

## AN ABSTRACT OF THE THESIS OF

Cheryl Anne Berry for the degree of Master of Science in Environmental Health

Management presented on May 1, 1998. Title: Asthma Prevalence in School Children in

Lane County, Oregon.

Abstract approved: \_\_\_\_\_

Redacted for Privacy

Catherine M. Neumann

Asthma prevalence in children under age 18 has risen steadily in the U.S. since the mid-1970s. Most researchers attribute this increase to air pollutants, socioeconomic status or urban residence. This study examined asthma prevalence in Lane County, Oregon, using children's asthma hospital discharges (1990–1995), and data from 10 of 16 school districts. The overall asthma prevalence in Lane County was 5.8%, ranging from 1.9% (coastal Siuslaw District) to 10% (urban, industrial Bethel District). Asthma prevalence during 1988–1997 was examined for the largest school district, Eugene 4J (43% of the total study population). Eugene 4J is divided into four regions: North, South, Churchill, and Sheldon. The largest asthma prevalence increase during this time was detected in North Region (2.5%-8%), while the overall district prevalence nearly doubled (3.3%-6.2%). No correlation was found between socioeconomic status and asthma prevalence, and there was not a consistent pattern between asthma prevalence and location of each school district (urban vs. rural). Two out of the three urban school districts had higher asthma prevalence than five of the rural school districts. However, three school districts did not follow this pattern, either being lower than expected for an urban area or higher than expected for a rural area. Asthma hospitalization rates increased for most children between 1990 and 1995, with a 20-fold increase for ages 15–19 years. The results of this study show an increase in prevalence and severity of asthma in school children in Lane County. Further studies are warranted to investigate the contribution that geography, meteorology, and proximity to industry have in asthma prevalence in Lane County.

©Copyright by Cheryl Anne Berry  
May 1, 1998  
All Rights Reserved

Asthma Prevalence in School Children in Lane County, Oregon

by

Cheryl Anne Berry

A THESIS

submitted to

Oregon State University

in partial fulfillment of  
the requirements for the  
degree of

Master of Science

Presented May 1, 1998  
Commencement June 1999

Master of Science thesis of Cheryl Anne Berry presented on May 1, 1998

APPROVED:

Redacted for Privacy

\_\_\_\_\_  
Major Professor, representing Environmental Health Management

Redacted for Privacy

\_\_\_\_\_  
Chair of Department of Public Health

Redacted for Privacy

\_\_\_\_\_  
Dean of Graduate School

I understand that my thesis will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my thesis to any reader upon request.

Redacted for Privacy

\_\_\_\_\_  
Cheryl Anne Berry Author

## ACKNOWLEDGMENTS

I would like to thank my major advisor, Cathy Neumann, for all her help and support throughout this project, and for her suggestion that I present the project findings to the Western Regional Epidemiology Network (WREN) conference in May 1998. I would like to thank Annette Rossignal for her help with my data figures, and both Cathy Neumann and Annette Rossignal for their co-authorship of the paper to submit for possible publication. Thanks to Susan Prows for her helpful suggestions on data collection, and Andrea Marks for her support and comments. I would like to thank Jan Bader, Marla London, and the American Lung Association of Oregon for the use of their Oregon asthma prevalence data. Acknowledgments also to Lane Regional Air Pollution Authority, Glenn Potter of the City of Eugene, Steve Slater of the Oregon Department of Education, and Tracy Lindsay of Oregon Health Plan Policy and Research, all of whom graciously provided me with important data. Of most importance, I would like to thank the Lane County school nurses, health clerks, secretaries, and administrators who compiled and presented valuable school data. I could not have completed this project without their assistance. Special thanks to: Sherri Morgan, Annette Miller, Patty Cupples, Barbara Daikh, Marcia Hanson, Susie Doyle, Barbara Arnold, Laurie Rowe, Pam Tibbitts, and Cindy Stultz from Eugene District 4J; Sharon Gayle, Pat Kelsay, and Pat Luce from Bethel District; Karen Brist from Springfield District; Patty Mays from Blachly District; Karen McClelland from Siuslaw District; Elizabeth Shaeffer from Fern Ridge District; Melinda Carlson from Junction City District; Becky Andrews from McKenzie District; Tudy Nystrom from Pleasant Hill District, and Margery Fisher and Ruth Ackley from South Lane District. Finally, thanks to all the professors and instructors who made my time at Oregon State University both an excellent learning experience and a lot of fun, with a special thanks to Anna Harding, who helped quell my first-term panic while I was making the transition from worker to student, and to Karel Leslie for her expertise and help.

## TABLE OF CONTENTS

	<u>Page</u>
INTRODUCTION.....	1
Significance of Study .....	3
Limitations of Study.....	4
Research Questions.....	5
Purpose and Objectives .....	5
LITERATURE REVIEW.....	6
Non-Environmental Asthma Risk Factors .....	6
Environmental Asthma Risk Factors.....	10
RESEARCH METHODS.....	22
Study Area.....	22
Study Population.....	23
Data Collection .....	27
RESULTS .....	31
DISCUSSION.....	41
RECOMMENDATIONS.....	46
BIBLIOGRAPHY .....	47
APPENDICES .....	53

## LIST OF FIGURES

<u>Figure</u>	<u>Page</u>
1. Lane County Asthma Prevalence by School District (1996/97).....	31
2. Eugene 4J Asthma Prevalence by Region (1996/97) .....	32
3. Eugene 4J Asthma Prevalence (1988–1997) .....	32
4. Eugene 4J Asthma Prevalence by Region and Year (1988–1997) .....	33
5. Lane County Asthma Prevalence by Grade (1996/97).....	34
6. Eugene District 4J Asthma by Grade and Year (1988–1997).....	34
7. Asthma Prevalence Cohort Study Eugene District 4J (1988–1997) .....	35
8. Lane County Asthma Prevalence by Gender and Grade (1996/97) .....	36
9. Lane County Elementary School Asthma and SES (1-764).....	38
10. Lane County Middle School Asthma and SES (1-355).....	39
11. Lane County High School Asthma and SES (1-250) .....	39
12. Lane County Asthma Hospital Discharges (1990-1995) .....	40

## LIST OF TABLES

<u>Table</u>	<u>Page</u>
1. Lane County Elementary School Asthma Prevalence and SES .....	37
2. Lane County Middle School Asthma Prevalence and SES .....	37
3. Lane County High School Asthma Prevalence and SES.....	38



## APPENDICES

	<u>Page</u>
Appendices .....	53
Appendix 1. Lane County, Oregon .....	54
Appendix 2. Lane County Physiography.....	55
Appendix 3. Lane County School Districts .....	56
Appendix 4. Eugene School District 4J Regions.....	57
Appendix 5. Letter to School District Superintendents .....	58
Appendix 6. Superintendent Consent Form.....	59
Appendix 7. School Enrollment Form.....	60
Appendix 8. Oregon School Health Record .....	61
Appendix 9. Lane County School Physical Form.....	62
Appendix 10. School Medication Form.....	63

## Asthma Prevalence in School Children in Lane County, Oregon

### INTRODUCTION

Asthma, a reversible, obstructive lung disease, is one of the most common chronic diseases of children in the United States. An estimated 4.8 million children under age 18 years are asthmatic, with boys having a higher prevalence than girls (American Lung Association [ALA], 1996a; Bates, 1996b). The ALA estimates that asthma accounts for 10.1 million lost school days annually, and 200,000 hospitalizations a year in children under 18 years of age. The yearly cost of treating children with asthma is estimated at \$1.9 billion (ALA, 1996a). In addition, asthmatic children can fall behind in school and may also show a delay in puberty, growth, or both (Merkus et al., 1993; Neville et al., 1996).

Asthma morbidity and mortality have risen steadily in the U.S. since the mid-1970s, with the greatest increase in children under the age of 18 years (Leikauf et al., 1995; Weiss et al., 1993). A study of 6–11-year-olds from 1971 to 1980 showed a 58% increase in asthma (Friebele, 1996). From 1980 to 1994, the number of children aged 5–14 years with asthma increased by 74%, from 42.8 to 74.4 per 1,000 people (Centers for Disease Control and Prevention [CDC], 1998). Total asthma prevalence increased 42% from 1985 to 1995, while asthma deaths increased 58% between 1979 and 1996 (CDC, 1996). The asthma death rate of children aged 5–14 years nearly doubled from 1983 to 1990.

Other developed countries are seeing similar increases (Weiss et al., 1993). In Sweden between 1971 and 1981, the prevalence of asthma in 18-year-old military inductees increased by 47%, from 1.9 to 2.8 per 100 people. In Finland between 1966 and 1989, the asthma prevalence of 19-year-old military inductees increased fivefold, from

0.29 to 1.79 per 100 people. Between 1964 and 1990, asthma prevalence in school children in Australia increased 141%, from 19.1 to 46 per 100 (Weiss et al., 1993).

U.S. data from the 1988 National Health Interview Survey (NHIS) on Child Health showed that while hospitalization rates for most childhood conditions had declined, hospitalization rates for childhood asthma increased between 1970 and 1987 (Halfon & Newacheck, 1993). Another study found that the annual hospitalization rate for asthma in people aged 0–24 years increased 28% from 1980 to 1993 (6.8 to 21.4 per 100,000 people) (CDC, 1996).

There is a well-documented excess prevalence (up to twofold) of asthma in boys compared to girls. Asthma has been shown to begin earlier in boys, and to be more severe (Gergen et al., 1988; Bates, 1995b). A National Center for Health Statistics survey conducted between 1976 and 1980 found that asthma prevalence in boys was highest at age 8 years, up to 10% (Gergen et al., 1988). By age 17 years, asthma prevalence in boys dropped to 6%. In contrast, asthma prevalence in girls begins increasing at 12–14 years, moving from 4% to 6% by age 17 years (Gergen et al., 1988).

Between 1983 and 1990, boys aged 5–14 years were 1.3 times more likely to be hospitalized or to die of asthma than girls (CDC, 1996). In contrast, adult asthma is more severe in women than in men (Seachrist, 1995). Females aged 15–24 years were 2.1 times more likely than males to be hospitalized (CDC, 1996).

Studies show a disproportionate rise in asthma incidence and mortality among ethnic minorities and among people who live in poverty or urban environments, or both. Most researchers attribute the rapid increase in asthma prevalence to environmental factors rather than to genetic, allergic, or viral factors (Etzel, 1995; Friebele, 1996; Leikauf et al., 1995; Pope et al., 1995).

In Oregon, the prevalence of asthma is not systematically tracked or assessed (Ertle & London, 1996). Data collected by the Oregon Health Division (OHD) in 1994 demonstrate that Oregon's overall asthma mortality rate of 2.6 per 100,000 people is

nearly twice the overall national average of 1.5 per 100,000 (Ertle & London, 1996). Recently, the American Lung Association of Oregon (ALAO) collected, reviewed, and analyzed asthma data for Oregon using statewide hospital discharge data from 1990–1993, OHD’s Behavioral Risk Factor Surveillance Survey (BRFSS), and 1995/1996 school registration health data from Multnomah Educational Service District, which includes Portland (Ertle & London, 1996).

Results of ALAO’s study indicated an overall asthma prevalence of 6% for children, and 7% for adults. Hospitalization rates, reported by decade of age for the entire state, appeared to be constant from 1990–1993, averaging 2,306 asthma discharges per year, giving an overall average of 75 per 100,000 people (Ertle & London, 1996). However, when data were extrapolated using asthma discharge rates from Lane County for ages 5–9 years, 10–14 years, and 15–19 years, asthma hospitalization rates between 1990 and 1993 increased in all ages. The largest rate increase occurred in children aged 15–19 years (9.6 to 42.2 asthma discharges per 100,000 person-years).

Using 1995/1996 school registration health data from Multnomah County, ALAO determined that 5.9% of the students had been diagnosed with asthma. Prevalence by district ranged from 2.5% in Sauvie Island, a small, rural farming area, to 6.2% in David Douglas District, a Portland suburb. The urban area of Portland, including Gresham, had an asthma prevalence of 6.1%. ALAO also found that asthma prevalence increased with age, with the lowest prevalence in kindergarten and first grade (4.9%) and the highest prevalence in 9th grade (7.5%) and 11th grade (6.8%). These findings were contrary to those of other studies that show asthma prevalence declining in adolescence (Ertle & London, 1996; Gergen, 1988; Halfon & Newacheck, 1993).

## **SIGNIFICANCE OF STUDY**

This research will examine asthma prevalence in school-aged children in Lane County, Oregon, in relation to several variables previously shown to influence asthma

prevalence and severity, including gender, socioeconomic status, urban vs. rural school districts, and asthma hospitalization rates.

This study is significant because Oregon has no systematic monitoring and analysis of asthma prevalence, and little has been done to evaluate asthma prevalence and severity in Oregon children. This information also is valuable because data on causes and risk factors for asthma are an important precursor to development of intervention strategies that will reduce both prevalence and severity of asthma.

## **LIMITATIONS OF STUDY**

Data were compiled for 10 of 16 public school districts in Lane County. The 10 school districts in the county providing data comprise 92% of the total school population. One limitation is the lack of data on asthma prevalence for school-aged children in Oakridge, a community that has been out of compliance with federal airborne particulate standards since 1992 (Lane Regional Air Pollution Authority [LRAPA], 1996). Also, the approximately 30 private elementary and high schools located in Lane County were not included in this study. Data from all Lane County schools may have provided a more complete representation of county asthma prevalence.

A second limitation of the study is the lack of complete data on asthma prevalence by gender. This data was available in 72 of the 102 schools sampled, including 28,468 students, or 65% of the population studied.

Because allergy continues to be the most common cause of asthma, another study limitation is lack of data on allergic/genetic backgrounds of individual asthmatic children. Data on ethnicity was not available, nor was information on parental smoking, both important factors in childhood asthma.

Finally, although locations of county industrial facilities were noted, lists of asthmagenic chemicals released from these facilities were not included in this study.

## **RESEARCH QUESTIONS**

The research questions that were addressed are:

- 1) What is the asthma prevalence in school-aged children in Lane County?
- 2) Is asthma prevalence in Lane County higher in specific school districts with lower socioeconomic status, as determined by the Oregon Department of Education?
- 3) What are the trends in urban vs. rural asthma prevalence in Lane County?
- 4) Have asthma hospitalizations for school-aged children in Lane County increased over the past 5–10 years?

## **PURPOSE AND OBJECTIVES**

The purpose of this study is to determine asthma prevalence in school-aged children in Lane County, and to assess differences in asthma prevalence by grade, gender, school and school district for the 1996/1997 school year. Differences in asthma prevalence for the Eugene School District 4J for the past nine years will be examined. District 4J is the only school district that had data available for 1988 to 1996. Asthma hospitalization trends in school-aged children in Lane County for 1990–1995 also will be examined.

Therefore, the objectives of this study are to:

- 1.) Determine asthma prevalence by grade, gender, geographic location, and socio-economic status for 10 of 16 public school districts in Lane County.
- 2.) Examine whether Eugene School District 4J asthma prevalence increased from 1988 to 1997.
- 3.) Report trends in asthma hospital discharges for Lane County school-aged children from 1990–1995.

This information will be shared with each school district and the American Lung Association of Oregon.

## **LITERATURE REVIEW**

Asthma is a multifactorial disease, associated with familiar, allergic, infectious, socioeconomic, psychosocial, and environmental factors (CDC, 1996). There is a growing concern regarding the role environmental factors play in the onset of childhood asthma (Bates, 1995a; Etzel, 1995; Friebele, 1995; Koren, 1995).

This review will be presented in two sections:

### Non-Environmental Asthma Risk Factors

- Allergic/Genetic Factors
- Viral Respiratory Infections
- Ethnic Differences
- Socioeconomic Status

### Environmental Asthma Risk Factors

- Indoor Asthmagens
- Outdoor Asthmagens
- Urban Asthmagens

## **NON-ENVIRONMENTAL ASTHMA RISK FACTORS**

### **ALLERGIC/GENETIC RISK FACTORS**

An estimated 40 to 50 million people in the U.S. suffer from allergies, including approximately 75% of children with asthma (ALA, 1996a). Data from the Second National Health and Nutrition Examination Survey (1976–1980) point to a slightly higher positive allergic skin test reaction rate in boys with asthma than in girls (Gergen et al., 1988).

The allergens most responsible for allergic/genetic asthma include house dust (dust mites), pets (e.g., dog and cat dander), ingested food (e.g., wheat and milk

products), mold spores, and pollen (e.g., grass, trees, ragweed). House dust mite exposure in early life seems to be linked to both an increase in childhood asthma and an earlier onset of the disease (Newman-Taylor, 1995; Weiss et al., 1995). Daily exposure to allergens may increase the possibility of asthma provocation, and interaction of common allergens with chemical pollutants such as tobacco smoke, nitrogen oxides, and formaldehyde can enhance sensitization to inhaled allergens (ALA, 1996; Newman-Taylor, 1995).

### **VIRAL RESPIRATORY INFECTIONS**

A longitudinal study by Johnston et al. (1994) showed that the human rhinovirus (HRV) is the respiratory virus that contributes most to asthma episodes in children. Bacterial respiratory infections do not precipitate asthma attacks, although chronic bacteria-caused sinusitis can trigger an attack (ALA, 1996a). Respiratory infections before the age of two have been reported to be associated with the later development of asthma, although no firm evidence exists that viral respiratory infections are responsible for asthma induction (Newman-Taylor, 1995). Viruses do, however, exacerbate asthma in childhood and often are cited as confounders of associations between asthma and other risk factors (Lipsett et al., 1997).

### **ETHNIC DIFFERENCES**

In a 1982–1984 study of children between 6 months and 11 years of age, Puerto Rican children in the U.S. showed an asthma prevalence of 11.2%, African-American children 5.9%, Cuban children 5.2%, Caucasian children 3.3%, and Mexican-American children 2.7% (Carter-Pokras & Gergen, 1993). In the 1988 NHIS on Child Health, African-American children under the age of 18 had a 20% higher asthma prevalence than Caucasian children (Halfon & Newacheck, 1993). Another study also found a higher



asthma prevalence (26%) in African-American children than in Caucasian children (Evans, 1992).

African-American children with asthma experience more severe disability than do Caucasian children, with higher hospitalization rates (Evans, 1992). In 1993, African-Americans aged 0–24 years were 3.4 times more likely to be hospitalized for asthma (CDC, 1996). Alarming, African-Americans aged 15–24 years were six times more likely to die from asthma than Caucasians in 1993 (CDC, 1996). Recent studies suggest this increase in asthma mortality and morbidity is related more to socioeconomic status than to ethnicity (Malveaux & Fletcher-Vincent, 1995).

### **SOCIOECONOMIC STATUS (SES)**

Research conducted by Columbia University National Center for Children in Poverty (1990) showed that in the U.S., 50% of African-American children, 42% of Latin-American children, and 10% of Caucasian children are low SES, living at or below the U.S. Poverty Level. Many of these low-income children live in urban inner-city areas (Malveaux & Fletcher-Vincent, 1995). Low-income children living in inner cities are at a higher risk for increased asthma-related hospitalization and death (Halfon & Newacheck, 1993). Between 1982 and 1986, hospitalizations for asthma in New York City showed East Harlem to have a 16% higher rate than Greenwich Village-Soho in Manhattan, a much higher SES area (Malveaux & Fletcher-Vincent, 1995). In East Harlem in 1992, the asthma death rate was 10 times higher than the average U.S. rate. In a 1992 study done in Cook County, Illinois, a high percentage of asthma deaths occurred in the inner city (Marder et al., 1992). This increase in asthma morbidity and mortality existing in urban centers may reflect the gap that exists between people of low SES and accessibility of preventive health care (Malveaux & Fletcher-Vincent, 1995).

Data from the 1988 NHIS on Child Health showed that asthmatic children living at or below the U.S. Poverty Level had 40% fewer doctor visits in the previous year than

children of higher SES. Low SES children were 40% more likely to have been hospitalized. More than 90% of all people surveyed reported having a usual source of medical care, but the locations of care differed by socioeconomic status. Low SES patients were more likely to receive care in a neighborhood health center or a hospital-based clinic than high SES patients, who were more likely to receive care in a doctor's office. For sick care, low SES people utilized emergency rooms more often than high SES people. Fewer preventive health medical visits and use of emergency room physicians unfamiliar with the children are factors affecting increased hospitalization rates in lower SES groups (Halfon & Newacheck, 1993).

Problems coordinating discharge planning and follow-up for children whose usual medical care is a community health center or public clinic are not uncommon. Community clinics are effective at providing health care, but may not be set up to provide the comprehensive services asthmatic children need, such as after-hours care, environmental control, and patient education (Halfon & Newacheck, 1993).

Other factors can contribute to higher asthma prevalence observed among low income children. For example, low SES children are more likely to be born prematurely with inadequate lung function; and low SES families often lack health insurance and may live in crowded, substandard homes with high exposure to allergens. These allergens include tobacco smoke, dust mites, and cockroach antigens, all known to affect the onset of asthma (Halfon & Newacheck, 1993; Malveaux & Fletcher-Vincent, 1995).

## ENVIRONMENTAL ASTHMA RISK FACTORS

### **INDOOR ASTHMAGENS**

Many researchers speculate that changes in the indoor environment over the past few decades have helped precipitate the recent asthma increase. Homes, schools, and offices are more “energy efficient” with increased thermal insulation and a reduced number of air exchanges with outdoor air, allowing a greater concentration of allergens and pollutants to remain inside (Newman-Taylor, 1995; Weiss et al., 1993). And the increased use of wall-to-wall carpeting in homes, schools, and offices also appears to impact the quality of indoor air (Weiss et al., 1993). Asthmatic children in schools with carpeting have more severe asthma symptoms than asthmatic children in schools without carpets (Godish, 1995). Carpets are often made of synthetic materials, many of which off-gas fumes from assorted volatile organic chemicals, including several known asthmagens. Other asthmagenic chemicals that are released from carpets include fumes from pesticides, herbicides, and fungicides applied to prevent fabric deterioration (Levy & Wegman, 1995). In addition to volatile organic compounds and pesticides, other major indoor asthmagens include cigarette smoke, cockroach antigen, and wood smoke.

### **Volatile Organic Compounds (VOCs)**

These are carbon-based chemicals that volatilize, or evaporate, at room temperature, and are emitted mainly from building materials and household products (Lu, 1996). VOCs are found indoors at up to five times the outdoor concentration, and are considered the most prevalent indoor air contaminant (Ashford & Miller, 1991; Lu, 1996).

VOCs known to either aggravate or induce asthma include:

- benzene, a respiratory irritant and known human carcinogen commonly found in solvents, adhesives, building materials, carpets, paints, pesticides, and gasoline

- styrene, a respiratory irritant found in rubber, vinyl products, plastics, carbonless copy paper, and carpets
- 4-phenylcyclohexane, a lung-damaging respiratory irritant found in carpets and adhesives
- xylene, a respiratory irritant known to cause pulmonary edema, found in paint, ink solvents, adhesives, floor coverings, computers, photocopiers, gasoline, and pesticides
- toluene, a respiratory irritant found in adhesives, solvents, paints, building materials, computers, photocopiers, gasoline, and pesticides
- toluene diisocyanate, one of the most common causes of occupational asthma, found in paints, adhesives, plastics, and polyurethanes

(Abrams et al., 1993; Dunford, 1994; Godish, 1995; Levy & Wegman, 1995).

The most widely used VOC is formaldehyde, an asthma inducer used as a preservative or disinfectant in upholstery and drapery fabric, cotton cloth and clothing, antiperspirants, detergents, shampoos, nail polish, diesel fuel, paper products, fertilizers, pesticides, soft drinks, and mouthwashes (Dunford, 1994). More than half of the 7 million pounds of formaldehyde produced annually is used as a preservative or disinfectant in building materials, such as plywood, particleboard, fiberboard, laminated lumber, vinyl panels, plaster, insulation, wall paper, concrete, and adhesives (Dunford, 1994). Because it is unstable, formaldehyde easily converts into an irritating toxic gas. Formaldehyde is also water soluble and readily enters the bloodstream (Dunford, 1994).

### **Pesticides**

Indoor pesticide exposure may come from several sources, including pest sprays or bombs; pet flea collars and shampoos; skin repellents (mosquitoes, fleas, lice, etc.); air fresheners; ingestion of food, including baby food; and drinking water (Landrigan et al., 1993; Wiles & Davis, 1995). A 1990 study by the U.S. Environmental Protection Agency (EPA), “The Non-Occupational Pesticide Exposure Study,” found that 85% of the total daily exposure to airborne pesticides comes from breathing inside the home (Immerman

& Schaum, 1990). Outdoor exposure to pesticides can occur in yards, gardens, schools, playgrounds, and agricultural areas (Landrigan et al., 1993; Wiles & Davis, 1995).

There is currently no link between pesticides and childhood asthma, but several studies do link pesticides to adult onset asthma. Among these studies, some show a relationship between asthma and exposure to synthetic organophosphates (agricultural workers), fungicides, and carbamate insecticides (Etzel, 1995). Roach sprays also have been indicated as asthmagens.

### **Cigarette Smoke**

Cigarette smoke is highly irritating and known to trigger asthma. According to ALA (1996a), environmental tobacco smoke (ETS) may be more significant to asthma than any allergen. ETS, or secondhand smoke, is a complex mix of more than 4,000 chemicals (Leikauf et al., 1995). Twenty-nine of the 49 major chemical components in ETS are listed under the Clean Air Act (CAA) as Hazardous Air Pollutants, dangerous to human health. Each component of ETS is a mixture of chemicals in both gas and particle phase (Leikauf et al., 1995).

ETS contains several respiratory carcinogens, including benzo-*a*-pyrene, benz-*a*-anthracene, and several polycyclic aromatic hydrocarbons. Also found in ETS are carbon monoxide and several respiratory irritants, including formaldehyde, acrolein (a primary ingredient in tear gas), other aldehydes, cadmium and other heavy metals. Nicotine is not suspected of inducing asthma (Leikauf et al., 1995).

In the U.S. in 1995, 27% of women of child-bearing age smoked (Andrews & DeAngelis, 1995). According to Oregon's BRFSS (1994), across Oregon 23% of women of child-bearing age smoke. In Lane County, 24% of the women of child-bearing age smoke. Maternal smoking has been more strongly correlated with children's respiratory dysfunction than paternal smoking (Weitzman et al., 1990). Fathers often work and spend less time at home than do mothers. Increases in asthma incidence and prevalence have

been noted when mothers smoke more than 10 cigarettes a day, while exposure of children to more than 20 cigarettes a day can produce 3.6 times more bronchial hyperactivity, a sign of asthma (Leikauf et al., 1995). Maternal smoking during pregnancy can limit lung development and cause diminished lung size and decreased pulmonary function in infants (Weitzman et al., 1990). Even if children exposed to ETS do not develop asthma, cigarette smoke exposure can cause long-term adverse effects in children's pulmonary function (Weitzman et al., 1990).

### **Cockroach Antigen**

Reductions in pulmonary function following a challenge with cockroach antigen strongly suggest a causative role in asthma symptoms (Malveaux & Fletcher-Vincent, 1995). Fletcher-Vincent et al. (1994) showed that 60% of children in Washington D.C. aged 2–10 years had positive allergic skin reactions to cockroach antigen. This was comparable to a 1976 Chicago study showing a 58% allergic sensitization to this antigen (Kang, 1976).

Of urban adults and children with asthma who had positive skin reactions to cockroach antigen, 91% had immediate bronchial reactivity, while 48% had late reactions (Malveux & Fletcher-Vincent, 1995). Fifty-two to 78% of asthmatics of low SES are allergic to cockroach antigen. The importance of cockroach-caused asthma may be related to the hypothesis that recurrent exposure to allergens is responsible for the airway inflammation in asthma (Malveaux & Fletcher-Vincent, 1995).

### **Wood Smoke**

During winter months, many neighborhoods across the U.S. are impacted by residential wood burning. Wood smoke can enter the house from outside, or from backdrafting from a fireplace or wood stove (Lipsett et al., 1997). Wood smoke

pollutants, including fine particulate matter released into the air, both indoors and out, are primarily products of incomplete combustion. Fine particulates, typically less than 1 micrometer ( $\mu\text{m}$ ) in diameter, are highly respirable. Wood smoke resembles environmental tobacco smoke, containing many of the same respiratory irritants as ETS, including formaldehyde, acrolein, acetaldehyde, acetic acid, phenol, and nitrous oxides (Koenig et al., 1993; Lipsett et al., 1997). Studies have shown a relationship between wood smoke air pollution and exacerbation of respiratory symptoms in asthmatic children.

In a 1988–1992 study done by Lipsett et al. (1997) in Santa Clara County, California, at least 45% of winter particulate matter less than or equal to  $10\ \mu\text{m}$  ( $\text{PM}_{10}$ ) was directly from residential wood burning. Lipsett et al.'s study results demonstrated an association between ambient winter-time  $\text{PM}_{10}$  and exacerbation of asthma in an area where one of the principal sources of fine particulate pollution is wood smoke. In Seattle, Washington, an area known to have substantial wood smoke pollution, Schwartz et al. (1993) found that  $\text{PM}_{10}$  concentrations were the best predictor of emergency room asthma visits. Also in Seattle, Koenig et al. (1993) found that fine particulate matter from wood burning is significantly associated with acute respiratory irritation in young asthmatic children.

## **OUTDOOR ASTHMAGENS**

A person inhales at least 8,000 liters of air each day, breathing in assorted chemicals (Brown, 1997). These chemicals include several criteria air pollutants that are regulated by the Clean Air Act of 1970. Standards for criteria air pollutants are set by the EPA, with particular concern for populations-at-risk. Criteria air pollutants are: carbon monoxide, ozone, sulfur oxides, nitrogen oxides, hydrocarbons (VOCs), particulate matter, and lead. Aside from hydrocarbons, four others are considered causative agents for asthma (Brown, 1997):

- Ozone
- Sulfur Dioxide
- Nitrogen Dioxide
- Particulate Matter

### **Ozone**

Ozone (O<sub>3</sub>) is a highly reactive and oxidative gas that is formed in the troposphere by photochemical reactions of sunlight on nitrogen oxides, sometimes called “ozone precursors,” and hydrocarbons. Nitrogen oxides are emitted from motor vehicles and power plants, while hydrocarbons come from activities such as house painting and construction, road paving, and refueling vehicles. High temperatures and stagnant air provide the right conditions for ozone formation (Breslin, 1995; Gong, 1992).

Ozone is a powerful respiratory tract irritant that constricts air passages, causing labored breathing, particularly in children and the elderly. Ozone can increase symptoms such as wheezing, coughing, and chest tightness, and can lead to lung inflammation and interfere with the lung’s ability to protect itself against viral and bacterial agents (ALA, 1996b; Breslin, 1995; Brown, 1997).

In 1995, the CDC reported that approximately 25% of children in the U.S. live in areas that exceed the federal standard for ozone. In the American Lung Association’s 1996 paper, “Danger Zones: Ozone Air Pollution and Our Children,” the following factors were identified that place children at risk to ozone exposure (ALA, 1996b):

- They spend more time outdoors in summertime when ozone levels are the highest.
- They spend more time engaged in vigorous exercise, increasing the amount of ozone inhaled deep into the lungs.
- They take in more air relative to their body weights and lung surface than do adults.
- Their lungs are still developing and their biological defenses against pollution are not fully mature.



- Their airways are narrower than those of adults, thus enhancing the inflammatory effects of ozone air pollution.
- Although they experience lung function losses comparable to adults, children do not seem to report symptoms to the same extent, making them less likely to reduce exposure (i.e., stopping exercise or moving indoors), thus increasing their risk of lung damage.

A review of epidemiological studies on ozone exposure done by Brunekreef et al. (1995) showed that ozone levels below the 1979 federal ozone standard (0.12 parts per million [ppm] over one hour) were linked to increased hospital admissions for asthma and other respiratory problems. Brunekreef et al. also found declines in lung function in children and adults that were related to low-level ozone exposure. Because of these and other studies that showed ozone injuries below the federal 0.12 ppm standard, EPA lowered ozone limits to 0.08 ppm over 8 hours, effective July, 1997.

Although ozone can aggravate and induce asthma episodes in asthmatics, there is debate whether ozone causes asthma. Philip Landrigan, director of the Mt. Sinai Environmental Health Sciences Center, and former director of The Children's Environmental Health Network, states that ozone is a potent cause of asthma by itself (Breslin, 1997). Others, such as George Thurston, a professor of environmental medicine at New York University School of Medicine, state that ozone aggravates, but does not cause, asthma (Brown, 1997).

### **Sulfur Dioxide**

Sulfur dioxide (SO<sub>2</sub>) has polluted the atmosphere for most of the earth's history. Concern about this pollutant was heightened by the London "fogs" in the middle of this century because SO<sub>2</sub> was a major component of those deadly pollution events (Folinsbee, 1992).

Sulfur dioxide is a colorless, water-soluble gas that reacts on the surface of airborne particles and with water to produce sulfuric acid and other sulfates. The primary

outdoor source of  $\text{SO}_2$  is the domestic, commercial, and industrial combustion of sulfur-containing fossil fuels. Coal-fired power plants are large point sources of  $\text{SO}_2$  pollution (Gong, 1992).

Because sulfur dioxide is highly water-soluble, it does not typically travel far in the respiratory tract, tending instead to deposit in the nasal tissues. Several studies have reported that nasal mucociliary transport, an imperfect defense mechanism, was slowed by exposure to as little as 1 ppm  $\text{SO}_2$  (Bascom et al., 1995; Folinsbee, 1992).

Even with this damage, the nasal mucosa is still able to remove most  $\text{SO}_2$ , even at high concentrations. But, this removal is dependent on breathing through the nose. When people breathe through their mouths,  $\text{SO}_2$  is not removed and enters the lungs. Mouth breathing occurs with heavy exercise or with nasal problems such as allergic rhinitis or a deviated nasal septum. However, even during heavy exercise, 40–60% of air is inhaled through the nose, although an asthmatic's absorption of  $\text{SO}_2$  during exercise is greater than that of a person without asthma (Folinsbee, 1992).

Asthmatics exposed to  $\text{SO}_2$  experience decreased lung function (Bascom et al., 1996). Nonasthmatics experience increased respiratory symptoms such as increased airway resistance caused by reflex bronchoconstriction, increased respiratory hospitalizations and mortality (Bascom et al., 1996; Folinsbee, 1992).

### **Nitrogen Dioxide**

Nitrogen ( $\text{N}_2$ ) is a major gaseous component in the atmosphere, accounting for 78% of the total air mass. Nitrous oxide ( $\text{NO}$ ) is a non-toxic, natural constituent of the atmosphere and is also formed by natural processes in soil. When combined with oxygen ( $\text{O}_2$ ), nitrous oxide produces nitrogen dioxide ( $\text{NO}_2$ ), a colored, odorous, toxic, corrosive gas (Godish, 1991). A major source of  $\text{NO}_2$  is the combustion of fossil fuels in stationary points (heating, power generation), and motor vehicles. Indoor sources of  $\text{NO}_2$  are

unvented combustion appliances such as gas stoves, kerosene heaters, and gas-fired water heaters (Gong, 1992). Nitrogen dioxide is the main precursor of ozone.

Nitrogen dioxide can cause an increased incidence of lower respiratory infections in children, and increased airway responsiveness in asthmatics (Bates, 1995b; Folinsbee, 1992). The small airways of the lungs are the primary site of NO<sub>2</sub> damage (Folinsbee, 1992). Some studies suggest a significantly increased risk of children under age 7 years experiencing more respiratory symptoms or disease in homes with gas stoves compared to homes with electric stoves (Godish, 1991). Studies of home kerosene heaters show that exposed children under age 7 years had a twofold higher risk of lower respiratory symptoms, including asthma, than children not exposed.

More recent studies are raising questions about the role of nitrogen dioxide in asthma prevalence and severity. Studies in Scandinavia have shown a rise in asthma hospital emergency visits when NO<sub>2</sub> levels were high (Bates, 1995b). An increased asthma prevalence in Great Britain has coincided with increasing exposure to levels of NO<sub>2</sub> in the general population (Bates, 1995a). The effects of nitrogen dioxide are probably the least understood effects of the criteria pollutants and more study is warranted.

### **Particulate Matter**

Airborne particulate matter is a complex mix of organic and inorganic solids and gaseous liquids, or aerosols. Respirable particles are emitted from a wide range of sources. Some sources are natural, such as dust storms, volcanic eruptions, pollen, bacterial decomposition, trees, and fungi. But, the more widespread and clinically relevant particulates come from man-made sources, commonly concentrated in heavily populated areas (Godish, 1991; Gong, 1992).

The major sources of man-made particulates are industry and fuel combustion. Godish (1991) reported that in 1987, industry was responsible for approximately 36% of

all particulate emissions, with fuel combustion contributing about 26%. Industrial sources include manufacturing plants, metal smelting, coal cleaning, rock crushing, agricultural grain milling, and agricultural burning. Motor vehicles provide most of the particulates from fuel combustion, with additions coming from industrial plants that burn fossil fuels (Godish, 1991).

Airborne particles may be fine or coarse. The larger, coarse particles over 10 microns in diameter ( $PM_{10}$ ), tend to stop in the nasal area. Fine particles, especially those less than  $PM_{2.5}$ , are inhaled, and retained, deep in lung tissue. Particles emitted from motor vehicles, agricultural and wood burning, fossil fuel burning and cigarette smoke tend to be in the fine particulate range, while particles emitted from industry are a combination of coarse and fine (Pope et al., 1995).

Until July 1997, the EPA under the CAA regulated particulate matter  $\leq 10 \mu m$  ( $PM_{10}$ ) in concentrations of 150 micrograms per cubic meter ( $\mu g/m^3$ ) daily. After numerous studies pointed to increased human illness and death from fine particulate matter  $\leq PM_{2.5}$ , the EPA amended the CAA and began regulating particles  $PM_{2.5}$  and smaller in concentrations of  $50 \mu g/m^3$  daily. The  $PM_{10}$  standard was not changed.

Recent studies have shown that exposure to airborne particulates contributes to cardiovascular and respiratory disease, including bronchitis, asthma, and airway obstructive disease (Abbey et al., 1995). An increase in  $PM_{10}$  equal to  $30 \mu g/m^3$  was associated with a 12% increase in hospital asthma visits (Pope et al., 1995). Pope (1989 and 1991) found statistically significant associations between  $PM_{10}$  and children's bronchitis and asthma hospital visits. Ransom and Pope (1992) showed significant grade-school absenteeism in Utah related to elevated  $PM_{10}$  lasting 3–4 weeks. Pope and Dockery (1992) found elevated  $PM_{10}$  levels in Utah associated with significant declines in lung function, increases in respiratory symptoms, and increased use of asthma medication.  $SO_2$  and  $NO_2$  were not confounders in this study.

Portney & Mullahy (1990), using data from the U.S. Annual Health Interview Survey, found particulate pollution associated with emphysema, chronic bronchitis, and asthma. Other studies showed that cough, bronchitis, and chest illness were associated with particulates (Pope et al., 1995). Associations were stronger for children with a history of wheezing or asthma. Abbey et al. (1995) showed that estimated concentrations of  $PM_{2.5}$  were associated with increasing severity of respiratory symptoms related to general airway obstructive disease, chronic bronchitis, and asthma.

Brown (1997) reported several studies on health effects related to  $PM_{2.5}$  pollution. These studies showed that fine particles are associated with changes in lung function, hospital admissions and mortality, and that day-to-day  $PM_{2.5}$  exposure leads to increased asthma attacks, chronic obstructive pulmonary disease, and pneumonia.

Researchers have found no clear evidence of a safe threshold level for airborne particulates (Pope et al., 1995). Respiratory health effects of particulate pollution can occur at low levels of exposure common to many U.S. cities (Brunekreef et al., 1995; Pope et al., 1995).

### **Urban Asthmagens**

Human commerce and recreation have led to the development and use of more than 50,000 chemicals (Leikauf et al., 1995). A chemical's toxicity depends on the amount of exposure, the dose to the target organ, and the biological response (Leikauf et al., 1995). The 1990 CAA mandates exposure standards for an initial list of 189 hazardous air pollutants. It also mandates identification of not fewer than 30 compounds that present the greatest threat to public health in urban areas.

Several compounds identified as urban air toxics (UATs), such as aldehydes, polyisocyanates/isocyanates, acidic anhydrides, and metals, have been classified as human asthmagens because they are known to cause occupational asthma in exposed workers (Leikauf et al., 1995). Seta et al. (1993) estimated that more than 6 million

workers are potentially exposed to chemical or metal asthmagens in U.S. industrial settings. Potential exposure to polyisocyanates, a major occupational asthmagen, exceeded 110,000 workers (Leikauf et al., 1995). This suggests that several point sources exist in industry than can potentially contribute to community air pollution.

Leikauf et al. (1995), using estimates from the EPA's 1990 Toxic Release Inventory (TRI), showed that 6,399 facilities released more than 55 million pounds of known asthmagens into the air across the U.S. This same study showed that 6,261 U.S. facilities emitted more than 193 billion pounds of UATs that are suspected of exacerbating the effects of known asthmagens. UATs that possibly exacerbate asthma include chlorine, hydrochloric acid, hydrogen fluoride, and dioctyl phthalate. The highest levels of release were for chlorine and hydrochloric acid, two known respiratory irritants (Leikauf et al., 1995).

## RESEARCH METHODS

### STUDY AREA

Lane County, stretching from the Pacific Ocean to the Cascade Mountains, is located in central Oregon at the southern end of the Willamette Valley (Appendix 1). The county's population (approx. 305,800) constitutes 10% of Oregon's total population. Eugene and Springfield in Lane County comprise the second largest urban area in the state (LRAPA, 1996). For this study, urban and rural classifications were determined using U.S. Bureau of Census standards, defining urban areas as continuously built-up areas with a population of 50,000 or more (U.S. Department of Commerce, 1994). Several school districts included small, incorporated cities surrounded by large, less densely populated areas. The U.S. Census Bureau urban density requirement of at least 1,000 people per square mile was utilized for these districts. Using these definitions, Eugene, Bethel and Springfield school districts were considered urban while Siuslaw, Blachly, Fern Ridge, Junction City, Pleasant Hill, South Lane and McKenzie school districts were considered rural.

In 1995, the population of Lane County was 92% Caucasian, 3.3% Latin-American, 2.4% Asian or Pacific Islander, 1.2% Native American, and 0.9% African-American (City of Eugene, 1997). Plentiful jobs are responsible for a low 1997 unemployment rate of 4.7%, although Lane County annual wages are close to \$3,000 per year lower than the state average, and \$6,000 per year lower than the Portland average (State of Oregon Employment Department, 1997a & 1997b). The climate is mild, with heavy rainfall and high pollen counts. Lane County is known for its natural beauty and recreational opportunities.

The central part of Lane County, which includes Eugene and Springfield, is found in a valley between the Coastal Mountain range to the west and the Cascade Mountains to the east (Appendix 2). This central valley is the most populated and industrialized area of

the county, with the greatest potential for air quality degradation. This inland area experiences periods of air stagnation in both summer and winter, when temperature inversion conditions trap air, pollen, and pollutants near the valley floor (LRAPA, 1996).

Residential wood burning, slash burning, and agricultural field burning contribute to air quality problems in all of Lane County except the coastal communities, which experience more air movement and fewer inversions (LRAPA, 1996). Because of excess airborne particulates, some areas of Lane County are not in compliance with the National Ambient Air Quality Standards (NAAQS)  $PM_{10}$  regulations. The cities of Eugene and Springfield have been considered  $PM_{10}$  “non-attainment” areas by the EPA since 1987. The small Cascade Mountains community of Oakridge has been out of compliance with  $PM_{10}$  regulations since 1992. It is highly unlikely that Lane County will be in compliance with the new NAAQS  $PM_{2.5}$  standards that went into effect July, 1997. Past studies showed that other criteria air pollutants, sulfur dioxide, and nitrogen dioxide have not been a problem in Lane County, and are therefore not currently monitored. The county also is currently in compliance with NAAQS carbon monoxide standards. Ozone is also in attainment, but ozone levels in the Eugene/Springfield area have been increasing in the past 10 years (LRAPA, 1996). Eugene/Springfield has been in compliance with the new federal ozone standard (0.08 ppm over 8 hours) in effect since July 1997, but equipment to monitor  $PM_{2.5}$  in Lane County was not received until March 1998, and no particulate reports were available.

## **STUDY POPULATION**

Lane County has 16 public school districts with a total 1996/1997 student population of 47,541 (Appendix 3). Ten of the 16 districts employ school nurses who gather and compile health problem information, including asthma statistics. Because asthma data are not compiled in the other six school districts, those districts (Creswell, Marcola, Lowell, Mapleton, Oakridge, and Crow-Applegate-Lorane) were not included



in this study. The 10 school districts contributing data have a combined student population of 43,790, or 92% of the county's total student population. The following data were gathered from individual school district superintendent's offices, the Oregon Department of Employment, and Chambers of Commerce in Eugene, Springfield, and Junction City.

The school districts included in this study are:

**Siuslaw School District**, with a student population of 1,698, has one primary school (K–2), one elementary school (3–5), one middle school, and one high school. Siuslaw District, situated along the Pacific coast, includes the communities of Florence and Dunes City. The district includes many recreational areas, with tourism as the main source of employment. Fishing and logging contribute a lesser extent to the local economy.

**Blachly School District**, with a student population of 65, has one elementary school (K–8) and one high school. This district, located in the Coastal Mountains, includes the communities of Blachly, Triangle Lake, Horton, and the Greenleaf area of Deadwood. A large number of residents are retired. There are several small local businesses, but many workers commute by automobile to Eugene/Springfield.

**Fern Ridge School District**, with a student population of 2,028, has four elementary schools, one middle school, and one high school. Fern Ridge District begins in the Coastal Mountains and stretches to Fern Ridge Reservoir, just west of Eugene. It includes the communities of Veneta, Elmira, Noti, and Walton. There are several small local businesses, and one lumber mill found in this district. The majority of workers commute to Eugene/Springfield by automobile.

**Junction City School District**, with a student population of 1,979, has two elementary schools, one middle school, and one high school. This district, located primarily in the central valley north of Eugene, includes the communities of Junction City and Cheshire. The district has a large agricultural area, including dairy and grass seed

farms. Junction City is home to two lumber mills, a large recreational vehicle manufacturing plant, and two veneer plants that laminate wood products. Many workers commute to Eugene/Springfield by automobile.

**Bethel School District**, with a student population of 4,418, has five elementary schools, two middle schools, and one high school. Bethel District is located in the northwest corner of Eugene, and includes an area known as the west Eugene industrial corridor. This section of Eugene is home to a large number of Lane County's manufacturing industries, including paint, wood products, metals, machinery, and rubber. In November 1996, the voters in Eugene passed a bill known as the City of Eugene Toxics Act, which goes into effect in April, 1999. It requires facilities that release state and federally listed hazardous substances into the environment to publicly identify these substances and the amount of each released. Of 42 facilities in Eugene known to release hazardous substances into the air, water, or soil, 25 are located within the Bethel School District boundaries, and six are found within two blocks of the district border (Eugene Toxics Board, 1997).

**Eugene School District 4J**, with a student population of 18,634, has 32 elementary schools, eight middle schools, and five high schools. Eugene District 4J includes all of Eugene but the northwest corner, adding the community of Coburg to the northeast. The district is divided into four regions (Appendix 4). North Region is the northwest area of the district and includes the unincorporated, primarily residential area of Santa Clara and some agricultural land. Sheldon Region is the northeast section of Eugene, which includes Coburg, a community with agricultural areas and two recreational vehicle manufacturing plants. South Region is the southeast area of Eugene and includes the campuses of the University of Oregon, Northwest Christian College and Lane Community College. Churchill Region is the southwest part of the city and includes some industry and agriculture. Eugene School District 4J has two school-based clinics run by nurse practitioners, located at North Eugene High School and South Eugene High

School. District employment opportunities are numerous and varied and include manufacturing, the service industry, government, and wholesale/retail trade. Some residents use an extensive system of bicycle trails or the bus system to commute to work, but most travel by automobile.

**Springfield School District**, with a student population of 10,532, has 15 elementary schools, four middle schools, and two high schools. Springfield District, located in the central valley adjacent to Eugene, includes the communities of Springfield, Goshen, and Walterville. Springfield is home to three wood products mills, a computer disc manufacturing plant, the main county U.S. Post Office, several shopping malls, and several agricultural areas. Like Eugene, this city has a large system of bicycle trails and a bus system, although most workers commute by automobile.

**Pleasant Hill School District**, with a student population of 1,285, has one primary school (K–3), one elementary school (4–6), one junior high school (7–8), and one high school. Pleasant Hill District, located southeast of Eugene/Springfield, stretches from the central valley into the Cascade Mountains, and includes the communities of Pleasant Hill, Dexter, Trent, and part of Jasper. Residents work in agriculture, forestry and small local businesses. The majority of workers commute by automobile to Eugene/Springfield.

**South Lane School District**, with a student population of 2,811, has six elementary schools, one middle school, and one high school. South Lane District, located south of Eugene in the Cascade Mountains, includes the communities of Cottage Grove and Dorena. Three lumber mills are located here along with two machinery plants, an industrial park, and a large nationally known discount store. There are many small local businesses and several recreational areas.

**McKenzie School District**, with a student population of 340, has one elementary school (K–8) and one high school. McKenzie District is located in the Cascade Mountains in the northeast corner of Lane County, and includes the communities of

McKenzie Bridge, Vida, Blue River, Nimrod, Finn Rock, and part of Leaburg. Many of the residents are retired. Some work in local businesses, forestry, and tourism, while many workers commute to Eugene/Springfield by automobile. This district includes large recreational areas.

## **DATA COLLECTION**

Data collection is divided into four areas: school data, socioeconomic data, hospitalization data, and demographic and school district data.

### **SCHOOL DATA**

The first phase in school data collection was to gain approval from Oregon State University's Institutional Review Board for the Protection of Human Subjects (IRB). After gaining this approval, a letter was sent to each school district superintendent describing the project and requesting consent to gather asthma information from district school nurses (Appendix 5). A consent form was signed and returned by each superintendent (Appendix 6).

School nurses in each district were contacted by phone to request asthma information from spring of the 1996/1997 school year on the number of children in their district with asthma by school, grade, and gender. Spring was chosen because it was the most recent information available when the request was made. Data were gathered by numbers in an aggregate fashion. Names were never seen, or used, by the researcher.

This asthma information was collected by school nurses in three ways. First, the state recommends that when a student is enrolled in school each year, parents provide health information on the school enrollment form. This form includes a box to check if the child has asthma (Appendix 7). Blachly District uses the state form, but the other

districts modify the form to their own needs. All districts do, however, include a request for asthma information.

School nurses or their health aides review enrollment forms for each new student, and record noted health problems, including asthma, on the child's health card, the second source of health information (Appendix 8). This mandated health card, used throughout Oregon, is part of permanent school records and is sent when a child changes schools.

The third information source is physicians or nurse practitioners, who report physical examination information on the Lane County school physical form (Appendix 9). This form, used by all Lane County schools, is a permanent part of each child's school record. Information may also come from health care providers in the form of an order for medication to be given at school. State law requires that a physician/nurse practitioner sign an order, including diagnosis, for any medication given at school (Appendix 10). Nurses often learn of a new asthma diagnosis when a signed medication order comes to school.

All school nurses in Lane County make lists of children with asthma by school. The larger districts, Eugene 4J, Springfield, and Bethel, put this information into a computer spreadsheet. Bethel and Springfield have computer programs capable of keeping this information for only one year, discarding it at the start of each new school year. Eugene 4J keeps the data in its system longer, and is the only district that had asthma prevalence data available for 1988, 1992, 1993, 1994, 1995, and 1997. Eugene 4J data by school and grade were available for 1988, 1995, and 1997.

Eugene 4J asthma data were available on computer printouts that included the total numbers of asthmatic children (without names listed). Because other districts' asthma listings included names, those nurses needed to transform these data for project use. Nurses in Siuslaw, Fern Ridge, Junction City, South Lane, and McKenzie Districts sent typed lists by mail. The Junction City nurse noted in her letter that her high school

asthma counts were lower than actual numbers, as she is only able to follow the most severely ill asthmatic high school students. Recent school budget cuts in Oregon have reduced nursing services in most Lane County school districts. Blachly, Springfield, and Pleasant Hill nurses reported their data by phone.

The researcher went in person to Bethel District and recorded asthma numbers read by one of the nurses from the health problem lists. The lists were not viewed by the researcher. Because someone had inadvertently removed all the 1996/1997 high school health data from the computer, extrapolations were made using data from Fall 1997. Fall 10th grade data were used for spring 9th grade counts; fall 11th grade data used for spring 10th grade; and fall 12th grade data used for spring 11th grade. Twelfth grade data for Spring 1997 were not available and not reported.

Gender information was not available in all school districts. Complete data were gathered from Bethel, Eugene 4J, Fern Ridge, Blachly, Siuslaw, and McKenzie districts.

### **SCHOOL SOCIOECONOMIC STATUS DATA**

School SES data were obtained from the “Guide to Interpreting 1997 Oregon Statewide Assessment Results” published yearly by the Oregon Department of Education in Salem. This assessment rates the socioeconomic status of students in each school in the state, using information based on the percent of students eligible for free and reduced-price lunch (FRL), student mobility (MOB) and attendance (ATT), and for middle and high schools, parent education (PED). SES analysis is done using data from grade 3 (754 schools) to represent elementary schools, grade 8 (355 schools) to represent middle schools, and grade 11 (254 schools) to represent high schools. The formulas for the SES indices are:

$$\text{Grade 3 SES index} = 354.4 - 0.146(\text{FRL}) + 0.536(\text{ATT}) - 0.123(\text{MOB})$$

$$\text{Grade 8 SES index} = 360.9 - 0.020(\text{FRL}) + 0.835(\text{ATT}) - 0.194(\text{MOB}) + 5.568(\text{PED})$$

Grade 11 SES index =  $407.1 + 0.043(\text{FRL}) + 0.350(\text{ATT}) - 0.196(\text{MOB}) + 9.701(\text{PED})$   
(Oregon Department of Education, 1997).

The schools are rated from the lowest SES (1) to the highest in each category. Therefore, SES ratings read as 1-764 for elementary schools, 1-355 for middle schools, and 1-254 for high schools.

### **HOSPITALIZATION DATA**

Hospital asthma discharge rates for Lane County were obtained from the office of Oregon Health Plan Policy and Research in Salem. Data collected included 1990–1995 hospital asthma discharges for ages 5–9 years, 10–14 years, and 15–19 years.

### **DEMOGRAPHIC AND SCHOOL DISTRICT DATA**

Lane Educational Service District (Lane ESD) in Eugene provided county school district maps and a comprehensive list of all county schools and superintendents. Lane Council of Governments in Eugene provided demographic information. LRAPA, a branch of the Department of Environmental Quality in Springfield, provided maps and county air quality information. The City of Eugene Toxics Board provided information on facilities in Eugene that release state and federally listed toxic chemicals into the environment. County employment data and school district characteristics were obtained from individual school district superintendent's offices, from the Oregon Department of Employment in Eugene, and from Chambers of Commerce in Eugene, Springfield, and Junction City.

### **DATA ANALYSIS**

Data were analyzed using SPSS software (primarily descriptive statistics).

## RESULTS

School nurses in 10 of 16 Lane County school districts identified 2,530 of 43,709 (5.8%) students as having asthma. The asthma prevalence for each district ranged from 1.9% (Siuslaw) to 10% (Bethel).

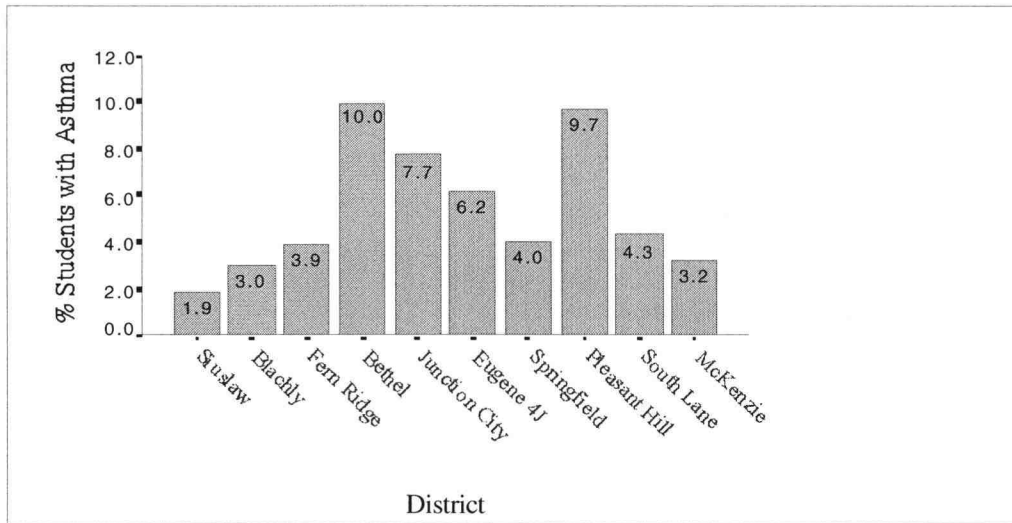


Figure 1. Lane County Asthma Prevalence by School District (1996/1997)

As shown in Figure 2, Eugene School District 4J, the largest district in Lane County, had variations in asthma prevalence in its four geographic regions: North, South, Churchill, and Sheldon. The highest asthma prevalence (8%) was in the North Region.



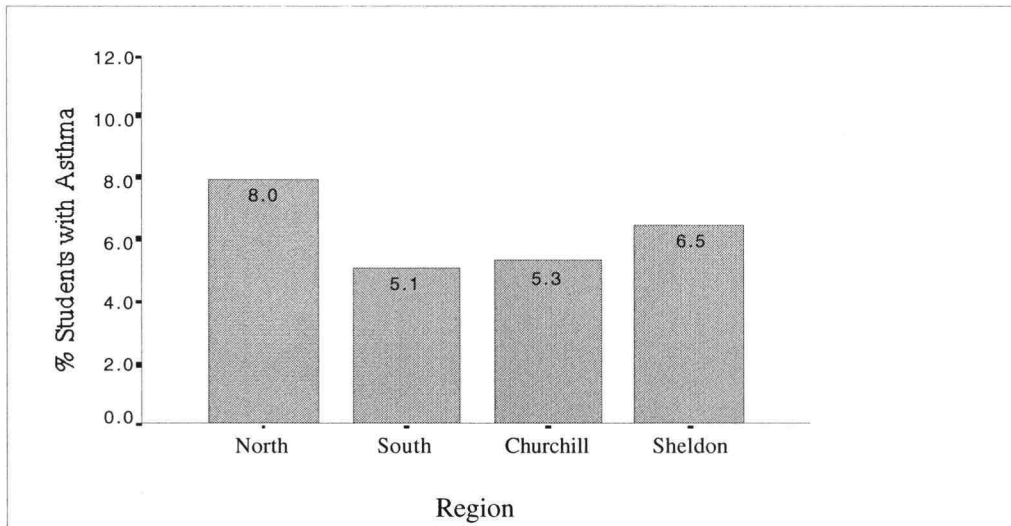


Figure 2. Eugene 4J Asthma Prevalence by Region (1996/1997)

As shown in Figure 3, the asthma prevalence in the Eugene School District 4J nearly doubled from 3.3% (1988) to 6.2% (1997).

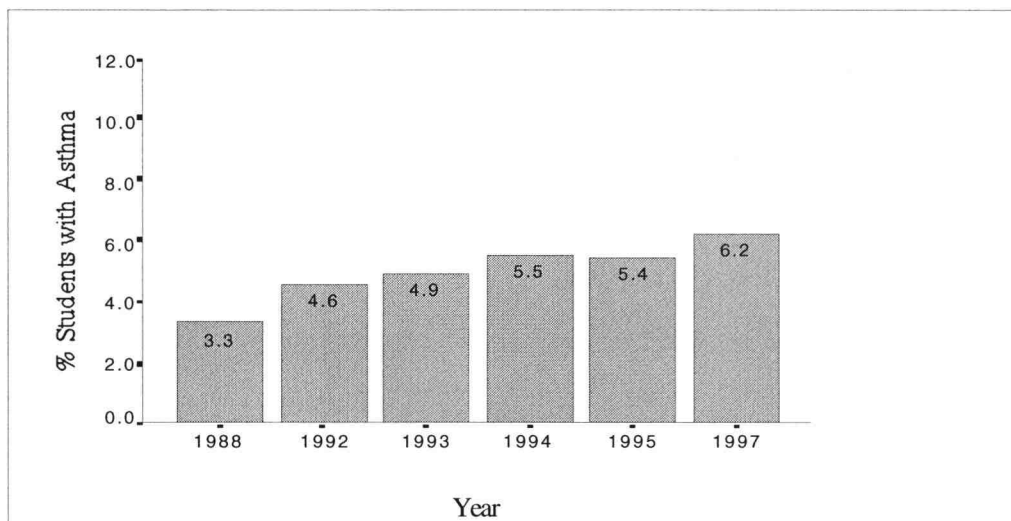


Figure 3. Eugene 4J Asthma Prevalence (1988–1997).

Asthma prevalence in the North Region of Eugene District 4J increased from 2.5% (1988) to 8% (1997), as shown in Figure 4. This degree of increase in asthma

prevalence was not seen in the other Eugene 4J regions. The asthma prevalence in South Region doubled during this period, while Churchill Region maintained the same asthma prevalence of 5.3% between 1995 and 1997. Sheldon Region increased its asthma prevalence from 4.6% (1988) to 7.3% (1995), then dropped to 6.5% (1997).

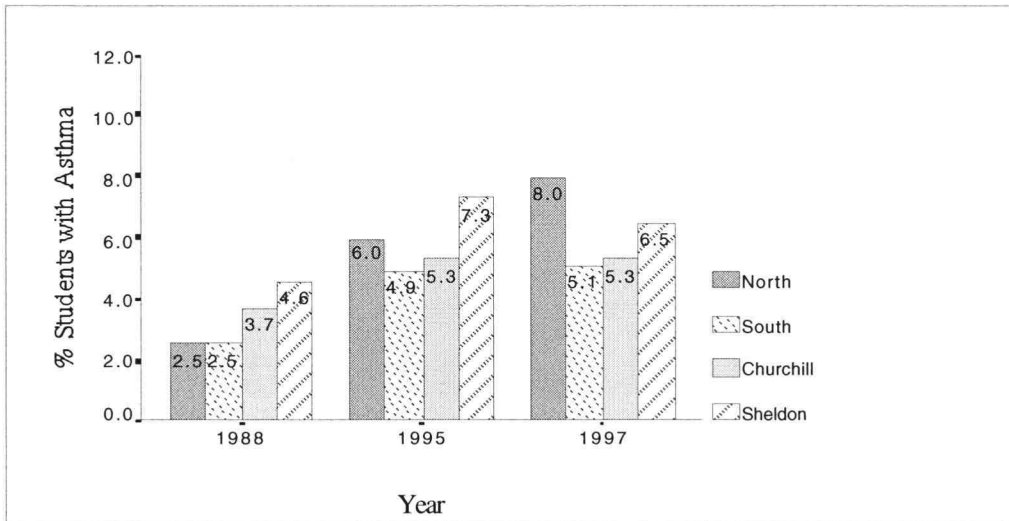


Figure 4. Eugene 4J Asthma Prevalence by Region and Year (1988–1997).

Asthma prevalence by grade level in Lane County ranged from 4.3% in 2nd grade to 7.5% in 8th grade (Figure 5). After 8th grade, the prevalence dropped to 5% by the 12th grade.

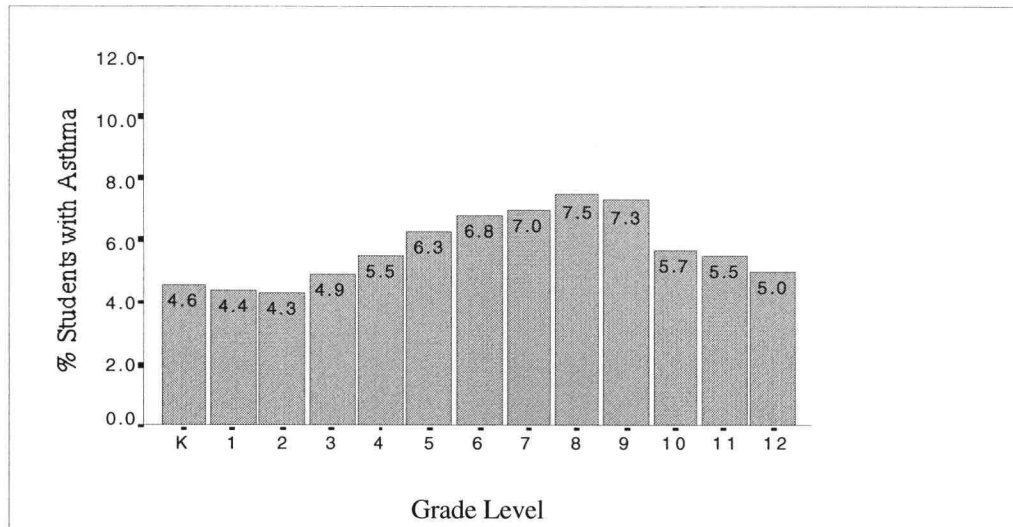


Figure 5. Lane County Asthma Prevalence by Grade (1996/1997).

As shown in Figure 6, the highest asthma prevalence in Eugene School District 4J (1988–1997) was found in 7th and 8th grades. In 1988 the highest asthma prevalence was in 8th grade (5.2%), in 1995 it was in 7th grade (7.4%), and in 1997 it was again in 8th grade (8.3%).

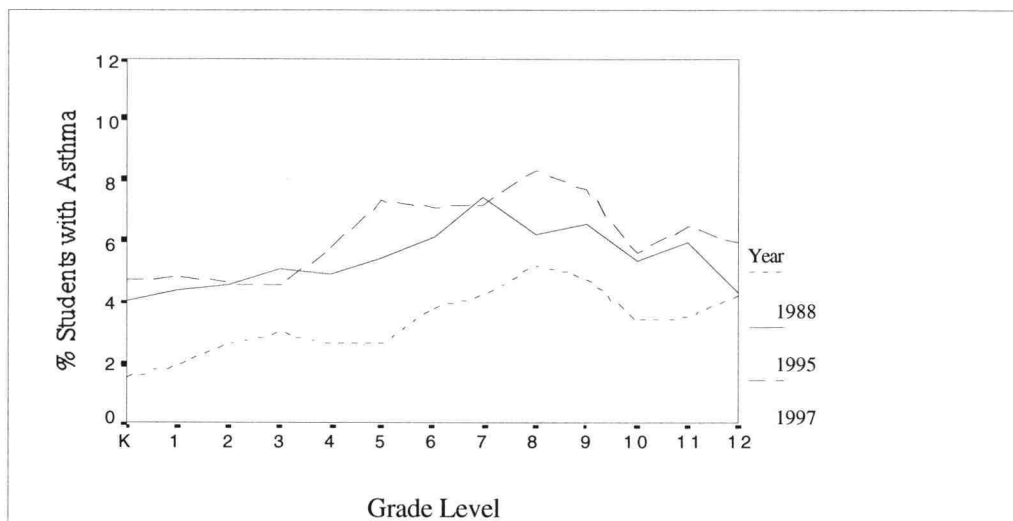


Figure 6. Eugene District 4J Asthma by Grade and Year (1988–1997).

A retrospective study of cohorts enrolled in Eugene School District 4J from 1988–1997 (Figure 7) showed an asthma prevalence increase in all cohorts. The largest increase was with kindergarten children in 1988, who had an asthma prevalence of 1.5%. This prevalence increased to 7.4% in 7th grade (1995) and 7.7% in 9th grade (1997).

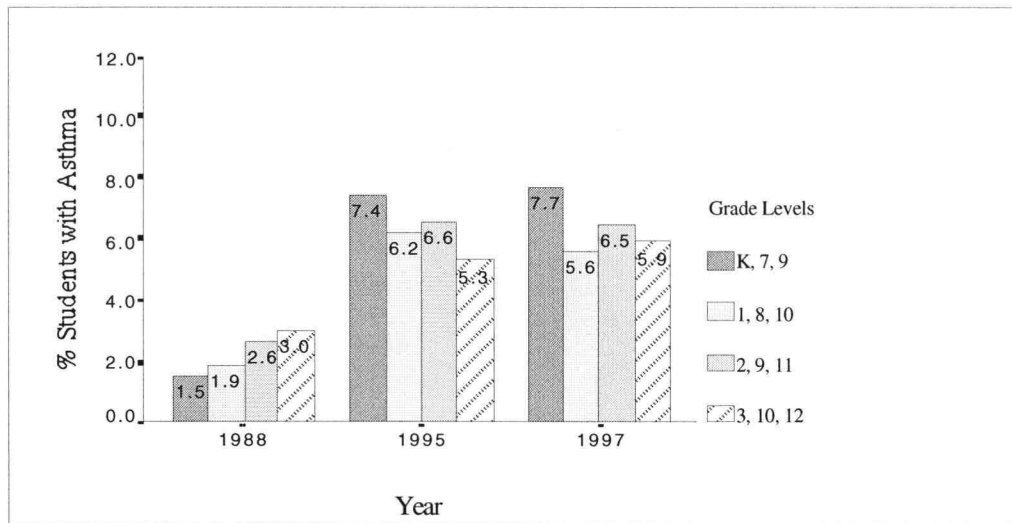


Figure 7. Asthma Prevalence Cohort Study Eugene District 4J (1988–1997).

As shown in Figure 8, Lane County boys had higher asthma prevalence than girls throughout elementary and middle school. In 10th grade, boys' asthma prevalence began to drop, and by 11th and 12th grades, girls' asthma prevalence was higher than boys'.

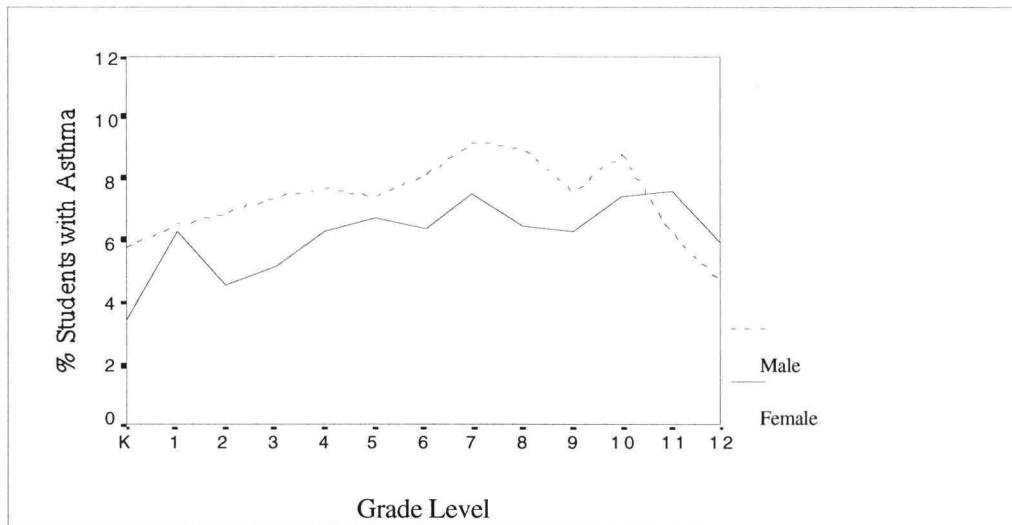


Figure 8. Lane County Asthma Prevalence by Gender and Grade (1996/1997).

As shown in Tables 1, 2, and 3, and Figures 7, 8, and 9, lower socioeconomic status was not strongly correlated with high asthma prevalence. The only school that showed this correlation was Willamette High School in Bethel District, which had the highest Lane County high school asthma prevalence (11.3%) and the lowest high school SES (88). The other schools in Bethel District had higher SES ratings than the high school. Many school districts with higher SES ratings had more asthma prevalence than lower SES school districts. For example, Blachly District, with one of the lowest socioeconomic standings, also had one of the lowest asthma prevalences (3.1%), while Pleasant Hill District had some of the highest SES ratings, with one of the highest asthma prevalences (9.7%).

**Table 1. LANE COUNTY ELEMENTARY SCHOOL ASTHMA PREVALENCE AND SES (State Ratings by School, 1-764)**

School District	% Asthma	School SES
Siuslaw	2.0%	399 & 414
Blachly	3.1%	77
Fern Ridge	3.5%	119–429
Bethel	8.9%	139–565
Junction City	7.2%	323 & 581
Eugene 4J	5.3%	4–763
Springfield	3.1%	74–710
Pleasant Hill	7.9%	570 & 651
South Lane	3.3%	45–467
McKenzie	2.8%	451

**Table 2. LANE COUNTY MIDDLE SCHOOL ASTHMA PREVALENCE AND SES (State Ratings by School, 1-355)**

School District	% Asthma	School SES
Siuslaw	3.2%	193
Blachly	0.0%	81
Fern Ridge	5.6%	189
Bethel	11.6%	115 & 221
Junction City	10.2%	228
Eugene 4J	7.5%	233–352
Springfield	4.8%	59 & 246
Pleasant Hill	10.2%	282
South Lane	5.2%	196
McKenzie	4.4%	202

**Table 3. LANE COUNTY HIGH SCHOOL ASTHMA PREVALENCE AND SES (State Ratings by School, 1-254)**

School District	% Asthma	School SES
Siuslaw	0.6%	116
Blachly	5.2%	127
Fern Ridge	1.4%	148
Bethel	11.3%	88
Junction City	5.4%	133
Eugene 4J	6.5%	177-250
Springfield	5.1%	105 & 194
Pleasant Hill	10.8%	221
South Lane	5.5%	108
McKenzie	2.9%	119

In Figures 9, 10 and 11, scatter plots using county data for individual elementary, middle, and high schools showed no relationship between SES and asthma rates in Lane County schools. R-square coefficients of determination and P values also showed no correlation.

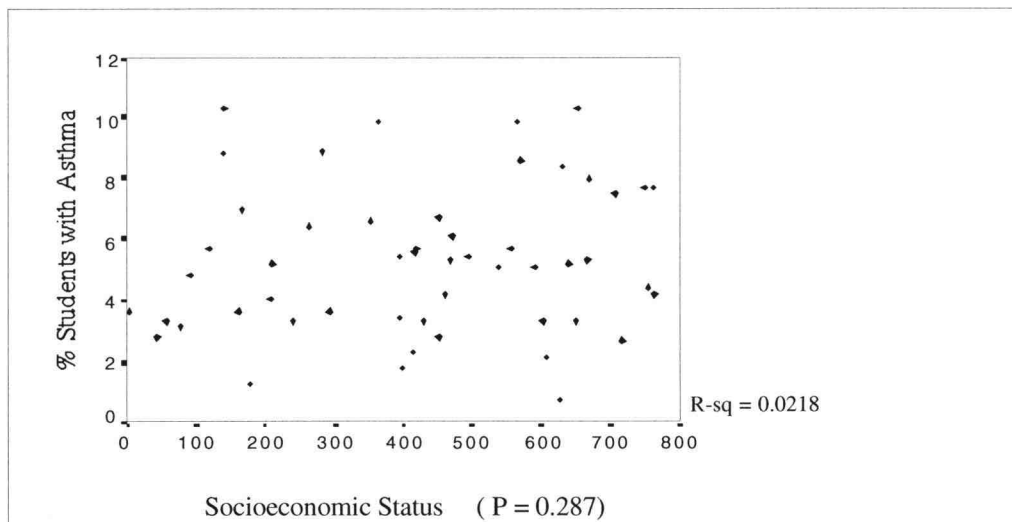


Figure 9. Lane County Elementary School Asthma and SES (1-764).

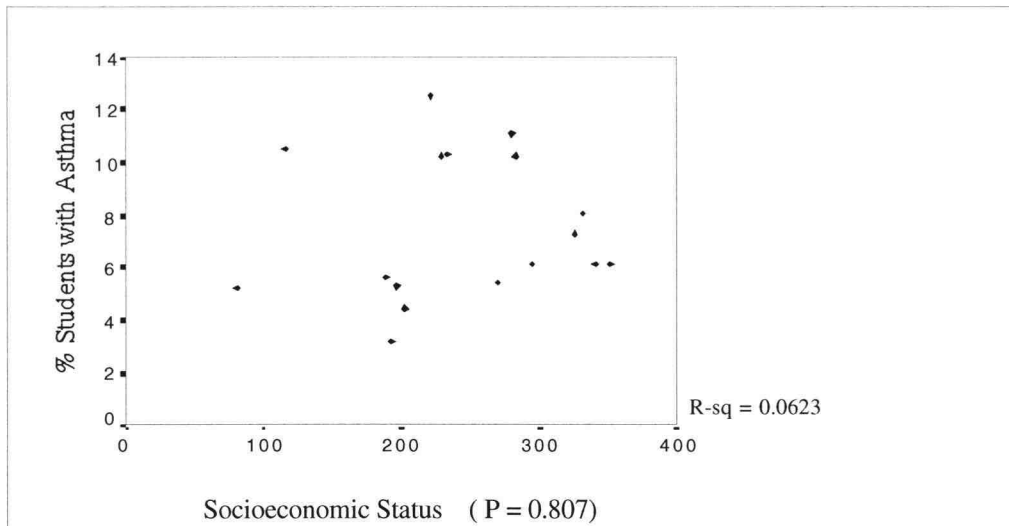


Figure 10. Lane County Middle School Asthma and SES (1-355).

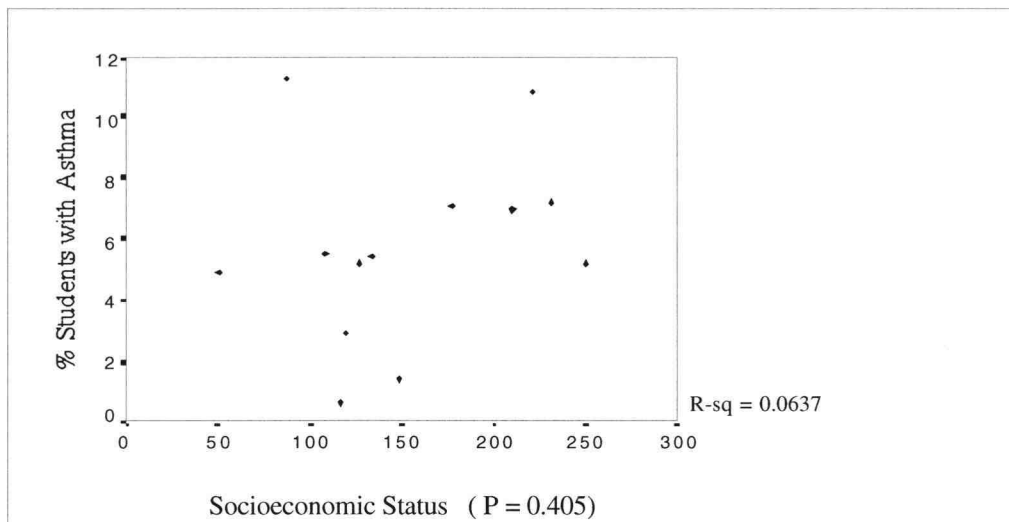


Figure 11. Lane County High School Asthma and SES (1-250).

As shown in Figure 12, Lane County asthma hospitalization rates exhibited a change between 1990 and 1995, particularly in children aged 15–19 years. Asthma hospital discharge rates in this age group increased from 9.6 to 189 per 100,000 person-years in this time. Asthma discharge rates in children aged 11–14 years also increased, from 54.6 to 115.6/100,000 person-years. Asthma hospitalization rates in children aged



5–9 years increased from 61.4 per 100,000 person-years (1990) to 83.7 per 100,000 person-years (1993), then decreased to 49.2 per 100,000 person-years (1995).

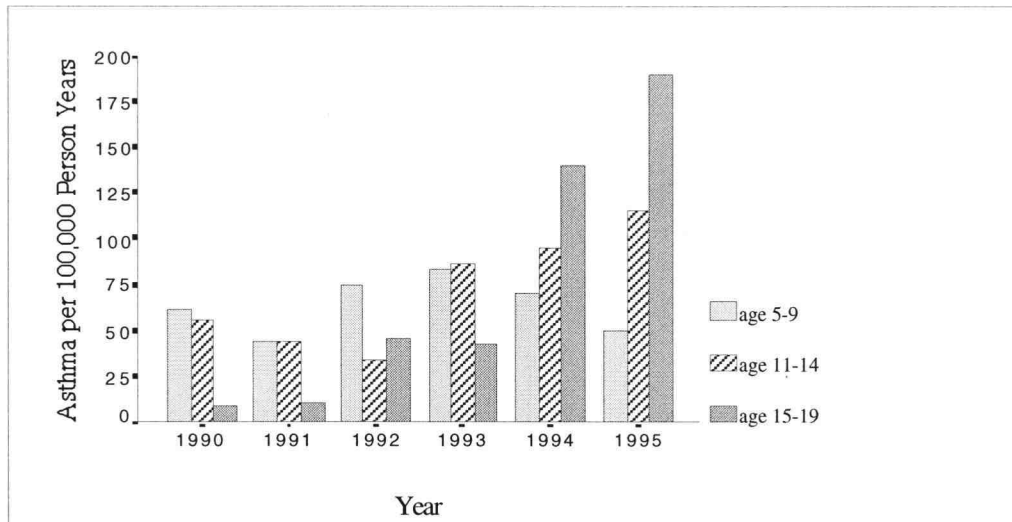


Figure 12. Lane County Asthma Hospital Discharges (1990–1995).

## DISCUSSION

The asthma prevalence in Lane County school districts (5.8%) for the school year 1996/1997 was similar to the rate in Multnomah County school districts (5.9%) in 1995/1996. However, there was a wider variation in asthma prevalence among individual school districts in Lane County than among those in Multnomah County. In Multnomah County, the lowest asthma prevalence (2.5%) was found in Sauvie Island District, a rural farming community, while the highest prevalence (6.2%) was found in David Douglas District, a Portland suburb. The urban area of Portland had a slightly lower asthma prevalence (6.1%) than David Douglas.

In Lane County, the lowest asthma prevalence (1.9%) was found in the coastal Siuslaw School District. This district is located in an area with little industry, good air movement, and minimal air inversions (LRAPA, 1996). The highest asthma prevalence was found in Bethel School District (10%), located in Eugene, an urban community situated in Lane County's central valley, an area known to have inland air stagnation and air inversions. Eugene and nearby Springfield have been out of compliance with NAAQS PM<sub>10</sub> standards since 1987. Ozone levels have been climbing in Eugene and Springfield since 1987, and are considered marginal by the Oregon Department of Environmental Quality (LRAPA, 1996).

Other school districts in the central valley of Lane County share the same air quality problems as Bethel, but have lower asthma prevalence; for example, Junction City (7.7%), Eugene District 4J (6.2%), and Springfield (4%). One possible explanation as to why Bethel has higher asthma prevalence than other schools in Lane County is the concentration of industrial facilities. According to the Eugene Toxics Board (1997), 25 of the industrial facilities found in Bethel District release state and federally listed hazardous or toxic chemicals into the air, water, or soil. Leikauf et al. (1995) point out that people who live in proximity to, or downwind from, an industrial point source should be

considered an important target population for asthma. Therefore, one possible explanation for higher asthma prevalence in Bethel may be the proximity of homes and schools to industrial facilities emitting asthmagenic chemicals. However, this hypothesis warrants further investigation.

Also of interest, four Lane County school districts had an asthma prevalence that exceeded the American Lung Association of Oregon's 1996 estimated state children's asthma prevalence of 6% (Ertle & London, 1996). Of these districts, Bethel (10%), and Eugene 4J (6.2%) are urban, while Junction City (7.7%), and Pleasant Hill (9.7%) are rural. Studies by Friebele, (1996) and Malveaux & Fletcher-Vincent, (1995) found higher asthma prevalence in urban areas than rural, while Gergen (1988) reports a U.S. urban asthma prevalence of 7.1%, and a rural prevalence of 5.7%. Comparing asthma prevalence from urban Bethel and Eugene 4J districts with rural rates from Siuslaw (1.9%), Blachly (3.1%), Fern Ridge (3.9%), South Lane (4.3%), and McKenzie (3.2%) districts supports the hypothesis that urban areas have higher asthma prevalence than rural areas do. However, asthma prevalence in urban Springfield (4%) and rural Pleasant Hill (9.7%) and Junction City (7.7%), and suburban David Douglas District in Multnomah County (6.2%) do not support this hypothesis.

Of interest is the asthma prevalence in Eugene School District 4J, which nearly doubled from 1988 to 1997 (3.3% to 6.2%). Data from previous years were not available from the other school districts; however Eugene 4J's 18,634 students comprise 43% of the total student population studied, thus providing support that asthma prevalence for a large number of Lane County children is increasing. This secular increase in asthma correlates with other research showing steady increases in asthma prevalence in the U.S., Australia, and Scandinavia since the 1970s (CDC, 1996; Friebele, 1996; Leikauf et al., 1995; Weiss et al., 1993).

The North Region of Eugene School District 4J, a heavily populated residential area bordering the Bethel District, had the most notable increase in asthma prevalence

(2.5% to 8%) from 1988–1997. No other region of Eugene District 4J had a comparable increase in asthma prevalence. One possible explanation for the high asthma prevalence in North Region is the proximity to industrial facilities found in the Bethel District, particularly those facilities which began operation after 1988. However, as mentioned before, the role of UATs and asthma in Lane County warrants further investigation.

In Lane County, as in Multnomah County, asthma prevalence increased by grade, with the highest prevalence (7.5%) found in 8th grade (13–14 years old) in Lane County, and 9th grade (14–15 years old) in Multnomah County. In Lane County from 1988 to 1997, Eugene District 4J showed the highest asthma prevalence in grades 7 and 8 (12–14 years old). During this time, four cohorts in Eugene District 4J showed an increase in asthma prevalence, with the 1988 kindergarten, 1st and 2nd grade cohorts showing highest asthma prevalence between grades 8 and 9 (13–15 years old), while the 3rd grade cohort in 1988 had the highest asthma prevalence in grade 12 (17–18 years old).

Gergen (1988), reporting from the National Health and Nutrition Examination Studies (1971–1980) for children 3–17 years old, showed the highest asthma prevalence at age 8 years (2nd–3rd grade), with a 10% prevalence for boys and a 6% prevalence for girls. Other researchers reporting data from the NHIS for Child Health (1988) for children 0–17 years old, showed the highest asthma prevalence (5.1%) in children aged 6–11 years (1st to 6th grades) (Bates, 1995b; Halfon & Newacheck, 1993; Weiss et al., 1993). These studies suggest that the age, or grade, of highest asthma prevalence in children may have increased between 1971 and 1997. Rich and Schneider (1996) call asthma the most common chronic disease of adolescents. Unfortunately, CDC's 1998 report "Surveillance for Asthma—United States, 1960–1995" does not address adolescent asthma prevalence because the age groups CDC used were 0–4 years, 5–14 years and 15–34 years; therefore adolescents were grouped either with young children or with adults.

Friebele (1996), Bates (1995a), Halfon & Newacheck (1993), and Malveaux & Fletcher-Vincent (1995) identified low SES as a significant risk factor in asthma, particularly for children living in urban centers. Children in low SES families often have less preventive medical care and more exposure to allergens, including cigarette smoke, than children in higher SES families. However, Lane County schools showed no correlation between asthma prevalence and SES. For example, Springfield District had similar SES ratings to Bethel District, but had a much lower asthma prevalence (4%) than Bethel (10%). Furthermore, Blachly District, with the lowest overall SES ratings, had one of the lowest asthma prevalences (3.1%), while Pleasant Hill District had some of the highest SES ratings, and one of the highest asthma prevalences (9.7%).

Gender patterns followed those reported by Gergen et al. (1988) and Bates (1995b), showing higher asthma prevalence in elementary school boys than in girls. The National Center for Health Statistics (1976–1980) found the highest male asthma prevalence at age 8 years old (2nd to 3rd grade), while Lane County's highest male prevalence was found at ages 12–14 years (7th and 8th grades). After 10th grade, Lane County male asthma prevalence began dropping, while female prevalence began to increase. Gergen's data reported similar changes in adolescent gender prevalence. By 11th and 12th grade in Lane County, a higher percentage of females (7.6%—11th grade; 6%—12th grade) had asthma than males (6.2%—11th grade; 4.6%—12th grade).

Asthma hospitalization rates in Lane County school children followed the national trend toward hospitalization rate increase discussed by Halfon & Newacheck (1993) and the CDC (1996). This Lane County rate change was most notable in children aged 15–19 years, who had a 20-fold increase in asthma hospital discharges. Children aged 10–14 years more than doubled their asthma hospitalization rate while children aged 5–9 years saw a decline in hospitalizations after 1993. While Bates (1995b) concludes that climbing U.S. asthma hospitalization rates indicate a recent increase in the severity of asthma, another factor in Lane County may be the Oregon Health Plan, which began operation in

March, 1994. This plan provides health care to low-income families who have no other source of assistance, and may have impacted access to hospital care for these families.

Other factors in adolescent hospitalization increase may include that teenagers are at high risk for poor asthma outcome due to lifestyle and development changes (Rich & Schneider, 1996). Adolescents have different treatment requirements than children or adults. Because of the rapid physical and hormonal changes that occur in adolescence, physicians need to encourage regular adolescent physical exams. Adolescents should be included in their own treatment plans, moving responsibility for care from parents to the teenagers themselves, while recognizing that asthma therapy has effects on both physical and psychological development (Gern et al., 1995; Price, 1996).

The results of this study are valuable because hospital discharge records and sample data from Eugene School District 4J indicate a likely increase in both prevalence and severity of asthma in school-aged children, particularly adolescents, in Lane County. Data from this study warrant further investigation into the role that geography, meteorology, and proximity to industry play in asthma prevalence in Lane County.

In order to reduce this rising asthma prevalence in children, and improve the health status of all asthmatics, it is necessary to take a public health approach that emphasizes disease prevention. To achieve success in asthma prevention, it is necessary to address the predominant social, cultural, and environmental conditions in which people, particularly children, live (Etzel, 1995). Education, controlling exposure to indoor antigens, and improving urban air quality are steps that could help reduce asthma prevalence and severity in children (Friebele, 1996).

## RECOMMENDATIONS

Three future areas of study should be considered:

1. Research should be done on causative factors of asthma in Lane County, including urban air toxics, and the role of geography, meteorology, and proximity to industry, especially in areas with the highest asthma prevalence (Bethel, Pleasant Hill, Junction City, North Region of Eugene 4J).
2. Further study of asthmatic children in the areas with the highest asthma prevalence, looking at age, gender, allergic history, family socio-economic status, and indoor/outdoor air quality, including parental cigarette smoking.
3. Initiate preventive public health measures such as asthma education for schools, parents, health care providers, legislators, and industry.

## BIBLIOGRAPHY

- Abbey, D. E., B. E. Ostro, F. Petersen, and R. J. Burchette. 1995. Chronic respiratory symptoms associated with estimated long-term ambient concentrations of fine particulates less than 2.5 microns in aerodynamic diameter (PM<sub>2.5</sub>) and other air pollutants. *Journal of Exposure Analysis and Environmental Epidemiology* 5(2): 137–159.
- Abrams, R., J. L. Amestoy, R. Blumenthal, and T. R. Kulongoski. 1993. *Carpets and Indoor Air: What You Should Know*. New York: Office of Public Information.
- American Lung Association. 1996a. *Childhood Asthma*.
- American Lung Association. 1996b. *Danger Zones: Ozone Air Pollution and Our Children*.
- Andrews J. S. and C. D. DeAngelis. 1995. Pediatrics. *The Journal of the American Medical Association* 273(21): 1708–1711.
- Ashford, N. A. and C. S. Miller. 1991. *Chemical Exposures, Low Levels and High Stakes*. New York: Van Nostrand Reinhold.
- Bascom, R., P. A. Bromberg, D. A. Costa, R. Devlin, D. W. Dockery, M. W. Frampton, W. Lambert, J. M. Samet, F. E. Speizer, and M. Utell. 1996. Health effects of outdoor air pollution. *American Journal of Respiratory Critical Care Medicine* 153: 3–50.
- Bates, D. V. 1995a. The effects of air pollution on children. *Environmental Health Perspectives* 103 (Suppl 6): 49–53.
- Bates, D. V. 1995b. Observations on asthma. *Environmental Health Perspectives* 103 (Suppl 6): 243–247.
- Brown, K. S. 1997. A decent proposal? EPA's new clean air standards. *Environmental Health Perspectives* 105(4): 378–383.
- Breslin, K. 1995. The impact of ozone. *Environmental Health Perspectives* 103(7–8): 660–664.



- Brunekreef, B., D. W. Dockery, and M. Krzyzanowski. 1995. Epidemiologic studies on short-term effects of low levels of major ambient air pollution components. *Environmental Health Perspectives* 103 (Suppl 2): 3–13.
- Carter-Pokras, O. D. & P. J. Gergen. 1993. Reported asthma among Puerto-Rican, Mexican-American, and Cuban children (1982 through 1984). *American Journal of Public Health* 83: 580–582.
- City of Eugene. 1997. *Community Profile*.
- Centers for Disease Control and Prevention. 1995. *Vital and Health Statistics, Current Estimates from the National Health Interview Survey, 1994*. DHHS Publication No. PHS: 96–1521.
- Centers for Disease Control and Prevention. 1996. Asthma mortality and hospitalization among children and young adults—United States, 1980–1993. *The Journal of the American Medical Association* 275(20): 1536–1537.
- Centers for Disease Control and Prevention. 1998. Surveillance for asthma—United States, 1960–1995. *MMWR* 47(SS-1), April 24.
- Columbia University National Center for Children in Poverty. 1990. *Data Sourcebook: Five Million Children*. New York.
- Dockery, D. W., C. A. Pope III, X. Xu, J. D. Spengler, J. H. Ware, M. E. Fay, B. G. Ferris Jr., F. E. Speizer. 1993. Mortality risks of air pollution: A prospective cohort study. *New England Journal of Medicine* 329: 1753–1759.
- Dunford, R. S. 1994. *Your Health and the Indoor Environment*. Dallas: NuDawn Publishing.
- Ertle, A. R. and M. R. London. 1996. *Insight into Asthma Prevalence in Oregon*. Portland: American Lung Association of Oregon.
- Etzel, R. A. 1995. Indoor air pollution and childhood asthma: effective environmental interventions. *Environmental Health Perspectives* 103( 6): 55–58.
- Eugene Toxics Board, City of Eugene Public Safety. 1997. Toxics List. *The Register-Guard*. October 4.

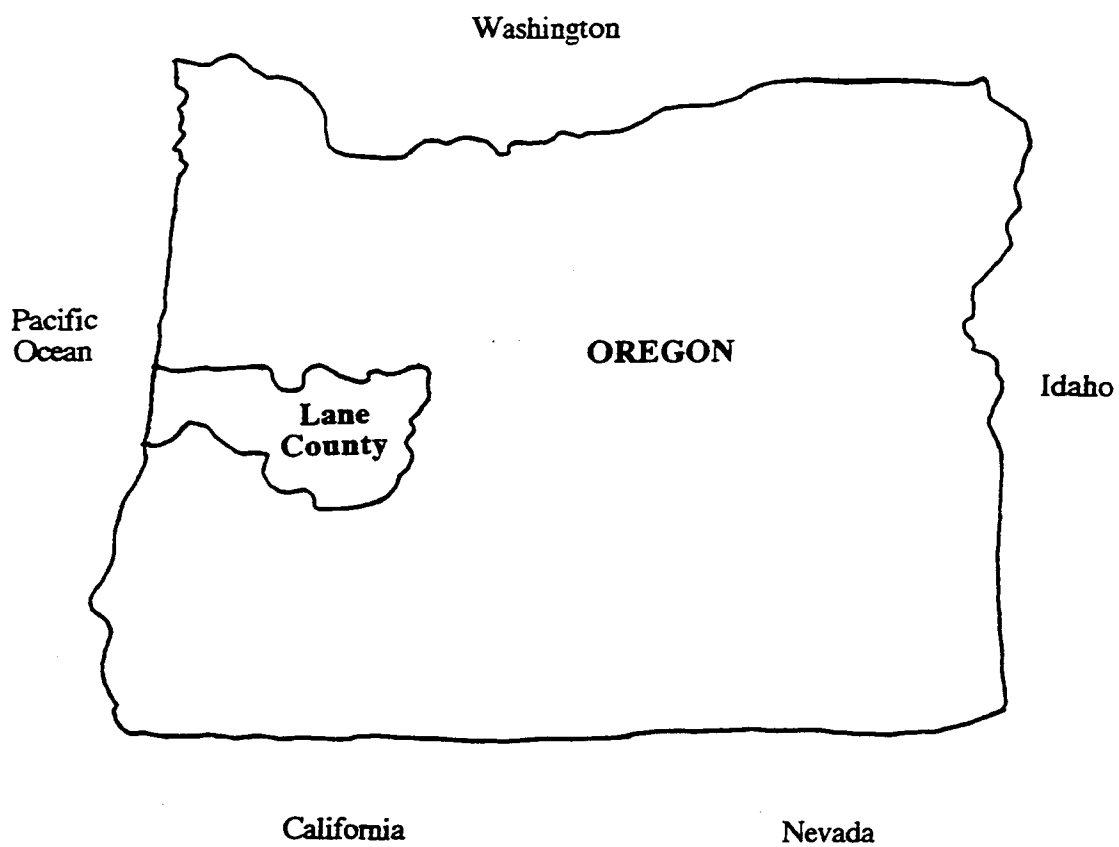
- Evans, R. 1992. Asthma among minority children: a growing problem. *Chest* 101(6): 368S–371S.
- Fletcher-Vincent, S. A., E. R. Reece, and R. J. Malveaux. 1994. Reactivity to cockroach and other allergens in an inner city population with rhinitis and asthma. *Journal of Allergy and Clinical Immunology* 93: 174.
- Folinsbee, L. J. 1992. Human health effects of air pollution. *Environmental Health Perspectives* 100: 45–56.
- Friebele, E. 1996. The attack of asthma. *Environmental Health Perspectives* 104(1): 22–25.
- Gergen, P. J., D. I. Mullally, and R. Evans. 1988. National survey of prevalence of asthma among children in the United States, 1976 to 1980. *Pediatrics* 81(1): 1–7.
- Gergen, P. J. and K. B. Weiss. 1990. Changing patterns of asthma hospitalization among children 1970–87. *The Journal of the American Medical Association* 264: 1688–1692.
- Gern, J. E., M. K. Schroth, and R. F. Lemanske Jr. 1995. Childhood asthma. Older children and adolescents. *Clinics in Chest Medicine* 16(4): 657–70.
- Godish, T. 1991. *Air Quality*. Ann Arbor: Lewis Publishers.
- Godish, T. 1995. *Sick Buildings Definition, Diagnosis and Mitigation*. Ann Arbor: Lewis Publishers.
- Goldman, L. R. 1995. Children—unique and vulnerable. Environmental risks facing children and recommendations for response. *Environmental Health Perspectives* 103(Suppl 6): 13–18.
- Gong, H. Jr. 1992. Health effects of air pollution; a review of clinical studies. *Clinics in Chest Medicine* 13(2): 201–214.
- Halfon, N. and P. W. Newacheck. 1993. Childhood asthma and poverty: Differential impacts and utilization of health services. *Pediatrics* 91(1): 56–61.

- Immerman, F. W. and J. L. Schaum. 1990. Nonoccupational pesticides exposure study. Document no. 600/S3-90/003. Washington: U.S. Environmental Protection Agency.
- Johnston, S. L., P. K. Pattemore, G. Sanderson, S. Smith, F. Lampe, L. Josephs, P. Symington, S. O'Toole, S. H. Myint, D. A. Tyrell. 1995. Community study of role of viral infections in exacerbations of asthma in 9-11 year olds. *BMJ* 310: 1225-1229.
- Kang, B. 1976. Study on cockroach antigen as possible causative agent in bronchial asthma. *Journal of Allergy and Clinical Immunology* 58: 357-365.
- Koenig, J. Q., T. V. Larson, Q. S. Hanley, V. Rebolledo, K. Dumler, H. Checkoway, S. Z. Wang, D. Lin, and W. E. Pierson. 1993. Pulmonary function changes in children associated with fine particulate matter. *Environmental Research* 63: 26-38.
- Koren, H. S. 1995. Associations between criteria air pollutants and asthma. *Environmental Health Perspectives* 103 (Suppl 6): 235-242.
- Landrigan, P. J., D. R. Mattison, H. B. Babich, B. Boardman, J. V. Bruckner, M. A. Gallo, D. E. Hutchings, R. J. Jackson, M. H. Karol, D. Krewski, G. A. Purvis, R. L. Rizek, J. N. Seiber, and W. B. Weil. 1993. *Pesticides in the Diets of Women and Children*. National Resource Council. Washington D.C.: National Academy Press.
- Lane Regional Air Pollution Authority. 1996 *Annual Report*. Springfield, Oregon.
- Leikauf, G. D., S. Kline, R. E. Albert, C. S. Baxter, D. I. Bernstein, J. Bernstein and R. Buncher. 1995. Evaluation of a possible association of urban air toxics and asthma. *Environmental Health Perspectives* 103(Suppl 6): 253-271.
- Levy, B. S. and D. H. Wegman. 1995. *Occupational Health; Recognizing and Preventing Work-Related Disease*. New York: Little Brown and Company.
- Lipsett, M., S. Hurley and B. Ostro. 1997. Air pollution and emergency room visits for asthma in Santa Clara County, California. *Environmental Health Perspectives* 105: 216-222.
- Lu, F. C. 1996. *Basic Toxicology*. Washington D.C.: Taylor and Francis.

- Malveaux, F. J. and S. A. Fletcher-Vincent. 1995. Environmental risk factors of childhood asthma in urban centers. *Environmental Health Perspectives* 103 (Suppl 6): 59–62.
- Marder, D., P. Targonsky, O. Orris, V. Persky, and W. Addington. 1992. Effect of racial and socioeconomic factors on asthma mortality in Chicago. *Chest* 101: 427S–430S.
- Mercus, P. J., E. E. van Essen-Zandvliet, E. J. Duiverman, H. C. van Houweilingen, K. F. Kerrebijn, and P. H. Quanjer. 1993. Long-term effect of inhaled corticosteroids on growth rate in adolescents with asthma. *Pediatrics* 91(6): 1121–1126.
- Neville, R. G., C. McCowan, G. Thomas, and I. K. Crombie. 1996. Asthma and growth—cause for concern? Asthma and growth in Tayside children. *Annals of Human Biology* 23(4): 323–331.
- Newman-Taylor, A. 1995. Environmental determinants of asthma. *The Lancet* 345(8945): 296–300.
- Oregon Department of Education. 1997. *Guide to Interpreting 1997 Oregon Statewide Assessment Results*. Salem.
- Oregon State Health Division. 1994. *Behavioral Risk Factor Surveillance Survey*. Salem.
- Pope, C. A. III. 1989. Respiratory disease associated with community air pollution and a steel mill, Utah Valley. *American Journal of Public Health* 79: 623–628.
- Pope, C. A. III. 1991. Respiratory hospital admissions associated with PM<sub>10</sub> pollution in Utah, Salt Lake and Cache Valleys. *Archives of Environmental Health* 46: 90–97.
- Pope C. A. III and D. W. Dockery. 1992. Acute health effects of PM<sub>10</sub> pollution in symptomatic and non-symptomatic children. *American Review of Respiratory Disease* 145: 1123–1128.
- Pope, C. A. III, D. V. Bates and M. E. Raizenne. 1995. Health effects of particulate air pollution: Time for reassessment? *Environmental Health Perspectives* 103: 472–480.
- Portney, P. R. and J. Mullahy. 1990. Urban air quality and chronic respiratory disease. *Regional Science and Urban Economics* 20: 407–418.

- Price, J. F. 1996. Issues in adolescent asthma: What are the needs? *Thorax* 51 (Suppl 1): S13–7.
- Ransom, M. R. and C. A. Pope III. 1992. Elementary school absences and PM<sub>10</sub> pollution in Utah Valley. *Environmental Research* 58: 204–209.
- Rich, M. and L. Schneider. 1996. Managing asthma with the adolescent. *Current Opinions in Pediatrics* 8(4): 301–309.
- Schwartz, J., D. Slater, T. V. Larson, W. E. Pierson, and J. Q. Koenig. 1993. Particulate air pollution and hospital emergency room visits in Seattle. *American Review of Respiratory Disease* 147: 826–831.
- Seachrist, I. 1995. Estrogen linked to adult asthma risk. *Science News* 48(18): 279.
- State of Oregon Employment Department. 1997a. *Oregon Labor Trends*. Salem.
- State of Oregon Employment Department. 1997b. *Resident Oregon Labor Force and Unemployment by Area*. Salem.
- Weitzman, M., S. Gortmaker, D. K. Walker and A. Sobol. 1990. Maternal smoking and childhood asthma. *Pediatrics* 85(4): 505–511.
- Weiss, K. B., P. J. Gergen and D. K. Wagener. 1993. Breathing better or wheezing worse? The changing epidemiology of asthma morbidity and mortality. *Annual Review of Public Health* 14: 491–513.
- Wiles R. and K. Davies. 1995. *Pesticides in Baby Food*. San Francisco: Environmental Working Group.

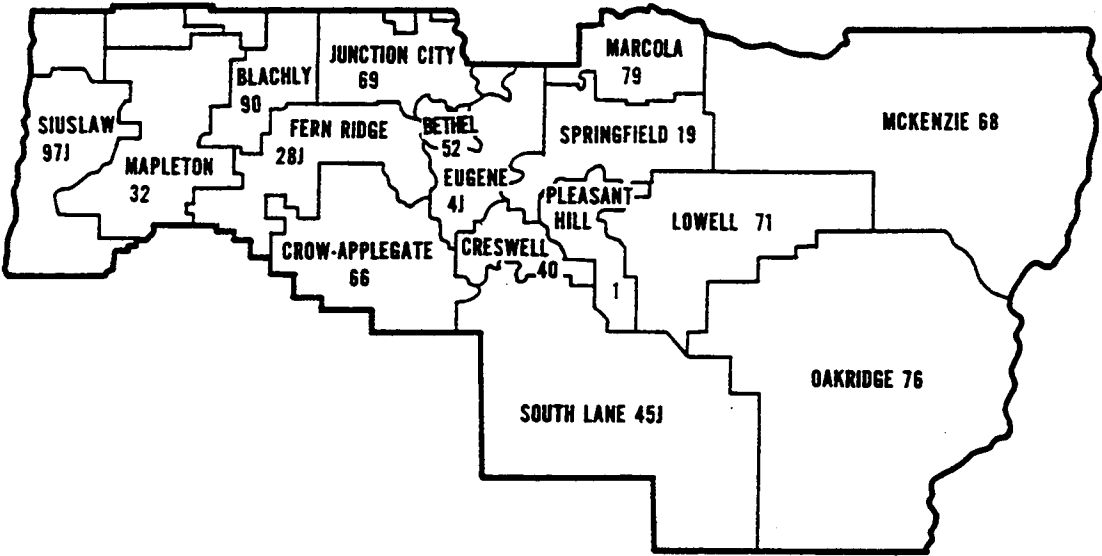
## APPENDICES

**APPENDIX 1. LANE COUNTY, OREGON**

**APPENDIX 2. LANE COUNTY PHYSIOGRAPHY (LRAPA, 1996)**

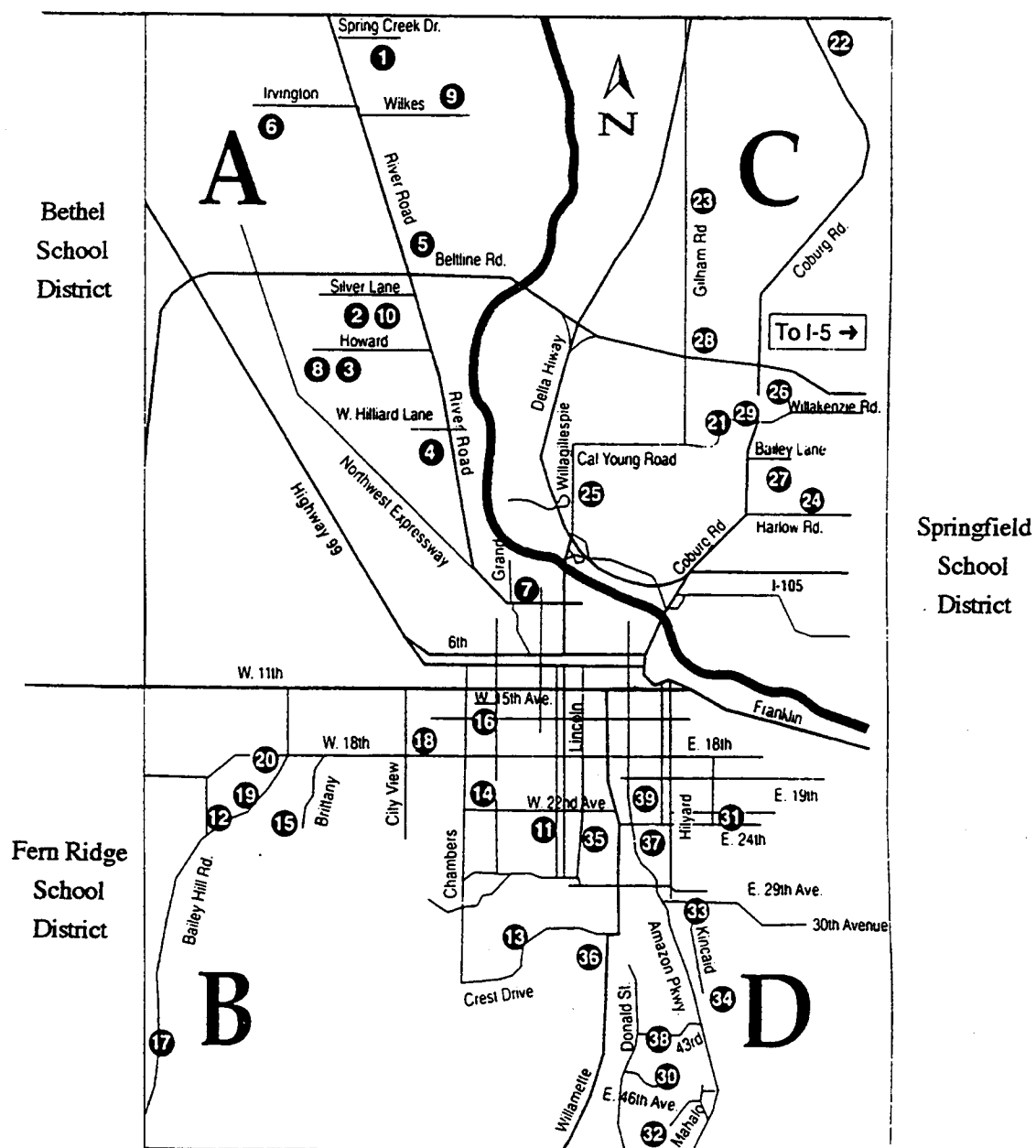


**APPENDIX 3. LANE COUNTY SCHOOL DISTRICTS  
(LANE ESD)**



# APPENDIX 4. EUGENE SCHOOL DISTRICT 4J REGIONS

- A. North Region
- B. Churchill Region
- C. Sheldon Region
- D. South Region



**APPENDIX 5. LETTER TO SCHOOL DISTRICT SUPERINTENDENTS**

May 27, 1997

Dear Dr.

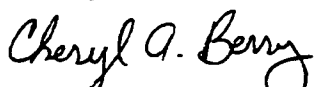
I am a graduate student in the Department of Public Health at Oregon State University (OSU). For my master's thesis research, I am working with the American Lung Association of Oregon (ALAO) to determine asthma prevalence over time in school-age children (K-12) in Lane County. Recent research shows that asthma rates have almost doubled in the U.S. in the past ten years, with higher rates in urban areas than in rural. I would like to gather and analyze asthma rates in all 16 districts, looking for rate increases in the past 5-10 years, noting any significant differences between urban and rural asthma rates. The ALAO compiled similar data from Multnomah County schools in 1996.

I am an Oregon certified school nurse and am aware of the importance of record confidentiality. This project, *Asthma Prevalence in School Children in Lane County, Oregon*, will first have to gain approval of OSU's Institutional Review Board for the Protection of Human Subjects. Names of children will not be collected or reported in any way. I would like a tally of the number of children reported by their parents to have asthma. This information is usually found on enrollment sheets and/or health cards. I am looking for asthma trends over time by age, gender, grade, school, and school district for 1996-1997 and for the past 5-10 years, if that data is available. The project will show the number of asthmatic children in each school, but to prevent linking identifiers to students, will not report age, grade, or gender by school. This demographic data will be compiled and analyzed by district. I am willing to assist with collection of the data if it is not computerized. This project should be completed by September, 1997.

No one is presently assessing or tracking asthma rates in Oregon and this research will help ALAO determine the prevalence and impact of asthma in the state. I am requesting permission to gather asthma information from your schools and have included a consent form with a stamped, addressed return envelope. If you have questions, I may be reached by phone in Eugene, 541/344-6828. You may also call Jeanette Bader, M.S.W., Assistant Executive Director, ALAO, 1/800/586-4872, or Catherine Neumann, PhD., Department of Public Health, OSU, 541/737-3833, with questions pertaining to this project.

Thank you for your time.

Sincerely,



Cheryl A. Berry, R.N., B.S.

**APPENDIX 6. SUPERINTENDENT CONSENT FORM****Asthma Prevalence in School Children  
in Lane County, Oregon**

Cheryl A. Berry, R. N., B.S.  
Investigator

I have read the procedures described and give informed consent to the project, *Asthma Prevalence in School Children in Lane County, Oregon*, to collect and analyze asthma rates over time by age, gender, grade, and school in this school district.

---

Superintendent

---

School District

---

Date Signed

## APPENDIX 7. SCHOOL ENROLLMENT FORM

### STUDENT REGISTRATION FORM

INSTRUCTIONS: Please print.

Please do not write in shaded areas

<b>SCHOOL NAME</b>				Providing your social security number (SSN) is voluntary. If you provide it, the school district will use your SSN for record-keeping, research, and reporting purposes only. The school district will not use your SSN to make any decision directly affecting you or any other person. Your SSN will not be given to the general public. If you choose not to provide your SSN, you will not be denied any rights as a student. Please read the statement on the back of this form that describes how your SSN will be used. Providing your SSN means that you consent to the use of your SSN in the manner described.			
<b>NEW STUDENT-10</b>							
STUDENT'S LAST NAME (known by) (1)		FIRST NAME, INITIAL (2)		SEX (3) M F		BIRTHDATE (4) / /	
				ETHNIC: CHECK ONE (OPT.) (5) WHITE <input type="checkbox"/> HISPANIC <input type="checkbox"/> BLACK <input type="checkbox"/> ASIAN <input type="checkbox"/> AMER. IND./ALASKA NATIVE <input type="checkbox"/>		SOCIAL SECURITY # (6) - -	
STUDENT'S LEGAL NAME (last, first, middle) (7)				HOME PHONE (8) Y N		BIRTH CITY (9)	
HOME ADDRESS (11)				HOME CITY (12)		STATE (13)	
MAILING ADDRESS (if different from Home Address) (15)				MAILING CITY (16)		STATE (17)	
MAILING NAME (if other than TO THE PARENTS OF) (19)				STUDENT CAR LICENSE(S) (20)			
LAST NAME HEAD-OF-HOUSEHOLD (21)		FIRST NAME HEAD-OF-HOUSEHOLD (22)		HEAD-OF-HOUSEHOLD WORK PHONE (23) Y N		EXT. (24)	
LAST NAME SPOUSE (25)		FIRST NAME SPOUSE (26)		SPOUSE WORK PHONE (27) Y N		EXT. (28)	
HEAD-OF-HOUSEHOLD OCCUPATION (30)		SPOUSE OCCUPATION (31)		LIVING WITH: (29) PARENTS PA FATHER FA STEPFATHER SF MOTHER MO STEPMOTHER SM FOSTER FO GUARDIAN CU GRANDPARENT GP BLENDED FAMILY BF		PLEASE CIRCLE	
If a specific caseworker is assigned to this student, please enter the following information:		CASEWORKER NAME (32)		CASEWORKER PHONE (33)		EXT. (34)	

### School Use Only

GRD (35)	ENR. CODE (36)	GRAD. CATEG (37)	ENTRY DATE (38)	ATT. CATEG (39)	FTE (40)	HOMEROOM (41)	LOCKER (42)	TEACHER ID (43)	TEACHER NAME
ATT. REF. (44)	MIG. REF. (45)	ESL (46)	LUNCH (47)	PIP (48)	PIC (49)	SPEC. ED (50)	TAG (51)	COLL. TESTS (52)	TITLE-I (53)
								DWELL (54)	MARK EXCL. (56)
COUNTY (59)	PRECINCT (60)	ELEM DIST (61)	ES-LAST (62)	MS-LAST (63)	SCH NEXT (64)	BUS ROUTE (65)	BUS STOP (67)	SCHOOL USE (66)	

### Previous School Information

PREVIOUS SCHOOL NAME, CITY, STATE (68, 69, 70)				
DATE ENROLLED (71) (MM/DD/YY)	DATE WITHDRAWN (72) (MM/DD/YY)	GRADE LEVEL (73)	SPECIAL ED TAG Y N	RECEIVED TAG Y N

### Emergency Information

EMERGENCY CONTACT #1 NAME/PHONE/RELATIONSHIP (74)				EMERGENCY CONTACT #2 NAME/PHONE/RELATIONSHIP (75)			
CHILD CARE PROVIDER NAME/PHONE (76)				PERSONS AUTHORIZED TO PICK UP STUDENT (77)			
DOCTOR NAME (78)		DOCTOR PHONE (79)		DENTIST NAME (80)		DENTIST PHONE (81)	
HOSPITAL (82)		PLEASE CHECK ANY CURRENT ON-GOING PROBLEMS:		Serious Allergy <input type="checkbox"/> (83) Asthma <input type="checkbox"/> (84) Heart Disease <input type="checkbox"/> (85) Diabetes <input type="checkbox"/> (86) Seizure Disorder <input type="checkbox"/> (87) Other <input type="checkbox"/> (88)		NURSING RECS (89)	
<input type="checkbox"/> I completed Health information last year when my child attended school (90)		HEALTH NOTES (list serious allergies, current medications, other health problems) (91)					
In case of accident or serious illness, if neither responsible guardian nor named physician can be reached, do you authorize and direct the school officials to send your child to a Doctor and/or Hospital for medical treatment?							
(92) YES <input type="checkbox"/> NO <input type="checkbox"/> PARENT SIGNATURE _____				DATE _____			



Last Name \_\_\_\_\_ First \_\_\_\_\_ MI \_\_\_\_\_  
 Social Security No. \_\_\_\_\_  
 (requested but not required)

Social Security No. \_\_\_\_\_  
(requested but not required)

(Please complete this section in pencil)

Sex \_\_\_\_\_ Birth Date \_\_\_\_\_ Birth Place \_\_\_\_\_

Street Address \_\_\_\_\_ Telephone No. \_\_\_\_\_

City \_\_\_\_\_ Zip Code \_\_\_\_\_

Father's Name \_\_\_\_\_ Day Phone \_\_\_\_\_

Mother's Name \_\_\_\_\_ Day Phone \_\_\_\_\_

Child Lives With \_\_\_\_\_ Phone No. \_\_\_\_\_

Emergency Name \_\_\_\_\_ Phone No. \_\_\_\_\_

Health Care Provider \_\_\_\_\_ Phone No. \_\_\_\_\_

[illegible][illegible][illegible]

Immunizations ☐  
Complete

Immunizations Incomplete (specify)	
---------------------------------------	--

Incomplete/ ☐ Up to date ☐ Date

Religious Exemption ☐ Date \_\_\_\_\_

Medical Exemption ☐ Date \_\_\_\_\_  
Specify \_\_\_\_\_

### TB Screening

[illegible]

61

# APPENDIX 9. LANE COUNTY SCHOOL PHYSICAL FORM

## MEDICAL REPORT FOR STUDENTS (GRADES K-12) LANE COUNTY SCHOOLS

THIS SECTION TO BE FILLED IN BY PARENT BEFORE PHYSICAL EXAMINATION: (Please print)

(School to be attending) \_\_\_\_\_ (Grade) \_\_\_\_\_  
 Pupil's Name \_\_\_\_\_ Sex: M F Birthdate \_\_\_\_\_  
 (Last) (First) (Month) (Day) (Year)  
 Address \_\_\_\_\_ Zip \_\_\_\_\_ Phone \_\_\_\_\_  
 (Street or Rural Route) (Town)  
 Parent/Guardian \_\_\_\_\_ Physician \_\_\_\_\_

Check the following information about your child:

1. Head/neck injuries	Yes* _____	No _____	Year _____	13. Kidney disease	Yes* _____	No _____	Year _____
2. Muscle, bone or joint disease	Yes* _____	No _____	Year _____	14. Mononucleosis	Yes* _____	No _____	Year _____
3. Scoliosis	Yes* _____	No _____	Year _____	15. Chickenpox	Yes* _____	No _____	Year _____
4. Loss or seriously impaired vision in one eye?	Yes* _____	No _____	Year _____	16. Insect/bee sting reaction	Yes* _____	No _____	Year _____
5. Hearing problem	Yes* _____	No _____	Year _____	17. Asthma	Yes* _____	No _____	Year _____
6. Pneumonia	Yes* _____	No _____	Year _____	18. Hay Fever	Yes* _____	No _____	Year _____
7. Hernia	Yes* _____	No _____	Year _____	19. Food allergy	Yes* _____	No _____	Year _____
8. Diabetes	Yes* _____	No _____	Year _____	20. Skin allergy	Yes* _____	No _____	Year _____
9. Fainting spells	Yes* _____	No _____	Year _____	21. Currently taking medication or shots	Yes* _____	No _____	Year _____
10. Epilepsy/seizures	Yes* _____	No _____	Year _____	22. Previous operations	Yes* _____	No _____	Year _____
11. Streptococcus infection	Yes* _____	No _____	Year _____	23. Any other serious problems	Yes* _____	No _____	Year _____
12. Rheumatic fever	Yes* _____	No _____	Year _____				

Comment on "Yes" \_\_\_\_\_

BEHAVIOR AND ANY PHYSICAL OR EMOTIONAL PROBLEMS: \_\_\_\_\_

## DOCTOR'S PHYSICAL EXAMINATION

Height _____	Vision with glasses/contacts <input type="checkbox"/>	<b>Immunization Summary</b>	
Weight _____	Vision without glasses <input type="checkbox"/>	Last Dose Month/Year	Given Today
Blood Pressure _____	R 20/_____ L/20 _____	Diphtheria _____	_____
		Whooping Cough _____	_____
		Tetanus _____	_____
		Polio _____	_____
		Sabin-oral _____	_____
		Salk _____	_____
		Measles(vaccine) _____	_____
		Mumps (vaccine) _____	_____
		Rubella (vaccine) _____	_____
<b>Examination</b>	<b>Satisfactory</b>	<b>Unsatisfactory</b>	
Teeth _____	_____	_____	
Hearing _____	_____	_____	
Cardiovascular _____	_____	_____	
Respiratory _____	_____	_____	
Liver, spleen, kidney _____	_____	_____	
hernia, genitals _____	_____	_____	
Extremities _____	_____	_____	
Orthopedic/posture _____	_____	_____	
Neurological _____	_____	_____	
Skin _____	_____	_____	
		Tuberculin _____	_____
		Chest X-Ray _____	_____
		Indicated lab tests _____	_____
		Urine _____	_____
		Blood _____	_____
		Other _____	_____

Significant illnesses or injuries \_\_\_\_\_

Diagnosis \_\_\_\_\_

I have on this date examined the above student and recommend him/her as being physically able to participate in regularly scheduled physical education classes and compete in the following supervised athletics: BASEBALL, BASKETBALL, CROSS COUNTRY, FOOTBALL, GOLF, SOCCER, SOFTBALL, SWIMMING, TENNIS, TRACK, VOLLEYBALL, WRESTLING\*, OTHER \_\_\_\_\_

\*This student may be permitted weight loss to make a lower weight class in WRESTLING. Yes \_\_\_\_\_ No \_\_\_\_\_ If "Yes" may lose \_\_\_\_\_ pounds (Grades 6-12)

Date \_\_\_\_\_ (Signature of Examining Physician)

NOTE: Physician is licensed by the Oregon State Board of Medical Examiners

**433.800 - PROGRAMS TO TREAT ALLERGENS**  
**"Policy. It is the purpose of ORS 433.800 to provide a means of authorizing certain individuals when a physician is not immediately available to administer lifesaving treatment to those persons who have severe adverse reactions to insect stings and other specific allergens.**