equivalent)**
	Body	**Liner**		
3-stone	N/A	N/A	Firewood	$0
Kuni Mbili	Metal	Ceramic	Firewood	$4 – $6
Upesi	Metal	Ceramic	Firewood	$4 – $6
Lira	Metal	Ceramic	Firewood	$4 – $6
Metal Jiko	Metal	N/A	Charcoal	$1.5 – $ 2
Kenya Ceramic Jiko (KCJ)	Metal	Ceramic	Charcoal	$4 – $6
Loketto	Metal	Metal	Charcoal	$4 – $6

[a] The price is for an average-size stove and depends on the quality and location of purchase.

[17] Ceramic wood stoves were introduced at Mpala in 1997 in workshops that were conducted by extension workers and community development group members from Mpala and the Mumias area of western Kenya.

[18] The term *jiko* means stove in Swahili.

(a)

(b)

(c)

(d)

Figure 4.6: Wood stoves used at Mpala Ranch. (a) 3-stone (open) fire. (b) 3-stone fire is often used together with a curved metal mesh that reduces the distance of the pot to the fire and makes it more stable. (c) *Kuni Mbili* ceramic stove. *Kuni Mbili* means two pieces of wood in Swahili indicating the fuel efficiency of the stove. (d) *Upesi* and *Lira* ceramic stoves. *Upesi* means fast-burning in Swahili. The ceramic liner is seen in orange in the picture and the body is painted black.

(a)

(b)

(c)

Figure 4.7: Charcoal stoves used at Mpala Ranch. (a) *Metal Jiko*. (b) *Kenya Ceramic Jiko* (*KCJ*) (c) *Loketto*. In each stove, charcoal burns in the upper container and the lower chamber is used for lighting the stove and collecting the ash.

Chapter 5 Data Collection

Data for this research were collected between August 1996 and August 1999 at Mpala Ranch. The first 6 – 8 months of field research were spent on becoming familiar with the study area and the residents through participating in their daily activities and collection of background data, including detailed demographic information for all the households residing on the ranch and surveys of energy use, energy technology, and related characteristics. Data collection throughout the rest of the field research can be divided into four broad categories based on the type of data and method of collection: monitoring of pollution and stove emissions, individual time-activity budget and exposure to pollutants, health data, and perceptions of technology and health.

Emission concentrations and time-activity budgets were monitored throughout the whole day – between the hours of approximately 6:30 and 20:30 – while normal household activities took place. A total of 210 days of sampling were conducted in 55 randomly-selected houses in both cattle-herding and maintenance villages. The visits were made on random days of the week. Approximately 20% of the households, randomly selected in both village types, were visited between 6 and 15 times to monitor the intra-household variation in emission concentrations as well as variations in time-activity budgets. Another 25% were visited once and the remaining households between 2 and 5 times. Included in these days were four nights of monitoring of activities of cattle guards and

the emissions from the fire that they use for warmth. The demographic characteristics of the individuals in the study households are given in Table 5.1.[19]

Table 5.1: Demographic characteristics of the study group. Numbers in brackets indicate standard deviations.

Age group [a]	**Number of individuals in the group**	**Fraction female**	**Mean age**
0 – 5 years	93	0.56	3.0 (1.4)
6 – 15 years	109	0.56	9.7 (2.7)
16 – 50 years	120	0.54	29.4 (10)
> 50 years	23	0.65	63.8 (9.4)
Total	345	0.56	18.3 (17.6)

[a] Children under the age of 5 have additional susceptibility to ARI and at higher ages chronic conditions begin to show. For those between the ages of 5 and 50, a division was made at the age of 15 when it is common for people to enter the work force or get married. Age is reported as the age of each individual in the last half-year period of data collection (in the spring of 1999). Therefore, individuals whose age crossed cut-off points during data collection period were allocated to the category characterizing their age in the last quarter of field research.

Data collection was performed by two field research assistants (one female and one male), accompanied by the principal researcher for the first six months of data gathering, with regular examination of data recording protocols after the first six months. Each person was assigned well defined tasks, especially in the first few minutes of each day when the pollution monitoring equipment was placed in the house. Information such as names and ages of household members were collected independently in the first few months of field research so that, on the days of monitoring, data sheets for activities for each individual could be prepared before arrival in the house. Test sessions were conducted and the protocols were adjusted to ensure minimal interference with household activities.

[19] The sample includes only people who resided in the household for a continuous period of six months or more. Therefore household members who were away at boarding school, worked in neighboring ranches, or lived on the reserve are not included. This group includes another 52 individuals.

5.1 Pollution Monitoring Equipment

Measurement of particulate matter was carried out using *personal*DataRAM manufactured by MIE, Inc. (Bedford, MA). *personal*DataRAM uses nephelometric (photometric) monitoring technology with passive sampling which minimizes interference with normal activities of the household. The particle size of maximum response is 0.1 μm to 10 μm. As a result of this response range, only a fraction of the measured concentration is due to particles below 2.5 μm ($PM_{2.5}$), which are believed to have the most important health impacts. Studies of particle pollution in both industrialized and developing countries has demonstrated correlation between PM_{10} and $PM_{2.5}$ concentrations (24, 139), but further research on this relationship in the case of biomass smoke is needed. Carbon monoxide concentration was measured using Enerac Pocket 100 manufactured by Energy Efficiency Systems, Inc. (Westbury, NY). Both instruments were sent to the factory approximately once per year for re-calibration of measurement range (span), and replacement of *personal*DataRAM measurement chamber and Enerac Pocket 100 sensors. The instruments were zeroed in clean air outside the village compound every day and the measurement chamber of *personal*DataRAM was cleaned using pressured air after every two days of measurement.

5.2 Temporal Variation of Suspended Particulate Emission

Particulate matter (PM_{10}) and carbon monoxide (CO) concentrations were recorded at a distance of approximately 0.5 m from the center of the stove, at a height of 0.5 m, where

the monitors were placed on a flat surface. Since cooking some of the common foods in the area and lighting and tending of fire are done with the user's head near the stove, sampling distance was chosen to be as close to the user's breathing area under such circumstances as possible. The other criteria used for choosing the sampling point were avoiding interference with household activities, ensuring that the instruments could be placed in a stable position and were not damaged due to heat, and ready standardization of measurement point. PM_{10} concentration was averaged over and recorded in one-minute intervals between the hours of 6:30 and 20:30. During the same period, carbon monoxide concentration was measured in five or ten minute intervals (depending on how stable the fire was) averaged over a period of 10-20 seconds. Figure 5.1 shows the PM_{10} and CO concentrations for one day of monitoring in a household that cooked inside using a wood-burning 3-stone fire. PM_{10} concentration was also monitored during the night when we could ensure that the equipment could be left in the house safely and without disturbing the household members. PM_{10} concentration data which were logged automatically by the *personal*DataRAM (PDR) were down-loaded into a personal computer after every set of sequential days of monitoring. The dates and memory locations of PDR were checked against the other data sheets.

5.3 Cooking and Energy Related Activities

During these 210 days of monitoring, we also recorded the status of fire (whether it was off, starting, burning, or smoldering), the type of food prepared, and other energy or cooking related behavior such as addition or moving of fuel or cooking pot, stirring food, and so on during the whole day. The status of the fire was recorded once every 5-10

minutes, depending on how stable the fire was. Sample data for one day of monitoring are seen in Figure 5.1.

5.4 Time-Activity Budget

Finally, we recorded the location and activities of all the household members who were present at home during the day. Location data were recorded as whether the person was inside or outside, and whether near fire (defined as within a distance of approximately 1 m from the stove) or far from fire. Activities and location were recorded as they occurred throughout the day.

We also conducted extensive interviews with household members and local extension workers on energy technology, cooking practices, and time-activity budgets. In each household, an adult member responsible for cooking was asked in detail about the stove and fuel used by the household, location and times of cooking, and the types of meals prepared. An adult member was also asked about the location and activities of each household member during six time periods in the day (morning, midday, early afternoon, late afternoon, evening, and night), with additional questions about location and activities during cooking. Extension workers were asked the same questions separately.

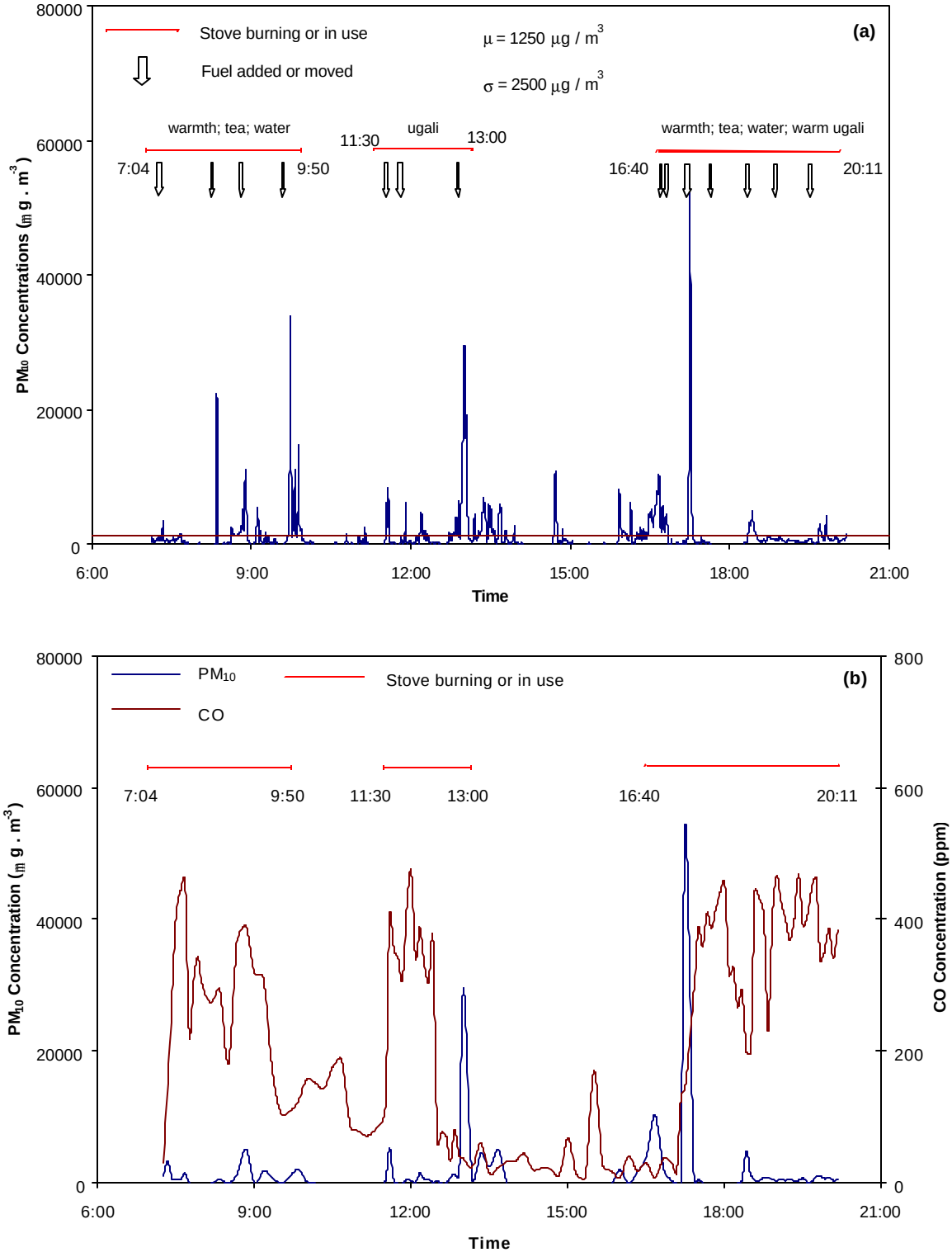


Figure 5.1: Day-long monitoring of pollution and cooking activities. (a) PM_{10} concentration in a household that used a 3-stone stove inside. The uses of the stove are indicated above the horizontal lines. The lower horizontal line indicates the mean pollution for the day. (b) PM_{10} and CO concentrations for the same day. PM_{10} concentration was sub-sampled at the moments when CO concentration was measured.

Household interviews were conducted in the language of choice of the respondent. The field research assistants had copies of the surveys in English and Swahili and had translated all questions into Turkana language with the supervision of field research directors. For each new interview, multiple days of dry-runs were conducted among the field research assistants and field research directors. On the few occasions when the respondents requested interviews in Samburu language, the interviews were conducted with the assistance of local residents who were fluent in Samburu and either Turkana or Swahili.

5.5 Spatial Variation of Indoor Air Pollution

We also collected data on the spatial distribution of indoor air pollution. These measurements were all conducted in two houses (one in each size group) while the residents were away. We ensured that the fire remained stable for a 15-minute period, during which we measured PM_{10} concentration sequentially at ten points inside the house. Eight of the points were at distances of 0.4, 0.8, 1.2, and 1.9 meters from the center of the stove, at heights of 0.5 and 1.0 meter. The ninth point was directly above the fire at a height of 1.0 meter and the tenth in the sleeping area. Together these points cover those parts of the house where household activities take place since, due to the small height of the roof, adults do not commonly stand in the house. Sampling took place once every second for a duration of one minute at each point. We repeated this experiment under different conditions with doors and windows open and closed, and with and without a cooking pot on the stove. A total of 78 repetitions of this experiment were conducted in the two houses. Any measurement during which the status of fire changed

(such as transition to smoldering phase) was discarded, resulting in 68 sets of measurements used in analysis.

5.6 Health Data

Two community nurses from Nanyuki District Hospital who had received the training provided by the National Acute Respiratory Infection (ARI) Programme (designed in consultation with and funded by the World Health Organization) on the WHO protocols for the clinical diagnosis of ARI visited all the households in the study group on a regular basis. In the initial months of the program each village was visited once every two weeks. The visits then increased to once per week. In the visits during the initial months, one of the coordinators of the National ARI Programme from the Department of Paediatrics of the Kenyatta National Hospital accompanied the visiting nurse to the village to ensure the proper execution of diagnosis protocols.

In each visit, at least one adult member from each household reported to the nurse on the health status of the household members, with specific emphasis on the presence of cough and other respiratory ailments. The responses were collected in the language of choice of the respondent and recorded in English by the nurse who spoke Swahili and Turkana. The nurse then clinically examined all those who were reported as having symptoms and recorded the relevant clinical information including symptoms and diagnosis. The reporting process also included information on visits to any other health facility since the nurse's last visit. Therefore the health data include a two-year array of weekly health records for each individual in the study group. Depending on the severity, the cases were

treated with the standardized treatment of the National ARI Programme, which also resulted in standardization of treatment in the study group. Treatments included drugs that are readily available in the town of Nanyuki (dispensed by the nurse) for more severe cases as well as providing assurance or recommending home remedies for minor cases. The extreme, and potentially fatal, cases were referred to one of the hospitals in Nanyuki. No information was recorded for those households from which no adult member was present or for household members who were away from home during the day of visit. Table 5.2 provides summary statistics on the number of health reports for the individuals in the study group.

Table 5.2: Number of health reports for the study group between early 1997 and June 1999.

Age group	**Mean**	**Standard deviation**	**Median**
0 – 5 years	72.2	23.9	85
6 – 15 years	82.2	16.3	88
16 – 50 years	80.5	17.7	87.5
> 50 years	73.9	19.1	82
Total	78.4	19.7	87

5.7 Interviews and Surveys

Finally, in a series of interviews in addition to those on time-activity budgets and energy use, individuals and groups were asked about their perceptions and preferences of energy technology, indoor air pollution, wood collection, and health.

Chapter 6 Exposure Assessment [20]

Assessment of human exposure to pollutants has been among the most controversial areas of risk assessment. Although theoretically exposure to air-borne pollutants is simply the integral of concentration over the period of exposure, obtaining the exposure concentrations is an extremely difficult task. Modeling of pollutant dispersion, in all but the simplest conditions, results in intractable mathematical complexity. Further, the set of physical variables that would characterize any model, such as air flow and deposition rates, are technically impossible or very costly to monitor continuously. Added to the problems of the measurement of concentration is the fact that, except for certain occupational conditions where mobility is restricted due to job constraints, people move from one microenvironment to another which further adds to the complexity of integration of concentrations (for a complete discussion see Chapter 3 in 140).

Some of the most extreme, and controversial, cases of data limitation in exposure assessment rise in research on carcinogenesis (141, 142). The health impacts of carcinogens are observed years or decades after exposure has occurred. Therefore, in carcinogenic risk assessment, personal exposures have been estimated using historical perspectives on time-activity budgets and the simplest indicators of concentrations in the work or living area, often assigning a single value to a whole workshop, factory, or (in

[20] A shorter version of this chapter has been published as the following article: Ezzati, M., H. Saleh, and D. M. Kammen (2000) "The Contributions of Emissions and Spatial Microenvironments to Exposure to Indoor Air Pollution from Biomass Combustion in Kenya," *Environmental Health Perspectives,* **108**.

the case of radiation) even city (for an example in the case of Benzene see 143, 144, 145, 146, 147, 148)

The expansion of environmental regulatory frameworks in industrialized countries has stimulated substantial progress in pollution monitoring technology and efforts. Motivated by environmental regulation and broader public health concerns, modeling and monitoring techniques for characterizing pollution dispersion have advanced considerably in various scales, from indoor tobacco smoke (149, 150) to regional or national transport of pollution from factories and refineries (151). In addition to characterizing physical dispersion, research in industrialized countries is making rapid progress in assessment of human exposure by taking into account people's time budgets and activity patterns (152).

In developing countries, on the other hand, exposure assessment has been among the weakest aspects of research on health risks associated with indoor air pollution (see also Chapter 2). Beyond the use of indirect measures of exposure such as fuel type or housing characteristics, exposures have often been calculated using average daily concentrations at a single point. Although useful for pollutants whose concentration has little temporal variation, average concentration is not appropriate for characterizing exposure to indoor smoke which fluctuates enormously throughout the day, as seen in Figure 5.1 for instance.[21] This simplifying attitude towards exposure assessment in research on indoor

[21] An alternative to the indirect exposure measures has been the use of personal monitors (see for example 7, 153). Although resolving the issue of exposure estimation, with most personal monitors exposure is aggregated over time and space. Therefore personal monitors limit a predictive assessment of various

air pollution is exemplified in the 1999 Air Quality Guidelines of the World Health Organization which states "although work on simple exposure indicators urgently needs to be encouraged, realistically it is likely to be some years before sufficient environmental monitoring can be undertaken in most developing countries" (13).

In this chapter, I integrate quantitative and qualitative data on individual time-activity budgets, household demographic characteristics, and continuous real-time monitoring of indoor air pollution to construct personal *profiles of exposure* to suspended particulate matter resulting from biofuel combustion. The exposure profiles in this analysis are constructed from fundamental components – the emission of the stove, and the location, time budget, and activities of household members – an approach gaining considerable strength in industrialized countries but still underutilized in developing nations. With continuous data on instantaneous pollution levels, I also move beyond the single measure of average daily pollution and develop exposure estimates using other descriptive statistics of emission data which better characterize human exposure.

In our 210 days of day-long home monitoring sessions, we collected data on pollution level at a single point (at a distance (x) of 0.4 – 0.5 m from the center of the stove, at a height (z) of 0.5 m). I first use the data on spatial distribution of pollution to predict PM_{10} concentration at other points inside the house, which in turn could be combined with data on location of household members to provide a complete *spatial and temporal profile of*

---

intervention strategies and do not allow incorporating the role of high-intensity emission episodes which happen commonly during combustion of biomass fuels.

pollution. Using these pollution profiles and data on time-activity budget – obtained from day-long monitoring as well as interviews – individual exposure is characterized while accounting for day-to-day variability of pollution and time-activity budgets. This process is schematically shown in Figure 6.1 and described in the following sections.

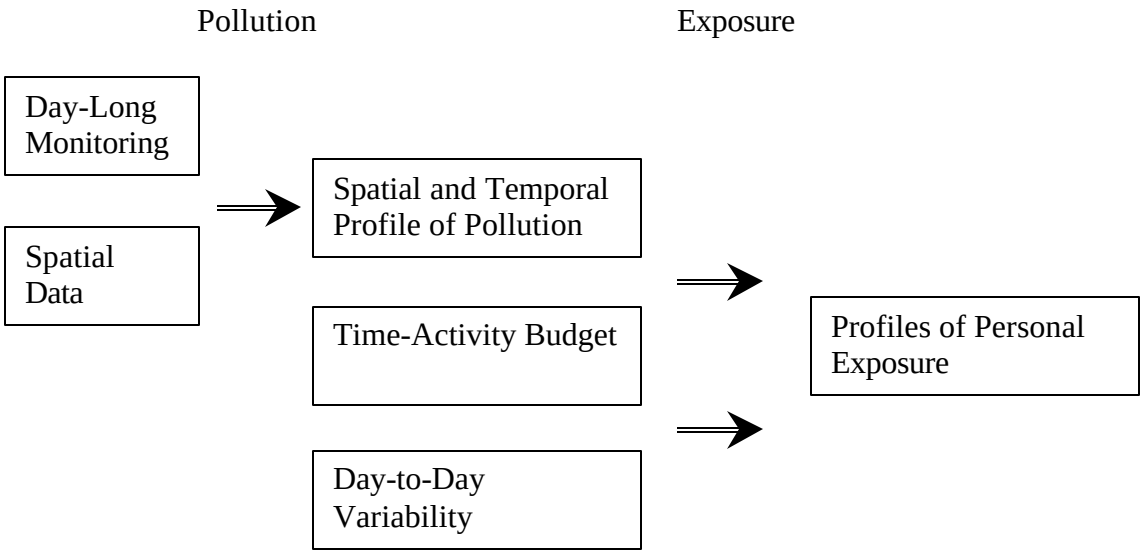


Figure 6.1: Exposure assessment process. Day-long monitoring of pollution (at one point) is combined with data on spatial dispersion of smoke to provide temporal and spatial profiles of pollution. Individual time-activity budget data illustrate which regions of this temporal-spatial pollution profile are occupied by each individual. Finally, day-to-day variability of pollution and time-activity budget are taken into account using data from houses that were visited multiple times as well as interviews.

6.1 Individual Exposure: The Role of Spatial Distribution of Pollution

Figure 6.2 plots the concentration of particulate matter against horizontal distance from the stove (x) for measurements at heights (z) of 0.5 m and 1.0 m for various measurement conditions corresponding to door or window being open/closed or cooking pot present/absent.

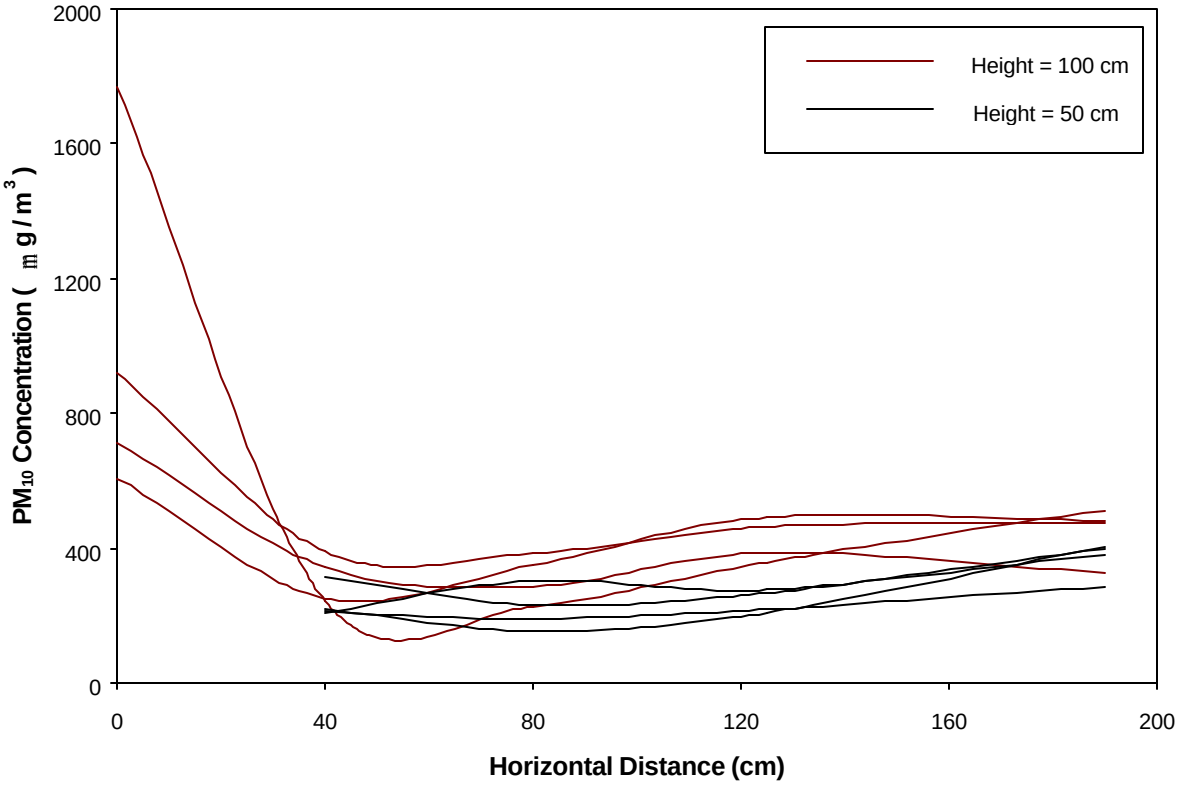


Figure 6.2: Spatial distribution of PM_{10} concentration. Each pair (at heights 100 cm and 50 cm) of curves corresponds to a measurement condition with combinations of window and/or door open/closed and cooking pot present/absent. The curves represent the average of 10-15 measurements for each measurement condition. Measurements took place for one minute each at distances of 0, 0.4, 0.8, 1.25, and 1.9 meters from a stable fire. See Chapter 5 for a complete description of measurements.

As seen in Figure 6.2, PM_{10} concentration initially drops rapidly with increasing distance from the stove, a pattern which can also be observed for visible smoke in actual conditions of use in Figure 6.3. Concentration then increases at a low rate after a distance of approximately 0.5 m. Further, points at a height of 1.0 m have slightly higher concentration than those at 0.5 m.[22] This pattern indicates that individual exposure to

[22] Higher concentration at a height of 1.0 m and the rise of concentration at horizontal distances above 0.5 m are consistent with a plume model of pollution dispersion.

smoke is dependent on the location of the individual relative to the fire, even in the houses as small as those described above.

Figure 6.3: There is considerably higher smoke directly above the fire before dispersion in the room.

There are few models for characterizing the indoor dispersion of particulate matter. Smith (49) describes and utilizes a steady-state model of pollutant dynamics which is based on the assumption of instant mixing, resulting in uniform concentration in the room. The works of (149, 150) however illustrate that the instantaneous mixing assumption is not applicable to a closed room with limited air flow, as also seen in Figure 6.2 and Figure 6.3.

I divide the indoor area of the houses in the study group into six exposure microenvironments. The six microenvironments include the area immediately around the stove where smoke rises and has the highest concentration, the sleeping area, and four additional areas from dividing the remainder of the house along a horizontal plane at a height of 0.5 – 1.0 m and a vertical plane at approximately 1.0 – 1.5 m (Figure 6.4). These divisions are based on incremental distances from the stove where various activities take place. Assuming that each of these microenvironments is well-mixed internally, pair-wise relationships among them can be expressed as the ratios of pollutant concentrations. The exact relationship between the microenvironment concentrations depends on the instantaneous air flow. Detailed measurements of this variable are however not possible in field data collection. I therefore use the average of the ratios obtained empirically under the different conditions of stove use to represent the relationship between the different exposure microenvironments. Using this method, the ratios of PM_{10} concentration in the microenvironments of Figure 6.4 relative to point (0.5, 0.5), where daily monitoring took place are: 7.0 – 7.5 for 1, 1.0 – 1.1 for 2, 1.7 – 1.8 for 3, 1.4 – 1.5 for 4, 2.0 – 2.2 for 5, and 1.2 – 1.3 for 6.

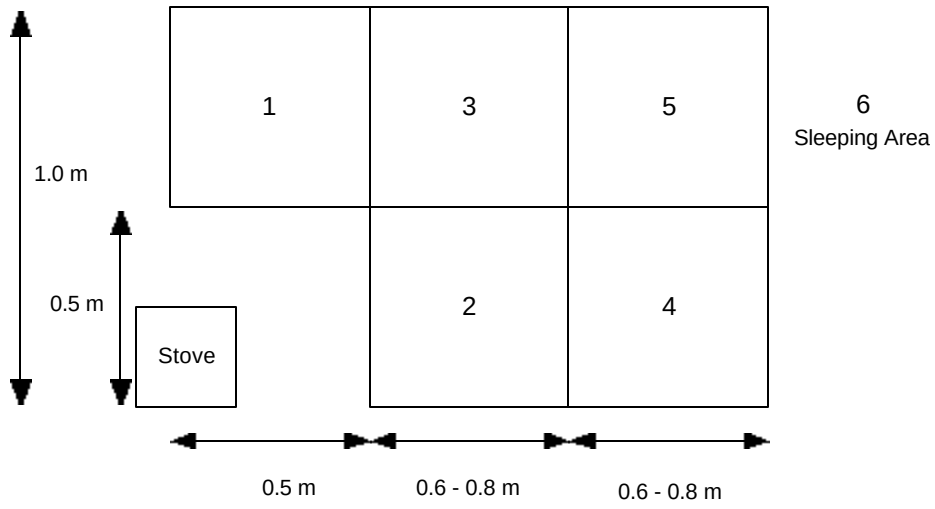


Figure 6.4: Schematic representation of indoor exposure microenvironments in the study houses. The divisions are based on incremental distances from the stove where various activities take place. A division, made of mud or plastic, separates the sleeping area (No. 6) from the rest of the house, but the division is not complete (i.e. there is an open entrance).

6.2 Individual Exposure: The Role of Time-Activity Patterns

Smoke emissions from a biomass stove exhibit very large variability throughout the day, including large peaks of short duration. This can be seen for example in the pollution profile of Figure 5.1 where PM_{10} concentration regularly exceeds the daily mean by large margins. In the day-long pollution data, PM_{10} concentration has average *coefficients of variation*[23] of 3.2 and 4.0 during burning and smoldering periods[24] respectively, indicating large daily variability around the mean.

---

[23] Coefficient of variation is defined as the ratio of standard deviation to mean and is a measure of the variability of data relative to its mean.

[24] A low background level of combustion takes place throughout the whole day. For the purpose of this analysis I define *burning* as the periods when the stove was used for cooking and/or it was in flame. *Smoldering*, therefore, refers to periods that the stove was neither in active use nor in flame. Active use while the stove is not in flame is included in the *burning* category because when cooking some foods, and

The quantitative and qualitative data on time-activity budgets also indicate that some household members are consistently closest to the fire when pollution level is the highest. These episodes typically occur when fuel is added or moved, the stove is lit, the cooking pot is placed on or removed from the fire, or food is stirred (in particular when cooking the common dish of *ugali*) as also seen in Figure 4.5, Figure 6.3, and Figure 6.5. Other individuals may be systematically outside/away from the house during some of these episodes, especially during the hours when the fire is lit or extinguished.

Figure 6.5: Household members involved in cooking are exposed to episodes of high pollution when they work directly above the fire. See Figure 6.3 for another example.

Systematic association between distance from the stove and emission peaks indicates that average daily concentration alone is not a sufficient measure of exposure. Therefore in

---

in particular *ugali*, there are moments that the flame is put out on purpose to control the heat. Yet this act is

addition to mean concentration (μ), I use the following two descriptive statistics (for both burning and smoldering phases) for the reasons stated:[25]

- Mean above the 75th percentile ($\mu_{>75}$): to account for the fact that some household members are closest to the stove during high-pollution episodes caused by cooking activities.
- Mean below the 95th percentile ($\mu_{<95}$): to eliminate the effect of large instantaneous peaks that especially occur when lighting or extinguishing the fire, or when fuel is added.

6.3 Individual Exposure: Day-to-Day Variability

In addition to the above daily variation, one may expect day-to-day variability in exposure to indoor smoke as a result of variation in both emissions and time-activity budget. Emission concentrations in a single household can vary from day to day because of fuel characteristics such as moisture content or density, air flow, type of food cooked, or if the household uses multiple stoves or fuels.[26] Table 6.1 shows the results for (sequential) decomposition of the variance of the above concentration data into inter-household and intra-household components.

---

a part of the cooking activity. I therefore classify it as *burning*.

[25] Such a break-down of exposure into high and low intensity episodes is used in settings where knowledge of variability of concentrations is available. See (148) for an example in the case of occupational exposure to Benzene. This also the standard approach for estimating exposure to toxic chemicals absorbed through digestion and dermal contact (see Chapter 7 in 154).

Table 6.1: Contributions of inter-household and intra-household days of sampling to the variance of emissions concentrations. The data consist of multiple days of observation for multiple households.

	Mean (μ) during *burning*	**Truncated mean ($\mu_{<95}$) during *burning***
Model 1 [a]	2.17×10^9	6.50×10^8
Inter-household	1.61×10^9 *	5.13×10^8 *
Intra-household (day-to-day)	5.63×10^8	1.37×10^8
Model 2 [a]	2.17×10^9	6.50×10^8
Intra-household (day-to-day)	2.46×10^8 *	5.69×10^7 *
Inter-household	1.92×10^9	5.93×10^8
Residual	5.79×10^8	1.95×10^8
N	188	188
R^2	0.79	0.77

[a] Sequential analysis of variance is used. In each model, the first sum-of-squares (marked by *) shows the portion of variance explained by that variable alone. The second sum-of-squares shows the additional portion of variance explained when the second variable is added the model. But some of this additional contribution is from simultaneous presence of both variables. For each household, data were used from a single cooking location. The case of multiple cooking locations is treated separately.

The fraction of variance of average burning-period emission concentrations (μ) (column 1) explained by inter-household variation is 6.5 times the fraction explained by day-to-day variability.[27] The corresponding ratio for $\mu_{<95}$ (column 2) (which is less sensitive to instantaneous peaks) equals 9.0. This comparison illustrates that, although considerably smaller than inter-household variation, pollution in individual households varies from day to day.

Activity patterns can also vary due to the seasonal nature of work and school, illness, market days, and so on. Therefore, in addition to the use of multiple descriptive statistics for characterizing daily exposure, I construct measures of exposure which are not solely

---

[26] There was no indication of systematic seasonal variation in emissions in our study area, which I attribute to the fact that storing and drying wood before use is a common practice among the households in the study group (in all but one of the monitoring days the firewood used was dry).

[27] The ratio is for the fraction of variances explained by each variable *alone*, since sequential analysis-of-variance (ANOVA) is used.

based on measurements and observations from individual days. Specifically, rather than using measurements of emission concentration directly, I assign *households* to emission concentration categories. This categorization is performed for the three descriptive statistics defined above (μ, $\mu_{<95}$, $\mu_{>75}$) for both burning and smoldering phases. A similar grouping is done for time budgets (including time spent inside, near fire, and inside during cooking) and activity (whether the person cooks regularly/sometimes/never and whether the person performs non-cooking household tasks regularly/sometimes/never).

The range of variability of time budgets is determined by people's activities. Information on both time budgets and activity types can be obtained using interviews. Therefore, the grouping of time budgets and activities is based on the data from the 210 days of direct observation of time-activity budgets as well as the supplemental interviews. For household pollution categories, on the other hand, no work on the thermodynamics of biomass combustion discusses the range and distribution of emissions from a single stove operated by the same user on different days. In addition to the fluctuation caused by the variations of the combustion parameters, including fuel characteristics and air flow, the specific use of stove may be different from day to day, even in a setting where the diet varies in a small range. In choosing the exposure categories, I have used criteria that were not statistically driven, but based on knowledge of physical characteristics of combustion as well as using the small subset of households that were visited multiple times as instructive cases. The following are the criteria motivating the choice of exposure categories. I discuss the research needs that can provide stronger statistically founded guidelines for this classification in Section 6.6.

- The width of concentration categories (i.e. bin size) is expected to be smaller in lower ranges to account for larger variability at higher concentrations.
- The two lowest concentration categories are selected to correspond to the "best-use" and "average-use" conditions of charcoal stoves. The "best-use" conditions, which are also similar to the pollution levels when kerosene stoves were used, are taken as the range of concentrations in 6 – 7 households that used improved charcoal stoves, with consistently low emission concentrations when using these stoves. "Average-use" conditions characterize the emissions in the other charcoal using households, except those that use the older *Metal Jiko* and had emission levels comparable to the lower-end of wood stove emissions.
- The third concentration category describes the extreme of pollution from charcoal stoves as well as the least-polluting use of wood stoves.
- The highest concentration category represents households that used 3-stone fire and consistently had very high pollution levels.
- Finally, I have selected three other overlapping categories between the third and the last ones to account for gradual transitions.[28]

---

[28] Concentration categories for mean PM_{10} (μ) during burning period are <200 $\mu g.m^{-3}$, 200 – 1000 $\mu g.m^{-3}$, 500 – 2000 $\mu g.m^{-3}$, 1000 – 3000 $\mu g.m^{-3}$, 2000 – 5000 $\mu g.m^{-3}$, 3000 – 7000 $\mu g.m^{-3}$, and 4000 – 10000 $\mu g.m^{-3}$. For $\mu_{<95}$ the categories are <150 $\mu g.m^{-3}$, 100 – 300 $\mu g.m^{-3}$, 250 – 1000 $\mu g.m^{-3}$, 500 – 2000 $\mu g.m^{-3}$, with the remaining categories being the same as those for μ. For $\mu_{>75}$ they are <500 $\mu g.m^{-3}$, 300 – 1000 $\mu g.m^{-3}$, 500 – 2000 $\mu g.m^{-3}$, 1000 – 5000 $\mu g.m^{-3}$, 2000 – 10000 $\mu g.m^{-3}$, 4000 – 20000 $\mu g.m^{-3}$, 6000 – 30000 $\mu g.m^{-3}$, and 10000 – 50000 $\mu g.m^{-3}$. The categories for mean PM_{10} (μ) during smoldering period are <150 $\mu g.m^{-3}$, 150 – 500 $\mu g.m^{-3}$, 250 – 1500 $\mu g.m^{-3}$, 500 – 2000 $\mu g.m^{-3}$, 1000 – 3000 $\mu g.m^{-3}$, 2000 – 5000 $\mu g.m^{-3}$, 3000 – 7000 $\mu g.m^{-3}$, and 4000 – 10000 $\mu g.m^{-3}$. For $\mu_{<95}$ the categories are <100 $\mu g.m^{-3}$, 50 – 300 $\mu g.m^{-3}$, 100 – 500 $\mu g.m^{-3}$, 250 – 1000 $\mu g.m^{-3}$, 500 – 2000 $\mu g.m^{-3}$, and 3000 – 7000 $\mu g.m^{-3}$. For $\mu_{>75}$ they are <500 $\mu g.m^{-3}$, 300 – 1000 $\mu g.m^{-3}$, 500 – 2000 $\mu g.m^{-3}$, 1000 – 5000 $\mu g.m^{-3}$, 2000 – 10000 $\mu g.m^{-3}$, 4000 – 20000 $\mu g.m^{-3}$, 6000 – 30000 $\mu g.m^{-3}$, and 10000 – 50000 $\mu g.m^{-3}$. The groups for time inside the house, as a fraction of the day, are < 0.2, 0.2 – 0.35, 0.3 – 0.45, 0.45 – 0.65, and > 0.6; for inside the house when stove was burning < 0.15, 0.15 – 0.3, 0.25 – 0.40, 0.35 – 0.60, and > 0.55; and for time spent near fire < 0.05, 0.05 – 0.1, 0.1 – 0.2, 0.2 – 0.4, and > 0.4.

Households that use multiple stoves or fuels necessarily span multiple categories. Further, those households that sometimes cook outside were assigned to two distinct categories, one for each cooking location. Similarly the time budget of individuals in the latter group of households is divided between the two locations accordingly. Table 6.2 provides a summary of the time spent inside the house and near the fire in demographic groups divided by gender and age.

Table 6.2: Time-activity budget for demographic sub-groups after assignment to time categories. The results are based on the mid-values for each category. In practice, the amount of time spent inside on different days is from a distribution around this mid-value.

Age group	**Fraction of time inside** [a]		**Fraction of time near fire** [b]		**Probability of cooking** [c]	
	Female	**Male**	**Female**	**Male**	**Female**	**Male**
0 – 5 years	0.43	0.44	0.20	0.20	0	0
6 – 15 years	0.40 *	0.26 *	0.23 *	0.13 *	0.39 *	0.02 *
16 – 50 years	0.54 *	0.24 *	0.38 *	0.06 *	0.98 *	0.11 *
> 50 years	0.39	0.30	0.24	0.13	0.27	0.19
Total	0.45 *	0.30 *	0.27 *	0.13 *	0.48 *	0.06 *

[a] Fraction of time is based on a 15-hour day from 6:00 to 21:00.
[b] Fraction of time is based on a 15-hour day from 6:00 to 21:00. Near fire refers to areas within a radius of approximately 1 meter from the stove.
[c] Average within the group, with a probability of 1 assigned to those who cook regularly, 0.5 to those who cook or look after fire sometimes, and 0 to those who do not perform cooking and energy related tasks.
* Difference between male and female rates significant with $p < 0.0001$.

6.4 Exposure Profiles as the Basis of Analysis

I construct *profiles of exposure* for each individual in the monitored households based on the combination of time-activity budgets, spatial dispersion, and daily and day-to-day exposure variability. In doing so, I divide the time budget of household members into the following activities: cooking, non-cooking household tasks, warming around the stove, playing, resting and eating, and sleeping. I also consider the set of potential locations

where each activity takes place. For example playing or resting may take place inside the house or outside, cooking activities directly above the fire or slightly farther away, other household tasks near the stove or closer to the sleeping area, and so on. The activity groups and their related parameters are described in Table 6.3

For each location-activity pair, I estimate an equivalent *conversion (or dilution) factor* which converts the emission concentration measurements (at point x = 0.5, z = 0.5) to concentrations at the microenvironment of exposure using the spatial distribution analysis (as described in Section 6.1 above). Daily exposure is then obtained using the following relationship:

$$E = \sum_{i=1}^{n} \sum_{j=1}^{6} w_j \, t_{ij} c_i \qquad (6.1)$$

where c_i is the emission concentration (at point x = 0.5, z = 0.5) in the i^{th} period of the day, t_{ij} time spent in the j^{th} microenvironment in the i^{th} period, and w_j the conversion factor for the j^{th} microenvironment.

Table 6.3: Activity groups, their location described by the microenvironments of Figure 3, and the descriptive statistics used to characterize emissions concentration while they occur. Dilution factors for the microenvironments are given in Section 6.1.

Activity Group	**Examples**	**Location (microenvironment)**	**Emissions concentration** [a]
Cooking 1	Lighting and tending fire; stirring food	1	Burning: $m_{>75}$
Cooking 2	Cutting and cleaning food items	3	Burning: m
Non-cooking work	Cleaning utensils, serving food, cleaning the house	3 and 5	Burning: m Smoldering: $m_{>75}$ [b]
Warming	N/A	2 and 3	Burning: m
Resting/Eating 1 (females and children)	N/A	4 and 5	Burning: m Smoldering: m
Resting/Eating 2 (adult males)	N/A	5	Burning: m [c] Smoldering: m
Playing (children)	N/A	3 and 5	Burning: m Smoldering: m
Playing (infants)	N/A	6	Burning: m Smoldering: m
Sleeping	N/A	6	Smoldering: $m_{<95}$ [d]

[a] In almost all houses, a low background level of combustion takes place throughout the whole day. For the purpose of this analysis we define *burning* as the periods when the stove is used for cooking and/or it is in flame. *Smoldering*, therefore, refers to periods that the stove is neither in active use nor in flame. By definition, cooking and warming over fire can take place only during *burning*. Other activities can in principle take place in both states, although in practice during sleeping at night the stove is not kept on.

[b] Non-cooking household tasks that take place during the smoldering phase often occur immediately before the fire is lit or after it is extinguished, therefore during the upper end of emission concentrations.

[C] For adult males, an alternative exposure profile would consider that they are systematically away when pollution is highest, especially during lighting and extinguishing times. With this characterization, their exposure concentrations will be based on $m_{<95}$ instead of m This choice has a very small effect on the outcome since first, adult males spend a very small fraction of the day indoors and second, they are consistently away from the fire where dilution reduces concentration the most.

[d] Since wood is rarely added or moved during the night but background combustion continues, pollution is described by the smoldering period concentration without its most polluted moments.

Figure 6.6 illustrates the average exposure concentration[29] for *total daily exposure* for various demographic groups obtained using the mid-point values[30] of emission

[29] *Average exposure concentration* is the PM_{10} concentration that if sustained for the whole day would result in exposure equal to total daily exposure of the individual.

concentration and time categories. These values were obtained using Equation 1 and dividing the day into burning and smoldering periods, further dividing each into high-intensity and low-intensity emission periods and dividing each component of the time budget among the possible location-activity pairs based on interviews, direct observation, and demographic characteristics of the household.

The comparison between female and male exposure shows that, in the exposure profile approach, the ratio of female to male total exposure is 0.91, 2.5, 4.8, and 1.2 for the four age groups. Therefore, young and adult women not only have the highest absolute exposure to particulate matter from biomass combustion (2795 $\mu g.m^{-3}$ and 4898 $\mu g.m^{-3}$ average exposure concentrations respectively), but also the largest exposure relative to that of males in the same age group.

In Figure 6.7 and Figure 6.8 the exposure values of Figure 6.6 are decomposed into exposure during high-intensity and low-intensity episodes.[31]

---

[30] Using lower and upper values of the pollution concentration range and time-inside range result in exposure estimates that are on average 30% and 170% of those using mid-point values respectively. Note that these are lower and upper bounds on day-to-day variability of exposure since they were calculated assuming that pollution and time spent inside are simultaneously at their lowest (or highest) levels. In practice day-to-day variability is likely to vary in a smaller range than 30% – 170% × mid-value .

[31] High-intensity exposure is defined as exposure during those times when: 1) a person is very close to the stove, either directly above it or within a distance of less than 0.4 – 0.5 m and 2) emissions are the highest, within the upper 25th percentile (i.e. moments when average emission concentration is characterized by $\mu_{>75}$).

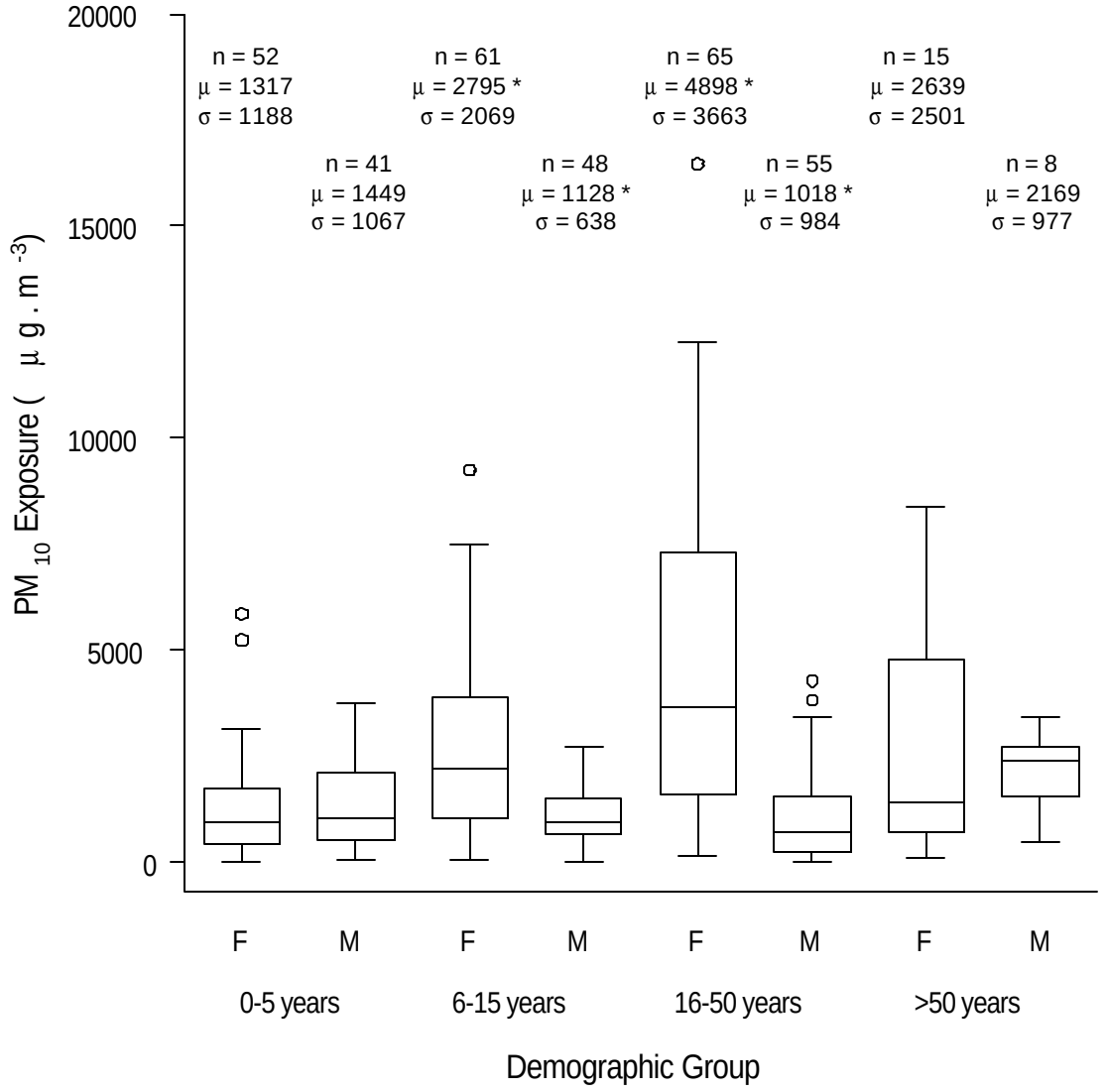
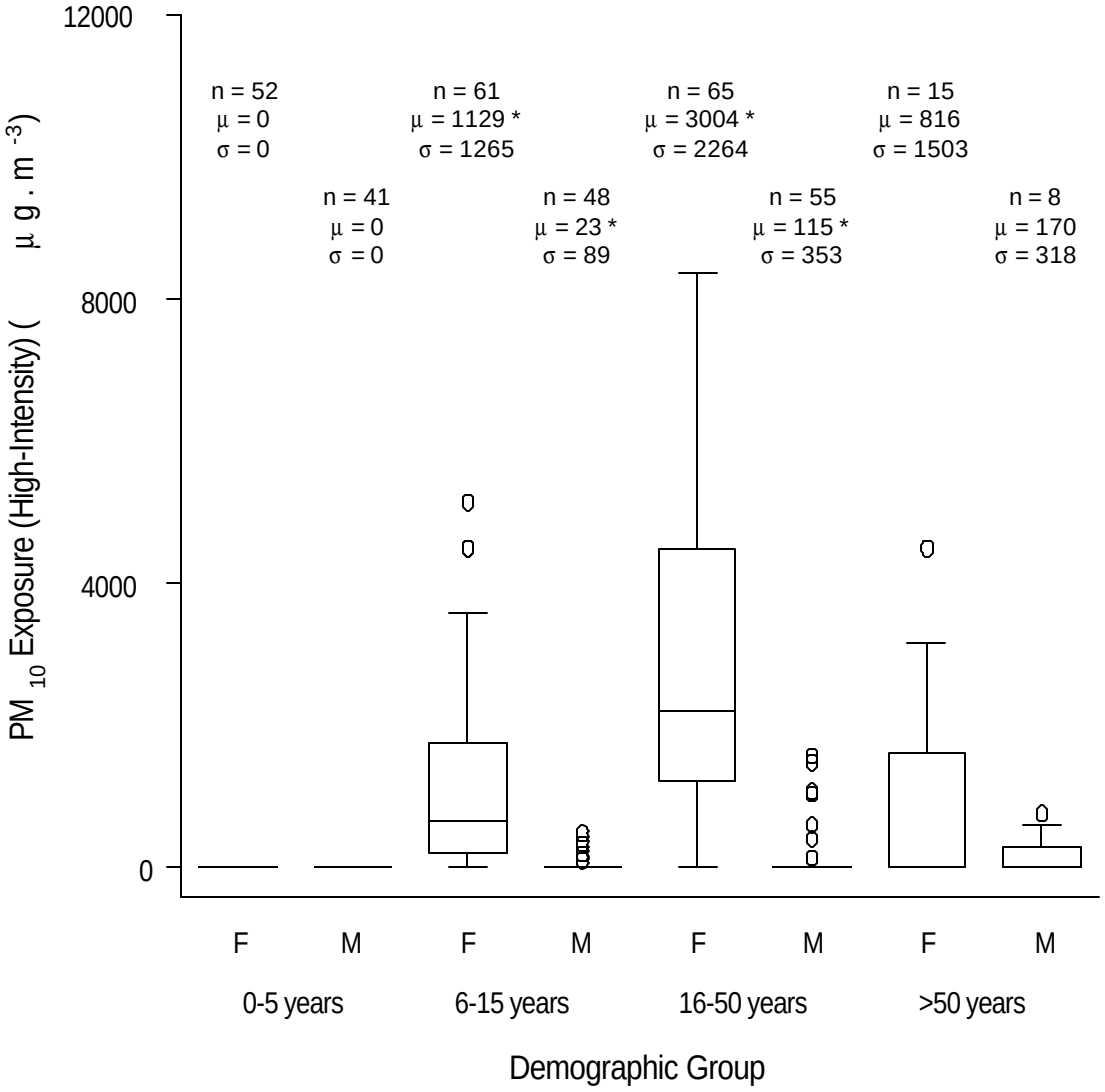
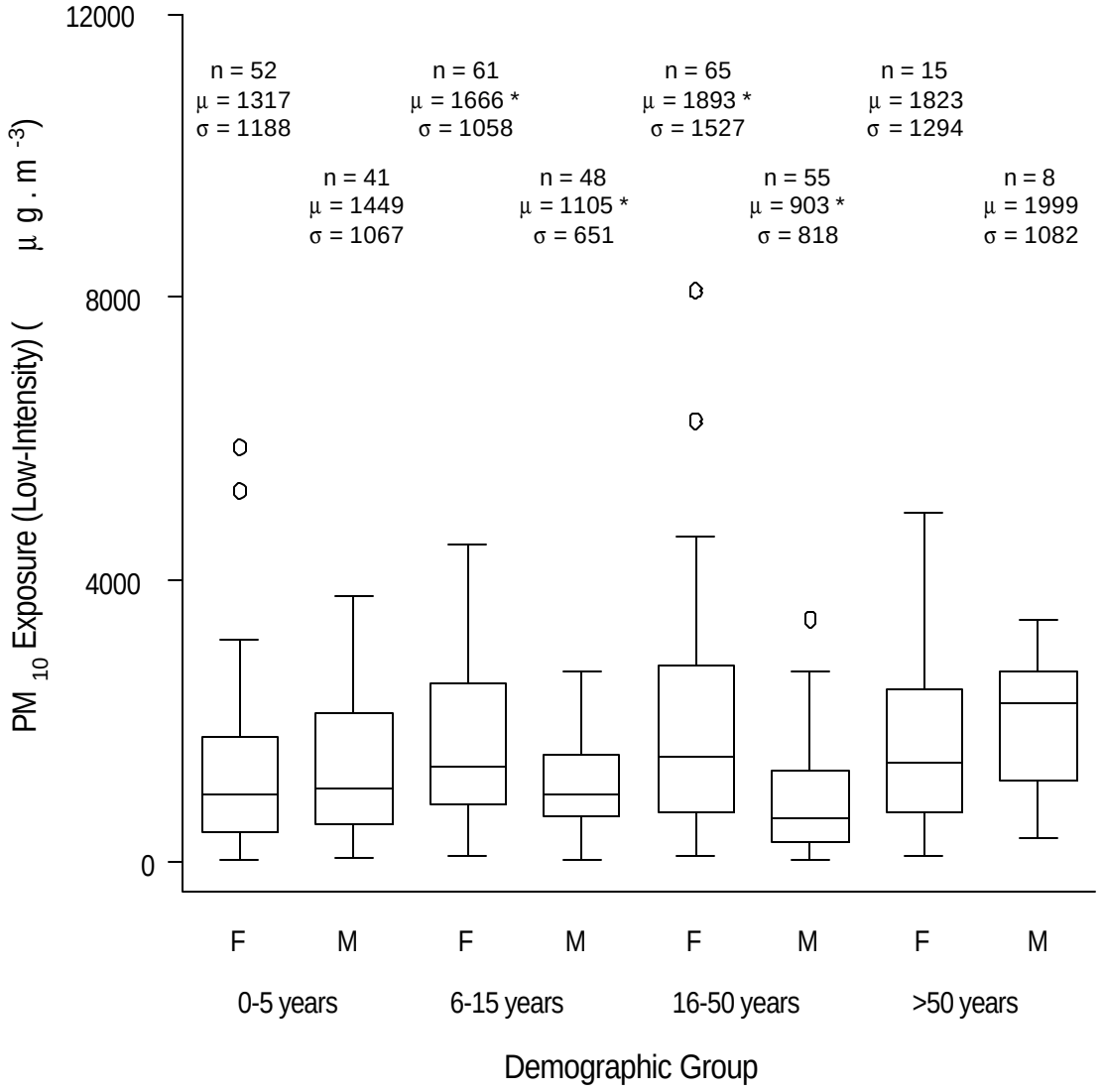


Figure 6.6: Average exposure concentration for total daily exposure to PM_{10} obtained using the exposure profile approach. *Average exposure concentration* is the PM_{10} concentration that if sustained for the whole (24-hour) day would result in exposure equal to total daily exposure of the individual. The box-plot, used in this and subsequent figures, shows a summary of the distribution of the variable. The lower and upper sides of the rectangle show the 25^{th} and 75^{th} percentiles and therefore enclose the middle one half of the distribution. The middle line, which divides the rectangle into two, is the median. The circles above (and below of which there are none in this figure) the outer two lines show the "outliers". *n* refers to the number of individuals in the demographic subgroup; *m* is the sample mean and σ the standard deviation. * indicates that the difference between male and female values is significant with $p < 0.0001$.


12000
8000
4000
0
PM 10 Exposure (High-Intensity) (μ g . m -3)
n = 52
μ = 0
σ = 0
n = 41
μ = 0
σ = 0
n = 61
μ = 1129 *
σ = 1265
n = 48
μ = 23 *
σ = 89
n = 65
μ = 3004 *
σ = 2264
n = 55
μ = 115 *
σ = 353
n = 15
μ = 816
σ = 1503
n = 8
μ = 170
σ = 318
F
M
F
M
F
M
F
M
0-5 years
6-15 years
16-50 years
>50 years
Demographic Group

(a)



(b)

Figure 6.7: Contribution of (a) high-intensity exposure episodes and (b) low-intensity exposure to total daily exposure to PM_{10} (i.e. Figure 6.6). Note that the high-intensity component of exposure occurs in less than one hour, emphasizing the intensity of exposure in these episodes. *n* refers to the number of individuals in the demographic subgroup; *m* is the sample mean and σ the standard deviation. * indicates that the difference between male and female values is significant with $p < 0.0001$.

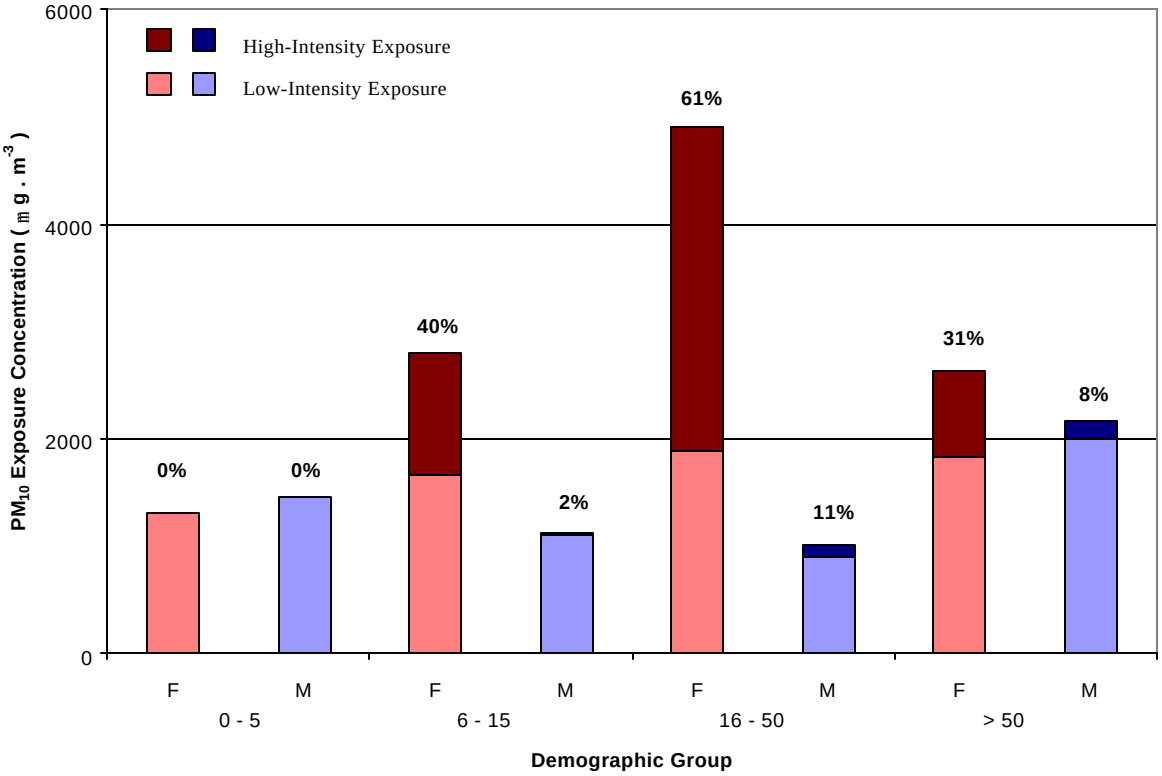


Figure 6.8: Breakdown of total daily exposure to PM10 (i.e. Figure 6.6) to high-intensity exposure and low-intensity exposure. For each demographic group the height of the column is the group average from Figure 6.6. The two (high- and low-intensity) components are the group averages from Figure 6.7a and Figure 6.7b. The numbers indicate the share of total exposure from high-intensity exposure. Note that the high-intensity component of exposure occurs in less than one hour, emphasizing the intensity of exposure in these episodes.

The ratios of high-intensity exposure to total exposure for the four age groups are 0, 0.40, 0.61, and 0.31 for females and 0, 0.02, 011, 0.08 for males. The larger value for young and adult women illustrates that high-intensity emission episodes account for a considerably larger fraction of exposure of those household members who are closest to fire at such times (and also much larger in absolute values since female exposure has larger base values).

6.5 Comparison with the Common Method of Exposure Estimation

Finally, in Figure 6.9, I compare the above exposure values to those obtained using only average emissions at a single point and time spent inside (i.e. without taking into account either the spatial distribution of pollution or the role of time-activity patterns on exposure).

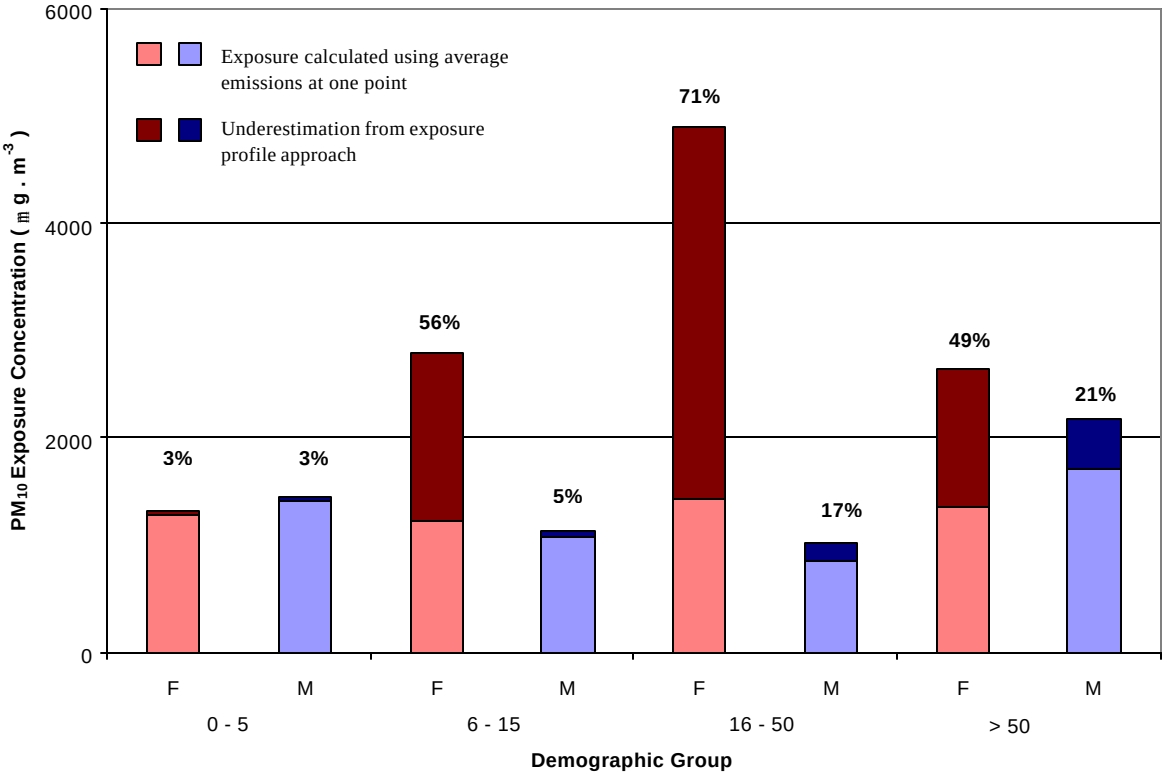


Figure 6.9: Comparison of exposure values using the exposure profile approach (i.e. Figure 6.6) to those using average emissions at a single point and time spent inside. For each demographic group the height of the column is the group average from Figure 6.5. The lower part is exposure calculated using average emissions at a single point. Therefore, the upper part is the underestimation of exposure using this method relative to the exposure profile approach, also shown as a percentage.

The ratios of exposure estimates using average emissions at only a single point to those using the exposure profile approach for the four age groups are 0.97, 0.44, 0.29, 0.51 for females and 0.97, 0.91, 0.83, 0.79 for males. The large variation of this ratio among the

different demographic groups indicates that ignoring the spatial distribution of pollution and the role of activity patterns on exposure not only results in inaccurate estimates of exposure but also – and possibly more importantly – biases the relative exposure levels for different demographic groups. The exposure of women, who cook and are most affected by high-intensity pollution episodes, is underestimated most severely by using average pollution alone. This would in turn result in systematic bias in assessment of the health impacts of exposure and benefits from any intervention strategy.

6.6 Verification of Exposure Estimates

Throughout this chapter, I have used quantitative and qualitative data on time-activity budgets and daily pollution profiles to construct measures of exposure for individual household members. This approach to exposure assessment, although more encompassing of the physical and social realities of exposure to indoor smoke, cannot be verified internally. Further, to be tractable, it continues to use simplifying assumptions such as specific cut-off points for the high intensity emissions episodes, assignment of individual time-activity budgets to activity categories, and assignment of households to pollution categories.

Rapid advances in monitoring technology will soon produce real-time particulate matter monitors that are small enough to be carried by individuals. Simultaneous use of personal and multiple stationary monitors will allow independent measurements of personal exposure and pollution, which will in turn provide the most reliable test for any exposure assessment methodology and an empirical guideline for the set of assumptions

that I have made. Finally, research is also needed on how exposure varies over time, at various scales. In particular, laboratory and field monitoring should focus on the variation of emission concentrations in individual households which will in turn result in plausible estimates of exposure variability from day to day or season to season.

Chapter 7 Exposure-Response Relationship [32]

Design and implementation of measures to reduce the adverse health impacts of exposure to indoor air pollution requires knowledge of the relationship between exposure and health outcomes, or the exposure-response relationship, along a continuum of exposure levels.

As I briefly discussed in Chapter 2, research on the health impacts of indoor air pollution in developing countries has been hindered by a lack of detailed data on both exposure and health outcomes. In these settings, many epidemiological studies have used indirect and often inaccurate measures, such as fuel or housing type, as proxies for personal exposure in cross-sectional studies (See for example 28, 29, 30, 31, 32, 33) (For a discussion of this issue see 34). Given the nearly universal use of biomass fuels in rural areas, this indirect approach to exposure estimation clusters numerous people into a single exposure category. But recent findings on large variations in emissions from individual stove types (14, 35) (see also Chapter 8) and in exposure profiles within individual households (Chapter 6) (36, 37, 38) demonstrate that aggregate analysis and grouping of individuals artificially reduces the variability of the explanatory variable in the exposure-response relationship, and therefore the reliability of the estimation of its parameters.

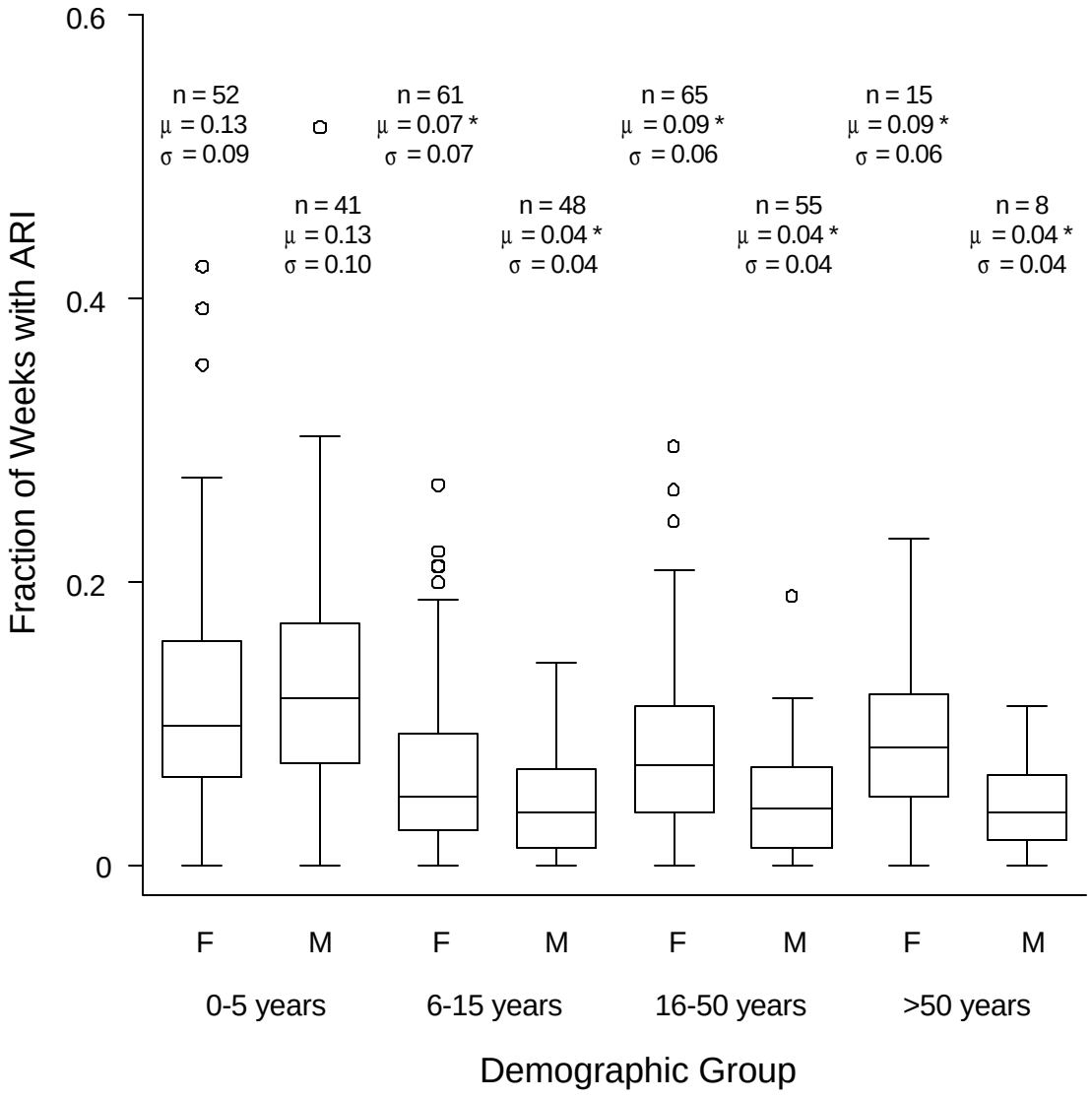
---

[32] Some of the material in this chapter has been published in the following articles: Ezzati, M. and D. M. Kammen (2000) "An Exposure-Response Relationship for Acute Respiratory Infections as a Result of Exposure to Particulates from Biomass Combustion," *The Lancet*, submitted. Ezzati, M., D. M. Kammen, and B. H. Singer (1999) "The Health Impacts of Exposure to Indoor Air Pollution from Biofuel Stoves in Rural Kenya," *The Proceedings of Indoor Air 99: the 8th International Conference on Indoor Air Quality and Climate; Edinburgh, Scotland; August 1999*, **3**, 130-135.

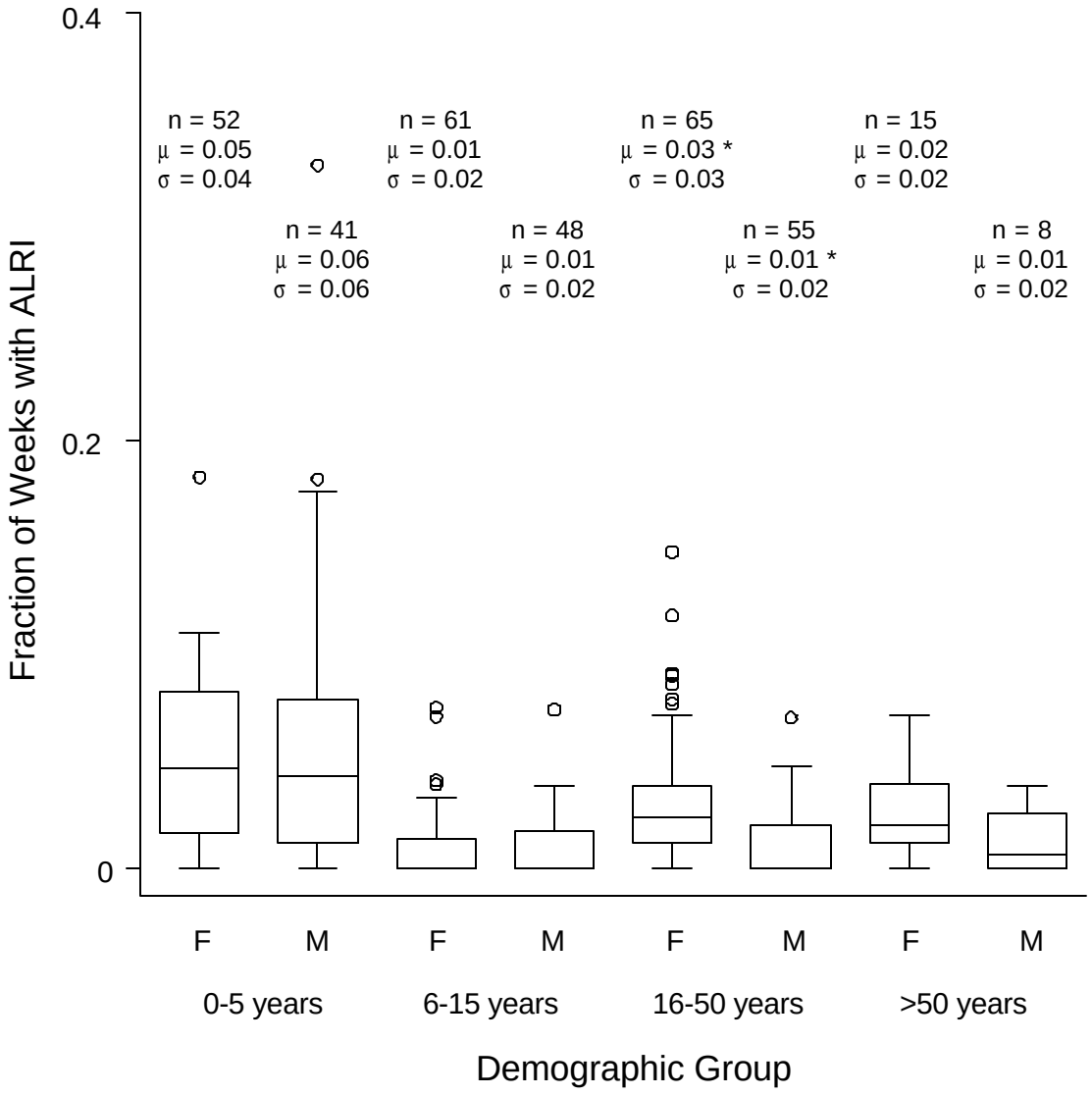
In this work, monitoring of both exposure to indoor air pollution and health status at the level of the individual permits quantifying the exposure-response relationship for indoor particulate matter along a continuum of exposure levels.

7.1 Demographic Distribution of Illness

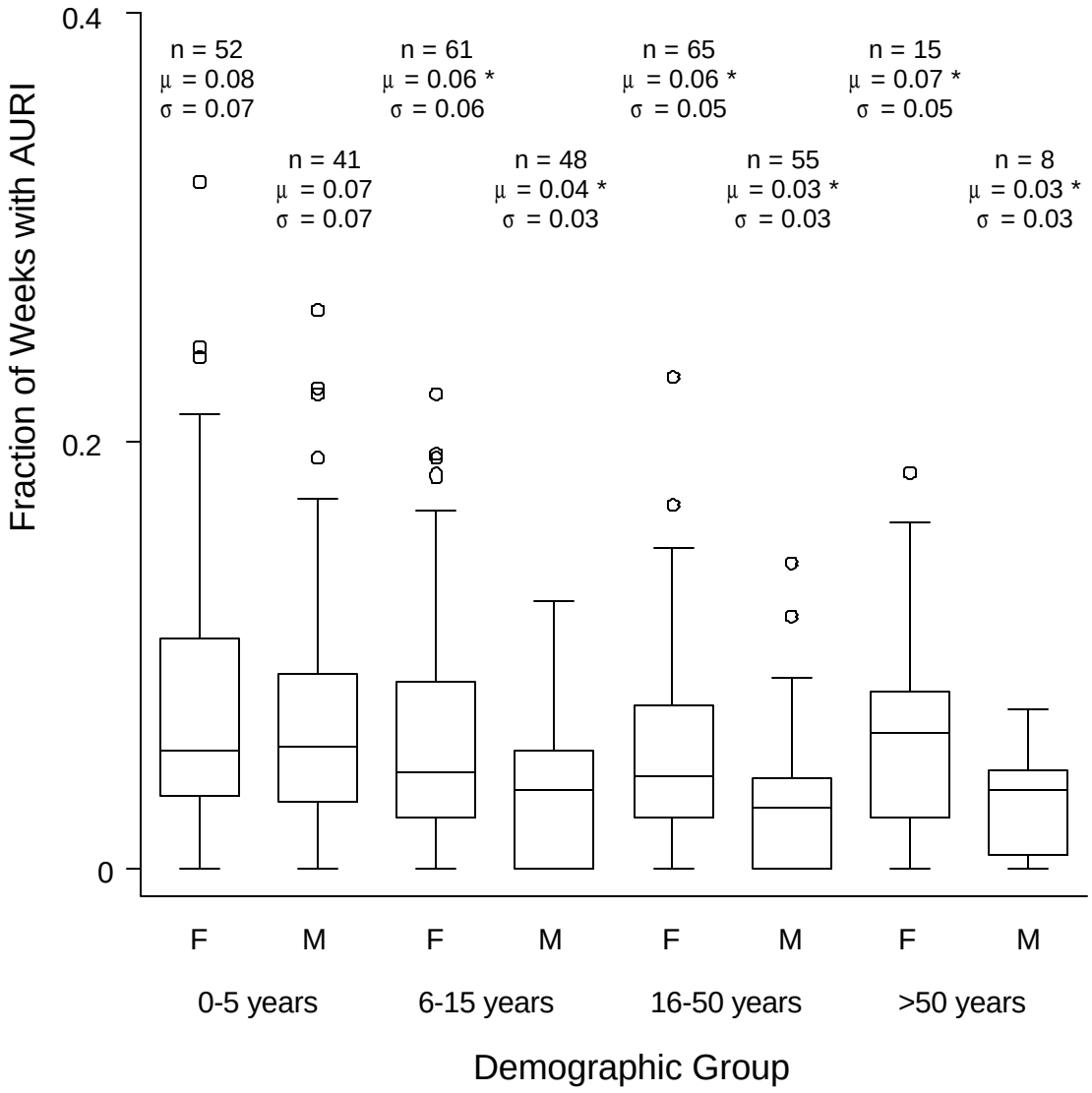
The health outcome used in this analysis is the fraction of weeks that an individual is diagnosed with an illness and is referred to as illness rate. Figure 7.1 provides summary statistics on acute respiratory infections (ARI), acute lower respiratory infections (ALRI), acute upper respiratory infections (AURI), and eye disease (including cataracts and conjunctivitis) rates for the different demographic groups. The female-male comparisons illustrate that after age 5 women are approximately twice as likely as men to be diagnosed with ARI or ALRI (see Figure for details).


0.6
0.4
0.2
0
Fraction of Weeks with ARI
n = 52
μ = 0.13
σ = 0.09
n = 41
μ = 0.13
σ = 0.10
n = 61
μ = 0.07 *
σ = 0.07
n = 48
μ = 0.04 *
σ = 0.04
n = 65
μ = 0.09 *
σ = 0.06
n = 55
μ = 0.04 *
σ = 0.04
n = 15
μ = 0.09 *
σ = 0.06
n = 8
μ = 0.04 *
σ = 0.04
F
M
F
M
F
M
F
M
0-5 years
6-15 years
16-50 years
>50 years
Demographic Group

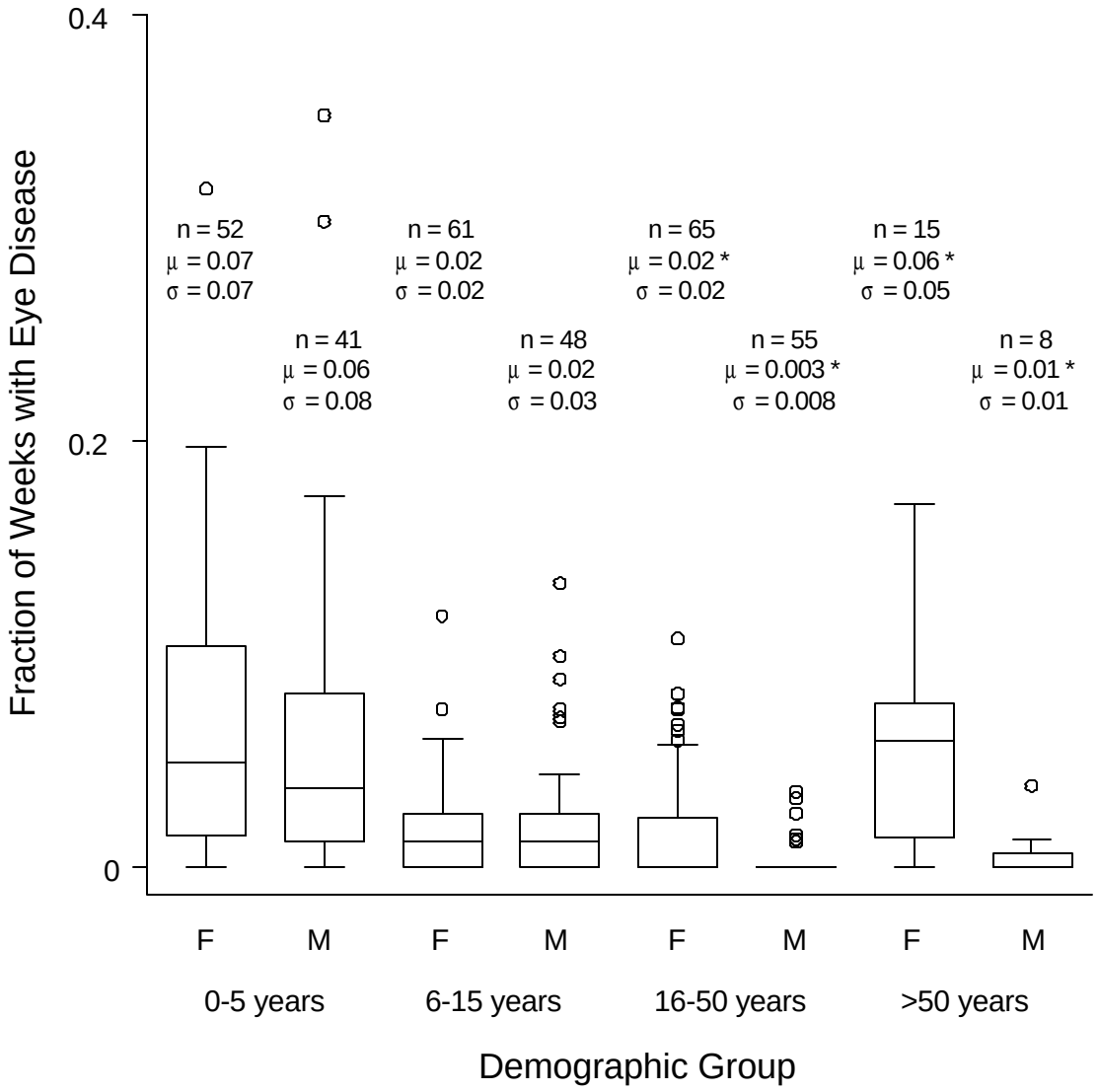
(a)


0.4
0.2
0
Fraction of Weeks with ALRI
n = 52
μ = 0.05
σ = 0.04
n = 41
μ = 0.06
σ = 0.06
n = 61
μ = 0.01
σ = 0.02
n = 48
μ = 0.01
σ = 0.02
n = 65
μ = 0.03 *
σ = 0.03
n = 55
μ = 0.01 *
σ = 0.02
n = 15
μ = 0.02
σ = 0.02
n = 8
μ = 0.01
σ = 0.02
F
M
F
M
F
M
F
M
0-5 years
6-15 years
16-50 years
>50 years
Demographic Group

(b)


0.4
0.2
0
Fraction of Weeks with AURI
n = 52
μ = 0.08
σ = 0.07
n = 41
μ = 0.07
σ = 0.07
n = 61
μ = 0.06 *
σ = 0.06
n = 48
μ = 0.04 *
σ = 0.03
n = 65
μ = 0.06 *
σ = 0.05
n = 55
μ = 0.03 *
σ = 0.03
n = 15
μ = 0.07 *
σ = 0.05
n = 8
μ = 0.03 *
σ = 0.03
F
M
F
M
F
M
F
M
0-5 years
6-15 years
16-50 years
>50 years
Demographic Group

(c)



(d)

Figure 7.1: Demographic distribution of illness rates in the study group. (a) Acute respiratory infections (ARI). (b) Acute lower respiratory infections (ALRI), including bronchitis, pneumonia and broncho-pneumonia. (c) Acute upper respiratory infections (AURI). (d) Eye disease (including cataracts and conjunctivitis). The health outcome is the fraction of weekly examinations in which an individual was diagnosed with the corresponding illness. *n* refers to the number of individuals in the demographic subgroup; *m* is the sample mean and *s* the standard deviation. * indicates that the difference between male and female values is significant with $p < 0.05$.

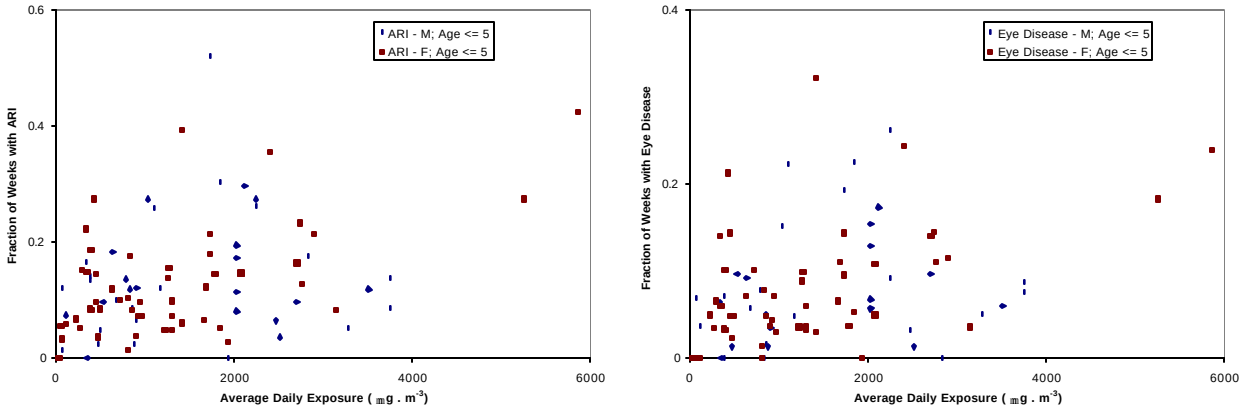
7.2 Exposure-Response Relationship: Modeling

Figure 7.2 and Figure 7.3 plot illness rates against daily exposure (as calculated in Chapter 6). The exposure values in these figures are those calculated using the mid-points of pollution concentrations and time-budget categories for each individual.

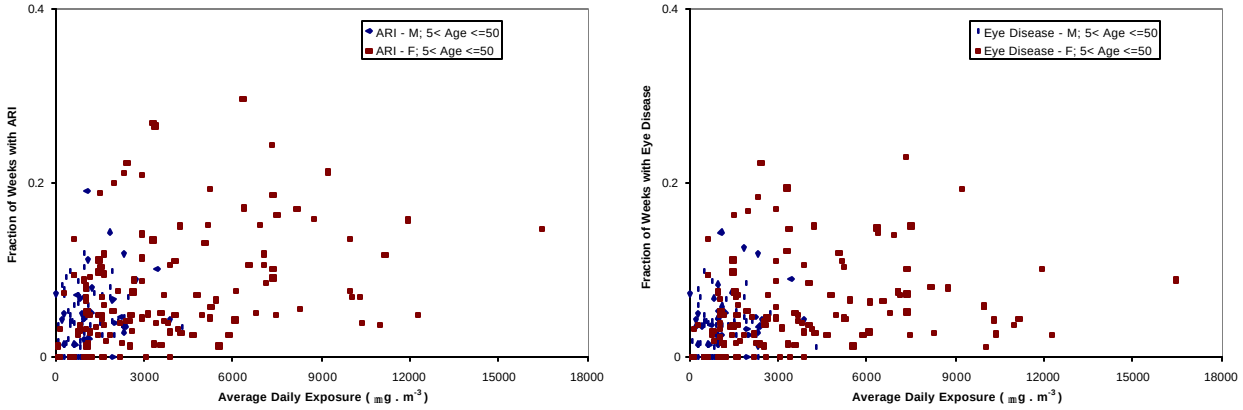
Using time-averaged exposure is a common practice in the literature on toxicity and health risk (especially for research on carcinogens) (140, 154). As I described in detail in Chapter 6, however, personal exposure to biomass smoke varies from day to day due to variation in both pollution levels and time-activity budget. To account for this variability, as well as error or uncertainty in the estimates of average exposure, in the remainder of this chapter, I assign individuals to exposure categories in addition to using exposure levels directly.

7.2.1 Exposure categorization

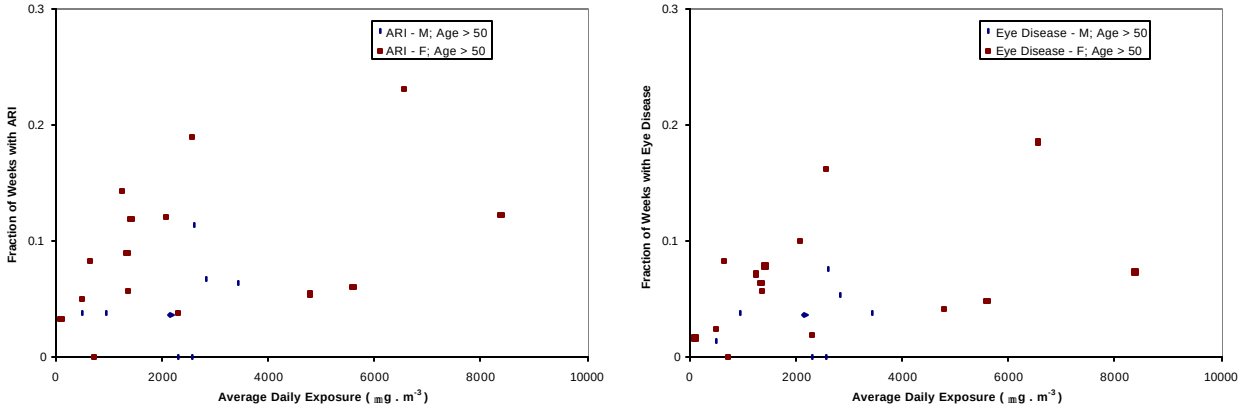
Division of exposure into discrete categories should be based on exposure ranges that satisfy the opposing criteria of being as large as possible to account for exposure variability but as small as possible to avoid grouping of individuals with characteristically different exposure patterns together. Given the larger absolute variability of exposure values at the higher levels, one would also expect an increasing size for exposure bins at higher exposure levels. Finally, the shape of the exposure-response curve should be robust to marginal changes in exposure categories.



(a)

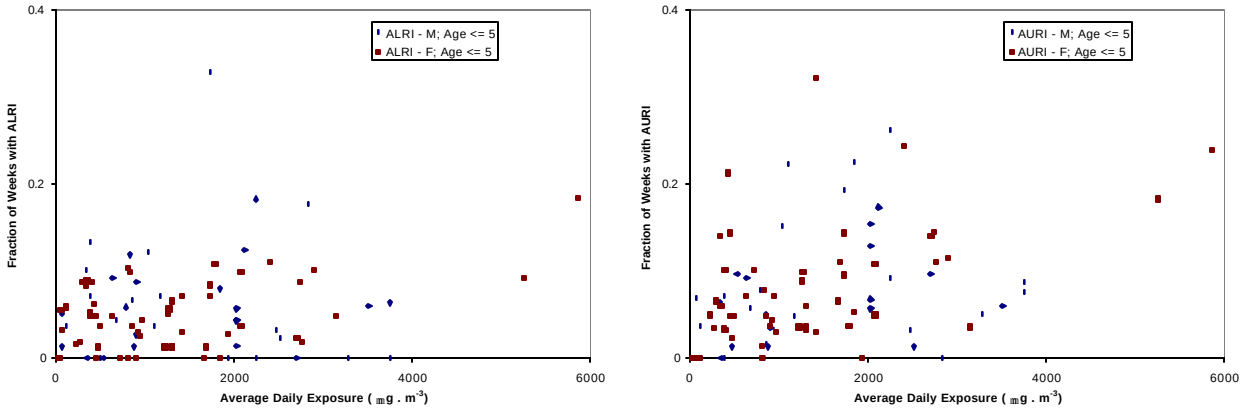


(b)

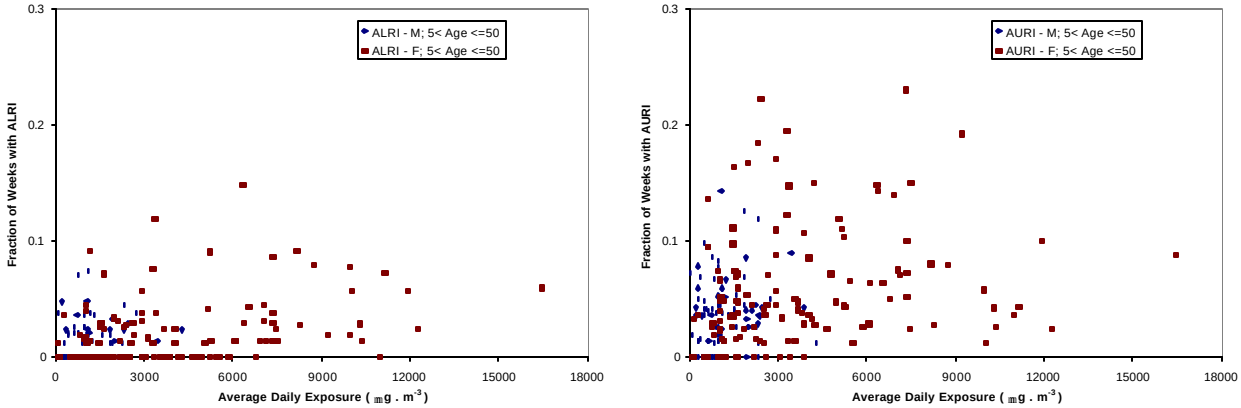


(c)

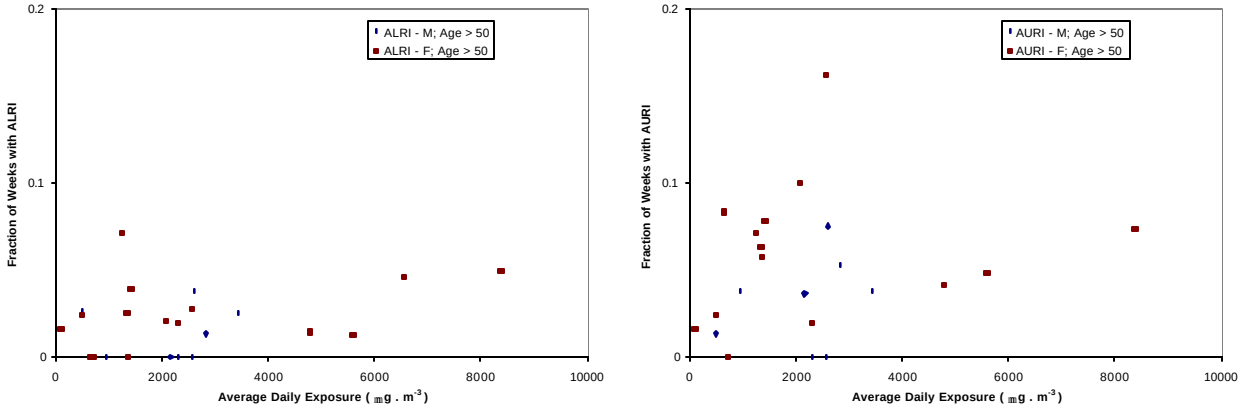
Figure 7.2: Exposure-illness plots for ARI and eye disease (including cataracts and conjunctivitis). (a) Age ≤ 5. (b) 5 < Age ≤ 50. (c) Age > 50. Exposure values are the mid-points of average daily exposure calculated in Chapter 6.



(a)



(b)



(c)

Figure 7.3: Exposure-illness plots for ALRI and AURI (break-down of ARI from Figure 7.2). (a) Age ≤ 5. (b) 5 < Age ≤ 50. (c) Age > 50. Exposure values are the mid-points of average daily exposure calculated in Chapter 6.

In Chapter 6, I found that the lower and upper bounds on exposure variability are on average 0.3 and 1.7 times the mid-values, resulting in exposure ranges that are 1.4 times the mid-value. Figure 7.4 plots the exposure-response relationship (for ARI and ALRI) using exposure categories that are approximately based on this value, as well as those that are fractions of this value.[33] For each exposure category, the mean and median of illness rates are plotted against the average exposure of individuals in the category.

Comparison of these figures shows that:

- The largest exposure categories (Figure 7.4 a) maintain the general shape of the exposure-response relationship but mask some of the changes in the slopes in each region.
- The smallest exposure categories (Figure 7.4 c) result in local fluctuations in the exposure-response curve. But this is often due to small sample size in some of the categories. In particular, the points where the curve for adults deviates from its overall trend (4000 – 5000 category and > 9000 category) have sample sizes of 9 and 11 respectively. For infants, there are 7 individuals above the exposure of 3000, only 2 of whom have exposures greater than 4000.
- The difference between the exposure-response curve obtained using mean and median illness rates is small.

[33] The plots are for age groups 0 – 5 and 6 – 50. I exclude those above the age of 50 from separate analysis since the number of individuals in this group is small and their exposure values have the highest uncertainty.

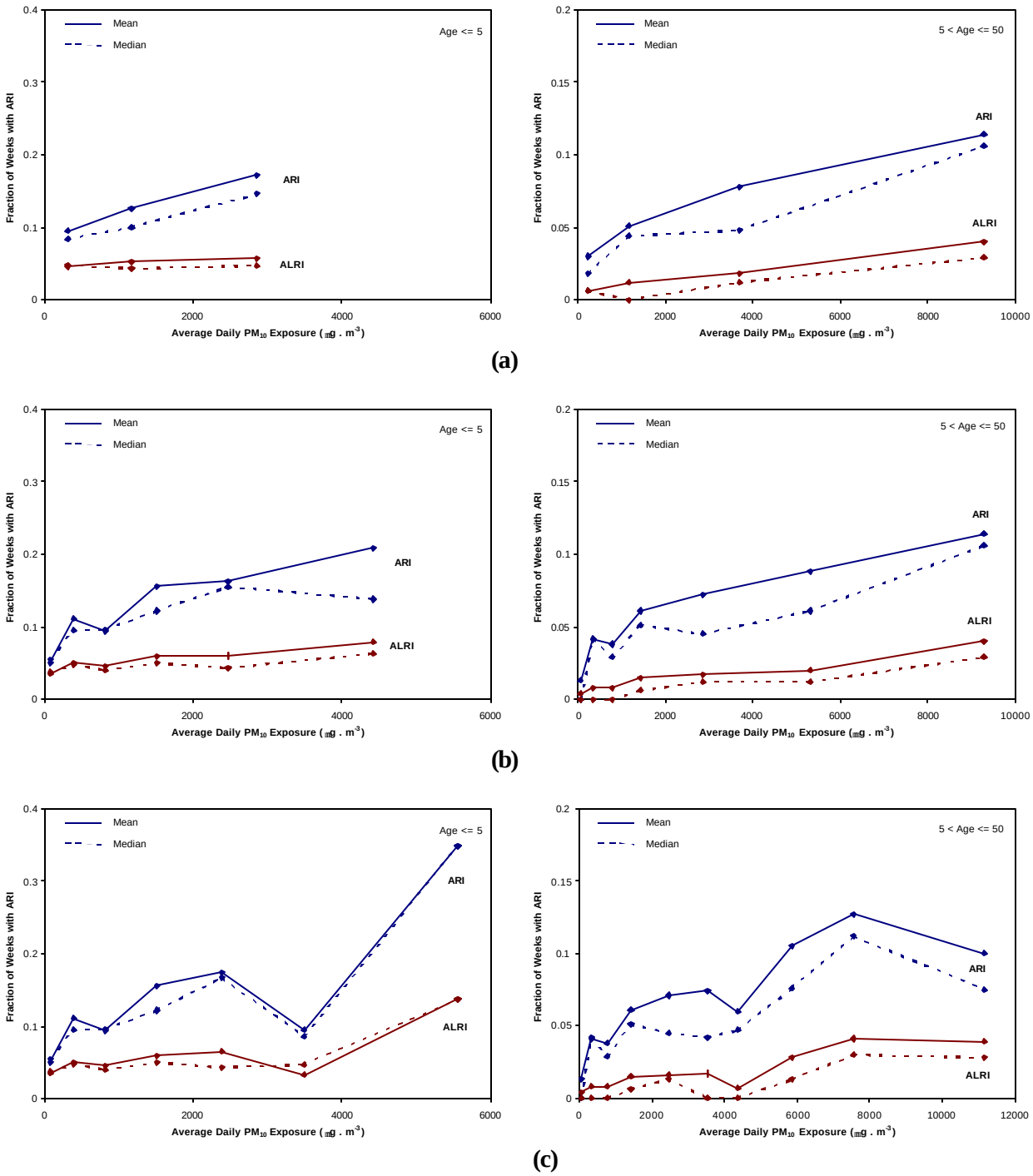


Figure 7.4: Exposure-response plots for ARI and ALRI after exposure categorizations for Age ≤ 5 (left column) and 5 < Age ≤ 50 (right column). The width of the exposure categories (with the exception of the first category which always has a width to mid-point value of 2) are approximately: (a) 1.1 – 1.2 times the mid-value: (0 – 500, 500 – 2000, > 2000 for (column 1) and 0 – 500, 500 – 2000, 2000 – 7000, and > 7000 for (column 2); (b) 0.55 – 0.86 times the mid-value: 0 – 200, 200 – 500, 500 – 1000, 1000 – 2000, 2000 – 3500, > 3500 for (column 1) and 0 – 200, 200 – 500, 500 – 1000, 1000 – 2000, 2000 – 4000, 4000 – 7000, > 7000 for (column 2);

and (c) 0.22 – 0.86 times the mid-value: and 0 – 200, 200 – 500, 500 – 1000, 1000 – 2000, 2000 – 3000, 3000 – 4000, > 4000 for (column 1) 0 – 200, 200 – 500, 500 – 1000, 1000 – 2000, 2000 – 3000, 3000 – 4000, 4000 – 5000, 5000 – 7000, 7000 – 9000, > 9000 for (column 2). For each category, mean and median of illness rates of all the individuals in the exposure category are plotted against the within-group exposure mean. The exposure of 6 – 50 age group reaches higher levels than the 0 – 5 group due to participation in cooking activities.

Based on these findings, and to satisfy the criteria of exposure ranges which are small enough to capture changes in the slope of the exposure-response curve and large enough not to be sensitive to statistical noise, I choose the categorization of Figure 7.4 b. Throughout the remainder of this chapter, I analyze the relationship between exposure to PM_{10} and illness based on these exposure categories as well as continuous treatment of exposure.

7.2.2 Exposure-response graphs

Figure 7.5 shows the exposure-response relationships for ARI (divided into AURI and ALRI) and eye disease for age groups 0 – 5 and 6 – 50 (using the categorization of Figure 7.4b). It can be seen that for both age groups, ARI, ALRI, and AURI rates are increasing functions of exposure but rise more rapidly for exposures below 2000 $\mu g.m^{-3}$. For age ≤ 5, ARI and ALRI rates in the 0 – 200 $\mu g.m^{-3}$ exposure category are respectively 0.11 ($p < 0.01$) and 0.024 ($p = 0.18$) lower than those in the 1000 – 2000 $\mu g.m^{-3}$ group. The increase between the latter group and the highest exposure category (> 3500 $\mu g.m^{-3}$) is

only 0.05 for ARI (p = 0.49) and 0.02 for ALRI (p = 0.57).[34] For the 6 – 50 age group, ARI and ALRI rates increase by 0.048 ($p < 0.0001$) and 0.011 ($p < 0.01$) between the lowest exposure group and 2000 $\mu g.m^{-3}$ compared to 0.053 ($p < 0.001$) and 0.025 ($p < 0.001$) between the latter group and the > 7000 $\mu g.m^{-3}$ category, in an exposure range four times as large. For eye diseases, the same patterns exists for age ≤ 5, but the change in slope occurs at a lower exposure compared to ARI, around 500 $\mu g.m^{-3}$. For 5 < age ≤ 50, no obvious relationship between eye diseases and exposure can be observed above the 500 $\mu g.m^{-3}$ exposure level.

7.2.3 Methodological issues in quantification of the exposure-response relationship

Confounding effects on exposure

An important concern in studies of indoor air pollution and health has been the role of confounding, especially in the form of correlation between exposure and other determinants of health such as socioeconomic status and nutrition (34). In particular, there is evidence that poorer households, who may have additional susceptibility to disease, cook using more polluting sources of energy and live in poorer housing conditions (26, 155). Although empirical research has demonstrated that household choice of energy technology is also determined by a set of social and cultural factors (156), income is indeed an important determinant of exposure (155).

[34] In this specific comparison, although the large p-values are partially due to the small fraction of children in the highest exposure category, they are also a reflection of the smaller slope of the exposure-response relationship.

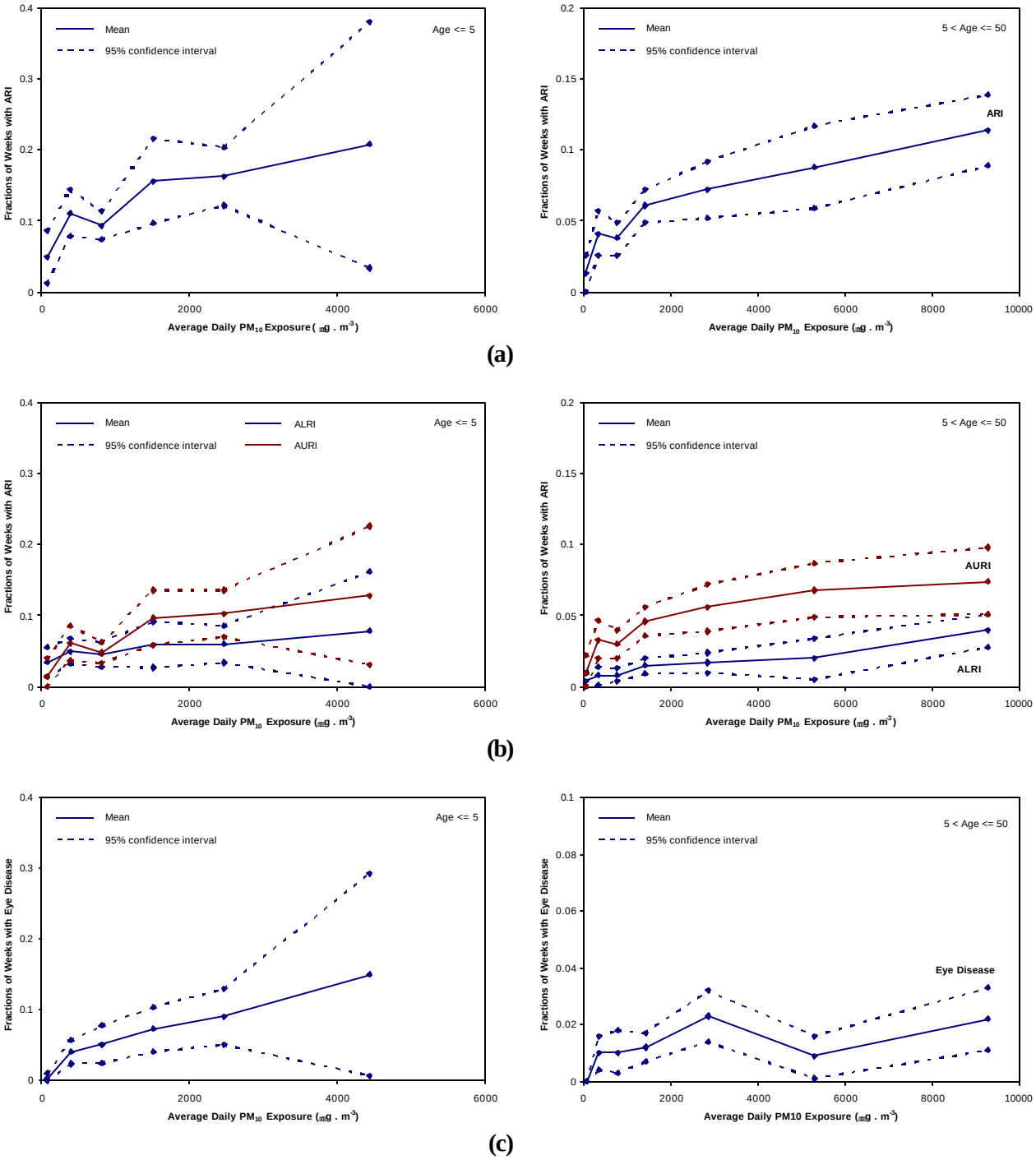


Figure 7.5: Exposure-response plots for age ≤ 5 (column 1) and 5 < Age ≤ 50 (column 2). (a) ARI. (b) ALRI and AURI. (c) Eye diseases. Exposure categories correspond to Figure 7.4b. Mean ARI and ALRI rates for each exposure category are plotted against the average exposure of the category.

Incomes vary in a small range among the residents of Mpala Ranch, except for a few skilled workers. Further, since part of the income is paid in-kind as food, the variation in nutrition is also smaller than many other communities. Incomes are similar between the two groups of villages (maintenance and cattle-herding) and people are moved between village types at the instruction of ranch management without changes in earning. Houses are assigned by the management and within each village type are nearly identical. Therefore, village type and housing are not endogenous variables and are not expected to be correlated with income.

As I discussed in Chapter 4, with the exception of occasional use of paraffin, firewood and charcoal are the exclusive fuels at Mpala Ranch. Further, access to the traders from the neighboring community of *Naibo* is an important determinant of access to charcoal. For this reason, charcoal consumption is mostly concentrated in the two maintenance villages as well as among those households who have regular contact with these villages because of their work. For example, some of the households who moved to a maintenance village from a *boma* did change their fuel consumption, despite maintaining constant income. Therefore, with the relatively small range of incomes, the use of charcoal or wood is partially determined by the location of the specific village that a family lives in, which as I have discussed above is exogenous.

It is nonetheless possible that other factors also influence the choice of fuel, especially since there is variation in fuel use within maintenance villages themselves. If these factors are not correlated with health (such as preference for a specific taste of food) then

the issue of endogenous exposure is not a concern. If some of the determinants of fuel use are correlated with health, such as the education of mother, then the problem of endogeneity remains. In our interviews on fuel use, the commonly stated reasons for choice of fuel were uncertainty about future access, the taste of food, cost of charcoal, and difficulty of wood collection. Since no household level variable which is correlated with health could be specified as the determinant of fuel choice and since very few households used charcoal exclusively (most charcoal users had a mixed fuel profile), in this analysis I treat the choice of fuel as exogenous. I nonetheless control for the type of village that a household lives in, to account for any unobservable differences between the two.

Clustering

The health and exposure data in this work may be characterized by two levels of clustering: clustering of individuals within households and clustering of households within villages. The determinants and outcome of health status are likely to exhibit similarity within a single household. Clustering within villages is a less likely phenomenon. No physical attribute, such as rainfall or temperature, is specific to individual villages or *bomas*. Further, *bomas* move and their size changes regularly depending on climate and other factors and households are moved among them. Therefore, consideration of clustering is limited to the household level.

7.2.4 Exposure-response models

The parameters of the exposure-response relationship are estimated for the following two models:

1) $$\mathbf{y} = \mathbf{X} \cdot \hat{\mathbf{a}} + \mathbf{u} \qquad (7.1)$$

where **y** is the (N × 1) vector of illness rates for all the individuals in the study group, **X** a (N × k) matrix of characteristics for the individuals in the study group, **b** the (k × 1) vector of coefficients, and **u** the (N × 1) vector of independent, normally distributed errors, and

2) $$\mathbf{y} = F\,(\mathbf{X} \cdot \hat{\mathbf{a}} + \mathbf{u}) \qquad (7.2)$$

where **y**, **X**, and **b** are defined as above, and F is the cumulative logistic distribution defined as:[35]

$$F(z) = \frac{\exp(z)}{1+\exp(z)} \qquad (7.3)$$

Model parameters are obtained using ordinary-least-squares (OLS) regression for model 1 with clustering in households and robust standard error estimates that account for outliers. For model 2, a *blogit* regression using maximum-likelihood estimation is used. *blogit* regression also allows accounting for the increasing confidence in illness rates with increasing number of visits.[36]

---

[35] In a logit or logistic regression model the left hand side of Equation 7.2 is the probability of an event **y** (such as illness), or Pr{**y**}. Here, since the outcome is defined as the fraction of time with illness, equivalent to probability of illness, the left hand side is simply **y**.

[36] The number of times that an individual is diagnosed with illness in n examinations has a binomial distribution. Illness rate, y, defined as the fraction of examinations with illness, is then an estimate for the probability of being diagnosed with illness, p. The confidence interval for p is obtained from an

7.2.5 Model variables

The characteristics of individuals considered in the analysis include:

- *Average exposure to PM_{10}*: as the main explanatory variable.
- *Gender*: to account for potential female-male susceptibility differences.
- *Age*: to account for impacts of age on immunity or the chronic impacts of long-term exposure.
- *Village type*: Although income and nutritional status are very similar between the residents of maintenance villages and *bomas*, differences that are unobservable to the researcher and can influence disease rates may exist.
- *Number of people residing in the house*: Due to the communicable nature of acute respiratory infections, living in more crowded environments would be expected to facilitate transmission. Since house sizes are standardized within each village type, the number of residents of each house is a proxy for crowding.
- *Smoking*: Smoking is a known causal agent of respiratory infections. The number of smokers at Mpala ranch is very low (13 in the sample of households used in this analysis), both because of the cost of cigarettes and the fact that *miraa* (described in chapter 4) provides a ready alternative.

Statistical summaries for exposure and demographic characteristics are provided in Chapters 5 and 6. The fraction of households and individuals living in the *bomas* are 0.56 and 0.66 respectively. The mean, median, and standard deviation of the number of

---

approximately normal distribution around y with variance $y\ (1 - y) / n$. The variance and the confidence interval are therefore decreasing functions of the number of visits, n (157).

people living in a house are 7.0, 7.0, and 2.2 in the cattle-herding villages and 5.3, 5.0, and 2.0 in the maintenance villages.[37]

In addition to these characteristics, I considered two exposure-related variables that would characterize individual exposure beyond its average daily value. These two variables are the level of participation of an individual in household tasks, with emphasis on cooking related tasks, and the intensity of exposure during the most intense exposure episodes.

- *Participation in household tasks* is a categorical variable which divides individuals into four groups: those who do not perform any household tasks; those who participate in some household tasks, such as water collection or cleaning the house, but none that involve the use of the stove; those who sometimes use or tend the stove but not on regular basis; and finally individuals who participate in cooking-related tasks regularly.
- *Exposure intensity* is defined as the concentration during an individual's most intense exposure episode. For those who participate in household tasks, this equals the pollution concentration in the area immediately around the stove during the times that stove has its highest pollution level (i.e. concentration is characterized by $\mu_{>75}$). For those who do not participate in cooking-related tasks, exposure intensity is simply their average daily exposure. I consider exposure intensity as both continuous and categorized variables. In the latter approach, exposure intensity is divided into four

[37] Four of the households in the *bomas* owned two huts because either a second wife or older unmarried children lived in the house. For these households, the number of people per house was counted based on two houses.

categories corresponding to concentrations of 0 – 20,000 $\mu g.m^{-3}$, 20,000 – 50,000 $\mu g.m^{-3}$, 50,000 – 100,000 $\mu g.m^{-3}$, and > 100,000 $\mu g.m^{-3}$.

Therefore, the above two variables are indicators of the length and intensity of exposure to high concentrations of PM_{10} respectively.

7.3 Exposure-Response Relationship: Parameter Estimation

7.3.1 OLS estimation

In Table 7.1 to Table 7.4, I report the parameter estimates using OLS regression, for both continuous and categorical treatments of average exposure and exposure intensity, for the 0 – 5 and 6 – 50 age groups.[38] In the continuous case, I have considered exposure in a linear and inverse quadratic manner to account for the declining slope of the exposure-response relationship observed in Figure 7.2 to Figure 7.4.[39] Model 1 (bivariate) corresponds to parameter estimates in which illness rates are regressed against each variable one at a time (i.e. gross effects).[40] Model 2 includes all the variables, except participation in household tasks and exposure intensity, simultaneously. Finally, Model 3

---

[38] Separate analysis is conducted for the two age groups in accordance with the literature on ARI in developing countries. This approach implicitly assumes that the two age groups are affected differently by exposure to indoor air pollution as well as the other variables in the system. Analysis of the sample as a whole, including dummy variables for age ≤ 5 (alone and interacted with exposure categories) shows that those below the age of 6 are 0.03 more likely to be diagnosed with ARI ($p = 0.01$) (0.08 without the interaction term) and 0.03 more likely to be diagnosed with ALRI ($p < 0.001$) (0.05 without the interaction term). Further the coefficient of the interaction terms between the dummy variable for age ≤ 5 and exposure categories are jointly significant for ARI and ALRI ($p < 0.001$). Therefore exposure does affect the 0 – 5 and 6 – 50 age groups differently.

[39] An alternative to the inverse quadratic relationship for a concave function would be a logarithmic function of exposure. But decline in the slope of the relationship occurs more rapidly for a logarithmic function than indicated in the relationships in Figure 7.2 to Figure 7.4.

includes all the explanatory variables described above. Therefore Models 2 and 3 (multivariate) show the effects of each variable, net of the other variables in the model. I have presented the results for total cases of acute respiratory infections (ARI), acute lower respiratory infections (ALRI), and eye diseases. Acute upper respiratory infections (AURI) are simply the difference between ARI and ALRI.

Table 7.1: OLS parameter estimates for illness rates using continuous exposure variables for 0 – 5 age group. (a) ARI. (b) ALRI. (c) Eye disease. Model 1 (bivariate) corresponds to parameter estimates in which illness rates are regressed against each variable one at a time, whereas in Models 2 and 3 (multivariate) all the variables are considered together. No one under the age of 6 participates in household tasks and therefor exposure intensity is equal to average exposure for all infants and children below 5. Therefore Models 2 and 3 are equivalent for this age group.

(a) ARI

	Model 1	**Model 2**	**Model 3**
Constant	0.13 ($p < 0.001$)	0.05 ($p = 0.48$)	0.05 ($p = 0.48$)
Exposure			
Average exposure ($mg.m^{-3}$)	1.6×10^{-5} ($p = 0.72$) **	-1.7×10^{-7} ($p = 0.99$) **	-1.7×10^{-7} ($p = 0.99$) **
(Average exposure)$^{0.5}$	0.0012 ($p = 0.70$) **	0.003 ($p = 0.38$) **	0.003 ($p = 0.38$) **
Female	-0.006 ($p = 0.79$)	0.001 ($p = 0.94$)	0.001 ($p = 0.94$)
Age	-0.01 ($p = 0.02$)	-0.008 ($p = 0.1$)	-0.008 ($p = 0.1$)
Maintenance village	-0.037 ($p = 0.09$)	0.02 ($p = 0.44$)	0.02 ($p = 0.44$)
Number residing in house	0.0002 ($p = 0.96$)	-0.0003 ($p = 0.95$)	-0.0003 ($p = 0.95$)
Smokes	N/A	N/A	N/A
Exposure intensity	3.2×10^{-5} ($p = 0.004$) [a]		N/A [a]
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
R^2	N/A	0.17	0.17
Sample size (N)	93	93	93
p > F	N/A	0.009	0.009

[a] For every member of this group exposure intensity and average exposure are the same by definition.
** Jointly significant ($p \leq 0.01$)

[40] Exposure variables (i.e. the linear and inverse quadratic terms in the continuous case and all the exposure categories in the categorical case) are included together in the bivariate model.

(b) ALRI

	Model 1	**Model 2**	**Model 3**
Constant	0.05 ($p < 0.001$)	0.07 ($p = 0.02$)	0.07 ($p = 0.02$)
Exposure			
Average exposure (mg.m^{-3})	9.3×10^{-6} ($p = 0.46$)	7.7×10^{-6} ($p = 0.73$)	7.7×10^{-6} ($p = 0.73$)
(Average exposure)$^{0.5}$	0.0007 ($p = 0.77$)	0.0001 ($p = 0.93$)	0.0001 ($p = 0.93$)
Female	-0.01 ($p = 0.35$)	-0.008 ($p = 0.37$)	-0.008 ($p = 0.37$)
Age	-0.013 ($p < 0.001$)	-0.01 ($p = 0.002$)	-0.01 ($p = 0.002$)
Maintenance village	-0.007 ($p = 0.52$)	0.009 ($p = 0.54$)	0.009 ($p = 0.54$)
Number residing in house	0.0005 ($p = 0.80$)	0.000 ($p = 0.85$)	0.000 ($p = 0.85$)
Smokes	N/A	N/A	N/A
Exposure intensity	9.3×10^{-6} ($p = 0.08$) [a]		N/A [a]
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
R^2	N/A	0.16	0.16
Sample size (N)	93	93	93
$p > F$	N/A	0.002	0.002

[a] For every member of this group exposure intensity and average exposure are the same by definition.

(c) Eye Disease

	Model 1	**Model 2**	**Model 3**
Constant	0.06 ($p < 0.001$)	0.13 ($p = 0.008$)	0.13 ($p = 0.008$)
Exposure			
Average exposure (mg.m^{-3})	3.0×10^{-5} ($p = 0.34$)	3.6×10^{-5} ($p = 0.17$)	3.6×10^{-5} ($p = 0.17$)
(Average exposure)$^{0.5}$	0.0022 ($p = 0.82$)	-0.0015 ($p = 0.36$)	-0.0015 ($p = 0.36$)
Female	0.007 ($p = 0.67$)	0.01 ($p = 0.38$)	0.01 ($p = 0.38$)
Age	-0.01 ($p = 0.02$)	-0.02 ($p = 0.001$)	-0.02 ($p = 0.001$)
Maintenance village	-0.017 ($p = 0.004$)	-0.044 ($p = 0.02$)	-0.044 ($p = 0.02$)
Number residing in house	0.003 ($p = 0.20$)	-0.0001 ($p = 0.96$)	-0.0001 ($p = 0.96$)
Smokes	N/A	N/A	N/A
Exposure intensity	3.0×10^{-5} ($p < 0.001$) [a]		N/A [a]
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
R^2	N/A	0.36	0.36
Sample size (N)	93	93	93
$p > F$	N/A	< 0.0001	< 0.0001

[a] for every member of this group exposure intensity and average exposure are the same by definition.

Table 7.2: OLS parameter estimates for illness rates using categorical exposure variables for 0 – 5 age group. (a) ARI. (b) ALRI. (c) Eye disease. Model 1 (bivariate) corresponds to parameter estimates in which illness rates are regressed against each variable one at a time, whereas in Models 2 and 3 (multivariate) all the variables are considered together. No one under the age of 6 participates in household tasks and they all belong to the lowest exposure intensity category. Therefore Models 2 and 3 are equivalent for this age group.

(a) ARI

	Model 1	**Model 2**	**Model 3**
Constant	0.13 ($p < 0.001$)	0.05 ($p = 0.45$)	0.05 ($p = 0.45$)
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	0.06 ($p = 0.005$) **	0.06 ($p = 0.002$) ***	0.06 ($p = 0.002$) ***
500 – 1000 mg.m^{-3}	0.04 ($p = 0.02$) **	0.06 ($p = 0.04$) ***	0.06 ($p = 0.04$) ***
1000 – 2000 mg.m^{-3}	0.11 ($p = 0.01$) **	0.13 ($p = 0.001$) ***	0.13 ($p = 0.001$) ***
2000 – 3500 mg.m^{-3}	0.11 ($p < 0.001$) **	0.14 ($p = 0.001$) ***	0.14 ($p = 0.001$) ***
> 3500 mg.m^{-3}	0.16 ($p = 0.04$) **	0.18 ($p = 0.04$) ***	0.18 ($p = 0.04$) ***
Female	-0.006 ($p = 0.79$)	-0.0007 ($p = 0.98$)	-0.0007 ($p = 0.98$)
Age	-0.01 ($p = 0.02$)	-0.009 ($p = 0.08$)	-0.009 ($p = 0.08$)
Maintenance village	-0.037 ($p = 0.09$)	0.03 ($p = 0.42$)	0.03 ($p = 0.42$)
Number residing in house	0.0002 ($p = 0.96$)	0.0005 ($p = 0.94$)	0.0005 ($p = 0.94$)
Smokes	N/A	N/A	N/A
Exposure intensity			
0 – 20,000 mg.m^{-3}	N/A [a]		N/A [a]
20,000 – 50,000 mg.m^{-3}	N/A		N/A
50,000 – 100,000 mg.m^{-3}	N/A		N/A
>100,000 mg.m^{-3}	N/A		N/A
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
R^2	N/A	0.19	0.19
Sample size (*N*)	93	93	93
p > F	N/A	0.0005	0.0005

[a] Every member of this group belongs to the lowest exposure intensity category.

*** Jointly significant ($p \leq 0.001$) ** Jointly significant ($p \leq 0.01$)

(b) ALRI

	Model 1	**Model 2**	**Model 3**
Constant	0.05 ($p < 0.001$)	0.07 ($p = 0.06$)	0.07 ($p = 0.06$)
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	0.01 ($p = 0.19$)	0.01 ($p = 0.16$)	0.01 ($p = 0.16$)
500 – 1000 mg.m^{-3}	0.01 ($p = 0.24$)	0.01 ($p = 0.24$)	0.01 ($p = 0.24$)
1000 – 2000 mg.m^{-3}	0.02 ($p = 0.20$)	0.03 ($p = 0.05$)	0.03 ($p = 0.05$)
2000 – 3500 mg.m^{-3}	0.03 ($p = 0.09$)	0.03 ($p = 0.16$)	0.03 ($p = 0.16$)
> 3500 mg.m^{-3}	0.04 ($p = 0.18$)	0.04 ($p = 0.31$)	0.04 ($p = 0.31$)
Female	-0.01 ($p = 0.35$)	-0.009 ($p = 0.43$)	-0.009 ($p = 0.43$)
Age	-0.013 ($p < 0.001$)	-0.012 ($p = 0.002$)	-0.012 ($p = 0.002$)
Maintenance village	-0.007 ($p = 0.52$)	0.006 ($p = 0.70$)	0.006 ($p = 0.70$)
Number residing in house	0.0005 ($p = 0.80$)	0.00005 ($p = 0.99$)	0.00005 ($p = 0.99$)
Smokes	N/A	N/A	N/A
Exposure intensity			
0 – 20,000 mg.m^{-3}	N/A [a]		N/A [a]
20,000 – 50,000 mg.m^{-3}	N/A		N/A
50,000 – 100,000 mg.m^{-3}	N/A		N/A
>100,000 mg.m^{-3}	N/A		N/A
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
R^2		0.16	0.16
Sample size (*N*)	93	93	93
p > F	N/A	0.0007	0.0007

[a] Every member of this group belongs to the lowest exposure intensity category.

(c) Eye Disease

	Model 1	**Model 2**	**Model 3**
Constant	0.06 ($p < 0.001$)	0.11 ($p = 0.005$)	0.11 ($p = 0.005$)
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	0.04 ($p < 0.001$) ***	0.03 ($p = 0.004$) *	0.03 ($p = 0.004$) *
500 – 1000 mg.m^{-3}	0.05 ($p < 0.001$) ***	0.02 ($p = 0.22$) *	0.02 ($p = 0.22$) *
1000 – 2000 mg.m^{-3}	0.07 ($p < 0.001$) ***	0.024 ($p = .15$) *	0.024 ($p = .15$) *
2000 – 3500 mg.m^{-3}	0.09 ($p < 0.001$) ***	0.03 ($p = 0.12$) *	0.03 ($p = 0.12$) *
> 3500 mg.m^{-3}	0.15 ($p = 0.01$) ***	0.08 ($p = 0.11$) *	0.08 ($p = 0.11$) *
Female	0.007 ($p = 0.67$)	0.007 ($p = 0.67$)	0.007 ($p = 0.67$)
Age	-0.01 ($p = 0.02$)	-0.01 ($p = 0.02$)	-0.01 ($p = 0.02$)
Maintenance village	-0.017 ($p = 0.004$)	-0.017 ($p = 0.004$)	-0.017 ($p = 0.004$)
Number residing in house	0.003 ($p = 0.20$)	0.003 ($p = 0.20$)	0.003 ($p = 0.20$)
Smokes	N/A	N/A	N/A
Exposure intensity			
0 – 20,000 mg.m^{-3}	N/A [a]		N/A [a]
20,000 – 50,000 mg.m^{-3}	N/A		N/A
50,000 – 100,000 mg.m^{-3}	N/A		N/A
>100,000 mg.m^{-3}	N/A		N/A
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
R^2		0.35	0.35
Sample size (*N*)	93	93	93
p > F	N/A	< 0.0001	< 0.0001

[a] Every member of this group belongs to the lowest exposure intensity category.

*** Jointly significant ($p \leq 0.001$) * Jointly significant ($p \leq 0.1$)

The results in Table 7.1 and Table 7.2 show that for infants and children below the age of 6, ARI and ALRI are increasing concave functions of average daily exposure to PM_{10}. In the continuous case, the exposure-response relationships have positive and decreasing slopes.[41] In the categorical treatment of exposure, the marginal increase in disease rates

[41] The relationship is of the form $y = ax^{0.5} - bx$ for ARI, where a and b are both positive. Therefore the slope is given by the relationship $\partial y / \partial x = 0.5ax^{-0.5} - b$ which is positive but decreasing for $0 < x \leq (2b \cdot a^{-1})^2$. The slope becomes negative after this maximum. But for the coefficients of Table

is smaller for the higher exposure categories, especially when the larger width of these categories in taken into account. The role of exposure is statistically significant only for ARI. Although lack of statistical significance for the effect of exposure on ALRI is partially due to small sample size, especially in the highest exposure category[42], it is also a reflection of the small slope of the exposure-response relationship for ALRI. This, in turn, would confirm a suspicion raised qualitatively by (34) that significant reductions in acute lower respiratory infections in children would require decreasing pollution to very low levels. For eye disease, exposure is not significant in the continuous case. In the categorical treatment of eye disease, exposure categories are jointly significant. At the same time, for all but the lowest and highest exposure groups, eye disease rates remain unchanged.

Female and male infants and children do not exhibit differential susceptibility to ARI, ALRI, or eye disease. For all three diseases, there is a decrease in illness rates with increasing age, possibly due to improved immunity. The number of people living in a house is not significantly associated with illness rates. This is attributed to the fact that, because of a pastoralist life-style, activity patterns and household roles are a more important determinant of the amount of time spent inside together than the number of household members. Therefore crowding as a result of household size is not an important factor in disease transmission.

7.1 this change does not occur in the exposure ranges observed in the data. For the relationship $y = ax^{0.5} + bx$ (ALRI) the slope is positive and decreasing for all $x > 0$.

[42] The number of children below 5 in the sample is 93, only 5 of whom have average daily exposures above 3500 $\mu g.m^{-3}$.

In Model 1 (gross effects), infants and children in *bomas* have higher rates of ARI and eye diseases, with differences in eye disease rates statistically significant ($p = 0.004$) and those in ARI rates weakly significant ($p = 0.09$). The additional impact of living in *bomas* on ARI is eliminated after controlling for exposure and other factors. The gross effect of living in a *boma* on ARI is in fact due to higher exposure levels in this group than those in the maintenance villages. Therefore, in the bivariate model, living in the *boma* is a proxy for the omitted variable of exposure, a role that is eliminated with accounting for exposure in the multivariate model. But the children in *bomas* continue to have higher rates of eye disease after accounting for exposure and other variables. Higher incidence of eye disease in the *bomas* is likely to be caused by the extremely high fly density as a result of proximity to cattle compounds (Figure 7.6) (158).

Figure 7.6: The large number of flies at the *bomas*, due to proximity to cattle, is an important factor in high rates of eye disease.

Table 7.3: OLS parameter estimates for illness rates using continuous exposure variables for 6 – 50 age group. (a) ARI. (b) ALRI. (c) Eye disease. Model 1 (bivariate) corresponds to parameter estimates in which illness rates are regressed against each variable one at a time, whereas in Models 2 and 3 (multivariate) all the variables are considered together.

(a) ARI

	Model 1	**Model 2**	**Model 3**
Constant	0.06 ($p < 0.001$)	0.02 ($p = 0.31$)	0.02 ($p = 0.45$)
Exposure			
Average exposure ($mg.m^{-3}$)	8.2×10^{-6} ($p = 0.23$) ***	-4.3×10^{-6} ($p = 0.36$) **	-3.5×10^{-7} ($p = 0.94$) **
(Average exposure)$^{0.5}$	0.001 ($p = 0.004$) ***	0.001 ($p = 0.02$) **	0.001 ($p = 0.03$) **
Female	0.035 ($p < 0.001$)	0.01 ($p = 0.20$)	0.007 ($p = 0.47$)
Age	-0.00002 ($p = 0.92$)	-0.0002 ($p = 0.28$)	-0.00005 ($p = 0.81$)
Maintenance village	-0.02 ($p = 0.02$)	-0.005 ($p = 0.64$)	-0.006 ($p = 0.63$)
Number residing in house	0.0002 ($p = 0.95$)	-0.002 ($p = 0.52$)	-0.003 ($p = 0.37$)
Smokes	-0.008 ($p = 0.31$)	0.02 ($p = 0.05$)	0.02 ($p = 0.04$)
Exposure intensity	3.3×10^{-7} ($p = 0.001$)		-3.7×10^{-7} ($p = 0.008$)
Household Tasks			
No household task	Omitted category		Omitted category
Some household task	0.03 ($p = 0.12$) ***		0.03 ($p = 0.09$)
Some cooking	0.02 ($p = 0.03$) ***		0.02 ($p = 0.31$)
Regular cooking	0.04 ($p < 0.001$) ***		0.007 ($p = 0.67$)
R^2	N/A	0.21	0.24
Sample size (N)	229	229	229
$p > F$	N/A	< 0.0001	< 0.0001

*** Jointly significant ($p \leq 0.001$) ** Jointly significant ($p \leq 0.01$)

(b) ALRI

	Model 1	**Model 2**	**Model 3**
Constant	0.02 ($p < 0.001$)	0.002 ($p = 0.75$)	0.01 ($p = 0.23$)
Exposure			
Average exposure ($mg.m^{-3}$)	3.2×10^{-6} ($p < 0.001$) ***	2.2×10^{-6} ($p = 0.19$) ***	4.5×10^{-6} ($p = 0.003$) **
(Average exposure)$^{0.5}$	0.0004 ($p < 0.001$) ***	0.0006 ($p = 0.73$) ***	-0.0001 ($p = 0.49$) **
Female	0.009 ($p = 0.001$)	0.003 ($p = 0.41$)	-0.005 ($p = 0.23$)
Age	0.0003 ($p = 0.001$)	0.0002 ($p = 0.03$)	0.0001 ($p = 0.25$)
Maintenance village	-0.008 ($p = 0.008$)	-0.002 ($p = 0.54$)	-0.007 ($p = 0.14$)
Number residing in house	0.0003 ($p = 0.67$)	-0.00004 ($p = 0.96$)	-0.0002 ($p = 0.78$)
Smokes	-0.001 ($p = 0.81$)	0.004 ($p = 0.42$)	0.007 ($p = 0.15$)
Exposure intensity	1.2×10^{-7} ($p < 0.001$)		-1.3×10^{-7} ($p = 0.06$)
Household Tasks			
No household task	Omitted category		Omitted category
Some household task	0.006 ($p = 0.11$) ***		0.01 ($p = 0.09$)
Some cooking	0.007 ($p = 0.11$) ***		0.01 ($p = 0.13$)
Regular cooking	0.02 ($p < 0.001$) ***		0.02 ($p = 0.04$)
R^2	N/A		
Sample size (N)	229	229	229
$p > F$	N/A	< 0.0001	< 0.0001

*** Jointly significant ($p \leq 0.001$) ** Jointly significant ($p \leq 0.01$)

(c) Eye Disease

	Model 1	**Model 2**	**Model 3**
Constant	0.01 ($p < 0.001$)	0.02 ($p = 0.006$)	0.02 ($p = 0.04$)
Exposure			
Average exposure ($mg.m^{-3}$)	1.2×10^{-6} ($p = 0.05$) **	-4.4×10^{-7} ($p = 0.78$)	-7.3×10^{-7} ($p = 0.67$)
(Average exposure)$^{0.5}$	0.0001 ($p = 0.02$) **	0.0002 ($p = 0.39$)	0.0003 ($p = 0.22$)
Female	0.005 ($p = 0.09$)	0.0007 ($p = 0.86$)	0.002 ($p = 0.78$)
Age	-0.0003 ($p = 0.04$)	-0.0003 ($p = 0.04$)	-0.0003 ($p = 0.1$)
Maintenance village	-0.01 ($p < 0.001$)	-0.009 ($p = 0.02$)	-0.008 ($p = 0.05$)
Number residing in house	0.0003 ($p = 0.67$)	-0.0006 ($p = 0.23$)	-0.0008 ($p = 0.19$)
Smokes	-0.01 ($p = 0.006$)	-0.002 ($p = 0.65$)	-0.002 ($p = 0.57$)
Exposure intensity	4.8×10^{-8} ($p = 0.20$)		-2.0×10^{-8} ($p = 0.66$)
Household Tasks			
No household task	Omitted category		Omitted category
Some household task	0.009 ($p = 0.33$)		0.005 ($p = 0.65$)
Some cooking	0.0002 ($p = 0.96$)		-0.004 ($p = 0.61$)
Regular cooking	0.002 ($p = 0.60$)		-0.003 ($p = 0.67$)
R^2	N/A	04.08	0.09
Sample size (N)	229	229	229
$p > F$	N/A	0.001	0.001

** Jointly significant ($p \leq 0.01$)

Table 7.4: OLS parameter estimates for illness rates using categorical exposure variables for 6 – 50 age group. (a) ARI. (b) ALRI. (c) Eye disease. Model 1 (bivariate) corresponds to parameter estimates in which illness rates are regressed against each variable one at a time, whereas in Models 2 and 3 (multivariate) all the variables are considered together.

(a) ARI

	Model 1	**Model 2**	**Model 3**
Constant	0.06 ($p < 0.001$)	0.03 ($p = 0.1$)	0.04 ($p = 0.07$)
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	0.03 ($p < 0.001$) ***	0.027 ($p = 0.003$) **	0.027 ($p = 0.01$) *
500 – 1000 mg.m^{-3}	0.025 ($p = 0.005$) ***	0.022 ($p = 0.06$) **	0.02 ($p = 0.16$) *
1000 – 2000 mg.m^{-3}	0.05 ($p < 0.001$) ***	0.04 ($p = 0.002$) **	0.04 ($p = 0.02$) *
2000 – 4000 mg.m^{-3}	0.06 ($p < 0.001$) ***	0.05 ($p = 0.001$) **	0.05 ($p = 0.007$) *
4000 – 7000 mg.m^{-3}	0.075 ($p < 0.001$) ***	0.06 ($p = 0.002$) **	0.08 ($p = 0.009$) *
> 7000 mg.m^{-3}	0.10 ($p < 0.001$) ***	0.09 ($p < 0.001$) **	0.1 ($p = 0.002$) *
Female	0.035 ($p < 0.001$)	0.01 ($p = 0.18$)	0.01 ($p = 0.34$)
Age	-0.00002 ($p = 0.92$)	-0.0003 ($p = 0.22$)	-0.0003 ($p = 0.23$)
Maintenance village	-0.02 ($p = 0.02$)	-0.007 ($p = 0.54$)	-0.008 ($p = 0.54$)
Number residing in house	0.0002 ($p = 0.95$)	-0.002 ($p = 0.45$)	-0.002 ($p = 0.36$)
Smokes	-0.008 ($p = 0.31$)	0.02 ($p = 0.04$)	0.02 ($p = 0.03$)
Exposure intensity			
0 – 20,000 mg.m^{-3}	Omitted category		Omitted category
20,000 – 50,000 mg.m^{-3}	0.025 ($p = 0.02$) ***		0.005 ($p = 0.74$)
50,000 – 100,000 mg.m^{-3}	0.04 ($p = 0.01$) ***		-0.009 ($p = 0.68$)
>100,000 mg.m^{-3}	0.06 ($p < 0.001$) ***		-0.01 ($p = 0.60$)
Household Tasks			
No household task	Omitted category		Omitted category
Some household task	0.03 ($p = 0.12$) ***		0.02 ($p = 0.49$)
Some cooking	0.02 ($p = 0.03$) ***		-0.0004 ($p = 0.97$)
Regular cooking	0.04 ($p < 0.001$) ***		0.001 ($p = 0.95$)
R^2	N/A	0.22	0.23
Sample size (*N*)	229	229	229
p > F	N/A	< 0.0001	< 0.0001

*** Jointly significant ($p \leq 0.001$) ** Jointly significant ($p \leq 0.01$)

* Jointly significant ($p \leq 0.1$)

(b) ALRI

	Model 1	**Model 2**	**Model 3**
Constant	0.02 ($p < 0.001$)	0.0003 ($p = 0.97$)	0.009 ($p = 0.32$)
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	0.004 ($p = 0.37$) ***	0.004 ($p = 0.48$) **	0.00005 ($p = 0.99$)
500 – 1000 mg.m^{-3}	0.004 ($p = 0.24$) ***	0.004 ($p = 0.32$) **	-0.0003 ($p = 0.95$)
1000 – 2000 mg.m^{-3}	0.01 ($p = 0.02$) ***	0.011 ($p = 0.03$) **	0.004 ($p = 0.46$)
2000 – 4000 mg.m^{-3}	0.013 ($p = 0.008$) ***	0.011 ($p = 0.03$) **	0.006 ($p = 0.37$)
4000 – 7000 mg.m^{-3}	0.016 ($p = 0.04$) ***	0.013 ($p = 0.09$) **	0.01 ($p = 0.43$)
> 7000 mg.m^{-3}	0.036 ($p < 0.001$) ***	0.031 ($p < 0.001$) **	0.023 ($p = 0.11$)
Female	0.009 ($p = 0.001$)	0.003 ($p = 0.40$)	-0.003 ($p = 0.47$)
Age	0.0003 ($p = 0.001$)	0.0002 ($p = 0.03$)	0.00008 ($p = 0.41$)
Maintenance village	-0.008 ($p = 0.008$)	-0.002 ($p = 0.53$)	-0.007 ($p = 0.16$)
Number residing in house	0.0003 ($p = 0.67$)	-0.0001 ($p = 0.87$)	-0.0001 ($p = 0.82$)
Smokes	-0.001 ($p = 0.81$)	0.004 ($p = 0.47$)	0.007 ($p = 0.15$)
Exposure intensity			
0 – 20,000 mg.m^{-3}	Omitted category		Omitted category
20,000 – 50,000 mg.m^{-3}	0.006 ($p = 0.11$) ***		-0.005 ($p = 0.43$)
50,000 – 100,000 mg.m^{-3}	0.007 ($p = 0.11$) ***		-0.012 ($p = 0.14$)
>100,000 mg.m^{-3}	0.02 ($p < 0.001$) ***		-0.009 ($p = 0.45$)
Household Tasks			
No household task	Omitted category		Omitted category
Some household task	0.006 ($p = 0.11$) ***		0.008 ($p = 0.22$)
Some cooking	0.007 ($p = 0.11$) ***		0.009 ($p = 0.12$)
Regular cooking	0.02 ($p < 0.001$) ***		0.02 ($p = 0.03$)
R^2	N/A	0.17	0.20
Sample size (*N*)	229	229	229
$p > F$	N/A	< 0.0001	< 0.0001

*** Jointly significant ($p \leq 0.001$) ** Jointly significant ($p \leq 0.01$)

For older children and adults up to the age of 50, the incidence of ARI and ALRI are significantly associated with average daily exposure to PM_{10}, in a concave, increasing relationship in both the continuous and categorical treatments of exposure. As for those below 6, the marginal increase in disease rates is lower for the higher (and wider) exposure categories. In the categorical treatment, however, exposure is no longer a significant determinant of ALRI when intensity and participation in household tasks are included in the model (Model 3).

(c) Eye Disease

	Model 1	**Model 2**	**Model 3**
Constant	0.01 ($p < 0.001$)	0.02 ($p = 0.002$)	0.02 ($p = 0.01$)
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	0.01 ($p = 0.001$) ***	0.009 ($p = 0.01$) **	0.01 ($p = 0.007$) ***
500 – 1000 mg.m^{-3}	0.01 ($p = 0.006$) ***	0.006 ($p = 0.28$) **	0.008 ($p = 0.06$) ***
1000 – 2000 mg.m^{-3}	0.012 ($p < 0.001$) ***	0.005 ($p = 0.16$) **	0.007 ($p = 0.13$) ***
2000 – 4000 mg.m^{-3}	0.023 ($p < 0.001$) ***	0.016 ($p = 0.003$) **	0.02 ($p = 0.01$) ***
4000 – 7000 mg.m^{-3}	0.009 ($p = 0.02$) ***	0.001 ($p = 0.87$) **	0.0003 ($p = 0.97$) ***
> 7000 mg.m^{-3}	0.022 ($p = 0.001$) ***	0.016 ($p = 0.09$) **	0.013 ($p = 0.28$) ***
Female	0.005 ($p = 0.09$)	0.001 ($p = 0.78$)	0.003 ($p = 0.67$)
Age	-0.0003 ($p = 0.04$)	-0.0003 ($p = 0.01$)	-0.0003 ($p = 0.05$)
Maintenance village	-0.01 ($p < 0.001$)	-0.008 ($p = 0.04$)	-0.007 ($p = 0.09$)
Number residing in house	0.0003 ($p = 0.67$)	-0.0008 ($p = 0.15$)	-0.0009 ($p = 0.13$)
Smokes	-0.01 ($p = 0.006$)	-0.002 ($p = 0.67$)	-0.003 ($p = 0.47$)
Exposure intensity			
0 – 20,000 mg.m^{-3}	Omitted category		Omitted category
20,000 – 50,000 mg.m^{-3}	0.003 ($p = 0.48$)		0.005 ($p = 0.51$)
50,000 – 100,000 mg.m^{-3}	0.005 ($p = 0.28$)		0.007 ($p = 0.32$)
>100,000 mg.m^{-3}	0.008 ($p = 0.14$)		0.011 ($p = 0.12$)
Household Tasks			
No household task	Omitted category		Omitted category
Some household task	0.009 ($p = 0.33$)		-0.002 ($p = 0.86$)
Some cooking	0.0002 ($p = 0.96$)		-0.01 ($p = 0.09$)
Regular cooking	0.002 ($p = 0.60$)		-0.007 ($p = 0.20$)
R^2	N/A	0.13	0.14
Sample size (*N*)	229	229	229
$p > F$	N/A	0.001	0.001

*** Jointly significant ($p \leq 0.001$) ** Jointly significant ($p \leq 0.01$)

In the bivariate model, being female and living in a *boma* both result in statistically significant increases in illness rates for both ARI and ALRI. These gross effects are, however, eliminated after accounting for exposure and other variables in the multivariate model. The gross effects of these variables in Model 1 are a result of their correlation with exposure – with higher exposure among the residents of *bomas* and women – and are eliminated in the multivariate models.

In this age group, age does not affect the incidence of ARI. Age has a positive effect on ALRI rates, but this effect is eliminated after accounting for participation in household

activities and the intensity of exposure. This may be either because long periods of daily exposure to high concentrations (which occurs among those who cook *and* are generally older) have harmful effects beyond that accounted for in average exposure, or that participation in cooking activities results in chronic respiratory conditions in higher ages which facilitate ALRI incidence.

Smoking does not have a statistically significant gross effect on ARI or ALRI. In fact, although statistically not significant, the sign of the coefficient of smoking in the bivariate model is negative for ARI, against the expected disease increasing effect of smoking. In the multivariate model the coefficient of smoking has its expected sign, resulting in an increase in ARI rates. This is largely because all of smokers are men, who also have lower exposure (and disease rates). As a result, while the gross effect of smoking was negative (albeit not significant), after exposure is accounted for it results in higher ARI rates. Smoking does not have a significant net impact on ALRI.

Exposure intensity is not a determinant of ARI and ALRI rates after average exposure has been accounted for. The level of participation in various household tasks does not affect ARI rates but the group that cooks regularly has additional susceptibility to ALRI, even after controlling for average exposure (although the participation variables are not jointly significant).[43] This result implies that either long periods of exposure to high levels of PM_{10} cause (either short-term or chronic) damage to the lower respiratory system beyond

[43] Recall from Chapter 6 that the average exposure values were calculated to include high-intensity exposure episodes. Because of exposure patterns, individuals with high exposure intensity generally have

that described by the average exposure-response relationship, or the exposure of this group is underestimated even by the exposure profile approach that accounts for higher exposure during cooking periods.

For eye disease, exposure categories are jointly significant in the categorical model. Despite this significance, except for the lowest and highest exposure groups, diseases of the eye do not show a monotonic relationship with exposure, as also reflected in the lack of statistical significance in the coefficients of exposure in the continuous model. Residence in *boma* is the only variable increasing the probability of eye disease, which decreases with age.

7.3.2 *blogit* estimation

In Table 7.5 and Table 7.6 the parameter estimates using *blogit* regression are reported for the categorical treatment of average exposure and exposure intensity for the 0 – 5 and 6 – 50 age groups. The odds ratios for exposure categories, obtained from Model 2, which controls for explanatory variables except intensity and participation in household tasks, are also shown in Figure 7.7.

---

high average exposures and vice versa. This is also seen in the gross effect of exposure intensity and participation in household tasks.

Table 7.5: *blogit* odds ratios for illness rates using categorical exposure variables for 0 – 5 age group. (a) ARI. (b) ALRI. (c) Eye disease. Model 1 (bivariate) corresponds to parameter estimates in which illness rates are regressed against each variable one at a time, whereas in Models 2 and 3 (multivariate) all the variables are considered together. No one under the age of 6 participates in household tasks and they all belong to the lowest exposure intensity category. Therefore Models 2 and 3 are equivalent for this age group. Numbers in brackets indicate 95% confidence interval.

(a) ARI

	Model 1	**Model 2**	**Model 3**
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	2.34 ($p < 0.001$) *** (1.48 – 3.69)	2.42 ($p < 0.001$) *** (1.53 – 3.83)	2.42 ($p < 0.001$) *** (1.53 – 3.83)
500 – 1000 mg.m^{-3}	1.78 ($p = 0.01$) *** (1.12 – 2.84)	2.15 ($p = 0.003$) *** (1.30 – 3.56)	2.15 ($p = 0.003$) *** (1.30 – 3.56)
1000 – 2000 mg.m^{-3}	3.40 ($p < 0.001$) *** (2.18 – 5.31)	4.30 ($p < 0.001$) *** (2.63 – 7.04)	4.30 ($p < 0.001$) *** (2.63 – 7.04)
2000 – 3500 mg.m^{-3}	3.70 ($p < 0.001$) *** (2.37 – 5.78)	4.72 ($p < 0.001$) *** (2.82 – 7.88)	4.72 ($p < 0.001$) *** (2.82 – 7.88)
> 3500 mg.m^{-3}	5.59 ($p < 0.001$) *** (3.34 – 9.38)	6.73 ($p < 0.001$) *** (3.75 – 12.06)	6.73 ($p < 0.001$) *** (3.75 – 12.06)
Female	0.97 ($p = 0.69$) (0.82 – 1.14)	0.99 ($p = 0.88$) (0.83 – 1.17)	0.99 ($p = 0.88$) (0.83 – 1.17)
Age [a]	0.86 ($p < 0.001$) (0.81 – 0.91)	0.88 ($p < 0.001$) (0.83 – 0.94)	0.88 ($p < 0.001$) (0.83 – 0.94)
Maintenance village	0.73 ($p < 0.001$) (0.61 – 0.86)	1.29 ($p = 0.06$) (0.99 – 1.67)	1.29 ($p = 0.06$) (0.99 – 1.67)
Number residing in house [a]	1.00 ($p = 0.85$) (0.96 – 1.03)	1.00 ($p = 0.99$) (0.95 – 1.05)	1.00 ($p = 0.99$) (0.95 – 1.05)
Smokes	N/A	N/A	N/A
Exposure intensity			
0 – 20,000 mg.m^{-3}	N/A [b]		N/A [b]
20,000 – 50,000 mg.m^{-3}	N/A		N/A
50,000 – 100,000 mg.m^{-3}	N/A		N/A
>100,000 mg.m^{-3}	N/A		N/A
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
Sample size (N)	93	93	93

[a] Odd ratios of age and household size, both continuous variables, represent the odds ratios for two subsequent units of these variables.

[b] Every member of this group belongs to the lowest exposure intensity category.

*** Jointly significant ($p \leq 0.001$)

(b) ALRI

	Model 1	**Model 2**	**Model 3**
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	1.39 (p = 0.25) ** (0.79 – 2.46)	1.48 (p = 0.18) ** (0.83 – 2.63)	1.48 (p = 0.18) ** (0.83 – 2.63)
500 – 1000 mg.m^{-3}	1.13 (p = 0.69) ** (0.63 – 2.01)	1.40 (p = 0.30) ** (0.74 – 2.67)	1.40 (p = 0.30) ** (0.74 – 2.67)
1000 – 2000 mg.m^{-3}	1.76 (p = 0.04) ** (1.01 – 3.04)	2.33 (p = 0.009) ** (1.23 – 4.38)	2.33 (p = 0.009) ** (1.23 – 4.38)
2000 – 3500 mg.m^{-3}	1.63 (p = 0.09) ** (0.93 – 2.86)	1.93 (p = 0.05) ** (0.99 – 3.78)	1.93 (p = 0.05) ** (0.99 – 3.78)
> 3500 mg.m^{-3}	2.84 (p = 0.002) ** (1.46 – 5.52)	2.93 (p = 0.007) ** (1.34 – 6.39)	2.93 (p = 0.007) ** (1.34 – 6.39)
Female	0.86 (p = 0.24) (0.68 – 1.10)	0.84 (p = 0.21) (0.65 – 1.10)	0.84 (p = 0.21) (0.65 – 1.10)
Age [a]	0.76 (p < 0.001) (0.70 – 0.82)	0.76 (p < 0.001) (0.70 – 0.84)	0.76 (p < 0.001) (0.70 – 0.84)
Maintenance village	0.92 (p = 0.50) (0.71 – 1.18)	1.18 (p = 0.41) (0.79 – 1.77)	1.18 (p = 0.41) (0.79 – 1.77)
Number residing in house [a]	0.97 (p = 0.27) (0.92 – 1.02)	0.98 (p = 0.70) (0.91 – 1.06)	0.98 (p = 0.70) (0.91 – 1.06)
Smokes	N/A	N/A	N/A
Exposure intensity			
0 – 20,000 mg.m^{-3}	N/A [b]		N/A [b]
20,000 – 50,000 mg.m^{-3}	N/A		N/A
50,000 – 100,000 mg.m^{-3}	N/A		N/A
>100,000 mg.m^{-3}	N/A		N/A
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
Sample size (*N*)	93	93	93

[a] Odd ratios of age and household size, both continuous variables, represent the odds ratios for two subsequent units of these variables.

[b] Every member of this group belongs to the lowest exposure intensity category.

** Jointly significant ($p \leq 0.01$)

(c) Eye Disease

	Model 1	**Model 2**	**Model 3**
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	10.84 (p = 001) *** (2.63 – 44.69)	8.87 (p = 003) ** (2.13 – 36.78)	8.87 (p = 003) ** (2.13 – 36.78)
500 – 1000 mg.m^{-3}	11.88 (p = 001) *** (2.89 – 48.87)	5.06 (p = 03) ** (1.16 – 22.06)	5.06 (p = 03) ** (1.16 – 22.06)
1000 – 2000 mg.m^{-3}	16.12 (p < 0.001) *** (3.95 – 65.79)	6.21 (p = 01) ** (1.44 – 26.74)	6.21 (p = 01) ** (1.44 – 26.74)
2000 – 3500 mg.m^{-3}	20.1 (p < 0.001) *** (4.93 – 81.92)	6.42 (p = 01) ** (1.48 – 27.85)	6.42 (p = 01) ** (1.48 – 27.85)
> 3500 mg.m^{-3}	35.73 (p < 0.001) *** (8.48 – 150.51)	10.31 (p = 0.002) ** (2.29 – 46.45)	10.31 (p = 0.002) ** (2.29 – 46.45)
Female	1.16 (p = 0.22) (0.92 – 1.46)	1.19 (p = 0.16) (0.93 – 1.53)	1.19 (p = 0.16) (0.93 – 1.53)
Age [a]	0.75 (p < 0.001) (0.69 – 0.81)	0.74 (p < 0.001) (0.68 – 0.80)	0.74 (p < 0.001) (0.68 – 0.80)
Maintenance village	0.32 (p < 0.001) (0.24 – 0.44)	0.33 (p < 0.001) (0.21 – 0.52)	0.33 (p < 0.001) (0.21 – 0.52)
Number residing in house [a]	1.08 (p = 0.001) (1.03 – 1.13)	1.02 (p = 0.65) (0.95 – 1.09)	1.02 (p = 0.65) (0.95 – 1.09)
Smokes	N/A	N/A	N/A
Exposure intensity			
0 – 20,000 mg.m^{-3}	N/A [b]		N/A [b]
20,000 – 50,000 mg.m^{-3}	N/A		N/A
50,000 – 100,000 mg.m^{-3}	N/A		N/A
>100,000 mg.m^{-3}	N/A		N/A
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
Sample size (*N*)	93	93	93

[a] Odd ratios of age and household size, both continuous variables, represent the odds ratios for two subsequent units of these variables.
[b] Every member of this group belongs to the lowest exposure intensity category.
*** Jointly significant ($p \leq 0.001$) ** Jointly significant ($p \leq 0.01$)

The estimated odds ratios for exposure categories in Table 7.5 show the same increasing, concave relationship between ARI and exposure (Figure 7.7a, column 1), and ALRI and exposure (Figure 7.7b, column 1) as the OLS model. Further, the coefficients of ALRI are statistically significant in the *blogit* estimation. Also similar to the OLS estimation, the slope of the relationship declines above the 1000 – 2000 μg.m^{-3} exposure category.

Exposure categories have a jointly significant impact on eye disease but, as also seen in the OLS model, for all but the lowest and highest exposure groups, the rates of eye disease remain unchanged.

Living in a *boma* remains an important determinant of eye disease rate, with those infants/children in *bomas* being 3 times as likely as those in maintenance villages to be diagnosed with this illness. The type of village however has no significant impact on the rates of ARI and ALRI after exposure and other factors have been controlled for. As in the OLS model, ARI, ALRI, and eye disease rates decrease with age for infants and children. The number of people residing in the house also has no effect on disease rates.

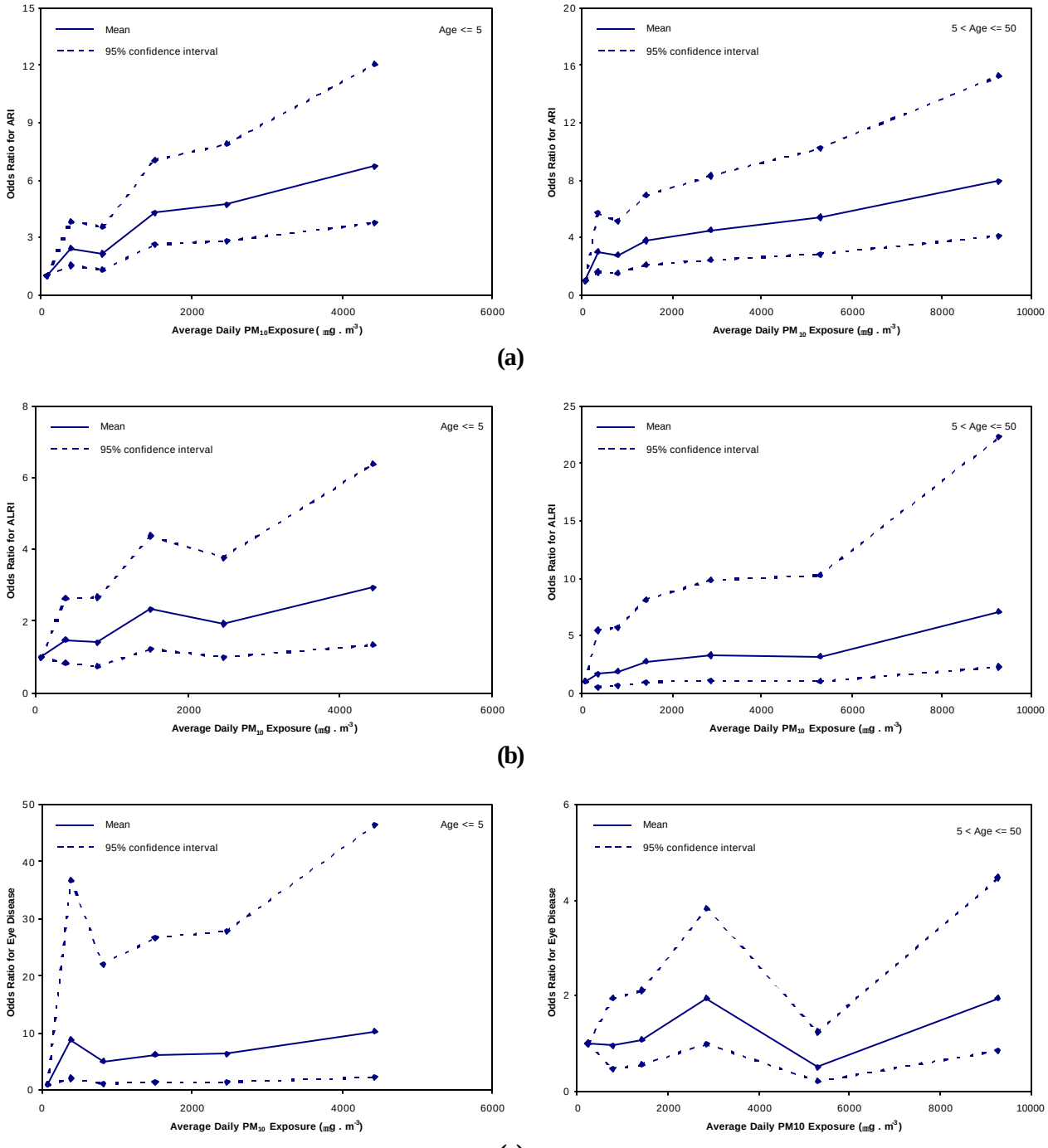


Figure 7.7: Exposure-response plots for age ≤ 5 (column 1) and 5 < Age ≤ 50 (column 2). (a) ARI. (b) ALRI and AURI. (c) Eye diseases. Adjusted odds ratios for each exposure category, obtained from *blogit* regression, are plotted against the average exposure of the category.

Table 7.6: *blogit* odds ratios for illness rates using categorical exposure variables for 6 – 50 age group. (a) ARI. (b) ALRI. (c) Eye disease. Model 1 (bivariate) corresponds to parameter estimates in which illness rates are regressed against each variable one at a time, whereas in Models 2 and 3 (multivariate) all the variables are considered together. Numbers in brackets indicate 95% confidence interval.

(a) ARI

	Model 1	**Model 2**	**Model 3**
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	3.18 ($p < 0.001$) *** (1.69 – 5.98)	3.01 ($p = 0.001$) *** (1.59 – 5.70)	3.03 ($p = 0.001$) *** (1.59 – 5.78)
500 – 1000 mg.m^{-3}	2.86 ($p = 0.001$) *** (1.56 – 5.22)	2.77 ($p = 0.001$) *** (1.49 – 5.13)	2.75 ($p = 0.002$) *** (1.46 – 5.17)
1000 – 2000 mg.m^{-3}	4.38 ($p < 0.001$) *** (2.44 – 7.87)	3.79 ($p < 0.001$) *** (2.07 – 6.92)	3.78 ($p < 0.001$) *** (2.01 – 7.11)
2000 – 4000 mg.m^{-3}	4.95 ($p < 0.001$) *** (2.75 – 8.90)	4.49 ($p < 0.001$) *** (2.43 – 8.30)	4.91 ($p < 0.001$) *** (2.58 – 9.35)
4000 – 7000 mg.m^{-3}	6.27 ($p < 0.001$) *** (3.45 – 11.37)	5.40 ($p < 0.001$) *** (2.85 – 10.22)	7.03 ($p < 0.001$) *** (3.40 – 14.53)
> 7000 mg.m^{-3}	8.87 ($p < 0.001$) *** (4.91 – 16.02)	7.93 ($p < 0.001$) *** (4.11 – 15.27)	10.72 ($p < 0.001$) *** (4.85 – 23.68)
Female	1.80 ($p < 0.001$) (1.55 – 2.07)	1.24 ($p = 0.04$) (1.01 – 1.52)	1.24 ($p = 0.11$) (0.95 – 1.62)
Age [a]	1.00 ($p = 0.52$) (0.99 – 1.00)	0.99 ($p = 0.02$) (0.99 – 1.00)	0.99 ($p = 0.10$) (0.99 – 1.00)
Maintenance village	0.68 ($p < 0.001$) (0.58 – 0.80)	0.92 ($p = 0.41$) (0.76 – 1.12)	0.93 ($p = 0.50$) (0.74 – 1.16)
Number residing in house [a]	1.00 ($p = 0.89$) (0.97 – 1.03)	0.96 ($p = 0.04$) (0.93 – 1.00)	0.95 ($p = 0.01$) (0.92 – 0.99)
Smokes	0.90 ($p = 0.48$) (0.68 – 1.20)	1.48 ($p = 0.02$) (1.07 – 2.04)	1.52 ($p = 0.02$) (1.08 – 2.12)
Exposure intensity			
0 – 20,000 mg.m^{-3}	Omitted category		Omitted category
20,000 – 50,000 mg.m^{-3}	1.53 ($p < 0.001$) *** (1.27 – 1.84)		1.15 ($p = 0.47$) (0.79 – 1.69)
50,000 – 100,000 mg.m^{-3}	1.84 ($p < 0.001$) *** (1.53 – 2.22)		0.91 ($p = 0.69$) (0.56 – 1.47)
>100,000 mg.m^{-3}	2.34 ($p < 0.001$) *** (1.96 – 2.81)		0.87 ($p = 0.62$) (0.51 – 1.50)
Household Tasks			
No household task	Omitted category		Omitted category
Some household task	1.90 ($p < 0.001$) *** (1.44 – 2.51)		1.30 ($p = 0.29$) * (0.80 – 2.13)
Some cooking	1.44 ($p < 0.001$) *** (1.18 – 1.76)		0.88 ($p = 0.55$) * (0.57 – 1.35)
Regular cooking	1.89 ($p < 0.001$) *** (1.61 – 2.21)		0.85 ($p = 0.48$) * (0.54 – 1.34)
Sample size (N)	229	229	229

[a] Odd ratios of age and household size, both continuous variables, represent the odds ratios for two subsequent units of these variables.

*** Jointly significant ($p \leq 0.001$) * Jointly significant ($p \leq 0.1$)

(b) ALRI

	Model 1	**Model 2**	**Model 3**
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	1.75 (p = 0.35) *** (0.55 – 5.60)	1.65 (p = 0.41) *** (0.50 – 5.45)	1.38 (p = 0.60) (0.42 – 4.54)
500 – 1000 mg.m^{-3}	1.86 (p = 0.26) *** (0.64 – 5.38)	1.87 (p = 0.27) *** (0.61 – 5.71)	1.48 (p = 0.49) (0.48 – 4.56)
1000 – 2000 mg.m^{-3}	2.68 (p = 0.06) *** (0.96 – 7.48)	2.74 (p = 0.07) *** (0.93 – 8.12)	1.90 (p = 0.26) (0.62 – 5.76)
2000 – 4000 mg.m^{-3}	3.40 (p = 0.02) *** (1.22 – 9.45)	3.28 (p = 0.03) *** (1.09 – 9.85)	2.59 (p = 0.11) (0.81 – 8.28)
4000 – 7000 mg.m^{-3}	3.52 (p = 0.02) *** (1.23 – 10.09)	3.21 (p = 0.05) *** (1.01 – 10.24)	3.47 (p = 0.07) (0.89 – 13.48)
> 7000 mg.m^{-3}	8.71 (p < 0.001) *** (3.16 – 24.04)	7.10 (p = 0.001) *** (2.26 – 22.32)	6.04 (p = 0.01) (1.52 – 24.00)
Female	1.87 (p < 0.001) (1.40 – 2.50)	1.21 (p = 0.39) (0.78 – 1.88)	0.88 (p = 0.64) (0.52 – 1.50)
Age [a]	1.02 (p < 0.001) (1.01 – 1.03)	1.01 (p = 0.02) (1.00 – 1.02)	1.00 (p = 0.45) (0.99 – 1.02)
Maintenance village	0.64 (p = 0.006) (0.47 – 0.88)	0.93 (p = 0.74) (0.62 – 1.40)	0.72 (p = 0.17) (0.45 – 1.15)
Number residing in house [a]	1.01 (p = 0.69) (0.95 – 1.08)	0.99 (p = 0.75) (0.92 – 1.07)	0.98 (p = 0.53) (0.90 – 1.05)
Smokes	0.97 (p = 0.90) (0.56 – 1.67)	1.53 (p = 0.18) (0.82 – 2.85)	1.82 (p = 0.07) (0.95 – 3.49)
Exposure intensity			
0 – 20,000 mg.m^{-3}	Omitted category		Omitted category
20,000 – 50,000 mg.m^{-3}	1.77 (p = 0.003) *** (1.22 – 2.56)		0.89 (p = 0.74) (0.44 – 1.78)
50,000 – 100,000 mg.m^{-3}	1.55 (p = 0.04) *** (1.03 – 2.32)		0.44 (p = 0.10) (0.16 – 1.17)
>100,000 mg.m^{-3}	3.28 (p < 0.001) *** (2.34 – 4.59)		0.59 (p = 0.29) (0.22 – 1.57)
Household Tasks			
No household task	Omitted category		Omitted category
Some household task	1.23 (p = 0.57) *** (0.61 – 2.49)		1.70 (p = 0.29) (0.64 – 4.53)
Some cooking	1.39 (p = 0.14) *** (0.90 – 2.15)		1.53 (p = 0.24) (0.76 – 3.11)
Regular cooking	2.83 (p < 0.001) *** (2.07 – 3.87)		2.40 (p = 0.03) (1.10 – 5.25)
Sample size (N)	229	229	229

[a] Odd ratios of age and household size, both continuous variables, represent the odds ratios for two subsequent units of these variables.

*** Jointly significant ($p \leq 0.001$) ** Jointly significant ($p \leq 0.01$)

(c) Eye Disease

	Model 1	**Model 2**	**Model 3**
Exposure			
0 – 500 mg.m^{-3} [a]	Omitted category	Omitted category	Omitted category
500 – 1000 mg.m^{-3}	1.51 (p = 0.23) *** (0.77 – 2.96)	0.95 (p = 0.89) *** (0.47 – 1.94)	1.00 (p = 0.99) *** (0.46 – 2.13)
1000 – 2000 mg.m^{-3}	2.19 (p = 0.01) *** (1.18 – 4.04)	1.08 (p = 0.82) *** (0.55 – 2.11)	1.12 (p = 0.76) *** (0.54 – 2.32)
2000 – 4000 mg.m^{-3}	3.87 (p < 0.001) *** (2.14 – 6.99)	1.94 (p = 0.05) *** (0.99 – 3.82)	2.02 (p = 0.07) *** (0.94 – 4.35)
4000 – 7000 mg.m^{-3}	1.06 (p = 0.89) *** (0.47 – 2.37)	0.50 (p = 0.14) *** (0.20 – 1.24)	0.44 (p = 0.14) *** (0.15 – 1.29)
> 7000 mg.m^{-3}	3.57 (p < 0.001) *** (1.88 – 6.77)	1.95 (p = 0.12) *** (0.85 – 4.47)	1.13 (p = 0.84) *** (0.35 – 3.67)
Female	1.53 (p = 0.004) (1.15 – 2.04)	1.22 (p = 0.31) (0.83 – 1.77)	1.53 (p = 0.08) (0.95– 2.46)
Age [b]	0.97 (p < 0.001) (0.96 – 0.99)	0.97 (p < 0.001) (0.96 – 0.99)	0.98 (p = 0.001) (0.96 – 0.99)
Maintenance village	0.34 (p < 0.001) (0.23 – 0.50)	0.41 (p < 0.001) (0.25 – 0.67)	0.43 (p = 0.005) (0.24 – 0.77)
Number residing in house [b]	1.03 (p = 0.25) (0.98 – 1.09)	0.96 (p = 0.18) (0.90 – 1.02)	0.94 (p = 0.11) (0.88 – 1.01)
Smokes	0.35 (p = 0.02) (0.14 – 0.85)	0.91 (p = 0.85) (0.37 – 2.27)	0.94 (p = 0.89) (0.36 – 2.41)
Exposure intensity			
0 – 20,000 mg.m^{-3}	Omitted category		Omitted category
20,000 – 50,000 mg.m^{-3}	1.15 (p = 0.48) * (0.78 – 1.68)		2.64 (p = 0.19) (0.61 – 11.43)
50,000 – 100,000 mg.m^{-3}	1.22 (p = 0.32) * (0.82 – 1.81)		2.78 (p = 0.19) (0.61 – 12.57)
>100,000 mg.m^{-3}	1.63 (p = 0.01) * (1.13 – 2.35)		4.40 (p = 0.06) (0.93 – 20.73)
Household Tasks			
No household task	Omitted category		Omitted category
Some household task	1.73 (p = 0.03) * (1.05 – 2.86)		0.32 (p = 0.15) * (0.07 – 1.48)
Some cooking	0.86 (p = 0.48) * (0.56 – 1.31)		0.19 (p = 0.03) * (0.04 – 0.82)
Regular cooking	1.06 (p = 0.71) * (0.77 – 1.46)		0.33 (p = 0.16) * (0.07 – 1.53)
Sample size (N)	229	229	229

[a] Eye disease rate in the 0 – 200 μg.m^{-3} is 0. Therefore in this analysis the lowest exposure group has been extended to the 0 – 500 μg.m^{-3} range to allow obtaining odds ratios.

[b] Odd ratios of age and household size, both continuous variables, represent the odds ratios for two subsequent units of these variables.

*** Jointly significant ($p \leq 0.001$) * Jointly significant ($p \leq 0.1$)

For those between 6 and 50, as for the younger group, the *blogit* regression also shows a statistically significant increasing concave relationship between ARI and ALRI with a

change in the slope of the exposure-response relationship above the 1000 – 2000 $\mu g.m^{-3}$ exposure category.

Also as in the OLS model, the gross increased probability of ARI and ALRI for the residents of *bomas* disappears after controlling for exposure. Similarly, the gross increased probability of ALRI for females disappears in the multivariate models. Women are however still 1.24 (95% C.I. 1.01 – 1.52) times more likely than men to be diagnosed with ARI after exposure is accounted for in Model 2. The odds ratio is no longer significant after exposure intensity and participation in household tasks have been accounted for. This, and also a statistically significant odds ratio for ALRI for the group who regularly take part in cooking, may imply a short-term or chronic impact from long periods of daily exposure. For ARI, this impact is captured by the odds ratio for the female variable when participation in cooking activities is not included in the model (i.e. Model 2).

The probability of being diagnosed with ALRI increases with age, but as above the effect is eliminated after controlling for participation in household tasks and exposure intensity. As discussed in Section 7.3.1 this may also be an indicator of, short-term or chronic, impacts of long daily periods of exposure. There is a decrease in ARI rates with age in Model 2 but not in Model 3. This effect cannot be explained using any physiological mechanism of toxicity, except increased immunity (which is not expected to continue at higher ages).

The number of people living in the household does not affect ARI or ALRI rates. Smokers are 1.48 (95% C.I. 1.07 – 2.04) times as likely as non-smokers to be diagnosed with ARI but not ALRI. Both results are consistent with the OLS parameters.

Also as with the OLS, exposure intensity is not a determinant of ARI and ALRI rates after average exposure has been accounted for. The level of participation in various household tasks does not affect ARI rates but the individuals who cook regularly have additional susceptibility to ALRI, even after controlling for average exposure (although the participation variables are not jointly significant). The possible reasons for this are discussed in Section 7.3.1.

For eye disease also the results are similar to the OLS model. Exposure categories are jointly significant but except for the lowest and highest exposure groups, diseases of the eye do not show any systematic relationship with exposure. Residence in *boma* is the only variable increasing the probability of eye disease, which decreases with age.

7.4 The Role of Exposure Estimation Methodology

The above parameter estimates were based on exposure values calculated using the exposure profile approach which account for patterns of exposure of individuals, including their time budget and activities, and the spatial dispersion of smoke in the house. In Chapter 6, I compared these with exposure estimates that were obtained using concentrations at a single point and time spent inside only. In Table 7.7 and Table 7.8, I use this latter measure of exposure in estimating the parameters of the exposure-response

relationship for the two age groups of 0 – 5 years and 5 – 50 years (for the categorical treatment of exposure, for ARI and ALRI).[44]

Table 7.7: OLS parameter estimates for illness rates using categorical exposure variables for 0 – 5 age group. (a) ARI. (b) ALRI. Exposure values are a product of average concentration at a single point and time spent inside. Model 1 (bivariate) corresponds to parameter estimates in which illness rates are regressed against each variable one at a time, whereas in Models 2 and 3 (multivariate) all the variables are considered together. No one under the age of 5 participates in household tasks. Therefore Models 2 and 3 are equivalent for this age group.

(a) ARI

	Model 1	**Model 2**	**Model 3**
Constant	0.13 ($p < 0.001$)	0.06 ($p = 0.21$)	0.06 ($p = 0.21$)
Exposure			
0 – 200 mg.m^{-3}	Omitted category	Omitted category	Omitted category
200 – 500 mg.m^{-3}	0.075 ($p < 0.001$)	0.07 ($p < 0.001$) **	0.07 ($p < 0.001$) **
500 – 1000 mg.m^{-3}	0.05 ($p = 0.02$)	0.05 ($p = 0.08$) **	0.05 ($p = 0.08$) **
1000 – 2000 mg.m^{-3}	0.1 ($p = 0.003$)	0.1 ($p = 0.001$) **	0.1 ($p = 0.001$) **
2000 – 3500 mg.m^{-3}	0.12 ($p = 0.006$)	0.12 ($p = 0.01$) **	0.12 ($p = 0.01$) **
> 3500 mg.m^{-3}	0.18 ($p = 0.06$)	0.17 ($p = 0.08$) **	0.17 ($p = 0.08$) **
Gender (*Female = 1*)	-0.006 ($p = 0.79$)	-0.003 ($p = 0.87$)	-0.003 ($p = 0.87$)
Age	-0.01 ($p = 0.02$)	-0.006 ($p = 0.18$)	-0.006 ($p = 0.18$)
Village (*maintenance village = 1*)	-0.037 ($p = 0.09$)	0.006 ($p = 0.81$)	0.006 ($p = 0.81$)
Number residing in house	0.0002 ($p = 0.96$)	0.0006 ($p = 0.92$)	0.0006 ($p = 0.92$)
Smokes	N/A	N/A	N/A
Exposure intensity [a]			
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
R^2	N/A	0.18	0.18
Sample size (*N*)	93	93	93
$p > F$	N/A	0.002	0.002

[a] When using average concentration, intensity of exposure is not defined.
** Jointly significant ($p \leq 0.01$)

[44] The results are presented for the OLS model only. The *blogit* results are similar.

(b) ALRI

	Model 1	**Model 2**	**Model 3**
Constant	0.05 ($p < 0.001$)	0.07 ($p = 0.008$)	0.07 ($p = 0.008$)
Exposure			
0 – 200 $mg.m^{-3}$	Omitted category	Omitted category	Omitted category
200 – 500 $mg.m^{-3}$	0.02 ($p = 0.12$)	0.017 ($p = 0.12$)	0.017 ($p = 0.12$)
500 – 1000 $mg.m^{-3}$	0.01 ($p = 0.18$)	0.016 ($p = 0.19$)	0.016 ($p = 0.19$)
1000 – 2000 $mg.m^{-3}$	0.01 ($p = 0.29$)	0.013 ($p = 0.18$)	0.013 ($p = 0.18$)
2000 – 3500 $mg.m^{-3}$	0.04 ($p = 0.08$)	0.04 ($p = 0.16$)	0.04 ($p = 0.16$)
> 3500 $mg.m^{-3}$	0.05 ($p = 0.18$)	0.04 ($p = 0.38$)	0.04 ($p = 0.38$)
Gender (*Female = 1*)	-0.01 ($p = 0.23$)	-0.006 ($p = 0.47$)	-0.006 ($p = 0.47$)
Age	-0.013 ($p < 0.001$)	-0.011 ($p = 0.002$)	-0.011 ($p = 0.002$)
Village (*maintenance village = 1*)	-0.007 ($p = 0.52$)	0.001 ($p = 0.90$)	0.001 ($p = 0.90$)
Number residing in house	0.0005 ($p = 0.80$)	-0.0003 ($p = 0.92$)	-0.0003 ($p = 0.92$)
Smokes	N/A	N/A	N/A
Exposure intensity [a]			
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
R^2		0.17	0.17
Sample size (*N*)	93	93	93
$p > F$	N/A	0.003	0.003

[a] When using average concentration, intensity of exposure is not defined.

(c) Eye Disease

	Model 1	**Model 2**	**Model 3**
Constant	0.06 ($p < 0.001$)	0.11 ($p = 0.01$)	0.11 ($p = 0.01$)
Exposure			
0 – 200 $mg.m^{-3}$	Omitted category	Omitted category	Omitted category
200 – 500 $mg.m^{-3}$	0.03 ($p = 0.001$)	0.023 ($p = 0.02$) *	0.023 ($p = 0.02$) *
500 – 1000 $mg.m^{-3}$	0.06 ($p < 0.001$)	0.036 ($p = 0.04$) *	0.036 ($p = 0.04$) *
1000 – 2000 $mg.m^{-3}$	0.07 ($p < 0.001$)	0.025 ($p = 0.11$) *	0.025 ($p = 0.11$) *
2000 – 3500 $mg.m^{-3}$	0.09 ($p < 0.001$)	0.033 ($p = 0.18$) *	0.033 ($p = 0.18$) *
> 3500 $mg.m^{-3}$	0.17 ($p = 0.01$)	0.098 ($p = 0.1$) *	0.098 ($p = 0.1$) *
Gender (*Female = 1*)	0.007 ($p = 0.67$)	0.01 ($p = 0.26$)	0.01 ($p = 0.26$)
Age	-0.01 ($p = 0.02$)	-0.016 ($p < 0.001$)	-0.016 ($p < 0.001$)
Village (*maintenance village = 1*)	-0.017 ($p = 0.004$)	-0.056 ($p = 0.008$)	-0.056 ($p = 0.008$)
Number residing in house	0.003 ($p = 0.20$)	-0.003 ($p = 0.46$)	-0.003 ($p = 0.46$)
Smokes	N/A	N/A	N/A
Exposure intensity [a]			
Household Tasks			
No household task	N/A		N/A
Some household task	N/A		N/A
Some cooking	N/A		N/A
Regular cooking	N/A		N/A
R^2		0.36	0.36
Sample size (*N*)	93	93	93
$p > F$	N/A	< 0.0001	< 0.0001

[a] When using average concentration, intensity of exposure is not defined.

* Jointly significant ($p \leq 0.1$)

Table 7.8: OLS parameter estimates for illness rates using categorical exposure variables for 6 – 50 age group. (a) ARI. (b) ALRI. Exposure values are a product of average concentration at a single point and time spent inside. Model 1 (bivariate) corresponds to parameter estimates in which illness rates are regressed against each variable one at a time, whereas in Models 2 and 3 (multivariate) all the variables are considered together.

(a) ARI

	Model 1	**Model 2**	**Model 3**
Constant	0.06 ($p < 0.001$)	0.01 ($p = 0.58$)	0.02 ($p = 0.31$)
Exposure			
0 – 200 mg.m^{-3}	N/A	N/A	N/A
200 – 500 mg.m^{-3}	0.035 ($p = 0.001$)	0.036 ($p = 0.001$) ***	0.031 ($p = 0.007$) ***
500 – 1000 mg.m^{-3}	0.034 ($p = 0.002$)	0.037 ($p = 0.02$) ***	0.033 ($p = 0.05$) ***
1000 – 2000 mg.m^{-3}	0.063 ($p < 0.001$)	0.062 ($p = 0.002$) ***	0.056 ($p = 0.006$) ***
2000 – 4000 mg.m^{-3}	0.066 ($p < 0.001$)	0.062 ($p = 0.003$) ***	0.057 ($p = 0.008$) ***
4000 – 7000 mg.m^{-3}	0.13 ($p < 0.001$)	0.12 ($p < 0.001$) ***	0.11 ($p < 0.001$) ***
> 7000 mg.m^{-3}	N/A	N/A	N/A
Gender (*Female = 1*)	0.035 ($p < 0.001$)	0.031 ($p < 0.001$)	0.017 ($p = 0.09$)
Age	-0.00002 ($p = 0.92$)	0.0001 ($p = 0.66$)	-0.0001 ($p = 0.68$)
Village (*maintenance village = 1*)	-0.02 ($p = 0.02$)	-0.002 ($p = 0.88$)	-0.006 ($p = 0.69$)
Number residing in house	0.0002 ($p = 0.95$)	-0.002 ($p = 0.44$)	-0.002 ($p = 0.41$)
Smokes	-0.008 ($p = 0.31$)	0.013 ($p = 0.14$)	0.017 ($p = 0.08$)
Exposure intensity [a]			
Household Tasks			
No household task	N/A		N/A
Some household task	0.03 ($p = 0.12$)		0.02 ($p = 0.26$)
Some cooking	0.02 ($p = 0.03$)		0.006 ($p = 0.49$)
Regular cooking	0.04 ($p < 0.001$)		0.02 ($p = 0.07$)
R^2	N/A	0.21	0.23
Sample size (*N*)	229	229	229
$p > F$	N/A	< 0.0001	< 0.0001

[a] When using average concentration, intensity of exposure is not defined.

*** Jointly significant ($p \leq 0.001$)

(b) ALRI

	Model 1	**Model 2**	**Model 3**
Constant	0.02 ($p < 0.001$)	-0.003 ($p = 0.57$)	0.005 ($p = 0.51$)
Exposure			
0 – 200 $mg.m^{-3}$	N/A	N/A	N/A
200 – 500 $mg.m^{-3}$	0.008 ($p = 0.20$)	0.009 ($p = 0.14$) ***	0.005 ($p = 0.38$) ***
500 – 1000 $mg.m^{-3}$	0.007 ($p = 0.04$)	0.007 ($p = 0.09$) ***	0.003 ($p = 0.49$) ***
1000 – 2000 $mg.m^{-3}$	0.015 ($p = 0.002$)	0.013 ($p = 0.006$) ***	0.007 ($p = 0.15$) ***
2000 – 4000 $mg.m^{-3}$	0.017 ($p = 0.001$)	0.013 ($p = 0.03$) ***	0.008 ($p = 0.23$) ***
4000 – 7000 $mg.m^{-3}$	0.053 ($p = 0.04$)	0.044 ($p < 0.001$) ***	0.034 ($p < 0.001$) ***
> 7000 $mg.m^{-3}$	N/A	N/A	N/A
Gender (*Female = 1*)	0.009 ($p = 0.001$)	0.009 ($p = 0.003$)	-0.002 ($p = 0.62$)
Age	0.0003 ($p = 0.001$)	0.0003 ($p = 0.001$)	0.0001 ($p = 0.21$)
Village (*maintenance village = 1*)	-0.008 ($p = 0.008$)	-0.003 ($p = 0.39$)	-0.007 ($p = 0.12$)
Number residing in house	0.0003 ($p = 0.67$)	-0.0002 ($p = 0.83$)	-0.0001 ($p = 0.88$)
Smokes	-0.001 ($p = 0.81$)	0.003 ($p = 0.59$)	0.006 ($p = 0.22$)
Exposure intensity [a]			
Household Tasks			
No household task	N/A		N/A
Some household task	0.006 ($p = 0.11$)		0.004 ($p = 0.46$) **
Some cooking	0.007 ($p = 0.11$)		0.002 ($p = 0.42$) **
Regular cooking	0.02 ($p < 0.001$)		0.02 ($p = 0.003$) **
R^2	N/A	0.14	0.18
Sample size (*N*)	229	229	229
$p > F$	N/A	< 0.0001	< 0.0001

[a] When using average concentration, intensity of exposure is not defined.

*** Jointly significant ($p \leq 0.001$) ** Jointly significant ($p \leq 0.01$)

(c) Eye Disease

	Model 1	**Model 2**	**Model 3**
Constant	0.01 ($p < 0.001$)	0.015 ($p = 0.03$)	0.014 ($p = 0.12$)
Exposure			
0 – 200 $mg.m^{-3}$	N/A	N/A	N/A
200 – 500 $mg.m^{-3}$	0.011 ($p = 0.02$)	0.01 ($p = 0.04$) *	0.01 ($p = 0.02$) *
500 – 1000 $mg.m^{-3}$	0.010 ($p < 0.001$)	0.006 ($p = 0.06$) *	0.006 ($p = 0.09$) *
1000 – 2000 $mg.m^{-3}$	0.019 ($p < 0.001$)	0.012 ($p = 0.02$) *	0.012 ($p = 0.03$) *
2000 – 4000 $mg.m^{-3}$	0.021 ($p < 0.001$)	0.014 ($p = 0.05$) *	0.015 ($p = 0.04$) *
4000 – 7000 $mg.m^{-3}$	0.022 ($p < 0.001$)	0.013 ($p = 0.08$) *	0.014 ($p = 0.09$) *
> 7000 $mg.m^{-3}$	N/A	N/A	N/A
Gender (*Female = 1*)	0.005 ($p = 0.09$)	0.002 ($p = 0.44$)	0.004 ($p = 0.53$)
Age	-0.0003 ($p = 0.04$)	-0.0002 ($p = 0.09$)	-0.0002 ($p = 0.14$)
Village (*maintenance village = 1*)	-0.01 ($p < 0.001$)	-0.007 ($p = 0.06$)	-0.007 ($p = 0.08$)
Number residing in house	0.0003 ($p = 0.67$)	-0.0007 ($p = 0.23$)	-0.0008 ($p = 0.20$)
Smokes	-0.01 ($p = 0.006$)	-0.003 ($p = 0.45$)	-0.003 ($p = 0.42$)
Exposure intensity [a]			
Household Tasks			
No household task	N/A		N/A
Some household task	0.009 ($p = 0.33$)		0.004 ($p = 0.66$)
Some cooking	0.0002 ($p = 0.96$)		-0.005 ($p = 0.48$)
Regular cooking	0.002 ($p = 0.60$)		-0.002 ($p = 0.75$)
R^2	N/A	0.10	0.11
Sample size (*N*)	229	229	229
$p > F$	N/A	0.001	0.001

[a] When using average concentration, intensity of exposure is not defined.

* Jointly significant ($p \leq 0.1$)

For both age groups, illness rates rise at a faster rate with increasing exposure compared to Table 7.2 and Table 7.4, especially in lower exposure ranges. This is because, as I described in Chapter 6, using average concentration at one point results in an underestimation of exposure compared to the exposure profile approach. This downward compression of the explanatory variable is equivalent to raising of the slope of the exposure response relationship, especially in lower exposure ranges.

The most important feature of the comparison between the two exposure estimation methods is the coefficient of gender in Table 7.8 and Table 7.4. In the latter, where patterns of male and female exposure to PM_{10} are accounted for by using the exposure

profile approach, males and females have similar response (i.e. the coefficient of gender is not significant). But if exposure is estimated from average daily PM_{10} concentration and time spent indoors only (Table 7.8 Model 2) females in the 6 – 50 range are found to have additional susceptibility to ARI by 0.03 ($p < 0.001$) and ALRI by 0.01 ($p = 0.003$). In Chapter 6, I demonstrated that this latter (and commonly used) method of exposure estimation underestimates the exposure of women – who cook – more than men. This comparison shows that this underestimation results in systematic bias in assessment of the exposure-response relationship.

This bias is further confirmed by noting that the role of gender appears only after the age of 5 when females actually take part in household activities. For age ≤ 5 (Table 7.7), the coefficient of gender remains insignificant ($p = 0.87$ for ARI and $p = 0.47$ for ALRI). Finally, in Table 7.8, controlling for the amount of cooking activity that a person performs (Model 3) eliminates the statistical significance of gender, further confirming that the role of gender is a substitute for exposure patterns (i.e. a proxy for the omitted variable of high intensity exposure) when average daily PM_{10} concentration is used.

7.5 Summary of Main Results

The analysis of this chapter illustrates that:

- ARI and acute lower respiratory infections (ALRI) are increasing, concave functions of average daily exposure to PM_{10}, with the rate of increase declining for exposures above approximately 2000 $\mu g.m^{-3}$. The result is robust to the choice of the exposure-response parameter estimation model (OLS or *blogit*).

- After controlling for other variables, in particular exposure, gender is not an important determinant of ARI. Only in the *blogit* estimation, females above 5 are 1.24 (95% C.I. 1.01 – 1.52) times more likely than men to be diagnosed with ARI. This effect disappears after accounting for participation in household activities and intensity of exposure.
- Disease rates decrease with age for infants and children below 6. Age is not a determinant of disease for older children and adults.
- In this setting, the type of the village that an individual resides in and the number of household members are not associated with disease incidence in a statistically significant manner.
- If exposure is estimated from average daily PM_{10} concentration and time spent indoors only (i.e. without accounting for the specific activities and movement patterns of individuals) young and adult females are found to have additional susceptibility to ARI by 0.03 ($p < 0.001$) and ALRI by 0.01 ($p < 0.01$). Once total exposure is calculated to appropriately include high-intensity exposure episodes, however, gender is no longer an effective indicator of ARI and ALRI rates.
- The intensity of exposure does not contribute to the incidence of disease, once its role is accounted for in total exposure. A similar results exists for participation in household tasks, except for the individuals that participate in cooking regularly, who are more likely to be diagnosed with ALRI.

Chapter 8 Energy Technology and Indoor Air Pollution [45]

"Improved" (high-efficiency and low-emission) have been the most celebrated tool in efforts to reduce indoor air pollution in developing countries in the past two decades (153, 159). Improved stoves were initially of interest to the international development community because of their potential to reduce fuel consumption and thus deforestation and land degradation (40, 160). Their public health benefits from reduction in exposure to indoor smoke became the subject of attention soon after. This "double-dividend" – improvements in public health while reducing adverse environmental impacts – focused a great deal of effort on the design and dissemination of improved stoves (45, 161, 162).

Initial works on the benefits of improved stoves were often marked by a lack of detailed data on stove performance. Efficiencies and emissions, for example, were often measured in controlled environments as the stoves were used by technical experts under conditions very dissimilar to those in the field (39, 40). More recently, the attention of the research community has shifted from such ideal operating conditions to monitoring stove performance under actual conditions of use, taking into account the various social and physical factors that would limit the use of these stoves all together or result in "sub-

[45] An earlier version of this chapter has been published as the following article: Ezzati, M., B. M. Mbinda, and D. M. Kammen (2000) "Comparison of Emissions and Residential Exposure from Traditional and Improved Cookstoves in Kenya," *Environmental Science and Technology*, **34**, 578-583. In that work, I also discuss the carbon monoxide (CO) emissions in detail.

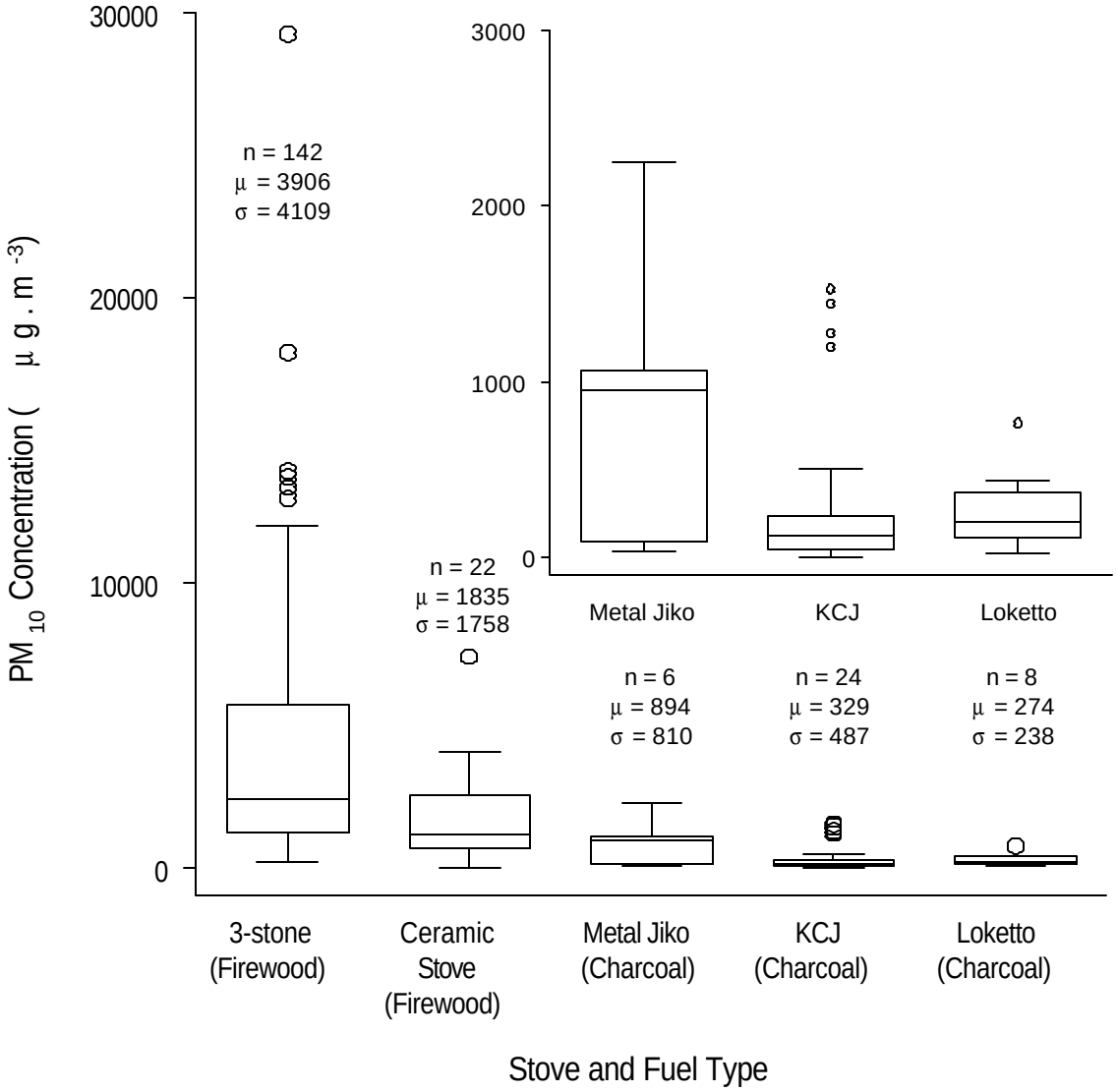
optimal" use (41, 42). As a result of these studies the initially-perceived high level of benefits from improved stoves has been called into question (35, 43).

In this chapter, I analyze the performance of an array of stove-fuel combinations used extensively by Kenyan households, specifically those at Mpala Ranch. The stoves, described in Table 4.3 and seen in Figure 1.6 and Figure 1.7, include the traditional open fire and *Metal Jiko* as well as a set of improved cookstoves. Data for analysis are from the 210 days of monitoring of emission concentrations under the actual conditions of use in 55 households. In this manner, this chapter complements the thorough work of Ballard-Tremeer and Jawurek (35) who compare the performance of five rural wood-burning stoves using standard tests.

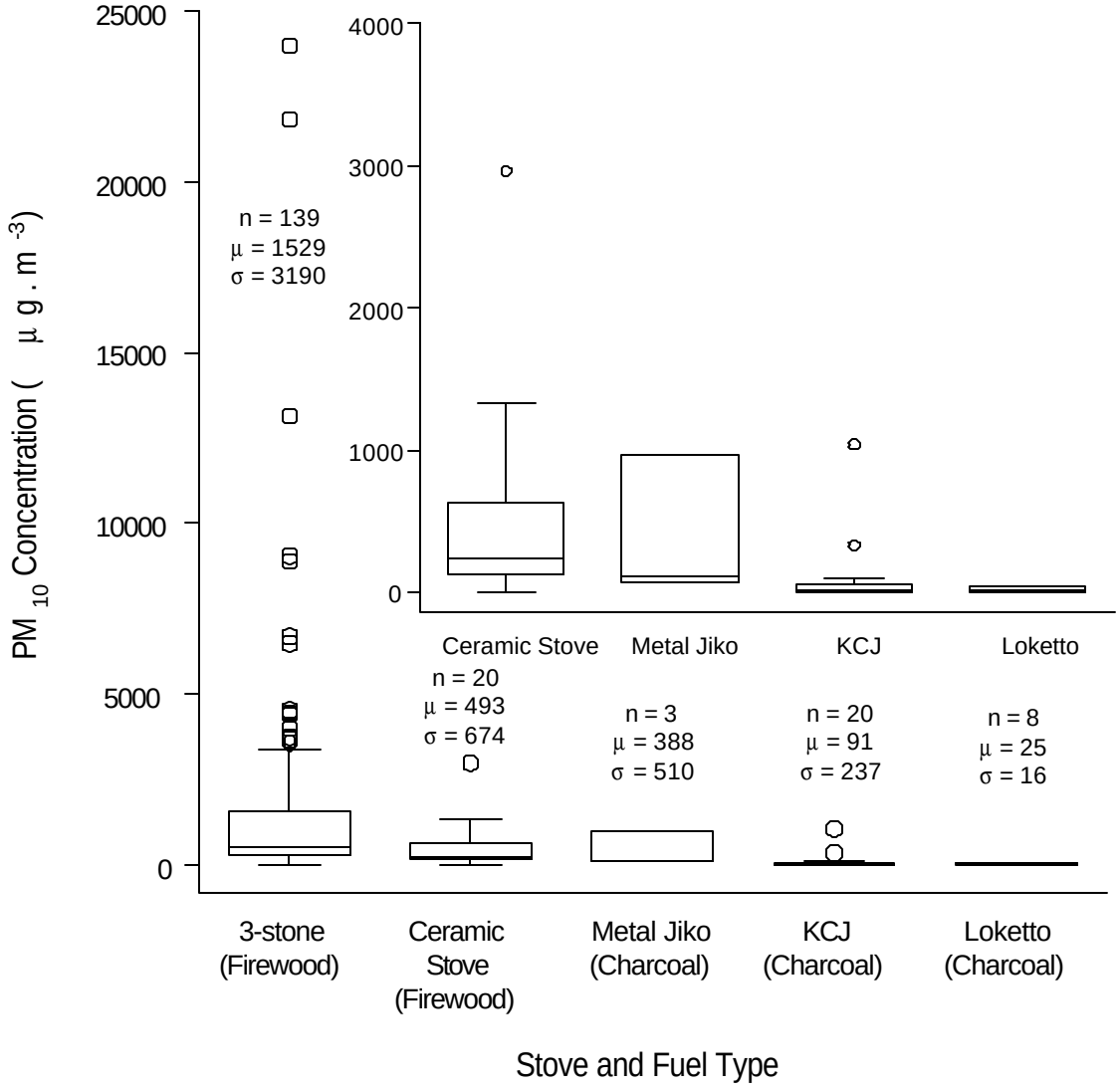
8.1 Comparison of average emission concentrations

Figure 8.1 illustrates the average suspended particulate (PM_{10}) concentration, averaged over the *burning* (panel a) and *smoldering* (panel b) periods respectively for various stove-fuel combinations. Quantitative comparison of these values using two-sided two-sample t-tests are given in Table 8.1 to Table 8.4.[46] None of the changes in carbon monoxide concentration is statistically significant.

[46] I assumed unequal variances in the t-tests, to account for possible differences in stove attributes.


30000
20000
10000
0
3000
2000
1000
0
PM 10 Concentration (µ g . m -3)
n = 142
µ = 3906
σ = 4109
n = 22
µ = 1835
σ = 1758
n = 6
µ = 894
σ = 810
n = 24
µ = 329
σ = 487
n = 8
µ = 274
σ = 238
3-stone (Firewood)
Ceramic Stove (Firewood)
Metal Jiko (Charcoal)
KCJ (Charcoal)
Loketto (Charcoal)
Metal Jiko
KCJ
Loketto
Stove and Fuel Type

(a)



(b)

Figure 8.1: Day-long average of PM_{10} concentration for various stove and fuel combinations, calculated over: (a) *burning* period and (b) *smoldering* period. The diagram on the upper right hand corner is a more detailed version of the plot for the last 3 or 4 stoves. *n* refers to the number individuals in the demographic subgroup; *m* is the sample mean and σ the standard deviation. See Table 8.1 to Table 8.4 for numerical comparison of emissions and emission reductions.

Table 8.1: Reduction in mean PM_{10} emission concentration (during the *burning* period) as a result of introduction of improved stoves.

Traditional Stove	**Improved Stove**	**Reduction in Average Emission** [a]
3-Stone	Ceramic Wood Stoves	2071 (53%) (p = 0.02)
Metal Jiko	Kenya Ceramic Jiko	442 (49%) (p = 0.13)
Metal Jiko	Loketto	620 (69%) (p = 0.31)

[a] The first number indicates the value of reduction in ($\mu g.m^{-3}$) and the number in brackets the reduction as a fraction of the emissions of the traditional stove. *p*-values were obtained using t-tests on the logarithms of concentrations. This transformation allows converting the (skewed) distribution of concentrations to a normal distribution.

Table 8.2: Reduction in mean PM_{10} emission concentration (during the *burning* period) as a result of fuel change.

Firewood	**Charcoal**	**Reduction in Average Emission**
All Stoves (Traditional and Improved)	All Stoves (Traditional and Improved)	3204 (89%) ($p < 0.0001$)
Best Case (Ceramic Wood Stoves)	Worst Case (Metal Jiko)	941 (51%) (p = 0.32)

Table 8.3: Reduction in mean PM_{10} emission concentration (during the *smoldering* period) as a result of introduction of improved stoves.

Traditional Stove	**Improved Stove**	**Reduction in Average Emission**
3-Stone	Ceramic Wood Stoves	1036 (68%) (p = 0.02)
Metal Jiko	Kenya Ceramic Jiko	297 (77%) (p = 0.07)
Metal Jiko	Loketto	363 (94%) (p = 0.08)

Table 8.4: Reduction in mean PM_{10} emission concentration (during the *smoldering* period) as a result of fuel change.

Firewood	**Charcoal**	**Reduction in Average Emission**
All Stoves (Traditional and Improved)	All Stoves (Traditional and Improved)	1289 (93%) ($p < 0.0001$)
Best Case (Ceramic Wood Stoves)	Worst Case (Metal Jiko)	105 (21%) (p = 0.89)

These comparisons show that improved wood-burning cookstoves reduce average daily particulate matter emission concentration during burning by 53% (2071 $\mu g.m^{-3}$). Average emission concentration during the smoldering period is reduced by 68% (1036 $\mu g.m^{-3}$). The larger relative reduction during smoldering compared to burning is because of the operation of the stove, not to the thermodynamics of combustion. 3-stone stove is often used with larger pieces of wood that remain in the stove for a longer period after cooking has taken place. Improved stoves, on the other hand, are used with smaller pieces of wood which stop burning shortly after the active use of the stove is terminated. Moreover, since ceramic stoves are portable, it is not uncommon for people to remove them from the house once cooking has taken place (see also the section on the comparison of intense emissions below).

For charcoal stoves, during the burning period the average suspended particulate emission concentrations of *KCJ* and *Loketto* are 49% (442 $\mu g.m^{-3}$) and 69% (620 $\mu g.m^{-3}$) lower than that of *Metal Jiko* respectively. This reduction is not statistically significant (potentially due to the small sample size). Although the absolute value of these reductions are small (relative to that between improved and traditional wood stoves), the improved charcoal stoves, with emission concentration levels of approximately 300 $\mu g.m^{-3}$, are the only biomass stoves in the study group that approach international standards. The USEPA standard for PM_{10} for example, requires a 24-hour average of no more than 150 $\mu g.m^{-3}$.

The difference between the 95th and 5th percentiles of emission concentrations (during burning) for the 3-stone, improved wood stoves, *Metal Jiko, KCJ*, and *Loketto* are 11376 $\mu g.m^{-3}$, 3797 $\mu g.m^{-3}$, 2223 $\mu g.m^{-3}$, 1436 $\mu g.m^{-3}$, 736 $\mu g.m^{-3}$ respectively (4.75, 3.26, 2.33, 11.31, 3.56 when normalized with respect to the median). All stove categories, therefore, exhibit large variability of emission concentrations. This variability illustrates that how a stove is used may be as important a determinant of emission as the stove type. This confirms under actual conditions of use the laboratory finding of Ballard-Tremeer and Jawurek (35) on the overlap between emission ranges of open fire and ceramic stoves.

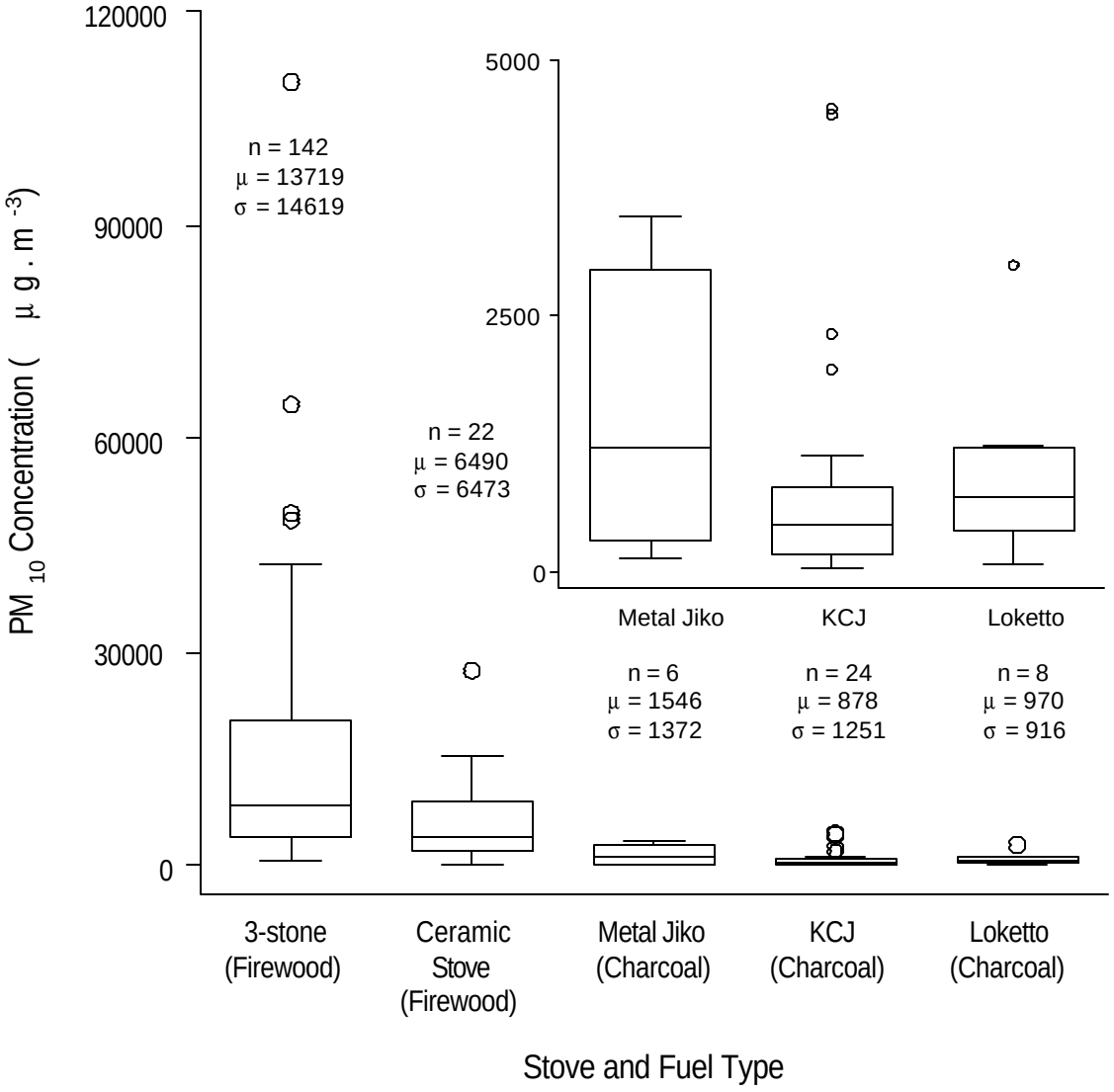
The largest reduction in suspended particulate emission concentration is achieved with transition from wood to charcoal in both burning and smoldering states. In the burning period, transition from wood to charcoal reduces average emission concentration by 3204 $\mu g.m^{-3}$ (89%), and in the smoldering period by 1289 $\mu g.m^{-3}$ (93%). During the burning period, even the comparison of the best-case scenario for wood stoves (improved wood stoves) and worst-case scenario for charcoal stoves (*Metal Jiko*) exhibits a drop in suspended particulate emission concentration of 51% (941 $\mu g.m^{-3}$) when charcoal is used. During the smoldering period, the best-case scenario for wood stoves (improved wood stoves) has comparable emission concentration to the worst-case scenario for charcoal stoves (*Metal Jiko*). As above, this relative improvement of improved wood stoves during the idle period is attributed to their operation, since they continue to smolder for a shorter period than the open fire.

8.2 Comparison of intense emission episodes

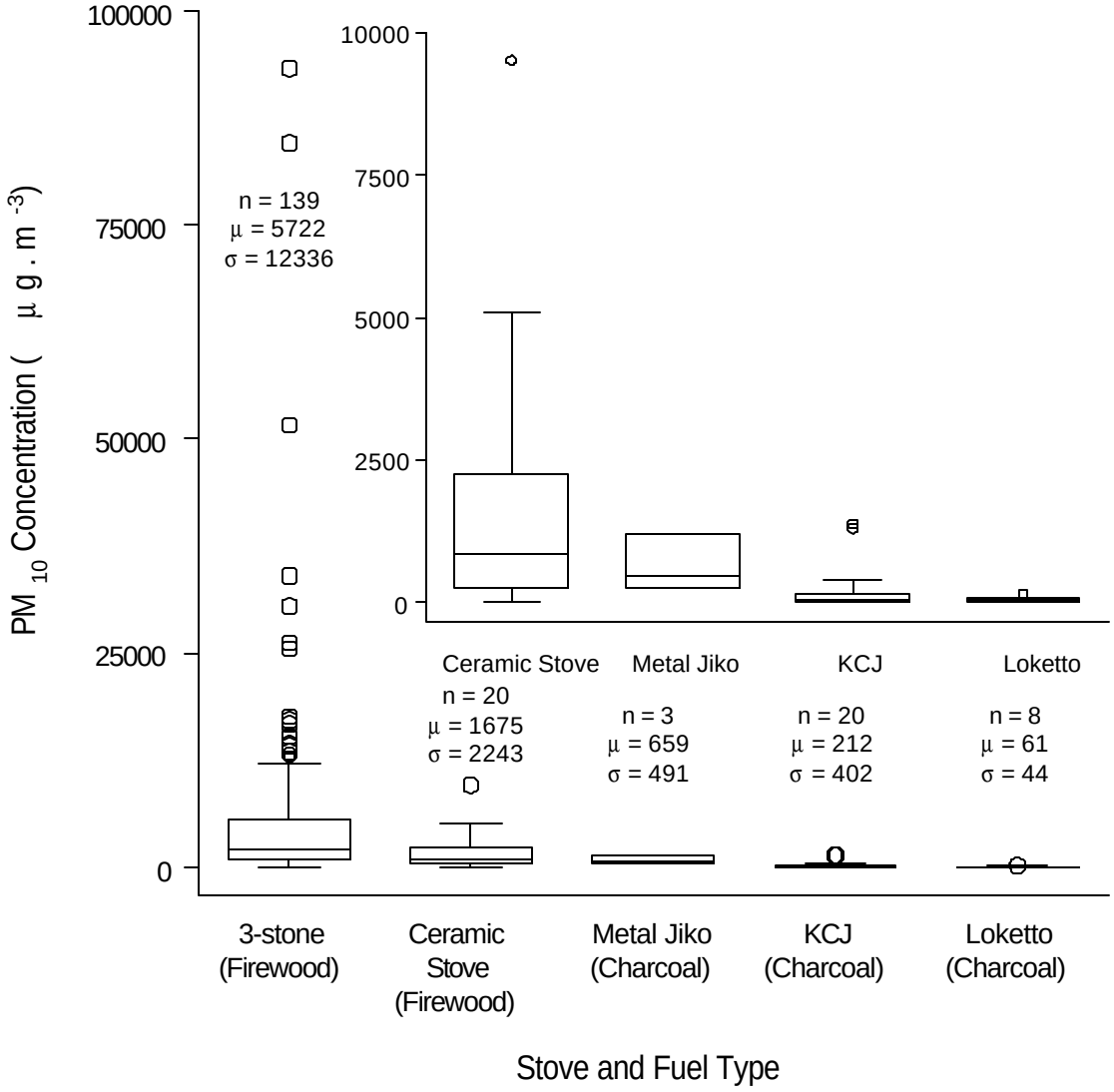
In Chapter 6, I argued that pollution levels vary a great deal throughout the day; and some household members, especially women who cook, are closest to fire when pollution level is the highest.

Therefore, average pollution level alone does not sufficiently explain the health impacts of household energy technology. I therefore go beyond this individual measure in comparing the various cookstoves and use other descriptive statistics, which may be better indicators of human exposure. Specifically, I compare stove emissions using the mean above the 75th percentile ($\mu_{>75}$) which, as described in Chapter 6, accounts for the important role of high-intensity exposure of women.

Figure 8.2 show the distribution of $\mu_{>75}$ for the burning period (panel a) and the smoldering period (panel b). Quantitative comparison of these values using two-sided two-sample t-tests are given in Table 8.5 to Table 8.8.


120000
90000
60000
30000
0
PM 10 Concentration (μ g . m -3)
n = 142
μ = 13719
σ = 14619
5000
2500
0
n = 22
μ = 6490
σ = 6473
Metal Jiko
KCJ
Loketto
n = 6
μ = 1546
σ = 1372
n = 24
μ = 878
σ = 1251
n = 8
μ = 970
σ = 916
3-stone (Firewood)
Ceramic Stove (Firewood)
Metal Jiko (Charcoal)
KCJ (Charcoal)
Loketto (Charcoal)
Stove and Fuel Type

(a)



(b)

Figure 8.2: Mean above the 75th percentile ($\mu_{>75}$) of PM_{10} concentration for various stove and fuel combinations, calculated over: (a) *burning* period and (b) *smoldering* period. The diagram on the upper right hand corner is a more detailed version of the plot for the last 3 or 4 stoves. *n* refers to the number individuals in the demographic subgroup; *m* is the sample mean and σ the standard deviation. See Table 8.5 to Table 8.8 for numerical comparison of emissions and emission reductions.

Table 8.5: Reduction in mean above the 75th percentile ($\mu_{>75}$) of PM_{10} emission concentration (during the *burning* period) as a result of introduction of improved stoves.

Traditional Stove	**Improved Stove**	**Reduction in Average Emission** [a]
3-Stone	Ceramic Wood Stoves	7229 (53%) (p = 0.02)
Metal Jiko	Kenya Ceramic Jiko	667 (43%) (p = 0.20)
Metal Jiko	Loketto	576 (37%) (p = 0.59)

[a] The first number indicates the value of reduction in ($\mu g.m^{-3}$) and the number in brackets the reduction as a fraction of the emissions of the traditional stove. p-values were obtained using t-tests on the logarithms of concentrations. This transformation allows converting the (skewed) distribution of concentrations to a normal distribution.

Table 8.6: Reduction in mean above the 75th percentile ($\mu_{>75}$) of PM_{10} emission concentration (during the *burning* period) as a result of fuel change.

Firewood	**Charcoal**	**Reduction in Average Emission**
All Stoves (Traditional and Improved)	All Stoves (Traditional and Improved)	11686 (92%) ($p < 0.0001$)
Best Case (Ceramic Wood Stoves)	Worst Case (Metal Jiko)	4944 (76%) (p = 0.07)

Table 8.7: Reduction in mean above the 75th percentile ($\mu_{>75}$) of PM_{10} emission concentration (during the *smoldering* period) as a result of introduction of improved stoves.

Traditional Stove	**Improved Stove**	**Reduction in Average Emission** [a]
3-Stone	Ceramic Wood Stoves	4047 (71%) (p = 0.01)
Metal Jiko	Kenya Ceramic Jiko	447 (68%) (p = 0.01)
Metal Jiko	Loketto	598 (91%) (p = 0.01)

Table 8.8: Reduction in mean above the 75th percentile ($\mu_{>75}$) of PM_{10} emission concentration (during the *smoldering* period) as a result of fuel change.

Firewood	**Charcoal**	**Reduction in Average Emission**
All Stoves (Traditional and Improved)	All Stoves (Traditional and Improved)	4968 (96%) ($p < 0.0001$)
Best Case (Ceramic Wood Stoves)	Worst Case (Metal Jiko)	1016 (61%) (p = 0.65)

The above comparisons illustrate that during the burning period, improved wood stoves provide an overall reduction in the emission concentration compared to 3-stone fire. In addition to mean concentration, the ceramic wood stoves also reduce the mean above the 75th percentile ($\mu_{>75}$) by 53% (7229 $\mu g.m^{-3}$). Therefore, these stoves shift the whole distribution of emission concentration downwards, thus reducing human exposure. In particular the reduction in $\mu_{>75}$ can be interpreted as lower emissions when people are closest to the stoves.[47]

When the stoves are not actively burning, $\mu_{>75}$ for improved wood stoves is 71% (4047 $\mu g.m^{-3}$ less than that of open fire. We saw earlier that ceramic wood stoves also reduce the mean daily emission concentration during the smoldering period compared to 3-stone fire. There is, however no reduction in the median emissions during smoldering as a result of transition to ceramic wood stoves (14). Simultaneous reductions in mean and $\mu_{>75}$, but not in median, emphasizes the idea that during the smoldering periods of the day most emissions occur in a short (but intense) period; for the rest of the time both stove types combust at low (and similar) levels.

The daily emission concentration profiles illustrate that this short period is often immediately before or immediately after combustion, when the stove is being lit or extinguished. Coupled with this reduction during non-cooking period is our quantitative

[47] The improved stoves in the study area were found not to offer significant reductions in carbon monoxide emission concentrations (14). Since these concentrations remain above the recommended WHO concentration of 87 ppm for more than 15 minutes (13), the public health benefits of these stoves is from the reduction in suspended particulate matter only.

and qualitative observation that at least some household members are likely to be in the house for a period after the completion of cooking, to serve/eat/drink food or tea for example, to clean the dishes used for cooking, or to sweep the house. With exposure extending beyond the active the burning period, the smoldering period reductions also provide benefits in lowering human exposure.

The improved charcoal stoves (*KCJ* and *Loketto*) offer only moderate emission reductions compared to the older *Metal Jiko.* *KCJ* and *Loketto* reduce $\mu_{>75}$ during the burning period by 667 $\mu g.m^{-3}$ (43%) and 576 $\mu g.m^{-3}$ (37%), but the results are not statistically significant. The lack of statistical significance in reductions during the burning period can be partially attributed to small sample size. At the same time, given the large similarity between *Metal Jiko* and the improved charcoal stoves (they both burn charcoal in a small compartment) and the physical attributes of charcoal combustion (relatively homogenous fuel with high carbon content) similar emission levels may be expected.

The largest reduction of high-intensity emission concentrations is also achieved through a transition from wood to charcoal. With this fuel transition, during the burning period $\mu_{>75}$ decreases by 92% (11686 $\mu g.m^{-3}$) and during smoldering by 96% (4968 $\mu g.m^{-3}$). These reductions imply a large overall downward shift in the pollution profile, and therefore human exposure, as a result of charcoal use.

Finally, similar to mean emission concentration, the large variations in daily median within each stove-fuel group illustrates that the benefits of improved stoves could theoretically be achieved through the best-mode operation of the traditional ones.

Chapter 9 Evaluation of Household Level Technology [48]

Numerous international development projects focus on facilitating the adoption of small-scale technologies among communities and households in the developing world. A perceived slow pace of adoption, typically accompanied by a lack of financial resources, has motivated the involvement of numerous development organizations in the process of technological change at the household level. Examples include high-efficiency cookstoves, rural electrification and other energy technologies, water and sanitation and other small-scale environmental projects, and agricultural techniques. Cost-benefit analysis has been used as a tool for evaluation of such technologies (see for example 1, 163, 164).

In this chapter, I examine the appropriateness of cost-benefit analysis for assessment of household technologies, from the perspective of *household level* decision making, and conclude that because of its mechanistic nature, cost-benefit analysis can account for neither the social context of technology and household preferences nor the fundamental transformations that new technology introduces in household life. Therefore, while the urge to adopt new technologies is high, proponents of adoption often lack methods suitable for determining to which technologies, if any, limited resources should be allocated. I end the chapter with a brief outline of the principles of a more appropriate

[48] An earlier version of this chapter has been published as the following article: Ezzati, M. (1999) "The Missing Costs and Benefits in Application of Cost-Benefit Assessment to Household Level Technology,"

framework for the evaluation of household level technologies. Throughout the chapter, I use some common household technologies – such as agricultural techniques, water technology, and "improved" (low-emission and high-efficiency) stoves – as examples of the issues that arise in household level technology assessment.[49]

9.1 Technology Assessment and Cost-Benefit Analysis

Many of the methods commonly used in technology assessment and technology policy were constructed and formalized in the context of the industrialized world, especially in established market economies. Moreover, the formalization of such methods took place after many common household technologies were believed to have been adopted in these nations. Therefore, not only did the underlying principle of these societies – that of individual choice – encourage relinquishing decisions regarding technology adoption to the households themselves, but also technology policy makers did not perceive an urgent need to transform household life drastically through policy intervention.

As a result, during the formation and evolution of the field of "technology assessment," much of its conceptual and methodological developments focused on the costs and benefits that are experienced through *societal channels*[50] rather than those at the household level. The vast literature, numerous case studies, and various regulatory

---

presented at the Conference on *The Cost-Benefit Analysis Dilemma: Strategies and Alternatives, Yale University, New Haven, CT, October 1999*.

[49] I draw extensively on "Taming Nature: An Agriculture of Legibility and Simplicity," Chapter 8 in (165) when discussing agricultural techniques.

[50] In these cases, costs and benefits are initiated and experienced by specific and often different groups in society which allows for a ready separation of the costs of a new technology from its benefits.

methods addressing the health risks of environmental pollution are probably the most important example of technology assessment tools at large scales. In other words, the application of cost-benefit analysis to technology assessment has evolved in a context in which costs and benefits are not only identifiable by the evaluator but also presumably separable from individual preferences and values (which subsequently allows their aggregation).[51]

Today, technology-based development, especially in rural regions of developing nations, often involves technology transfer at the level of individual households, therefore unfolding under conditions different from those which formed the context for the construction of technology assessment methods in the industrialized world. The initial experiences of failure in technology-based development raised concerns about the blind transfer of technology, resulting in the "appropriate technology" movement in the international development community. The notion of appropriate technology however has so far mostly addressed technical characteristics and complexity of new technology, stopping short of a challenge to other underlying principles and methods of technology evaluation. Therefore, even with such concerns, economic cost-benefit analysis, accompanied by the "rational utility-seeking actor" norm, continues to be the major prescriptive vehicle to guide the allocation of resources.[52]

---

[51] See (166) (Chapter 7) for a more accurate description of the evolution of cost-benefit analysis in technology assessment. Taking Porter's historical perspective into account, even in the case of apparently well-defined and separable costs and benefits, CBA evolved as a tool for rationalization rather than evaluation.

[52] As explained in one World Bank report on rural water projects "all decisions are based on some form of benefit-cost assessment, even when the comparison is more intuitive than calculated. The question is not whether to compare benefits with costs, but how" (167). Another report, in developing a conceptual

At the same time, while new technologies – modern agricultural techniques, new medical practices, rural electrification, new water and sanitation projects, and so forth – are playing an increasingly important role in the realm of international development, reports on their failures are also extensively documented. According to a World Bank study for example, "out of 183 surveyed donor-supported rural water supplies in developing countries around the world, some 40 percent were out of order five years after commissioning. After seven and ten years the figures were 70 and 85 percent, respectively" (168). The same study also found that most water supplies in rural areas are out of order at any given time. Similar experiences exist in the context of agricultural techniques intended to increase food production or household income and welfare (165, 169, 170) and ceramic stoves designed for lowering wood consumption (43, 171). When these apparently beneficial efforts fail in actual implementation or when they cannot be sustained over time, the expert planners often enter a state of conceptual confusion.[53] In the following sections I consider the inconsistencies which arise when cost-benefit analysis – with its assumptions of well-identified costs and benefits which are separable

framework for cost-benefit analysis of water projects, focuses on the cost of medicine to treat water-borne diseases or attempts to "estimate the value of this [saved] time to users, in light of the evidence on the behavior for these households" (163). The 1993 *World Development Report*, which is devoted to the issue of health, despite its strengths on promoting environmental preventive management of public health, in discussing the cost-effectiveness of intervention strategies focuses solely on mechanical calculation of costs and benefits, leaving out the issue of the context of intervention technique (see 1, pp. 59 – 107).

[53] The two above mentioned reports on water and sanitation projects both refer to "past disappointments" in implementation of water projects blaming "too many untested assumptions". They then propose further analytical frameworks for the calculation of costs and benefits of new projects. In the case of green revolution technologies, initial reports had difficulty in explaining failure to observe predicted increases in crop production and/or household welfare (169).

from the context and preferences – is applied to household technology evaluation, before outlining the principles of an alternative strategy.

9.2 The Unknowable Impacts of New Technology

In a deterministic description, technology is defined as the solution to a specific problem and a tool "created by humans to carry out tasks they could not otherwise accomplish" (172). This definition however, especially when applied to central aspects of day-to-day life, ignores the reality that some of the consequences of a new technology are inherently *unknowable* before their formation. The problem of unknowable costs and benefits arises from attempts to measure impacts which are not yet defined, as they have never existed before. More fundamentally, such attempts stem from a paradigm which defines social development as linear process with a clear final goal or direction. But unlike the well-defined world of economic theory, interests and preferences are as much formed by the process of development as their expression determines the direction and outcome of the process.

The classic example of *unknowable consequences* comes from green revolution technologies that can fundamentally modify peasant life (see 173, 174, 175 for detailed discussion of the various social and environmental impacts of green revolution technologies) (see 165 for an epistemological analysis of modern agriculture). The potential annual increase in crop yield from the new high-yielding varieties (HYV) can provide extra income for the family. The large year-to-year variations in the yield or in crop price, on the other hand, increase the uncertainty associated with income and

undermine the existing practices of ensuring food and income security. New agricultural technology is likely to alter the distribution of work and income among the members of family or in entire regions (176). Export of superfluous food to external markets as well as import of the needed fertilizer and pesticide will require new socioeconomic practices and institutions. All in all, many such new technologies for rural households required altogether new household practices for their successful implementation (165, 170, 177).

9.3 The Issue of Uncertainty in Impacts of Technology

Beyond the issue of unforeseeable consequences, uncertainty about those which can potentially be foreseen limits the determination of the impacts of new household technology. In the presence of uncertainty about possible outcomes, the "rational actor" of economic theory would choose the alternative with the greatest expected utility. In addition to the problems associated with the calculation of expected utility[54], its very definition also limits its usefulness as a foundation for household decisions. Statistical expectation of random variables is a construct which describes the "average" – or expected – outcome of an event over a (large) number of trials, but its own occurrence in any one experience is no more likely than that of any other outcome. Working with statistical expectation therefore is working "... with an 'average' future, which may be an unlikely future indeed" (178, 11). The problem of "unlikely expectation" gains

[54] The use of statistical expectation as the basis of choice, inherently assumes that the possible outcomes of each action constitute a set of statistical random variables. At a conceptual level, however, despite a lack of certainty in occurrence, the events that make up the day-to-day life differ from statistical random variables in two fundamental ways. First, the probabilities associated with world events are themselves often unknown – or estimated "subjectively" – and second, these probabilities constitute dynamic entities which are in constant change (178).

tremendous significance at the household level, since households often bear all the consequences of any decision about new technology alone once aggregation is removed. Therefore, if some of the likely outcomes of an option entail adverse consequences which can drastically threaten the livelihood of the household, the option may have to be avoided altogether. In an alternative (and more appropriate) approach to the statistical expectation methodology for dealing with uncertainty, one would consider the set of *all* events with plausible likelihood, especially if they can influence life adversely and drastically.

Many green revolution techniques, for instance, were found to be extremely sensitive to the exact amount and the timing of the application of fertilizers and pesticides, beyond the levels implementable outside the ideal laboratory environment. In this manner, while the aggregate result of green revolution technology experiments illustrated apparent success in the laboratories of the International Rice Research Institute, their effects on the livelihood of many rice farmers were far from ideal due to uncertain yields, a potential reason for "slow" adoption by small land-holders (177).

The Social and Cultural Context of Household Technology

Determining the effects and consequences of household technology – and labeling them as costs and benefits – is further complicated when one remembers that the impacts of new technology are defined in a social and cultural context. Going beyond its mechanical interpretation, and defining technology not as a machine, but as how tasks are done, household technology is intimately tied to local practices and customs (179).

"Improved" (high-efficiency and low-emission) cookstoves provide an example of the central role of social and cultural factors in defining the appropriateness and success of household technologies. Improved stoves have become a celebrated means in the development community for reducing both fuel consumption and exposure to indoor air pollution (14, 153, 159). This "double-dividend" – improvements in public health while reducing adverse environmental impacts – focused a great deal of effort on the design and dissemination of improved stoves (45, 161, 162). Although the initially-perceived high level of advantages of improved stoves has been called into question (35, 43) many of the benefits for which they were designed have been documented under actual conditions of use (14, 153, 159), tilting the outcome of any cost-benefit assessment in their favor.

Nonetheless, for reasons often traceable to the social and cultural contexts of household energy, initial attempts in dissemination of improved stoves met with limited success. Some of the early cookstove programs focused so much on the criteria of increased efficiency and decreased emissions that the most basic principles of design – such as the need to incorporate the size of the pot used locally or the durability of stove under conditions of use – were ignored (171). Some of the new stoves required additional efforts – such as cutting wood into smaller pieces or moving the fuel more frequently – which further limited their use since cooking and other household tasks were often performed simultaneously in the busiest part of the day. Similarly, heat retention – the very purpose of the new stoves – became a problem when families used their stoves for the dual task of cooking and heating the house.

Other obstacles to the success of the new stoves were even more subtle. Most fundamental was that the performance of the stoves was dependent on how they were used. "Optimal use," defined as the practice of the designer, at times differed from that of the actual users. Not surprisingly, in an experience similar to that of green revolution technologies, the wood-saving and/or emission reduction capabilities of the stoves dropped when people *adopted* and then *adapted* the new technologies to their purposes and ways (42). For instance, when large pieces of wood, commonly used with 3-stone fire, are used with Kenyan ceramic wood stoves their emissions may increase to levels comparable to open fire's. In brief, the mechanistic cost-benefit assessment outcome did not account for social factors which govern how often, and how, new stoves are used in the same manner that the goal of maximizing yield failed to account for taste or other locally important crop characteristics.

9.4 Rigor in a Local Context

I have argued that in the assessment of household technology, posing the question as one which solely focuses on the mechanical impacts, rather than on the consequences *as perceived and experienced by those who adopt the technology*, cannot provide adequate basis for sound evaluation. For this reason, although cost-benefit analysis may provide a valuable tool under a limited set of circumstances – when all the outcomes and processes of the alternatives are indeed similar and already agreed upon – one should be critical of its application to the assessment of new household technology, where a clear definition of costs and benefits may not exist.

With this criticism, the development of a rigorous and systematic methodology for the evaluation of household technologies may seem to come into an apparent conflict with the notion of social (and also ecological) context. The basis for reconciliation between a need for tools for systematic assessment of household technologies and the important issue of the social and cultural context of technology lies in an approach that

> ... replaces the "stratigraphic" conception of the relations between the various aspects of human existence with a synthetic one; that is, one in which biological, psychological, sociological, and cultural factors can be treated as variables within unitary systems of analysis (180).

Such an approach should recognize that technology is not a socially neutral phenomenon, but a way of life whose attributes and impacts are intimately tied to the societies where it originates or is applied. In contrast to initial experiences, for example, successful stove programs were those where design and function were based on technologies and practices already in place, such as the charcoal burning Kenya Ceramic Jiko (KCJ) of which an estimated 800,000 units have been sold in Kenya (42, 171). In the case of the agricultural techniques, the social and environmental consequences of the initial high-yielding varieties motivated the creation of MASIPAG, a communal and less technology-oriented but more locally-engineered effort among the farmers and scientists in the area. MASIPAG's efforts resulted in the production of more reliable strains of rice with "good – though not necessarily the highest possible – yields" (170). In a new framework for technology assessment, the underlying social objectives and technical attributes of technological development are negotiated and constructed in the context of the society in which the adoption takes place.

Chapter 10 Conclusions, Policy Implications, and Future Research

10.1 Conclusions and Implications for Public Health and Technology Transfer Policy

Acute respiratory infections (ARI) and chronic respiratory diseases, which are causally linked to exposure to indoor air pollution in developing countries, are the leading cause of global morbidity and mortality. In Chapter 1 and Chapter 2, I argue that, despite its central role in public health and household welfare in developing countries, mitigation of health risks caused by exposure to indoor air pollution has been undermined by a lack of systematic research and data. In particular, little in known about the determinants of human exposure, the exposure-response relationship, and the performance of intervention strategies, such as improved cookstoves, in a detailed, quantitative manner.

Despite increasing awareness of the important gaps in our understanding of exposure to particulate matter as a causal agent of ARI, an expert panel of the World Health Organization (WHO) focusing on the new air quality guidelines stopped short of encouraging detailed and systematic research on exposure to indoor air pollution and its health impacts. Rather, the panel concluded that "although work on deriving simple exposure indicators urgently needs to be encouraged, realistically it is likely to be some years before sufficient environmental monitoring can be undertaken in most developing countries" (13).

The field research that underlies this dissertation is among the first studies to examine human exposure to indoor air pollution from biomass combustion and its impact on the incidence of acute respiratory infections (ARI) at the level of the individual. A unique data set, including detailed monitoring of individual-level exposure to indoor PM_{10} from biomass combustion, longitudinal data on ARI, and data on stove emissions under the actual conditions of use, has enabled me to conduct quantitative analysis of the questions fundamental for mitigating this health risk. In particular, I derive, for the first time, the exposure-response relationship for acute respiratory infections that result from exposure to particulates from biomass combustion.

I use continuous monitoring of PM_{10} concentration, data on spatial dispersion of indoor smoke, and detailed quantitative and qualitative time-activity budget data to construct profiles of exposure to indoor particulate matter which account for individual exposure patterns, including daily and day-to-day variability. Including these factors, beyond the commonly-used single measure of average pollution concentration, illustrates that average pollution alone is not a sufficient measure of human exposure in situations where a large fraction of exposure occurs during high-intensity emission episodes, such as the case for individuals who cook using biomass stoves. Consequently, intervention schemes, such as new stove technology, should pay as much attention to "*worst-scenario*" emissions – such as emissions during lighting, extinguishing, or moving of fuel – as to average emission levels. Furthermore, this result indicates the importance of

detailed exposure assessment in quantifying the exposure-response relationship for indoor particulate emissions that exhibit episodic characteristics.

My analysis of the exposure-response relationship shows that the fraction of time that a person has ARI, or the more severe ALRI, is an increasing, concave function of daily exposure to indoor PM_{10}. The rate of increase is higher for daily exposures below approximately 2000 $\mu g.m^{-3}$. An important implication is that public health programs designed to reduce the adverse impacts of indoor air pollution in developing countries should focus on measures that result in larger reductions in pollution, especially those that bring average exposure below 2000 $\mu g.m^{-3}$, confirming a concern that was raised qualitatively in (34).

Exposure assessment methodology has commonly focused on average pollution levels. In the case of indoor smoke, where exposure occurs in an episodic manner, using average concentrations results in a systematic gender-based bias in assessment of exposure (Chapter 6) and health impacts (Chapter 7). I find that once total exposure is calculated to appropriately include high-intensity exposure episodes, gender is no longer an effective indicator of ARI and ALRI. I also find that exposure intensity does not contribute to the incidence of disease, once its role is accounted for in total exposure. At the same time, since combustion of biomass results in highly volatile pollution profiles, for the highest exposure groups (notably the individuals who cook), approximately one half of daily exposure occurs during high-intensity episodes. This implies an important role for measures that reduce total exposure by reducing peak emissions.

In comparing stove performance, I find that improved wood stoves provide an overall reduction in the emission concentration compared to the traditional 3-stone fire. In addition to mean concentration, ceramic wood stoves reduce the high-intensity emission episodes, characterized by mean above the 75th percentile ($\mu_{>75}$). Therefore, these stoves shift the whole distribution of emission concentration downwards, thus reducing human exposure.

The largest reduction of emission concentrations and human exposure is achieved through a transition from wood to charcoal. The concave, increasing exposure-response relationship for PM_{10} and ARI suggests that the marginal health benefits as a result of additional pollution reduction achieved by charcoal stoves is larger than those from the initial reduction gained from transition to ceramic wood stoves. In fact, charcoal stoves can conveniently reduce average exposure to levels below 2000 $\mu g.m^{-3}$, where public health benefits of marginal reduction in exposure are the largest.

In addition to its public health implications, the benefits of transition to charcoal raise an important environmental policy question. Although charcoal production causes more environmental damage than fuelwood harvesting (160), public health benefits are likely to be considerable. This tension reminds us of the need for integrated approaches to technology, environment, and health in designing successful intervention strategies.

10.2 Directions for Future Research

This dissertation has significantly advanced our understanding of exposure to indoor air pollution from biomass combustion in developing countries and its impacts on human health. In particular, using multi-day, continuous monitoring of pollution and time-activity budgets, I have characterized daily and day-to-day variability of exposure. At the same time, due to the resource-intensive nature of day-long monitoring of pollution and time-activity budgets, I have used a small sub-set of the households in the study group (10 – 12 households that were monitored for 6 – 15 days) in describing day-to-day exposure variability. In a larger study, considerably more resources should be devoted to understanding variations in human exposure from day to day, or season to season. This includes monitoring of pollution in the same households for a large number of days over a period of 1 – 2 years (for example once per week). A more robust and accurate understanding of "low-frequency" variations in exposure will also clarify the temporal relationship between exposure and respiratory diseases (i.e. the delay between exposure and health impacts).

In this dissertation, I have *constructed* exposure measures using detailed continuous data on pollution and time-activity budgets. In addition to discussing the advantages of this process in accounting for patterns of exposure, I have demonstrated that this approach eliminates the gender-based bias in health impacts suggested by the traditional method of using average daily pollution levels (Chapter 7). The most convincing validation of the results of exposure estimation would, nonetheless, be comparison with *direct external measurements* of individual exposure. Since our monitoring was conducted for 14 – 15

hours per day and multiple days we could not justify direct exposure measurement for both ethical (carrying heavy monitors for long periods) and logistical (heavy monitors may affect activity patterns; monitors are sensitive and subject to damage; purchasing one monitor per household member would have exceeded our financial resources) reasons. Rapid advances in monitoring technology are likely to result in compact real-time personal monitors, which will allow precise comparison between the exposure estimation process used in this dissertation and actual exposures. Despite logistical difficulties, exposure monitoring in developing countries should not be put aside as an unrealistic goal, as portrayed by the WHO Air Quality Guidelines, but should rather become a central focus of research on indoor air pollution and heath in these settings. Design of successful intervention requires thorough understanding of human exposure.

The particle size of maximum response of our particulate monitoring instrument was 0.1 μm to 10 μm. As a result of this response range, only a fraction of the measured concentration is due to particles below 2.5 μm, which are believed to have the most important health impacts. Studies of particle pollution in both industrialized and developing countries have demonstrated correlation between PM_{10} and $PM_{2.5}$ concentrations (24, 139), but further research on this relationship in the case of biomass smoke is needed.

I find that the exposure intensity does not contribute to disease incidence, once its role is accounted for in total exposure. At the same time, since combustion of biomass results in highly volatile pollution profiles, for the highest exposure groups (notably the individuals

who cook) approximately one half of daily exposure occurs during high-intensity episodes. This correlation suggests that further investigation of the role of high-intensity exposure beyond its contribution to average exposure is needed. In particular, the role of high-intensity exposure raises a research question about inhalation and pulmonary deposition of particulate matter under different exposure conditions. Important recent work has shed light on the dispersion of aerosol bolus in human airways (52). New research that integrates modeling, laboratory testing, and field trials is needed to consider dispersion, deposition, and health impacts as a function of pollution intensity.

Due to data limitations, current exposure is the only environmental explanatory variable which I have considered directly in my analysis of ARI incidence. Birth weight and perinatal diseases, nutrition (including breast-feeding), child care practices, and the education of mother have been documented as determinants of ARI (181, 182). In a larger study, a "life-history" approach that also accounts for these factors can provide a more complete picture of ARI incidence. Similarly, more detailed treatment of previous exposure and crowding are also likely to contribute to our understanding of the environmental determinants of ARI.

Finally, I have described some of the important issues in successful dissemination of technologies that are central to household life. Analytical and empirical research on valuation of household level technology is needed. In particular, we must develop methods that can represent the complexity of technology choice in a quantitative manner without reducing them to a single metric, such as cost. Such methods would be a first

step in reconciling local technology preferences with the goals of public health, environmental, and development policies.

10.3 A Final Note on International Public Health and Technology Transfer Policies

Technology transfer programs and public health initiatives provide a variety of benefits in developing nations. With more than two billion people worldwide relying on biomass as their primary source of energy, efforts to introduce new energy technologies should also pay detailed attention to health outcomes. A long record of national, multilateral, and private donor efforts to promote improved (high-efficiency and low-emissions) stoves exists (45). Many of these programs, although lowering average emissions, may not have reduced exposure below the 2000 $\mu g.m^{-3}$ level, let alone to several hundreds of $\mu g.m^{-3}$, that provide important health benefits. The results of the analysis in this dissertation, for example, indicate that although improved wood stoves substantially reduce exposure, in many cases they offer smaller health benefits than a transition to charcoal which can reduce exposure to very low levels. Other transitions through the "energy ladder", from wood to charcoal, or to kerosene, gas, and electricity also require an evaluation of public health and environmental tradeoffs (such as impacts on vegetation and greenhouse gas emissions) of various energy technologies. In particular, armed with a richer quantitative understanding of health impacts of particulate matter, development, public health, and energy R&D efforts that aim to reduce disease burden can effectively address acute respiratory infections.

References

1. World Bank (1993) *World Development Report: Investing in Health*. New York: Oxford University Press.

2. K. R. Smith (1999) "The National Burden of Disease from Indoor Air Pollution in India," *The Proceedings of Indoor Air 99: The 8th International Conference on Indoor Air Quality and Climate*, Edinburg, Scotland.

3. World Health Organization (WHO) (1999) *World Health Report*. Geneva: WHO.

4. World Health Organization (WHO) (1998) *World Health Report*. Geneva: WHO.

5. B. H. Chen, C. J. Hong, M. R. Pandey, K. R. Smith (1990) "Indoor Air Pollution in Developing Countries," *World Health Statistics Quarterly*, **43**, 127-138.

6. A. de Francisco, J. Morris, A. J. Hall, J. R. M. Armstrong Schellenberg, B. M. Greenwood (1993) "Risk Factors for Mortality from Acute Lower Respiratory Tract Infections in Young Gambian Children," *International Journal of Epidemiology*, **22**, 1174-1182.

7. A. Ellegard (1996) "Cooking Fuel Smoke and Respiratory Symptoms among Women in Low-Income Areas in Maputo," *Environmental Health Perspectives,* **104**, 980-985.

8. A. Ellegard (1997) "Tears while Cooking: An Indicator of Indoor Air Pollution and Related Health Effects in Developing Countries," *Environmental Research,* **75**, 12-22.

9. M. R. Pandey (1984) "Domestic Smoke Pollution and Chronic Bronchitis in a Rural Community of the Hill Region of Nepal," *Thorax*, **39**, 337-339.

10. M. R. Pandey, R. P. Neupane, A. Gautam, I. B. Shrestha (1989) "Domestic Smoke Pollution and Acute Respiratory Infections in a Rural Community of the Hill Region of Nepal," *Environment International*, **15**, 337-340.

11. K. R. Smith, J. M. Samet, I. Romieu, N. Bruce (2000) "Indoor Air Pollution in Developing Countries and Acute Lower Respiratory Infections in Children," *Thorax,* **55**, 518-532.

12. K. R. Smith (1996) "Indoor Air Pollution in developing Countries: Growing Evidence of its Role in the Global Burden of Disease," *The Proceedings of Indoor Air 96: The 7th International Conference on Indoor Air Quality and Climate*, Nagoya, Japan.

13. World Health Organization (WHO) (1999) *WHO Air Quality Guidelines*. Geneva: WHO.

14. M. Ezzati, B. M. Mbinda, D. M. Kammen (2000) "Comparison of Emissions and Residential Exposure from Traditional and Improved Biofuel Stoves in Rural Kenya," *Environmental Science and Technology*, **34**, 578-583.

15. J. D. Spengler, K. Sexton (1983) "Indoor Air Pollution: A Public Health Perspective," *Science,* **221**, 9-17.

16. D. W. Dockery, et al. (1989) "Effects of Inhalable Particles on Respiratory Health of Children," *American Review of Respiratory Disease,* **139**, 587-594.

17. C. A. Pope, III, D. W. Dockery, J. D. Spengler, M. E. Raizenne (1991) "Respiratory Health and PM_{10} Pollution: A Daily Time-Series Analysis," *American Review of Respiratory Disease,* **144**, 668-674.

18. J. V. Hall, et al. (1992) "Valuing the Health Benefits of Clean Air," *Science,* **255**, 812 - 817.

19. C. A. Pope, III, D. W. Dockery (1992) "Acute Health Effects of PM_{10} Pollution on Symptomatic and Asymptomatic Children," *American Review of Respiratory Disease,* **145**, 1123-1128.

20. D. W. Dockery, et al. (1993) "An Association between Air Pollution and Mortality in Six U.S. Cities," *New England Journal of Medicine,* **329**, 1753-1759.

21. D. W. Dockery, C. A. Pope, III (1994) "Acute Respiratory Effects of Particulate Air Pollution," *Annual Review of Public Health,* **15**, 107-132.

22. G. Hoek, B. Brunekreef (1994) "Acute Effects of a Winter Air Pollution Episode on Pulmonary Function and Respiratory Symptoms of Children," *Archives of Environmental Health,* **48**, 328-335.

23. C. A. Pope, III, D. V. Bates, M. E. Raizenne (1995) "Health Effects of Particulate Air Pollution: Time for Reassessment," *Environmental Health Perspectives,* **103**, 472-480.

24. R. Wilson, J. D. Spengler, Eds. (1996) *Particles in Our Air: Concentrations and Health Effects*. Cambridge, MA: Harvard University Press.

25. P. H. Abelson (1998) "Airborne Particulate Matter," *Science,* **281**, 1609.

26. K. R. Smith (1988) "Air Pollution: Assessing Total Exposure in Developing Countries," *Environment,* **30**, 16-34.

27. K. R. Smith (1993) "Fuel Combustion, Air Pollution Exposure, and Health: Situation in Developing Countries," *Annual Review of Energy and Environment,* **18**, 529-566.

28. J. R. M. Armstrong, H. Campbell (1991) "Indoor Air Pollution Exposure and Respiratory Infections in Young Gambian Children," *International Journal of Epidemiology,* **20**, 424-429.

29. S. Berman (1991) "Epidemiology of Acute Respiratory Infections in Children in Developing Countries," *Review of Infectious Diseases,* **13 (Suppl 6)**, S454-S462.

30. E. J. Stanek III, E. M. Wafula, F. E. Onyango, J. Musia (1994) "Characteristics Related to the Incidence and Prevalence of Acute Respiratory Infection in Young Children in Kenya," *Environmental Health Perspectives,* **18**, 639-647.

31. S. Awasthi, H. A. Glick, R. H. Fletcher (1996) "Effect of Cooking Fuels on Respiratory Diseases in Preschool Children in Lucknow, India," *Science,* **55**, 48-51.

32. H. Campbell (1997) "Indoor Air Pollution and Acute Lower Respiratory Infections in Young Gambian Children," *Health Bulletin,* **55**, 20-31.

33. S. Sharma, et al. (1998) "Indoor Air Quality and Acute Lower Respiratory Infections in Indian Urban Slums," *Environmental Health Perspectives,* **106**, 291-297.

34. N. Bruce, L. Neufeld, E. Boy, C. West (1998) "Indoor Biofuel Air Pollution and Respiratory Health: The role of Confounding Factors among Women in Highland Guatemala," *International Journal of Epidemiology,* **27**, 454-458.

35. G. Ballard-Tremeer, H. H. Jawurek (1996) "Comparison of Five Rural, Wood-Burning Cooking Devices: Efficiencies and Emissions," *Biomass and Bioenergy,* **11**, 419-430.

36. J. S. M. Boleij, et al. (1989) "Domestic Air Pollution from Biomass Burning in Kenya," *Atmospheric Environment,* **23**, 1677-1681.

37. S. Saksena, R. Prasad, R. C. Pal, V. Joshi (1992) "Patterns of Daily Exposure to TSP and CO in the Garhwal Himalaya," *Atmospheric Environment,* **26A**, 2125-2134.

38. M. Ezzati, H. Saleh, D. M. Kammen (2000) "The Contributions of Emissions and Spatial Microenvironments to Exposure to Indoor Air Pollution from Biomass Combustion in Kenya," *Environmental Health Perspectives,* **108**.

39. H. Krugmann (1987) *Review of Issues and Research Relating to Improved Cookstoves.* International Development Research Centre (IDRC-MR152e), Ottawa.

40. F. R. Manibog (1984) "Improved Cooking Stoves in Developing Countries: Problems and Opportunities," *Annual Review of Energy,* **9**, 199-227.

41. N. H. Ravindranath, J. Ramakrishna (1997) "Energy Options for Cooking in India," *Energy Policy,* **25**, 63-75.

42. B. Agarwal (1983) "Diffusion of Rural Innovations: Some Analytical Issues and the Case of Wood-burning Stoves," *World Development,* **11**, 359-376.

43. K. Wallmo, S. K. Jacobson (1998) "A Social and Environmental Evaluation of Fuel-Efficient Cook-stoves and Conservation in Uganda," *Environmental Conservation,* **25**, 99-108.

44. S. F. Baldwin (1986) *Biomass Stoves: Engineering Design, Development, and Dissemination.* Centre for Energy and Environmental Studies, Princeton University (224), Princeton.

45. D. F. Barnes, K. Openshaw, K. R. Smith, R. van der Plas (1994) *What Makes People Cook with Improved Biomass Stoves? A Comparative International Review of Stove Programs.* The World Bank, Washington, DC.

46. A. K. N. Reddy, R. H. Williams, T. B. Johansson (1996) *Energy after Rio: Prospects and Challenges*. New York: United Nations Publications.

47. S. Arungu-Olende (1984) "Rural Energy," *Natural Resources Forum,* **8**, 117-126.

48. United Nations Development Programme (UNDP) (1999) *Human Development Report 1999*. New York: United Nations Development Programme (UNDP).

49. K. R. Smith (1987) *Biofuels, Air Pollution, and Health: A Global Review*. New York: Plenum Press.

50. K. R. Smith (1993) *The Most Important Chart in the World*. United Nations University Lecture Series (6), Tokyo.

51. M. Utell, J. Samet (1996) "Airborne Particles and Respiratory Disease: Clinical and Pathogenetic Considerations," *Particles in Out Air: Concentrations and Health Effects*. R. Wilson, J. D. Spengler. Cambridge, MA: Harvard University Press.

52. R. Sarangapani, A. S. Wexler (1999) "Modeling Aerosol Bolus Dispersion in Human Airways," *Journal of Aerosol Science*, **30**, 1345 - 1362.

53. B. M. Mbinda, M. Ezzati, D. M. Kammen (2000, forthcoming) "Ecological and Social Analysis of Wood Use in Central Kenya".

54. World Health Organization (WHO) (1990) *Acute Respiratory Infections in Children: Case Management in Small Hospitals in Developing Countries: A Manual for Doctors and Other Senior Health Workers*. World Health Organization, Programme for Control of Acute Respiratory Infections (WHO/ARI/90.5), Geneva.

55. N. M. H. Graham (1990) "The Epidemiology of Acute Respiratory Infections in Children and Adults: A Global Perspective," *Epidemiologic Reviews*, **12**, 149-178.

56. G. D. Maynard (1913) *An Enquiry into the Etiology, Manifestations, and Prevention of Pneumonia amongst Natives on the Rand, Recruited from Tropical Areas*. Johannesburg: The South African Institute for Medical Research.

57. M. Gelfand (1957) *The Sick African*. Cape Town: Juta and Company Limited.

58. Admiralty and the War Office (1923) *Hygiene and Disease in Eastern Africa.* London: Her Majesty's Stationary Office.

59. G. C. Gould, Ed. (1971) *Health and Disease in Africa*. Nairobi: East African Literature Bureau.

60. D. F. Owen (1973) *Man's Environmental Predicament: An Introduction to Human Ecology in Tropical Africa.* Oxford: Oxford University Press.

61. G. W. Hartwig, K. D. Patterson, Eds. (1978) *Disease in African History: An Introductory Survey and Case Studies.* Durham, NC: Duke University Press.

62. E. E. Sabben-Clare, D. J. Bradley, K. Kirkwood, Eds. (1980) *Health in Tropical Africa during Colonial Period.* Oxford: Clarendon Press.

63. M. H. Dawson (1983) *Socio-economic and Epidemiological Change in Kenya: 1880-1925*. Ph.D. Dissertation, University of Wisconsin, Madison.

64. J. K. van Ginneken, A. S. Muller, Eds. (1984) *Maternal and Child Health in Rural Kenya: An Epidemiological Study*. London: Croom Helm.

65. A. D. Charters (1985) *Reminiscences of East Africa and Western Australia in the Milestones of a Doctor's Life, 1903-1984*.

66. R. N. O. K'Okul (1991) *Maternal and Child Health in Kenya*. Uppasala, Sweden: Scandinavian Institute of African Studies.

67. L. M. Manderson (1996) *Sickness and the State: Health and Illness in Colonial Malay, 1870 - 1940*. Cambridge: Cambridge University Press.

68. K. Kirkwood (1980) "Questions to Answer," *Health in Tropical Africa during Colonial Period*. E. E. Sabben-Clare, D. J. Bradley, K. Kirkwood, Eds. Oxford: Clarendon Press.

69. P. D. Curtin (1998) *Disease and Empire: The Health of European Troops in the Conquest of Africa.* Cambridge: Cambridge University Press.

70. W. M. MacMillan (1938) *Africa Emergent.* Penguin Books.

71. F. L. Lambrecht (1991) *In the Shade of an Acacia Tree: Memoirs of a Health Officer in Africa, 1945-1959.* Philadelphia: American Philosophical Society.

72. G. W. Hartwig (1978) "The Disease Factor: An Introductory Overview," *Disease in African History: An Introductory Survey and Case Studies.* G. W. Hartwig, K. D. Patterson. Durham, NC: Duke University Press.

73. H. Kloos, Z. Ahmed Zein (1993) "Other Diseases," *The Ecology of Health and Disease in Ethiopia*. H. Kloos, Z. Ahmed Zein, Eds. Boulder, CO: Westview Press.

74. J. Bonte (1974) "Patterns of Mortality and Morbidity," *Health and Disease in Kenya*. L. C. Vogel, A. S. Muller, R. S. Odingo, Z. Onyango, A. De Geus, Eds. Nairobi: Kenya Literature Bureau.

75. O. Odhiambo, A. M. Voorhoeve, J. K. van Ginneken (1984) "Age-Specific Infant and Childhood Mortality and Causes of Death," *Maternal and Child Health in Rural Kenya: An Epidemiological Study*. J. K. van Ginneken, A. S. Muller, Eds. London: Croom Helm**,** 213-222.

76. A. M. Voorhoeve, H. J. Nordbeck, S. A. Lakhani (1984) "Factors Related to Infant Mortality," *Maternal and Child Health in Rural Kenya: An Epidemiological Study.* J. K. van Ginneken, A. S. Muller, Eds. London: Croom Helm**,** 213-222.

77. World Health Organization (WHO) (1990) *Antibiotics in the Treatment of Acute Respiratory Infections in Young Children.* World Health Organization, Programme for Control of Acute Respiratory Infections (WHO/ARI/90.10), Geneva.

78. World Health Organization (WHO) (1991) *Technical Basis for the WHO Recommendations on the Management of Pneumonia in Children at First-Level Health Facilities.* World Health Organization, Programme for Control of Acute Respiratory Infections (WHO/ARI/91.20), Geneva.

79. World Health Organization (WHO) (1996) *Division of Diarrhoeal and Acute Respiratory Disease Control: 1994-1995 Report.* World Health Organization (WHO/CHD/96.1), Geneva.

80. R. A. LeVine, et al. (1994) *Child Care and Culture: Lessons from Africa*. Cambridge: Cambridge University Press.

81. P. Stanfield, B. Balldin, Z. Versluys, Eds. (1997) *Child Health: A Manual for Medical and Health Workers in Health Centres and Rural Hospitals.* Rural Health Series. Nairobi: African Medical and Research Foundation.

82. C. A. Pope, III, D. W. Dockery, J. Schwartz (1995) "Review of Epidemiological Evidence of Health Effects of Particulate Air Pollution," *Inhalation Toxicology,* **7**, 1-18.

83. D. T. Rice (1960) "Less Smoke in the Cook-House," *Rural Health Digest,* **2**, 214.

84. G. J. Clearly, R. B. Blackburn (1968) "Air Pollution in Native Huts in the Highlands of New Guinea," *Archives of Environmental Health,* **17**, 785-794.

85. G. O. Sofoluwe (1968) "Smoke Pollution in Dwellings of Infants with Bronchopneumonia," *Archives of Environmental Health,* **16**, 670-672.

86. A. J. Woolcock, R. B. Blackburn (1967) "Chronic Lung Disease in the Territory of Papua and New Guinea - An Epidemiological Study," *Australasian Annals of Medicine,* **16**, 11-19.

87. H. R. Anderson (1978) "Respiratory Abnormalities in Papua New Guinea Children: The Effects of Locality and Domestic Wood Smoke Pollution," *International Journal Epidemiology,* **7**, 63-72.

88. H. R. Anderson (1979) "Chronic Lung Disease in the Papua New Guinea Highlands," *Thorax,* **34**, 647-653.

89. World Health Organization (WHO) (1991) *Epidemiological, Social, and Technical Aspects of Indoor Air Pollution from Biomass Fuel: Report of a WHO Consultation.* World Health Organization (WHO/PEP/92.3A), Geneva.

90. D. A. Collings, S. D. Sithole, K. S. Martin (1990) "Indoor Woodsmoke Pollution Causing Lower Respiratory Disease in Children," *Tropical Doctor,* **20**, 151-155.

91. H. Hu, Y. Liu (1989) "Evaluation of Indoor Air Pollution and its Effects on Human Health in Beijing's Rural Areas," *Environment International,* **15**, 321-335.

92. W. Jedrychowski, B. Tobiasz-Adamczyk, E. Flak, E. Mroz, K. Gomola (1990) "Effect of Indoor Air Pollution Caused by Domestic Cooking on Respiratory Problems of Elderly Women," *Environment International,* **16**, 57-60.

93. A. W. B. R. Johnson, W. I. Aderele (1992) "The Association of Household Pollutants and Socio-Economic Risk Factors with the Short-Term Outcome of Acute Lower Respiratory Infections in Hospitalized Pre-School Nigerian Children," *Annals of Tropical Paediatrics,* **12**, 421-432.

94. T. Norboo, M. Yahya, N. G. Bruce, H. J. A., K. P. Ball (1991) "Domestic Pollution and Respiratory Illness in a Himalayan Village," *International Journal of Epidemiology,* **20**, 749-757.

95. T. J. D. O'dempsey, et al. (1996) "A Study of Risk Factors for Pneumococcal Disease among Children in a Rural Area of West Africa," *International Journal of Epidemiology,* **25**, 885-893.

96. L. F. Robin, et al. (1996) "Wood-Burning Stoves and Lower Respiratory Illnesses in Navajo Children," *The Pediatric Infectious Disease Journal,* **15**, 859-865.

97. C. J. L. Murray, A. D. Lopez, Eds. (1994) *Global Comparative Assessments in the Health Sector: Disease Burden, Expenditures, and Intervention Packages.* Geneva: World Health Organization.

98. C. J. L. Murray, A. D. Lopez (1999) "On the Comparable Quantification of Health Risks: Lessons from the Global Burden of Disease," *Epidemiology,* **10**, 594-605.

99. U.S. Geological Survey (1999) "Greater Horn of Africa Region," U.S. Geological Survey (http://edcintl.cr.usgs.gov/adds/c1/r2/r2.html).

100. E. A. Brett (1973) *Colonialism and Underdevelopment in Africa: The Politics of Economic Change 1919-1939*. London: Heinemann.

101. D. Kennedy (1987) *Islands of White: Settler Society and Culture in Kenya and Southern Rhodesia, 1890-1939.* Durham, NC: Duke University Press.

102. P. Mosley (1983) *The Settler Economies: Studies in the Economic History of Kenya and Southern Rhodesia 1900-1963.* Cambridge: Cambridge University Press.

103. W. R. Ochieng (1985) *A History of Kenya*. London: MacMillan.

104. W. R. Ochieng, Ed. (1990) *Themes in Kenyan History.* Nairobi: Heinemann.

105. M. G. Schatzberg, Ed. (1987) *The Political Economy of Kenya.* New York: Praeger.

106. R. L. Tignor (1976) *The Colonial Transformation of Kenya: The Kamba, Kikuyu, and Maasai from 1900 to 1939.* Princeton: Princeton University Press.

107. R. H. Bates (1981) *Markets and States in Tropical Africa: Political Basis of Agricultural Policies*. Berkeley: University of California Press.

108. J. Herbst (1993) "The Politics of Sustained Agricultural Reform in Africa," *Hemmed in: Responses to Africa's Economic Decline*. T. M. Callaghy, J. Ravenhill, Eds. New York: Columbia University Press**,** 398-462.

109. M. F. Lofchie (1993) "Trading Places: Economic Policy in Kenya and Tanzania," *Hemmed in: Responses to Africa's Economic Decline*. T. M. Callaghy, J. Ravenhill, Eds. New York: Columbia University Press**,** 398-462.

110. C. Ake (1996) *Democracy and Development in Africa*. Washington, DC: The Brookings Institution.

111. National Council for Population and Development, Central Bureau of Statistics (1994) *Kenya Demographic and Health Survey 1993.* Calverton, MD: Macro International Inc.

112. National Council for Population and Development, Central Bureau of Statistics (1999) *Kenya Demographic and Health Survey 1998.* Calverton, MD: Macro International Inc.

113. Central Intelligence Agency (CIA) (1999) "The World Factbook 1999: Kenya," Central Intelligence Agency (CIA) (http://www.odci.gov/cia/publications/factbook/ke.html).

114. World Bank (1999) "Kenya at a Glance," World Bank (http://www.worldbank.org/data/countrydata/aag/ken_aag.pdf).

115. World Bank (2000) "African Development Indicators 2000," World Bank (http://www.worldbank.org/data/countrydata/adi/adi.html).

116. United Nations Children's Fund (UNICEF) (2000) "UNICEF Statistics: Kenya," United Nations Children's Fund (UNICEF) (http://www.unicef.org/statis/).

117. HealthNet (1999) "Kenya at a Glance: Health Statistics," SATELLIFE (HealthNet) (http://www.healthnet.org/hnet/ken.html#stats).

118. World Bank (1999) "Country Data: Country at a Glance Tables," World Bank (http://www.worldbank.org/data/countrydata/aag.htm).

119. Laikipia Research Programme (LRP) (1996) "Laikipia District, Kenya: Dominant Landuse and Population Density," Nanyuki: Laikipia Research Programme (LRP).

120. Laikipia Research Programme (LRP) (1997) "Laikipia District, Kenya: Mean Annual Precipitation and Agro-Ecological Zones," Nanyuki: Laikipia Research Programme (LRP).

121. R. S. Odinga (1971) *The Kenya Highlands: Land Use and Agricultural Development.* Nairobi: East African Publishing House.

122. Weblink Services Ltd. (2000) "Kenya, Provinces and Districts," Weblink Services Ltd. (http://www.kenyaweb.com/ourland/#provinces).

123. M. Kituyi (1990) *Becoming Kenyans: Socio-Economic Transformation of the Pastoral Maasai.* Nairobi: African Centre for Technology Studies.

124. R. K. Langat (1986) "Commercial Ranches in Kenya," *Range Development and Research in Kenya.* R. M. Hansen, B. M. Woie, R. D. Child, Eds. Morrilton, AR: Winrock International Institute for Agricultural Development.

125. Laikipia Research Programme (LRP) (1996) "Laikipia District, Kenya: Landuse & Land Ownership 1996," Nanyuki: Laikipia Research Programme (LRP).

126. Kenya Colonial Office (1946-1961) *Annual Report on the Colony and Protectorate of Kenya*. Kenya Colonial Office, London and Nairobi.

127. African Medical and Research Foundation (AMREF) (1982) *Turkana 1982 Survey Report*. African Medical and Research Foundation (AMREF), Nairobi.

128. E. Nordberg, H. Oranga (1996) "Health Information for District Level Planning: A Cross-Sectional Household Survey in Rural Kenya," *East African Medical Journal,* **73**, 364-369.

129. D. M. Kammen, M. R. Dove (1997) "The Virtues of Mundane Science," *Environment,* **39**, 10-15; 38-41.

130. Mpala Research Centre (MRC) (1999) Mpala Research Centre (MRC) (http://www.nasm.edu/ceps/mpala/main.html).

131. S. W. Taiti (1992) *The Vegetation of Laikipia District, Kenya.* Laikipia Research Program (Laikipia - Mount Kenya Papers, B - 2), Nanyuki.

132. T. P. Young, N. Patridge, A. Macrae (1995) "Long-Term Glades in Acacia Bushland and Their Effects on Laikipia, Kenya," *Ecological Applications,* **5**, 97-108.

133. G. S. Were (1986) *Samburu District: Socio-Cultural Profile.* The Institute of African Studies, University of Nairobi and The Ministry of Planning and National Development, Nairobi.

134. R. C. Soper (1985) *Socio-Cultural Profile of Turkana District.* The Institute of African Studies, University of Nairobi and The Ministry of Planning and National Development, Nairobi.

135. M. Brenzinger, B. Heine, I. Heine (1994) *The Mukogodo Maasai: An Ethnobotanical Survey*. Koln: Rudiger Koppe Verlag.

136. M. Ezzati (1996-1999) Personal Field Notes.

137. K. A. Galvin, M. A. Little (1999) "Dietary Intakes and Nutritional Status," *Turkana Herders of the Dry Savanna: Ecology and Biobehavioral Response of Nomads to an Uncertain Environment.* M. A. Little, P. W. Leslie, Eds. Oxford: Oxford University Press.

138. N. Hall (1999) "A Recipe for Ugali," Personal Communication.

139. L. P. Naeher, et al. (1996) "CO as a tracer for assessing exposure to particulates in wood and gas cookstove households of Highland Guatemala," *The Proceedings of Indoor Air 96: The 7th International Conference on Indoor Air Quality and Climate*, Nagoya, Japan.

140. National Research Council (NRC) (1994) *Science and Judgment in Risk Assessment.* Washington, DC: National Academy Press.

141. T. R. Bartman (1982) "Regulating Benzene," *Quantitative Risk Assessment in Regulation*. L. B. Lave. Washington, DC: The Brookings Institution.

142. J. D. Graham, L. C. Green, M. J. Roberts (1988) *In Search of Safety: Chemicals and Cancer Risk*. Cambridge: Harvard University Press.

143. H. Austin, E. Delzell, P. Cole (1988) "Benzene and Leukemia: A Review of the Literature and A Risk Assessment," *American Journal of Epidemiology*, **127**, 419 - 438.

144. R. A. Rinsky, et al. (1987) "Benzene and Leukemia," *The New England Journal of Medicine,* **316**, 1044-1050.

145. R. A. Rinsky (1989) "Benzene and Leukemia: An Epidemiologic Risk Assessment," *Environmental Health Perspectives,* **82**, 189-191.

146. S. M. Brett, J. V. Rodricks, V. M. Chinchilli (1989) "Review and Update of Leukemia Risk Potentially Associated with Occupational Exposure to Benzene," *Environmental Health Perspectives,* **82**, 27-281.

147. D. M. Byrd, E. T. Barfield (1989) "Uncertainty in the Estimation of Benzene Risks: Application of an Uncertainty Taxonomy to Risk Assessments Based on an Epidemiology Study of Rubber Hydrochloride Workers," *Environmental Health Perspectives,* **82**, 283-287.

148. D. J. Paustenbach, et al. (1992) "Reevaluation of Benzene Exposure for the Pliofilm (Rubber Worker) Cohort (1936-1976)," *Journal of Toxicology and Environmental Health,* **36**, 177-231.

149. A. C. Dresher, C. Lobascio, A. J. Gadgil, W. W. Nazaroff (1995) "Mixing of a Point Source Pollutant by Forced Convection," *Indoor Air: International Journal of Indoor Air Quality and Climate,* **5**, 204-214.

150. A. V. Baughman, A. J. Gadgil, W. W. Nazaroff (1994) "Mixing of a Point Source Pollutant by Natural Convection Flow Within a Room," *Indoor Air: International Journal of Indoor Air Quality and Climate,* **4**, 114-122.

151. J. Spengler, R. Wilson (1996) "Emissions, Dispersion, and Concentration of Particles," *Particles in Out Air: Concentrations and Health Effects*. R. Wilson, J. D. Spengler. Cambridge, MA: Harvard University Press.

152. H. Ozkaynak, J. Spengler (1996) "The Role of Outdoor Particulate Matter in Assessing Total Human Exposure," *Particles in Out Air: Concentrations and Health Effects*. R. Wilson, J. D. Spengler. Cambridge, MA: Harvard University Press.

153. H. F. Reid, K. R. Smith, B. Sherchand (1986) "Indoor Smoke Exposures from Traditional and Improved Cookstoves: Comparisons among Rural Nepali Women," *Mountain Research and Development,* **6**, 293-304.

154. D. M. Kammen, D. M. Hassenzahl (1999) *Should we Risk it? Exploring Environmental, Health, and Technological Problem Solving.* Princeton, NJ: Princeton University Press.

155. A. K. N. Reddy, R. H. Williams, T. B. Johansson, Eds. (1996) *Energy after Rio: Prospects and Challenges.* New York: United Nations Publications.

156. B. D. Saatkamp, O. Masera, D. M. Kammen (1998) "Social versus technical visions of the energy ladder: Fuels, stoves, and indoor air pollution in Jarácuaro, México," *Boiling Point,* (40), 16-18.

157. R. J. Larsen, M. L. Marx (1986) *An Introduction to Mathematical Statistics and Its Applications*. Englewood Cliffs, NJ: Prentice Hall.

158. C. C. Rich (1999) *Insects in the Native Homes of Kenya: Variations in Abundance and Diversity, Health Effects, and Reduction Strategies.* Senior Thesis, Princeton University.

159. J. P. McCracken, K. R. Smith (1998) "Emissions and Efficiency of Improved Woodburning Cookstoves in Highland Guatemala," *Environment International,* **24**, 739-747.

160. D. R. Ahuja (1990) "Research Needs for Improving Biofuel Burning Cookstove Technologies: Incorporating Environmental Concerns," *Natural Resources Forum,* **14**, 125-134.

161. D. M. Kammen (1995) "From Energy Efficiency to Social Utility: Improved Cookstoves and the *Small is Beautiful* Model of Development," *Energy as an Instrument for Social Change.* J. Goldemberg, T. B. Johansson. New York: United Nations Development Programme.

162. K. R. Smith, G. Shuhua, H. Kun, Q. Daxiong (1993) "One Hundred Million Improved Cookstoves in China: How Was it Done?," *World Development,* **21**, 941-961.

163. A. A. Churchill (1987) *Rural Water Supply and Sanitation: Time for a Change.* The World Bank, Washington, DC.

164. A. Kim, B. Benton (1995) *Cost-Benefit Analysis of Onchecerciasis Control Program (OCP).* The World Bank (282), Washington, DC.

165. J. C. Scott (1998) *Seeing Like a State: How Certain Schemes to Improve the Human Condition Have Failed*. New Haven: Yale University Press.

166. T. Porter (1994) *Trust in Numbers: The Pursuit of Objectivity in Science and Public Life.* Princeton: Princeton University Press.

167. J. Briscoe, D. deFerranti (1988) *Water for Rural Communities: Helping People Help Themselves.* Washington, DC: The World Bank.

168. J.-O. Drangert (1993) *Who Cares About Water? A study of household water development in Sukumaland, Tanzania*. Linkoping, Sweden: Linkoping University.

169. K. G. Cassman, P. L. Pingali (1995) " Extrapolating Trends from Long-term Experiments to Farmers' Fields: The Case of the Irrigated Rice Systems in Asia," *Agricultural Sustainability: Economic, Environmental, and Statistical Considerations.* V. Barnett, R. Payne, S. R, Eds. London: John Wiley and Sons.

170. D. Frossard (1994) *Peasant Science: Farmer Research and Philippine Rice Development.* Ph.D. Thesis, University of California, Irvine.

171. D. M. Kammen (1995) "Cookstoves for the Developing World," *Scientific American,* **273**, 63-67.

172. S. J. Kline, D. E. Kash (1992) "Do We Need a Technology Policy?," *IEEE Technology and Society Magazine,* **11**, 18-25.

173. J. M. Antle, C. C. Crissman (1990) "Risk, efficiency, and the adoption of modern crop varieties: evidence from the Philippines," *Economic Development and Cultural Change*, **38**, 517-537.

174. V. Barnett, R. Payne, J. R. Steiner, Eds. (1995) *Agricultural Sustainability in Economic, Environmental, and Statistical Terms*. London: John Wiley and Sons.

175. H. Bernstein, B. Crow, M. MacKintosh, M. C. (1990) *The Food Question: Profits Versus People*. New York: Monthly Review Press.

176. A. Whitehead (1990) "The Food Crisis and Gender Conflict in the African Countryside," *The Food Question: Profits Versus People*. H. Bernstein, B. Crow, M. MacKintosh, C. Martin, Eds. New York: Monthly Review Press.

177. P. B. R. Hazell, C. Ramasamy (1991) *The Green Revolution Reconsidered: The Impacts of High-Yielding Rice Varieties in South India*. Baltimore: The Johns Hopkins University Press.

178. M. Edesess, G. A. Hambrecht (1980) "Scenario Forecasting: Necessity, Not Choice," *The Journal of Portfolio Management*, (Spring Issue), 10-15.

179. U. Franklin (1990) *The Real World of Technology*. Toronto: CBC Enterprises.

180. C. Geertz (1973) *The Interpretation of Cultures*. Basic Books.

181. J. R. Cruz, et al. (1990) "Epidemiology of Acute Respiratory Tract Infections Among Guatemalan Ambulatory Preschool Children," *Review of Infectious Diseases*, **12**(Supplement 8), S1029-S1034.

182. M. C. Cerqueiro, P. Murtagh, A. Halac, M. Avila, M. Weissenbacher (1990) "Epidemiologic Risk Factors for Children with Acute Lower Respiratory Tract Infection in Buenos Aires, Argentina: A Matched Case-Control Study," *Review of Infectious Diseases*, **12**(Supplement 8), S1021-S1028.