

Ekrem YAZICIO LU

**AIR “CRITERIA POLLUTANTS” AND HEALTH INDICATORS:
BRIDGING THE GAP FROM SOURCES TO HEALTH
OUTCOMES AS A CASE STUDY IN MU LA CITY**

M.S. Thesis in Environmental Engineering

by

Ekrem YAZICIO LU

December - 2011

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Ekrem YAZICIO LU

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in

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APPROVAL PAGE

I certify that this thesis satisfies all the requirements as a thesis for the degree of Master of Science.

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This is to certify that I have read this thesis and that in my opinion it is fully adequate, in scope and quality, as a thesis for the degree of Master of Science.

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December 2011

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Supervisor: Assist. Prof. Dr. Ekrem UZONUR

ABSTRACT

The U.S. Environmental Protection Agency (EPA) has established National Ambient Air Quality Standards (NAAQS) for six principal air pollutants (“criteria” pollutants): Carbon Monoxide (CO), Lead (Pb), Nitrogen Dioxide (NO₂), Particulate Matter (PM) in two size ranges [$< 2.5 \mu\text{m}$ (PM_{2.5}) and $< 10 \mu\text{m}$ (PM₁₀)], Ozone (O₃), and Sulfur Dioxide (SO₂). Although associations have been identified between these pollutants and adverse health effects, considerable uncertainty remains regarding a) methods and approaches to understanding relationships between air pollution and health effects; b) which components (gas and/or aerosol) and sources are most toxic; c) the mechanisms of actions of the pollutants and causal relationships; d) effect of confounding factors, and e) which populations are susceptible etc.

Global Alliance Against Chronic Respiratory Diseases (GARD) work on the above issues and in this thesis the data of GARD in Mu la city will be evaluated from the academical perspective to fulfill and bridge the gaps between the data available (criteria air pollutants, sources and airway diseases’ epidemiological, in Mu la city) with the health outcomes.

Keywords: Air Pollution and Health Effects, SO₂, PM₁₀, Chronical Airway Diseases, GARD, Mu la.

HAVA KİRLİLİĞİ VE SAĞLIK BAZI HASTALIKLARINI SORGULAMADA MÜLAKAT KRONİK HAVA YOLU HASTALIKLARI ÖRNEĞİ

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Yüksek Lisans Tezi – Çevre Mühendisliği
Aralık 2011

Tez Danışmanı: Yrd. Doç. Dr. Mehmet UZONUR

ÖZ

Mülakatlı, mevcut verilere göre hava kirliliğinin yoğunlaştığı yerleşim yerlerinden biridir. Hava kirliliği ve kronik hava yolu hastalıklarının ilişkisi bilinse de, bunun tüm yönleriyle ortaya konulabilmesi söylenemez. Kronik hava yolu hastalıklarının önlenmesi veya sıkıntılarının azaltılması yönündeki çabaların yetersizliği ve bu yönde yeni araştırmalara ihtiyaç duyulduğu da çok açıktır.

Tezimizde, Mülakatlı mevcut kirlilik verileri kaynaklarından alınmış; ilin doğu ve insan kaynaklı hava kirlleticileri harita üzerinde belirlenmiş; kronik solunum yolu hastalık ham verileri de değerlendirilmiş ve ArcView 10 programıyla harita üzerinde yerleştirilmiştir. Mevcut veriler (hastalık verileri: toplam ve nüfusa oranlı hasta sayısı; kirlilik parametreleri: SO₂ ve PM₁₀; meteorolojik veriler; Mülakatlı maden yatakları ve hava kirleticisi kaynakları) uygun sitelerden güncellenerek bar ve pasta grafiklerinde gösterilmiştir. Tez çalışmamızda mevcut ham veriler yorumlanabilir, üzerinde bilimsel değerlendirme ve tartışma yapılabilir, faydaya yönelik verilere dönüştürülmüştür; Türkiye ve dünyadaki durumların karşılaştırılabilmesi için grafikler haline getirilmiştir.

Anahtar Kelimeler: Hava kirliliği ve Sağlık Sonuçları, Kronik Solunum Sistemi Hastalıkları, SO₂, PM₁₀, GARD, Mülakatlı.

DEDICATION

To my dear, precious, and esteemed family and

To my parents

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LIST OF SYMSBOLS AND ABBREVIATIONS

SYMBOLS/ABBREVIATIONS

AAAR	American Association of Aerosol Research
GARD	Global Alliance Against Chronic Respiratory Diseases
VOC	Volatile Organic Compound
EPA	Environmental Protection Agency
ICD-10	International Statistical Classification of Diseases and Related Health Problems 10th Revision
NGOs	Universities and Non-Governmental Organizations
OAQPS	The EPA Office of Air Quality Planning and Standards
AQI	Air Quality Index
API	Air Pollution Index
PSI	Pollutant Standard Index
PM	Particulate Matter
PM ₁₀	Combination of Coarse and Fine Particulate Matter
PM _{2.5}	Fine Particulate Matter
SO ₂	Sulfur dioxide
O ₃	Ozone
CO	Carbon Monoxide
NO	Nitric oxide
NO ₂	Nitrogen dioxide
NO _x	Nitric oxide and Nitrogen dioxide
SO _x	Sulfur Oxides
VOC	Volatile Organic Compounds
NAAQS	National Ambient Air Quality Standards
O ₂	Oxygen
OH	Hydroxyl radical

GPS	Global Positioning System
GIS	Geography Information System
VPN	Virtual Private Network
COPD	Chronic Obstructive Pulmonary Disease
AIDS	Acquired Immunodeficiency syndrome
CRD	Chronic Respiratory Disease
CAD	Chronic Airway Diseases
HIV	Human Immunodeficiency Virus
AQMNC	Air Quality Monitoring Network Center
MDG	Millennium Development Goal
NCD	Non Communicable Disease
NCDnet	Global Noncommunicable Disease Network
NGO	Non-governmental organization
V/Q	Ventilation/Perfusion
TB	Tuberculosis
PAH	Polycyclic Aromatic Hydrocarbon
UN	United Nations
EU	European Union
USA	United States of America
WHO	World Health Organization

CHAPTER 1

INTRODUCTION

1.1 BACKGROUND

The problems and solutions discussed in this thesis requires the management of an environmental scientist that should have all capabilities, knowledge, skills and capacities of making scientific research, following technological innovations and to be informed with and involved in governmental policies and effective with decision and policy makers.

In March 2010, the American Association of Aerosol Research (AAAR) held a conference titled “Air Pollution and Health: Bridging the Gap from Sources to Health Outcomes,” which was sponsored by the U.S. EPA and the Health Effects Institute. This conference brought together more than 500 scientists from across the air pollution disciplines, along with risk assessors and policy makers, to look at air pollution through new perspectives. The conference was designed to help disseminate and integrate results from scientific studies that cut across the range of air pollution– and health-related disciplines of the source-to-health effects continuum. The conference addressed the science of air pollution and health within a multi pollutant framework, focusing on five key science areas—sources, atmospheric sciences, exposure, dose, and health effect. Eight key policy-relevant science questions that integrated across various parts of these science areas formed the basis of the meeting, and a ninth question addressed the policy implications of the findings.

In this thesis work we have done a research in urge of answering the above mentioned questions addressed in reference titled “Air pollution and health: bridging the gap from sources to health outcomes.” [1].

Dated 11.11.2010 and No. 602-24509 Mu la Governorship Approval Authority as a Chemical Engineer with the Provincial Environment and Forestry Directorate, on

GARD (Global Alliance Against Chronic Respiratory Diseases) Turkey (CAD) Chronic Airway Diseases was given to me. I am in charge due to the Provincial Executive Committee for the Prevention and Control Program to reason start up this thesis. To our knowledge there exists no thesis related with deep health issues exist until now related with Mu la city with our motivation and viewpoints. See: <http://tez2.yok.gov.tr/>.

Unfortunately during the thesis work there occurred some unwanted and unambiguous consequences of the work that will be a part of answers to the above questions; I have been withdrawn from my position in GARD Mu la (03.11.2011) without any reason, but still with some evidence based work will be discussed in Chapter 4, Results and Discussions with reference figure 4.28 as evidence.

This thesis is a case work about air “criteria pollutants” and health indicators to bridge the gap from sources to health outcomes in Mu la city. No expert or heuristic system, no statistical or mathematical evaluations, models or simulations could have done what an expert eye has done for this case. Machines can learn but they need a real expert to teach them. In this respect the huge raw data (health and air quality data, from Mu la, Yata an Air Quality Monitoring Network Centers (AQMNC)) with all supportive materials and methods by a near expert eye still in learning process is evaluated to open a new era in GARD work [1, 2].

1.2 PURPOSE OF STUDY

This thesis aims the evaluation of air criteria pollutants (SO_2 and PM_{10}), air pollution and Global Alliance against chronic Respiratory Diseases (GARD) work on the above mentioned background issues and motivations in Mu la city. In this thesis the data of GARD in Mu la city will be evaluated from the academical perspective to fullfill and bridge the gaps between the data available with the health outcomes based on scenarios and policies for Mu la.

CHAPTER 2

LITERATURE REVIEW

2.1 AIR POLLUTION AND CRITERIA AIR POLLUTANTS STANDARDS, LEGISLATIONS

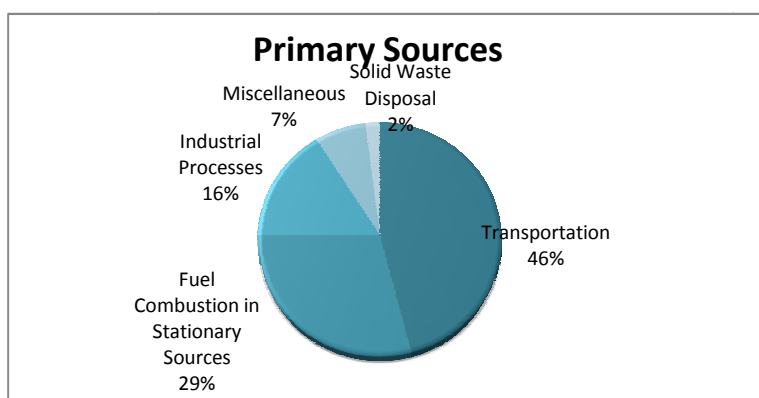
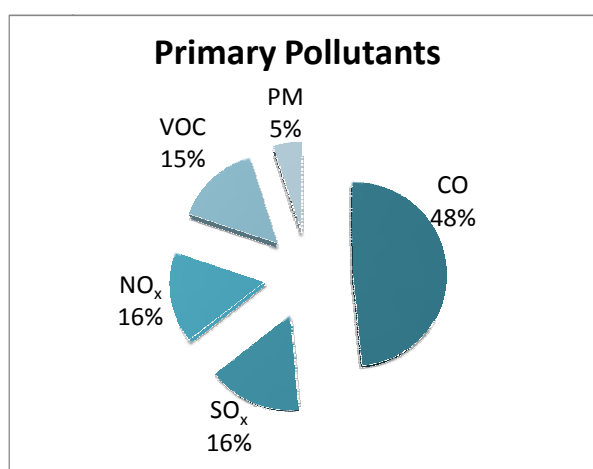
In this thesis the evaluation of air pollution will be health of population centered. Air pollution can have both acute and chronic health effects latter is cumulative and progressive. In this cumulative and progressive, chronic affect respect the literature will be evaluated with special emphasis for the studies of populations residing near point sources of air pollution.

Residential proximity to point sources of air pollution is a potential source of exposure to chemicals with various health impacts and known or suspected carcinogens. Fossil fuel-fired (i.e., coal, oil, natural gas) electrical power plants emit known or suspected carcinogens, including metals such as chromium and nickel, radionuclides such as radon and uranium, and PAH such as benzo[a]pyrene. Nonferrous metal smelters emit inorganic arsenic and other metals and SO₂ [2, 3]. Investigation of the etiologic mechanisms that might underlie air pollution's role in lung cancer occurrence, including its role in the association of lung cancer with preexisting respiratory disease led to the risk of lung cancer is increased among people with preexisting lung disease (e.g., chronic obstructive pulmonary disease and asthma) and among those who have exhibited accelerated rates of decline in pulmonary function. Long-term exposure to air pollution has also been associated with low levels of lung function and chronic respiratory symptoms in several cross-sectional and longitudinal studies of children. These reported associations suggest mechanisms other than, or in addition to, the direct initiation or promotion of lung tumors, by which prolonged exposure to air pollution could increase lung cancer risk [2, 3].

Table 2.1 Selected known carcinogens in ambient air [3].

Substance	Urban air	Rural air
Inorganic particulates (ng/m³)		
Arsenic	2-130	<0.5-5
Asbestos	10-100	-
Chromium	5-120	<1-10
Nickel	10-1000	<10
Radionuclides (Ci/m³)		
²¹⁰ Pb	1x10 ⁻¹⁵ -30x10 ⁻¹⁵	5.5x10 ⁻¹⁵ -10x10 ⁻¹⁵
²¹² Pb	0.1x10 ⁻¹⁵ -4x10 ⁻¹⁵	0.03x10 ⁻¹⁵ -0.06x10 ⁻¹⁵
²²² Rn	20x10 ⁻¹²⁵ -1.000x10 ⁻¹²	0.1x10 ⁻¹² -20x10 ⁻¹²
Gaseous and particulate organic species (ng/m³)		
Benzene	5-90	-
Benzo(a)pyrene	1-50	-
Benzene-soluble organics	1.000-2.000	200-300

Pollutants can be classified primary and secondary. Primary pollutants are substances directly emitted from a process. See: <http://apollo.lsc.vsc.edu/classes/met130/notes/chapter18/primary.html>.

**Figure 2.1** Primary sources for air pollutants.**Figure 2.2** Primary air pollutants.

Primary pollutants are

- Carbon monoxide (CO): odorless, colorless, poisonous gas, created by incomplete combustion (especially bad with older cars), generates headaches, drowsiness, fatigue, can result in death.
- Nitrogen oxides (NO_x): emitted directly by autos, industry.
- Sulfur Oxides (SO_x): SO₂ - sulfur dioxide, produced largely through coal burning, responsible for acid rain problem.
- Volatile organic compounds (VOC's): highly reactive organic compounds, release through incomplete combustion and industrial sources.
- Particulate Matter (PM): bad for lungs and respiratory system.

(See: <http://apollo.lsc.vsc.edu/classes/met130/notes/chapter18/primary.html>) [4].

Secondary pollutants occur as a result of a reaction of primary pollutants. These are not emitted directly to the air, water, or soil.

The best known of the secondary pollutants are nitrogen dioxide, ozone, hydrogen peroxide, aldehydes etc. (See: <http://science.jrank.org/pages/6028/Secondary-Pollutants.html>), [5].

Photochemical oxidants

- Photochemical oxidants: products of secondary atmospheric reactions driven by solar energy.
- One of the most important reactions involves formation of singlet (atomic) oxygen by splitting nitrogen dioxide (NO₂).
- Then the atomic oxygen reacts with another molecule of O₂ to make ozone (O₃).
- Ozone formed in the stratosphere provides a valuable shield for the biosphere by absorbing incoming ultraviolet radiation.
- In ambient air, however, O₃ is a strong oxidizing reagent and damages vegetation, building materials, and sensitive tissues.

See: http://zoology.muohio.edu/oris/cunn06/cs6_18.htm

Atmospheric oxidant production:

1. $\text{NO} + \text{VOC} \longrightarrow \text{NO}_2$
2. $\text{NO}_2 + \text{UV} \longrightarrow \text{NO} + \text{O}$
3. $\text{O} + \text{O}_2 \longrightarrow \text{O}_3$
4. $\text{NO}_2 + \text{VOC} \longrightarrow \text{PAN, etc. (peroxyacetyl nitrate)}$

Net results:

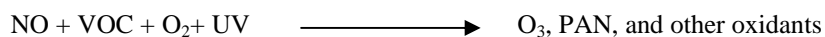


Figure 2.3 Secondary atmospheric oxidant productions by photochemical reactions.

2.1.1 Air Quality Standards and Regulations

The Air Quality Index (AQI) (also known as the Air Pollution Index (API) or Pollutant Standard Index (PSI) is a number used by government agencies to characterize the quality of the air at a given location.

See: http://en.wikipedia.org/wiki/Air_Quality_Index#Limitations_of_the_AQI.

2.1.2 API Mechanics

An individual score is assigned to the level of each pollutant and the final API is the highest of those 5 scores. The pollutants can be measured quite differently. SO_2 , NO_2 and PM_{10} concentrations are measured as average per day. CO and O_3 are more harmful and are measured as average per hour. The final API value is calculated per day.

The scale for each pollutant is non-linear, as is the final API score. Thus an API of 100 does not mean twice the pollution of API at 50, nor does it mean twice as harmful. While an API of 50 from day 1 to 182 and API of 100 from day 183 to 365 does provide an annual average of 75, it does *not* mean the pollution is acceptable even if the benchmark of 100 is deemed safe. This is because the benchmark is a 24 hour target. The annual average must match against the annual target. It is entirely possible to have safe air every day of the year but still fail the annual pollution benchmark.

Table 2.2 API and health implications indicating daily targets, with related colored air pollution levels as a sample work. See:

http://en.wikipedia.org/wiki/Air_Quality_Index#Limitations_of_the_AQI for further references.

API	Air Pollution Level	Health Implications
0 - 50	Excellent	No health implications
51 -100	Good	No health implications
101-150	Slightly Polluted	Slight irritations may occur, individuals with breathing or heart problems should reduce outdoor exercise.
151-200	Lightly Polluted	Slight irritations may occur, individuals with breathing or heart problems should reduce outdoor exercise.
201-250	Moderately Polluted	Healthy people will be noticeably affected. People with breathing or heart problems will experience reduced endurance in activities. These individuals and elders should remain indoors and restrict activities.
251-300	Heavily Polluted	Healthy people will be noticeably affected. People with breathing or heart problems will experience reduced endurance in activities. These individuals and elders should remain indoors and restrict activities.
300+	Severely Polluted	Healthy people will experience reduced endurance in activities. There may be strong irritations and symptoms and may trigger other illnesses. Elders and the sick should remain indoors and avoid exercise. Healthy individuals should avoid out door activities.

	SO2	NO2	CO	O3	PM10
Hava Kalitesi İndeksi	1 saatlik ortalama	24 saatlik ortalama	24 saatlik ortalama	1 saatlik ortalama	24 saatlik ortalama
	[$\mu\text{g}/\text{m}^3$]	[$\mu\text{g}/\text{m}^3$]	[mg/m^3]	[$\mu\text{g}/\text{m}^3$]	[$\mu\text{g}/\text{m}^3$]
1 (çok iyi)	0 -50	0 - 45	0 - 2,9	0 - 35	0 - 55
2 (iyi)	51-199	46 - 89	3,0 - 8,9	36 - 89	56-109
3 (yeterli)	200-399	90 - 179	9,0 - 15,9	90 - 179	110-159
4 (orta)	400-899	180 - 299	16 - 21,9	180 - 239	160-219
5 (kötü)	900-1499	300- 699	22,0 - 49,9	240 - 359	220-799
6 (çok kötü)	>1500	> 700	> 50,0	> 360	> 800

Figure 2.4 Air quality index/Air pollution index as accepted by Turkish authorities (See: <http://www.havaizleme.gov.tr/Default.ltr.aspx>).

The Clean Air Act, which was last amended in 1990, requires EPA to set National Ambient Air Quality Standards (40 CFR part 50) for pollutants considered harmful to

public health and the environment. The Clean Air Act established two types of national air quality standards. Primary standards set limits to protect public health, including the health of "sensitive" populations such as asthmatics, children, and the elderly. Secondary standards set limits to protect public welfare, including protection against decreased visibility, damage to animals, crops, vegetation, and buildings.

The EPA Office of Air Quality Planning and Standards (OAQPS) has set National Ambient Air Quality Standards for six principal pollutants are listed in Table 2.3 without any distortion of the original material. Units of measure for the standards are parts per million (ppm) by volume, parts per billion (ppb - 1 part in 1,000,000,000) by volume, milligrams per cubic meter of air (mg/m^3), and micrograms per cubic meter of air ($\mu\text{g}/\text{m}^3$) [5-9].

Table 2.3 Ambient Air Quality EPA Standards.

Pollutant	Primary Standards		Secondary Standards	
	Level	Averaging Time	Level	Averaging Time
Carbon Monoxide	9 ppm (10 mg/m^3)	8-hour ⁽¹⁾	None	
	35 ppm (40 mg/m^3)	1-hour ⁽¹⁾		
Lead	0.15 $\mu\text{g}/\text{m}^3$ ⁽²⁾	Rolling 3-Month Average	Same as Primary	
Nitrogen Dioxide	53 ppb ⁽³⁾	Annual (Arithmetic Average)	Same as Primary	
	100 ppb	1-hour ⁽⁴⁾	None	
Particulate Matter (PM₁₀)	150 $\mu\text{g}/\text{m}^3$	24-hour ⁽⁵⁾	Same as Primary	
Particulate Matter (PM_{2.5})	15.0 $\mu\text{g}/\text{m}^3$	Annual (Arithmetic Average) ⁽⁶⁾	Same as Primary	
	35 $\mu\text{g}/\text{m}^3$	24-hour ⁽⁷⁾	Same as Primary	
Ozone	0.075 ppm (2008 std)	8-hour ⁽⁸⁾	Same as Primary	
	0.08 ppm (1997 std)	8-hour ⁽⁹⁾	Same as Primary	
	0.12 ppm	1-hour ⁽¹⁰⁾	Same as Primary	
Sulfur Dioxide	0.03 ppm	Annual (Arithmetic Average)	0.5 ppm	3-hour ⁽¹⁾
	0.14 ppm	24-hour ⁽¹⁾		
	75 ppb ⁽¹¹⁾	1-hour	None	

(1) Not to be exceeded more than once per year.

(2) Final rule signed October 15, 2008. The 1978 lead standard (1.5 $\mu\text{g}/\text{m}^3$ as a quarterly average) remains in effect until one year after an area is designated for the 2008 standard, except that in areas designated nonattainment for the 1978 standard, the 1978 standard remains in effect until implementation plans to attain or maintain the 2008 standard are approved.

(3) The official level of the annual NO_2 standard is 0.053 ppm, equal to 53 ppb, which is shown here for the purpose of clearer comparison to the 1-hour standard.

(4) To attain this standard, the 3-year average of the 98th percentile of the daily maximum 1-hour average at each monitor within an area must not exceed 100 ppb (effective January 22, 2010).

(5) Not to be exceeded more than once per year on average over 3 years.

(6) To attain this standard, the 3-year average of the weighted annual mean PM_{2.5} concentrations from single or multiple community-oriented monitors must not exceed 15.0 µg/m³.

(7) To attain this standard, the 3-year average of the 98th percentile of 24-hour concentrations at each population-oriented monitor within an area must not exceed 35 µg/m³ (effective December 17, 2006).

(8) To attain this standard, the 3-year average of the fourth-highest daily maximum 8-hour average ozone concentrations measured at each monitor within an area over each year must not exceed 0.075 ppm. (effective May 27, 2008)

(9) (a) To attain this standard, the 3-year average of the fourth-highest daily maximum 8-hour average ozone concentrations measured at each monitor within an area over each year must not exceed 0.08 ppm.

(b) The 1997 standard—and the implementation rules for that standard—will remain in place for implementation purposes as EPA undertakes rulemaking to address the transition from the 1997 ozone standard to the 2008 ozone standard.

(c) EPA is in the process of reconsidering these standards (set in March 2008).

(10) (a) EPA revoked the 1-hour ozone standard in all areas, although some areas have continuing obligations under that standard ("anti-backsliding").

(b) The standard is attained when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 ppm is < 1.

(11) (a) Final rule signed June 2, 2010. To attain this standard, the 3-year average of the 99th percentile of the daily maximum 1-hour average at each monitor within an area must not exceed 75 ppb.

The U.S. Environmental Protection Agency (EPA) has established National Ambient Air Quality Standards (NAAQS) for six principal air pollutants "criteria pollutants": carbon monoxide (CO), lead (Pb), nitrogen dioxide (NO₂), particulate matter (PM) in two size ranges [< 2.5 µm (PM_{2.5}) and < 10 µm (PM₁₀)], ozone (O₃), and sulfur dioxide (SO₂).

2.1.3 EU Standards and Regulations

The European Union has also developed an extensive body of legislation which establishes health based standards and objectives for a number of pollutants in air. These standards and objectives are given as it is originally in the Table 2.4. These apply over differing periods of time because the observed health impacts associated with the various pollutants occur over different exposure times. See: <http://ec.europa.eu/environment/air/quality/standards.htm>, [5-9].

Table 2.4 Ambient Air Quality EU Standards.

Pollutant	Concentration	Averaging period	Legal nature	Permitted exceedences each year
Fine particles (PM2.5)	25 µg/m ³ ***	1 year	Target value enters into force 1.1.2010 Limit value enters into force 1.1.2015	n/a
Sulphur dioxide (SO₂)	350 µg/m ³	1 hour	Limit value enters into force 1.1.2005	24
	125 µg/m ³	24 hours	Limit value enters into force 1.1.2005	3
Nitrogen dioxide (NO₂)	200 µg/m ³	1 hour	Limit value enters into force 1.1.2010	18
	40 µg/m ³	1 year	Limit value enters into force 1.1.2010*	n/a
PM₁₀	50 µg/m ³	24 hours	Limit value enters into force 1.1.2005**	35
	40 µg/m ³	1 year	Limit value enters into force 1.1.2005**	n/a
Lead (Pb)	0.5 µg/m ³	1 year	Limit value enters into force 1.1.2005 (or 1.1.2010 in the immediate vicinity of specific, notified industrial sources; and a 1.0 µg/m ³ limit value applies from 1.1.2005 to 31.12.2009)	n/a
Carbon Monoxide (CO)	10 mg/m ³	Maximum daily 8 hour mean	Limit value enters into force 1.1.2005	n/a
Benzene	5 µg/m ³	1 year	Limit value enters into force 1.1.2010**	n/a
Ozone	120 µg/m ³	Maximum daily 8 hour mean	Target value enters into force 1.1.2010	25 days averaged over 3 years
Arsenic (As)	6 ng/m ³	1 year	Target value enters into force 1.1.2012	n/a
Cadmium (Cd)	5 ng/m ³	1 year	Target value enters into force 1.1.2012	n/a
Nickel (Ni)	20 ng/m ³	1 year	Target value enters into force 1.1.2012	n/a
Polycyclic Aromatic Hydrocarbons	1 ng/m ³ (expressed as concentration of Benzo(a)pyrene)	1 year	Target value enters into force 1.1.2012	n/a

*Under the new Directive the member State can apply for an extension of up to five years (i.e. maximum up to 2015) in a specific zone. Request is subject to assessment by the Commission. . In such cases within the time extension period the limit value applies at the level of the limit value + maximum margin of tolerance (48 µg/m³ for annual NO₂ limit value).

**Under the new Directive the Member State can apply for an extension until three years after the date of entry into force of the new Directive (i.e. May 2011) in a specific zone. Request is subject to assessment by the Commission. In such cases within the time extension period the limit value applies at the level of the limit value + maximum margin of tolerance (35 days at 75µg/m³ for daily PM₁₀ limit value, 48 µg/m³ for annual Pm₁₀ limit value).

***Standard introduced by the new [Directive](#).

Under EU law a limit value is legally binding from the date it enters into force subject to any exceedances permitted by the legislation. A target value is to be attained as far as possible by the attainment date and so is less strict than a limit value.

The new [Directive](#) is introducing additional PM2.5 objectives targeting the **exposure** of the population to fine particles. These objectives are set at the national level and are based on the average exposure indicator (AEI).

AEI is determined as a 3-year running annual mean PM2.5 concentration averaged over the selected monitoring stations in agglomerations and larger urban areas, set in urban background locations to best assess the PM2.5 exposure to the general population.

<i>Title</i>	<i>Metric</i>	<i>Averaging period</i>	<i>Legal nature</i>	<i>Permitted exceedences each year</i>
PM2.5 Exposure concentration obligation	20 µg/m ³ (AEI)	Based on 3 year average	Legally binding in 2015 (years 2013,2014,2015)	n/a
PM2.5 Exposure reduction target	Percentage reduction* + all measures to reach 18 µg/m ³ (AEI)	Based on 3 year average	Reduction to be attained where possible in 2020, determined on the basis of the value of exposure indicator in 2010	n/a

* Depending on the value of AEI in 2010, a percentage reduction requirement (0,10,15, or 20%) is set in the Directive. If AEI in 2010 is assessed to be over 22 µg/m³, all appropriate measures need to be taken to achieve 18 µg/m³ by 2020.

2.1.4 "Particulate Matter" PM₁₀

"Particulate Matter," also known as particle pollution or PM, is a complex mixture of extremely small particles and liquid droplets. Particle pollution is made up of a number of components, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil or dust particles [6-9].

The size of particles is directly linked to their potential for causing health problems (Figure 2.5). EPA is concerned about particles that are 10 micrometers in diameter or smaller because those are the particles that generally pass through the throat and nose and enter the lungs. Once inhaled, these particles can affect the heart and lungs and cause serious health effects. EPA groups particle pollution into two categories:

- "Inhalable coarse particles," such as those found near roadways and dusty industries, are larger than 2.5 micrometers and smaller than 10 micrometers in diameter.
- "Fine particles," such as those found in smoke and haze, are 2.5 micrometers in diameter and smaller. These particles can be directly emitted from sources such as forest fires, or they can form when gases emitted from power plants, industries and automobiles react in the air.

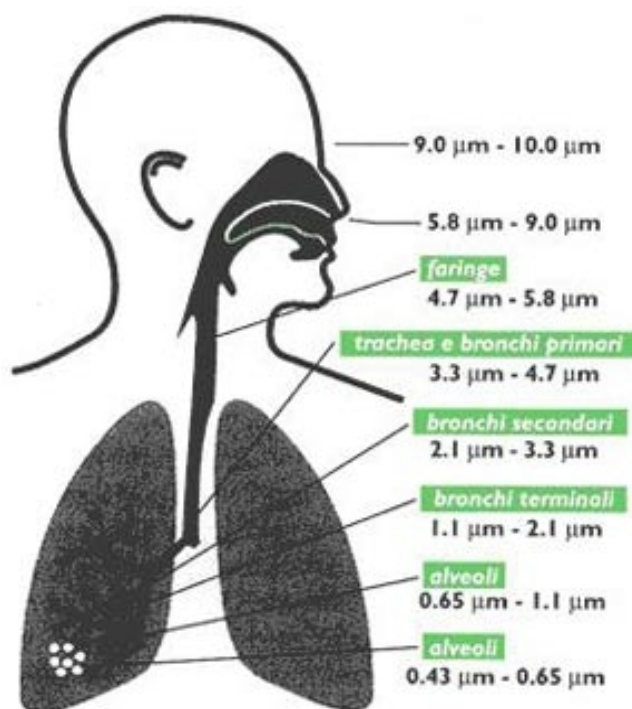


Figure 2.5 The comparative sizes for various particulate matter in respiratory system.

Non-mortality effects of PM_{10} are covered under two categories: chronic obstructive pulmonary diseases (COPD), and respiratory admissions to hospital.

The incidence of PM_{10} pollution also affects a number of chronic obstructive pulmonary diseases and allied conditions [6-9]. These include:

- bronchitis
- chronic bronchitis
- emphysema
- bronchiectasis
- extrinsic allergic alveolitis
- chronic airways obstruction.

2.1.5 Sulfur Dioxide SO_2

(SO_2) is one of a group of highly reactive gases known as “oxides of sulfur”. The largest sources of SO_2 emissions are from fossil fuel combustion at power plants (73%) and other industrial facilities (20%). Smaller sources of SO_2 emissions include industrial processes such as extracting metal from ore, and the burning of high sulfur

containing fuels by locomotives, large ships, and non-road equipment. SO₂ is linked with a number of adverse effects on the respiratory system.

Sulphur dioxide (SO₂) is a pungent gas that causes sore throat and eyes, and can have an effect on mortality. Sulphur oxides (primarily SO₂ and lesser quantities of sulphur trioxide) are gases formed by the oxidation of sulphur contaminants in fuel on combustion. SO₂ is a potent respiratory irritant, and has been associated with increased hospital admissions for respiratory and cardiovascular disease, as well as mortality. Asthmatics are a particularly susceptible group [7, 8].

2.2 CHRONIC RESPIRATORY DISEASES

Exposure and genetic predisposition should be assessed together to fill the gaps between exposure to air pollution and the expected health outcomes (Figures 2.6, 2.7). Table 2.5 lists examples of environmentally caused diseases. The two most important determinants in one's risk of developing an environmental disease are: a. exposure; and b. one's unique genetic make-up, usually neglected because of economic and lack of knowledge and practice. The first six examples in Table 2.5 represent large doses of an environmental agent that can be quite easily documented by a good medical history e.g. pack-years of smoking. The next three examples represent exposures to sun and to the outdoors and chemicals in the work place; quantitation in these cases is generally more difficult than the first six examples e.g. what is the actual number of days worked? Was the exposure identical for all of these days? The last four examples of Table 2.5 depict even more blurred cases in which a cause-and-effect correlation might be demonstrated by large human populations, but a cause-and-effect in a particular individual is often difficult to prove medically [6-12].

Figure 6 summarizes the whole pollution exposure related biomarker assessment levels to relate with any kind of pollutant exposure and health related issue. In this respect evaluation of air pollution and ambient air criteria pollutants and their health effects on various levels of health indicators: tissue, systemic, organism, population, community can be done [10-11].

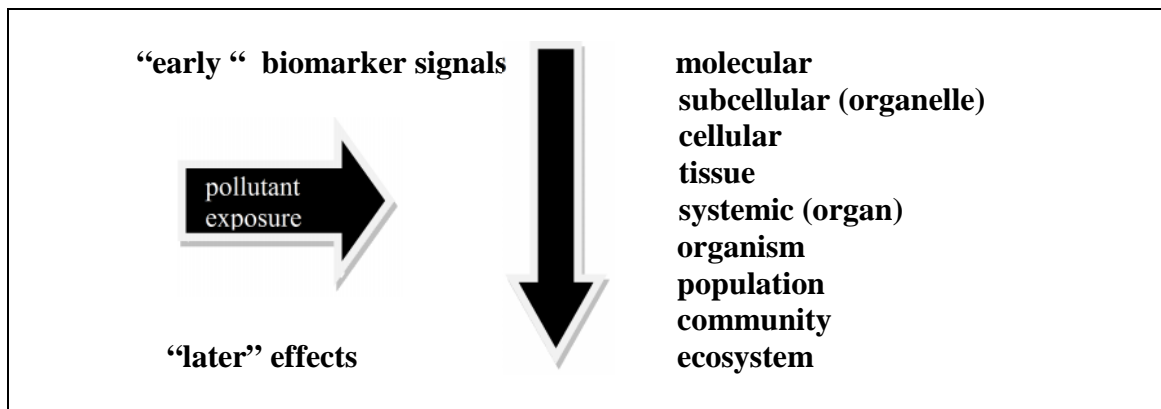


Figure 2.6 Schematic representation of the sequential order of responses to pollutant stress within a biological system [10, 11].

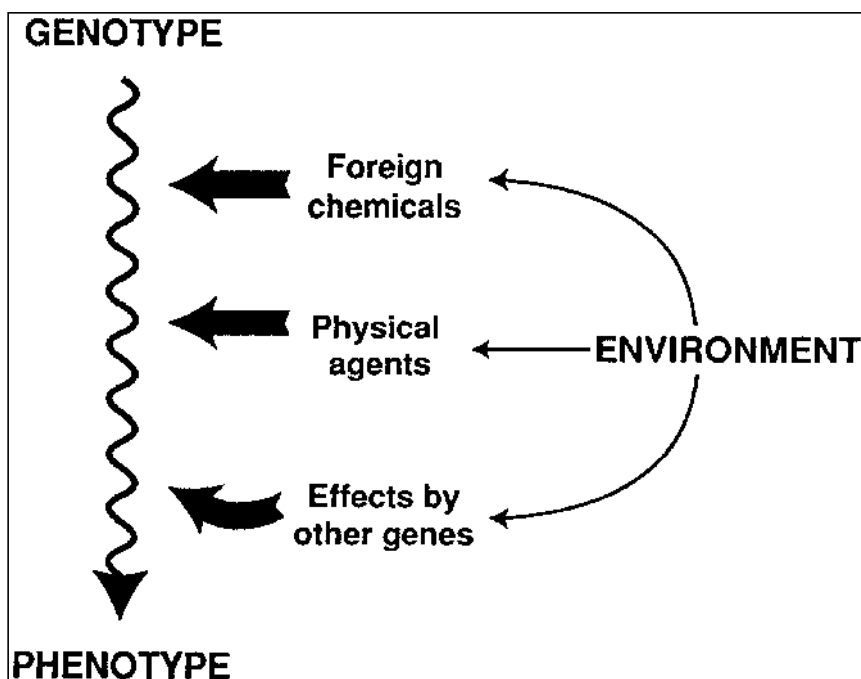


Figure 2.7 Environmental and epigenetic effects on an organism's genotype, by which the phenotype can be changed [11].

Table 2.5 Environmentally caused disease examples [11].

1. Bronchogenic carcinoma in cigarette smokers
2. Chronic bronchitis, emphysema, heavy wrinkles in cigarette smokers
3. Liver fibrosis, cirrhosis in alcoholics
4. Drug-related lupus syndrome in patients taking procainamide
5. Dangerously lowered blood pressure in patients taking debrisoquine or sparteine
6. Lung cancer in people exposed to radon
7. Malignant melanoma, other skin cancers, heat stroke, sunburn in persons exposed to excessive sunlight
8. Lung cancer in uranium mine workers
9. Chloracne, porphyria cutanea tarda in workers exposed to dioxin and other halogenated hydrocarbons
10. Ataxia, lowered mentality in persons exposed to high levels of lead
11. Increased risk of chronic myelogenous leukemia in workers exposed to benzene, of urinary bladder cancer in chemical dye workers
12. Asthma in children and adults exposed to indoor or outdoor air pollution
13. Toxicity or malignancy in persons living near a hazardous toxic waste dump site

Chronic respiratory diseases (CRDs) are firmly on the political and health-care agendas of the WHO 2008–2013 Action Plan for the Global Strategy for the Prevention and Control of Noncommunicable Diseases (NCD Action Plan), and that GARD is also in line with this plan. The Millennium Development Goals (MDGs) focused on maternal and perinatal conditions such as HIV/AIDS, tuberculosis (TB), malaria and other infectious diseases. However, in the next decade, the push will be to prioritize noncommunicable diseases (NCDs), making chronic disease the most important [12].

The Global Noncommunicable Disease Network (NCDnet) is a new network to combat NCDs in developing countries. Its goals are to raise awareness through advocacy, increase resource availability and catalyse country-level implementation [12].

The 193 Member States of WHO have been surveyed and analysed. The data collected has led to the first Global Status Report on NCDs that is released in early 2011 [12].

Aligning the GARD 2008–2013 Work Plan with the WHO 2008–2013 Action Plan for the Global Strategy for the Prevention and Control of Noncommunicable

Diseases (NCD Action Plan) is an important strategy of the works mentioned in the thesis [2-12].

2.2.1 Chronic Respiratory Diseases Worldwide

Map in Figure 2.8 shows the age-standardized estimate of mortality by chronic respiratory diseases per 100.000 people.

Chronic respiratory diseases are chronic diseases of the airways and other structures of the lung. Some of the most common are asthma, chronic obstructive pulmonary disease (COPD), respiratory allergies, occupational lung diseases and pulmonary hypertension.

Chronic respiratory diseases were responsible for 4.2 million deaths globally in 2008 and are on the increase. There is a great need for more and better care of these diseases [9, 13].

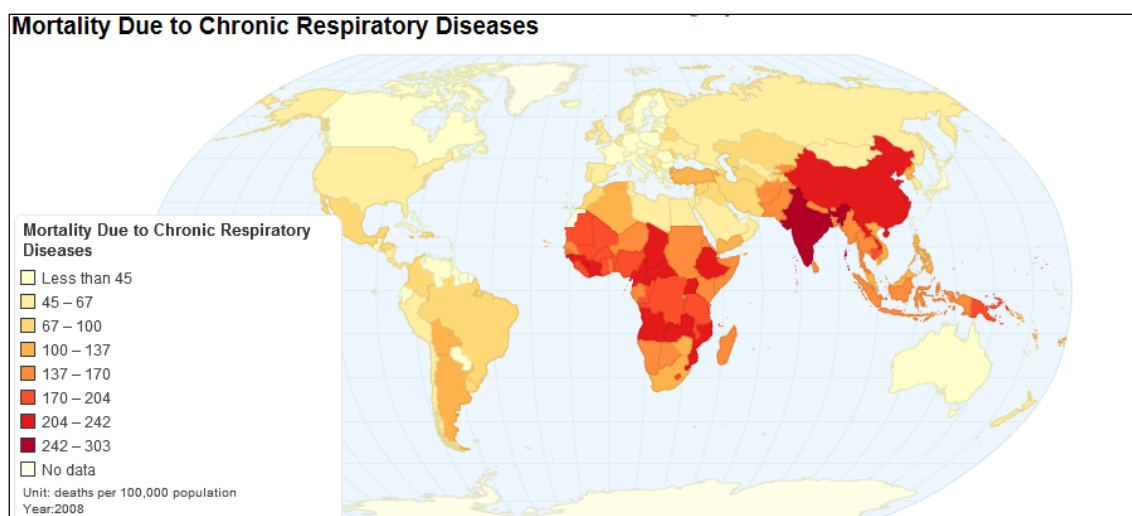


Figure 2.8 Mortality due to Chronic Respiratory Disases See: <http://chartsbin.com/view/3754>

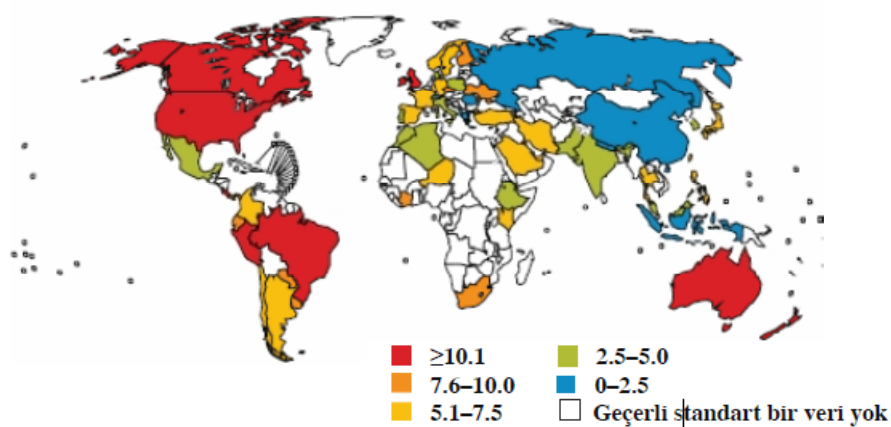


Figure 2.9 Asthma prevalence world map [14].

For cases in which chronic airways disease is suspected, a complete medical history should be taken and physical and laboratory examinations performed. There are several key points that need to be evaluated during this process such as characterizing the problem. Cough, wheeze, breathlessness, shortness of breath, chest tightness, watery runny nose, and itchy nose (including exercise-related symptoms) are common symptoms of chronic airways diseases among patients presenting in general practice. Once a presentation consistent with airways disease has been established, further characterize the problem in the following three ways:

- Establish Chronicity
- Exclude Non-Respiratory or Other Causes
- Exclude Infectious Diseases

2.2.2 Chronic Respiratory Disases in Turkey

In Turkey, chronic diseases accounted for 79 % of all deaths in 2002 (Figure 2.10) that is 437.000. Total deaths related to chronic disease in Turkey in 2002 is 346.000 and chronic respiratory diseases make up 6% of these.

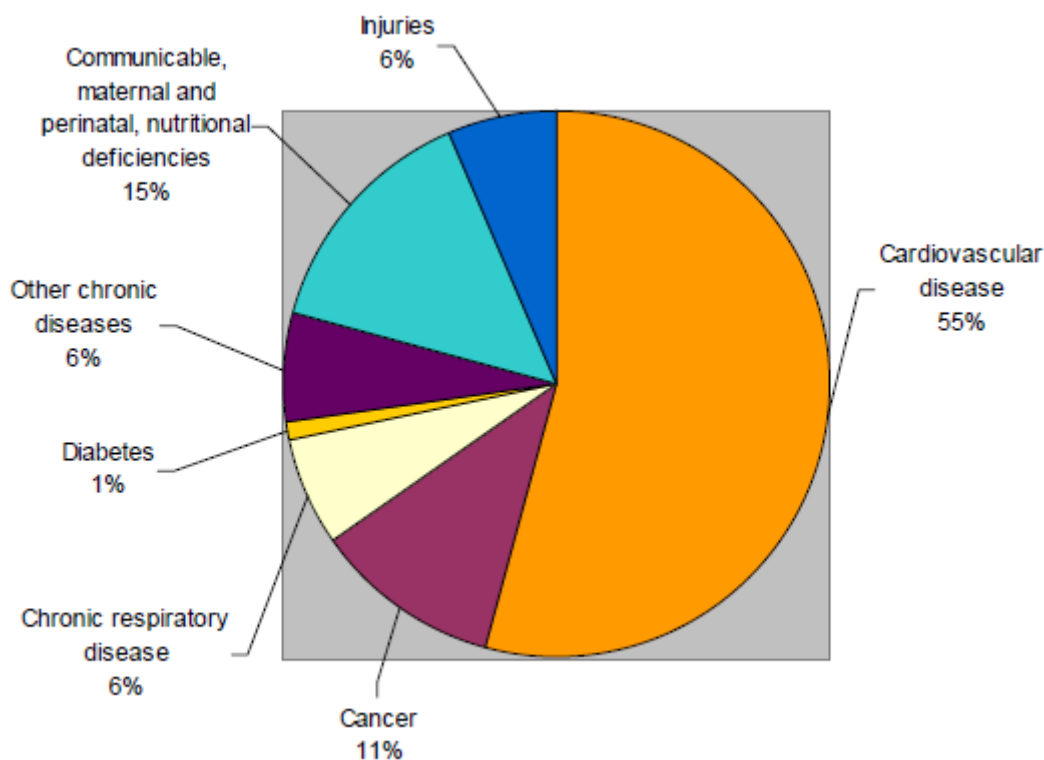


Figure 2.10 Deaths by cause in 2002 in Turkey.

See: http://www.who.int/chp/chronic_disease_report/turkey.pdf

2.2.2.1 Asthma

Asthma is the common chronic inflammatory disease of the airways characterized by variable and recurring symptoms, reversible airflow obstruction, and bronchospasm. Many environmental risk factors have been associated with asthma development and morbidity in children.

Maternal tobacco smoking during pregnancy and after delivery is associated with a greater risk of asthma-like symptoms, wheezing, and respiratory infections during childhood. Low air quality, from traffic pollution or high ozone levels, has been repeatedly associated with increased asthma morbidity and has a suggested association with asthma development that needs further research.

Recent studies show a relationship between exposure to air pollutants (e.g. from traffic) and childhood asthma. This research finds that both the occurrence of the disease and exacerbation of childhood asthma are affected by outdoor air pollutants. High levels of endotoxin exposure may contribute to asthma risk. As of 2009, 300 million people worldwide were affected by asthma leading to approximately 250,000

deaths per year. It is estimated that asthma has a 7-10 % prevalence worldwide. As of 1998, there was a great disparity in prevalence worldwide across the world (as high as a 20 to 60-fold difference), with a trend toward more developed and westernized countries having higher rates of asthma. Westernization however does not explain the entire difference in asthma prevalence between countries, and the disparities may also be affected by differences in genetic, social and environmental risk factors. See: <http://en.wikipedia.org/wiki/Asthma>.

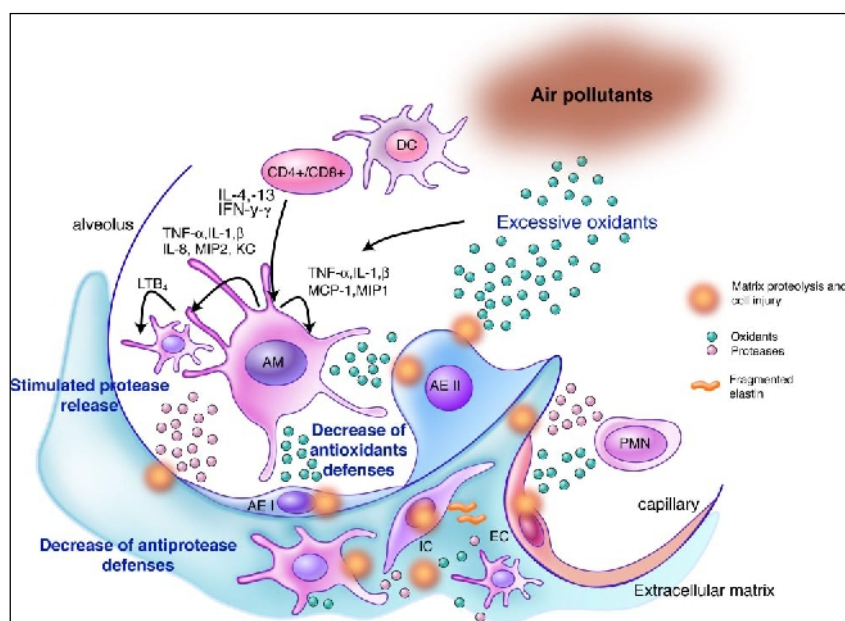


Figure 2.11 Inflammation, extracellular matrix proteolysis, and oxidative stress: the classical hypothesis. Air pollutants, including cigarette smoke, are a rich source of oxidants (i.e., O_2^- , NO), leading to the recruitment of macrophages and neutrophils with a series of complex mechanisms [15].

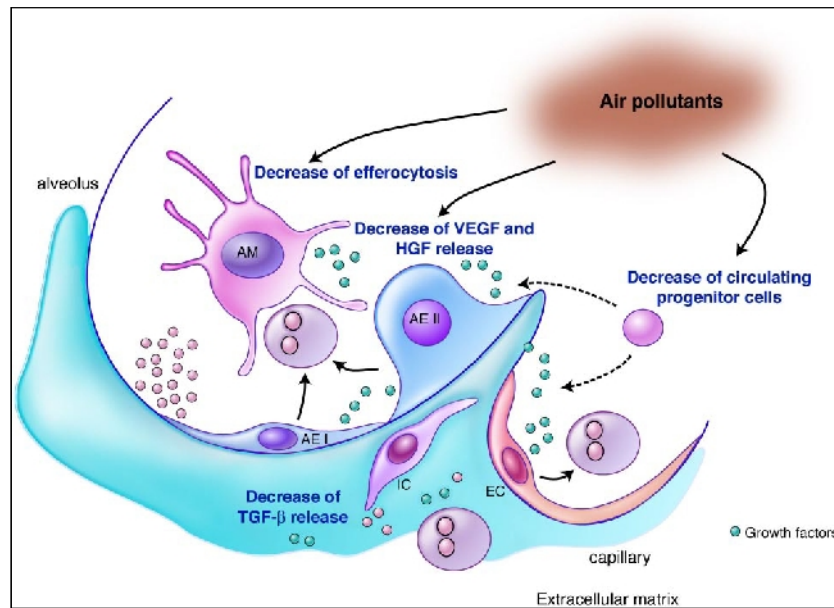


Figure 2.12 Breakdown of cellular and molecular maintenance program in emphysema. Air pollutants, including cigarette smoke, might inhibit the production and release of growth and survival factors from alveolar epithelial cells, endothelial cells, and interstitial cells resulting in apoptosis [15].

2.2.2.2 Chronic Obstructive Pulmonary Disease (COPD)

Chronic obstructive pulmonary diseases (COPD) are comprised of pulmonary emphysema, chronic bronchitis, and structural and inflammatory changes of small airways, is a leading cause of morbidity and mortality in the world. A better understanding of the pathobiology of COPD is critical for the developing of novel therapies, as the majority of patients with the disease have little therapeutic options at the present time. The pathobiology of COPD encompasses multiple injurious processes including inflammation (excessive or inappropriate innate and adaptive immunity), cellular apoptosis, altered cellular and molecular alveolar maintenance program, abnormal cell repair, extracellular matrix destruction (protease and anti-protease imbalance), and oxidative stress (oxidant and antioxidant imbalance). These processes are triggered by urban and rural air pollutants and active and/or passive cigarette smoke and modified by cellular senescence and infection. A series of receptor-mediated signal transduction pathways are activated by reactive oxygen species and tobacco components, resulting in impairment of a variety of cell signaling and cytokine networks, subsequently leading to chronic airway responses with mucus production, airway remodeling, and alveolar destruction. The authors Toshinori Yoshida and Rubin

M. Tudor provide an updated insight into the molecular and cellular pathobiology of COPD based on human and/or animal data [15].

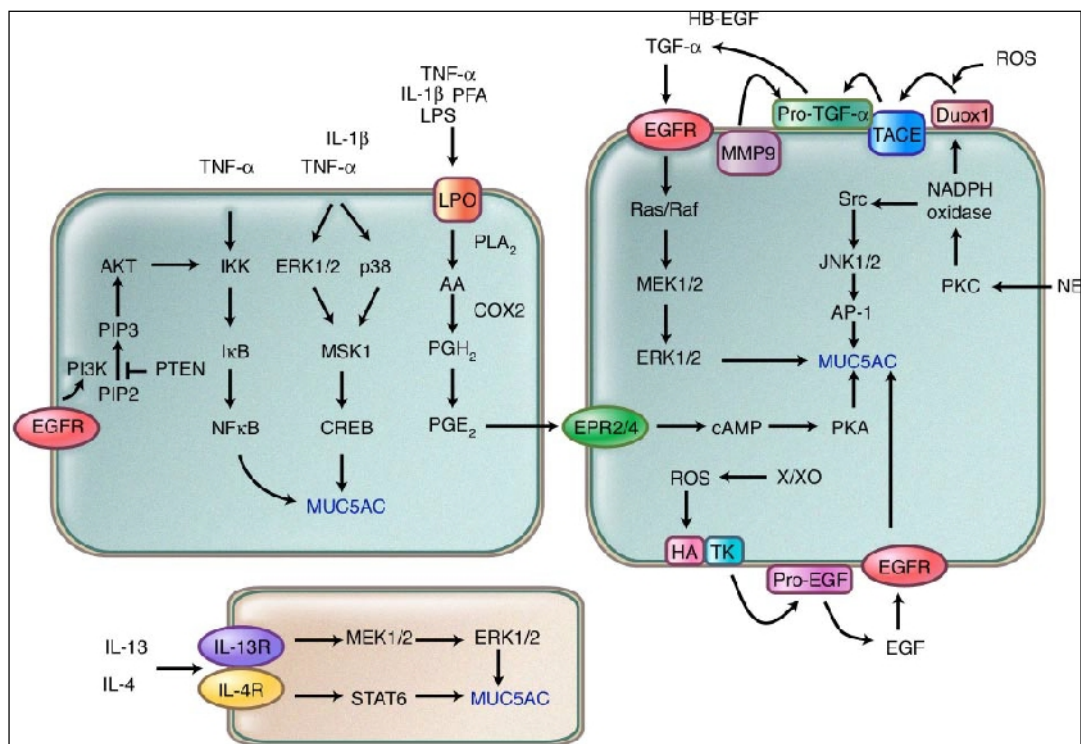


Figure 2.13 Oxidative stress responses in chronic obstructive pulmonary disease (COPD). Oxidative stress has a central role in the pathogenesis of COPD as oxidants mediate inflammation, extracellular degradation, and failure of alveolar maintenance [15].

Chronic obstructive pulmonary disease (COPD) is also known as chronic obstructive lung disease (COLD) is the co-occurrence of chronic bronchitis and emphysema, a pair of commonly co-existing diseases of the lungs in which the airways become narrowed. It is a progressive disease that makes it hard to breathe. "Progressive" means the disease gets worse over time. COPD can cause coughing that produces large amounts of mucus, wheezing, shortness of breath, chest tightness, and other symptoms. Cigarette smoking is the leading cause of COPD. Most people who have COPD smoke or used to smoke. Long-term exposure to other lung irritants, such as air pollution, chemical fumes, or dust, also may contribute to COPD. To understand COPD, it helps to understand how the lungs work. The air that you breathe goes down the windpipe into tubes in lungs called bronchial tubes or airways. Within the lungs, bronchial tubes branch into thousands of smaller, thinner tubes called bronchioles.

These tubes end in bunches of tiny round air sacs called alveoli. Small blood vessels called capillaries run through the walls of the air sacs. When air reaches the air sacs, the oxygen in the air passes through the air sac walls into the blood in the capillaries. At the same time, carbon dioxide moves from the capillaries into the air sacs. This process is called gas exchange. The airways and air sacs are elastic. In COPD, less air flows in and out of the airways because of one or more of the following:

- The airways and air sacs lose their elastic quality.
- The walls between many of the air sacs are destroyed.
- The walls of the airways become thick and inflamed.
- The airways make more mucus than usual, which tends to clog them.

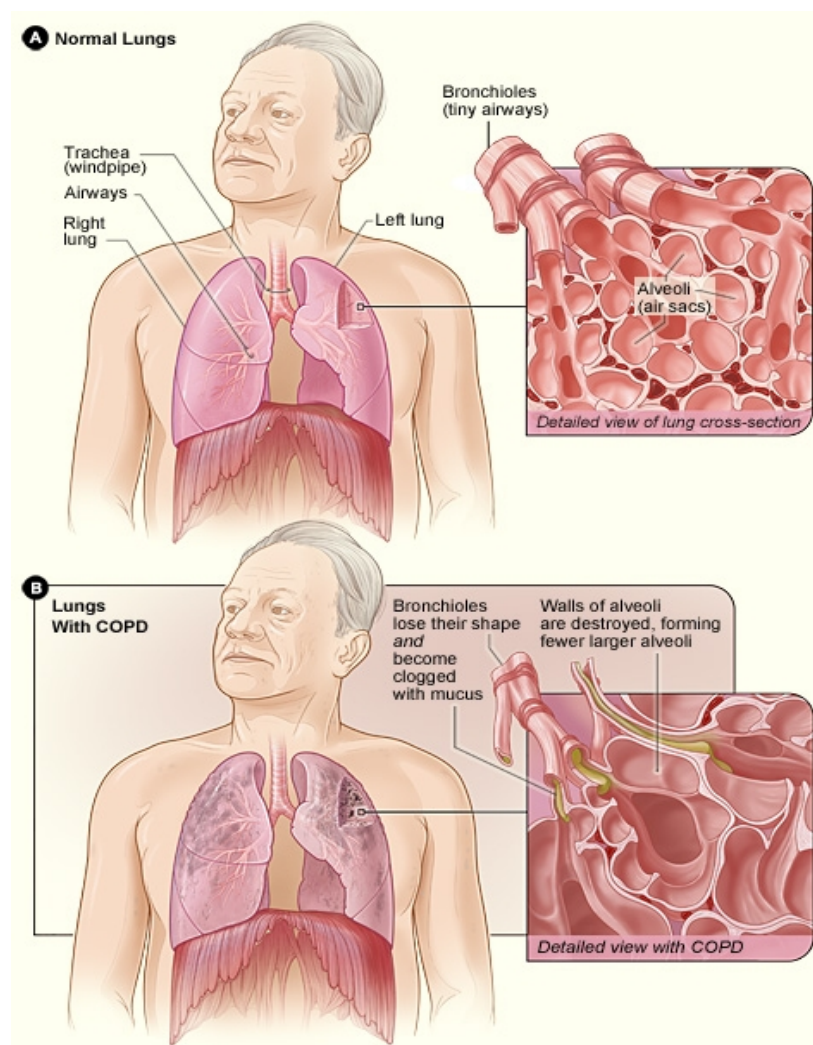


Figure 2.14 Normal lungs and lungs with COPD. A shows the location of the lungs and airways in the body. The inset image shows a detailed cross-section of the bronchioles and alveoli. Figure B shows lungs damaged by COPD. The inset image shows a detailed cross-section of the damaged bronchioles and alveolar walls. See: <http://www.nhlbi.nih.gov/>.

COPD occurs in 34 out of 1000 greater than 65 years old. In England, an estimated 842.100 of 50 million people have a diagnosis of COPD; approximately one person in 59 receiving a diagnosis of COPD at some point in their lives. In the United States, the prevalence of COPD is approximately 1 in 20 or 5 %, approximately 13,5 million people in USA, or possibly approximately 25 million people if undiagnosed cases are included [12-16].

2.2.2.3 Bronchiectasis

Bronchiectasis is a disease state defined by localized, irreversible dilation of part of the bronchial tree caused by destruction of the muscle and elastic tissue. It is classified as an obstructive lung disease, along with emphysema, bronchitis, asthma, and cystic fibrosis. Involved bronchi are dilated, inflamed, and easily collapsible, resulting in airflow obstruction and impaired clearance of secretions. Bronchiectasis is associated with a wide range of disorders, but it usually results from necrotizing bacterial infections, such as infections caused by the *Staphylococcus* or *Klebsiella* species or *Bordetella pertussis*. See: Gibraltar WebMD. <http://www.emedicine.com/radio/topic116.htm>. Retrieved 2007-06-22, [12-18].

2.2.2.4 Emphysema

Emphysema is a long-term, progressive disease of the lungs that primarily causes shortness of breath. In people with emphysema, the tissues necessary to support the physical shape and function of the lungs are destroyed. It is included in a group of diseases called chronic obstructive pulmonary disease or COPD (pulmonary refers to the lungs). Emphysema is called an obstructive lung disease because the destruction of lung tissue around smaller sacs, called alveoli, makes these air sacs unable to hold their functional shape upon exhalation. It is often caused by smoking or long-term exposure to air pollution.

Emphysema can be classified into primary and secondary. However, it is more commonly classified by location into panacinary and centroacinary (or panacinar and centriacinar, or centrilobular and panlobular).

- Panacinar (or panlobular) emphysema: The entire respiratory acinus, from respiratory bronchiole to alveoli, is expanded. Occurs more commonly in the lower lobes, especially basal segments, and anterior margins of the lungs.
- Centriacinar (or centrilobular) emphysema: The respiratory bronchiole (proximal and central part of the acinus) is expanded. The distal acinus or alveoli are unchanged. Occurs more commonly in the upper lobes.

The majority of all emphysema cases are caused by smoking tobacco. Emphysema cases that are caused by other etiologies are referred to as secondary emphysema.

Some types of emphysema are considered a normal part of aging and are found in the elderly whose lungs have deteriorated due to age. At about 20 years of age, people stop developing new alveoli tissue. In the years following the cessation of the development of new alveoli, lung tissue can start to deteriorate. This is a normal, natural part of aging in healthy people. Alveoli will die, the number of lung capillaries will decline and the elastin of the lungs will begin to break down causing a loss of pulmonary elasticity. As people age, they will also lose strength and mass in their chest muscles causing these muscles to become weaker. In addition, bones can start to deteriorate and a person's posture can change. Together, all of these age-related manifestations can cause the development of emphysema. Though not all elderly people will develop emphysema, they are all at risk of having decreased respiratory function.

Other causes of emphysema can be anything that causes the body to be unable to inhibit proteolytic enzymes in the lung. This could be exposure to air pollution, second hand smoke or other chemicals and toxins See: <http://en.wikipedia.org/wiki/Emphysema>, [12-18].

2.2.2.5 Respiratory Failure

The term respiratory failure, in medicine, is used to describe inadequate gas exchange by the respiratory system, with the result that arterial oxygen and/or carbon dioxide levels cannot be maintained within their normal ranges. A drop in blood oxygenation is known as hypoxemia; a rise in arterial carbon dioxide levels is called hypercapnia. The normal reference values are: oxygen PaO₂ greater than 80 mmHg

(11 kPa), and carbon dioxide PaCO_2 less than 45 mmHg (6.0 kPa). Classification into type I or type II relates to the absence or presence of hypercapnia respectively.

Type 1 respiratory failure is defined as hypoxemia without hypercapnia, and indeed the PaCO_2 may be normal or low. It is typically caused by a ventilation/perfusion (V/Q) mismatch; the volume of air flowing in and out of the lungs is not matched with the flow of blood to the lungs. The basic defect in type 1 respiratory failure is failure of oxygenation characterized by:

PaO_2	low (< 60 mmHg (8.0 kPa))
PaCO_2	Normal or low
PA-aO ₂	Increased

This type of respiratory failure is caused by conditions that affect oxygenation such as: Parenchymal disease (V/Q mismatch)

Diseases of vasculature and shunts: right-to-left shunt, pulmonary embolism, interstitial lung diseases: ARDS, pneumonia, emphysema. The basic defect in type 2 respiratory failure is characterized by:

PaO_2	Decreased
PaCO_2	Increased
PA-aO ₂	Normal
pH	Decreased

Type 2 respiratory failure is caused by increased airway resistance; both oxygen and carbon dioxide are affected. Defined as the build up of carbon dioxide levels (PaCO_2) that has been generated by the body. The underlying causes include: Reduced breathing effort (in the fatigued patient)

A decrease in the area of the lung available for gas exchange (such as in emphysema) [12-18].

2.2.2.6 Chronic Bronchitis

Chronic bronchitis, a type of chronic obstructive pulmonary disease, is characterized by the presence of a productive cough that lasts for three months or more per year for at least two years. Chronic bronchitis most often develops due to recurrent injury to the airways caused by inhaled irritants. Cigarette smoking is the most common cause, followed by air pollution and occupational exposure to irritants. Chronic bronchitis is treated symptomatically. Inflammation and edema of the respiratory epithelium may be reduced with inhaled corticosteroids. Wheezing and shortness of breath can be treated by reducing bronchospasm (reversible narrowing of smaller bronchi due to constriction of the smooth muscle) with bronchodilators such as inhaled -Adrenergic agonists (e.g., salbutamol) and inhaled anticholinergics (e.g., ipratropium bromide). Hypoxemia, too little oxygen in the blood, can be treated with supplemental oxygen. However, oxygen supplementation can result in decreased respiratory drive, leading to increased blood levels of carbon dioxide and subsequent respiratory acidosis [12-18].

2.2.2.7 Acute Bronchitis

Acute bronchitis is most often caused by viruses that infect the epithelium of the bronchi, resulting in inflammation and increased secretion of mucus. Cough, a common symptom of acute bronchitis, develops in an attempt to expel the excess mucus from the lungs. Other common symptoms include sore throat, runny nose, nasal congestion (coryza), low-grade fever, pleurisy, malaise, and the production of sputum.

Acute bronchitis often develops during the course of an upper respiratory infection (URI) such as the common cold or influenza See: <http://en.wikipedia.org/wiki/Bronchitis> - cite note-ID-0 About 90% of cases of acute bronchitis are caused by viruses, including rhinoviruses, adenoviruses, and influenza. Bacteria, including *Mycoplasma pneumoniae*, *Chlamydia pneumoniae*, and *Bordetella pertussis*, account for about 10% of cases [12-18].

Only about 5–10% of bronchitis cases are caused by a bacterial infection. Most cases of bronchitis are caused by a viral infection and are "self-limiting" and resolve themselves in a few weeks [16].

As most cases of acute bronchitis are caused by viruses, antibiotics should not be used, since they are effective only against bacteria. Using antibiotics in patients without bacterial infections promotes the development of antibiotic-resistant bacteria, which may lead to greater morbidity and mortality. Antibiotics should be prescribed only if examination of gram-stained sputum shows large numbers of bacteria present.

2.2.3 ICD-10

The International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10) is a coding of diseases and signs, symptoms, abnormal findings, complaints, social circumstances and external causes of injury or diseases, as classified by the World Health Organization (WHO) ICD-10 Chapter X: Diseases of the respiratory system.

(J40–J47) Chronic lower respiratory diseases

- (J40) [Bronchitis](#), not specified as acute or chronic
- (J41) [Simple](#) and [mucopurulentchronic bronchitis](#)
- (J42) Unspecified [chronic bronchitis](#)
 - Chronic [bronchitis](#) NOS
 - Chronic [tracheitis](#)
 - Chronic [tracheobronchitis](#)
- (J43) [Emphysema](#)
- (J44) Other [chronic obstructive pulmonary disease](#)
- (J45) [Asthma](#)
- (J46) [Status asthmaticus](#)
- (J47) [Bronchiectasis](#)

2.3 GARD

2.3.1 Gard Worldwide

GARD is a WHO alliance. It is a voluntary alliance and not a legal entity. The legal identity of GARD emanates from WHO. In view of the legal status of GARD, all GARD projects and activities are de facto WHO projects and activities and are therefore subject to WHO review and approval in line with WHO priorities and work plans. In this regard, GARD is an important tool in advocating for the implementation of WHO-approved guidelines and work plans [13, 14].

•All GARD activities should be in compliance with an integrated approach to NCDs and not focus on one disease only (e.g. allergic rhinitis). While WHO advocates for integrated national NCD policies and plans to ministries of health in low- and middle-income countries, GARD advocates for national plans on respiratory diseases, which is confusing to ministries of health in low- and middle-income countries.

•GARD activities and efforts should focus on (i) WHO priorities, such as primary care in low- and middle-income countries; (ii) prioritized NCDs, such as asthma and chronic obstructive pulmonary disease (COPD); and (iii) access to equitable care [12-14].

2.3.2 GARD in Turkey

GARD Turkey has more than 50 collaborating partners. Short-, medium- and long-term activities have been established within its six working groups. The working groups report to the Executive Committee, which meets once per year. The activities of GARD Turkey include:

- The Ministry of Health has been restructured and its action plan for 2010–2014 has been prepared.
- Public institutions, universities and nongovernmental organizations (NGOs) participated in the action plan.
- Sharing official information has motivated the groups. A questionnaire for COPD and asthma public awareness has been developed. Awareness and advocacy materials have been prepared, and integration with other advocacy plans is under way.
- Efforts are being made to reduce the modifiable risk factors for NCDs, such as tobacco and obesity.
- An expert panel drawn from a variety of national groups and programmes has prepared a report on the evaluation of indoor and outdoor pollution with respect to climate change.
- A workshop for education in primary care settings is being planned.
- Educators will focus on asthma, COPD, home care, pulmonary rehabilitation and tobacco control guidelines.

- Regarding the effective treatment of disease and the prevention of complications, a home-care workshop is being planned, along with integration with other NCD home-care and rehabilitation programmes.
- Reimbursement of the items for pulmonary rehabilitation and home care are being discussed.
- The monitoring group is trying to renovate the recording system and plan a new data collection system throughout the country.
- A manuscript detailing the accomplishments of GARD Turkey has been published and is available on MEDLINE [19].

2.3.3 GARD in Mu la

Mu la city has a GARD program and the updated information and activities can be followed on-line by the page in Figure 2.15. See: <http://www.muglasm.gov.tr/gard/gardanasayfa.asp>

MUĞLA İL SAĞLIK MÜDÜRLÜĞÜ
Türkiye Kronik Hava Yolu Hastalıkları Önleme ve Kontrol Programı

GARD Muğla

29.06.2009 tarih ve 2009/39 sayılı Genelge doğrultusunda Türkiye Kronik Hava Yolu Hastalıkları Önleme ve Kontrol Programı kapsamında İl GARD Kurulu (Global Alliance Against Chronic Respiratory Diseases- Kronik Solunum Hastalıklarına Karşı Küresel Birlik) oluşturuldu.

- Muğla GARD 1.Olağan Kurulu Toplantısı 18.01.2011 tarihinde Muğla ASH; Komuta Merkezi Toplantı Salonunda toplam 24 üyenin 22 sinin katılımıyla ilk kurul yapıldı, ilk kararlar alındı.
- Muğla GARD 1.Yürütme Kurulu Toplantısı 10.03.2011 tarihinde İl Sağlık Müdürlüğü Toplantı Salonunda toplam 10 üyeden oluşan yürütme kurulu üyelerinin 9 unun katılımıyla yapıldı ve ilk kararlar alındı.
- Muğla GARD 2.Olağan Kurulu Toplantısı 19.04.2011 tarihinde İl Sağlık Müdürlüğü Toplantı Salonunda 19 üye ile birlikte yapıldı ve ikinci kararları aldı. Toplantı Fotoğrafları için [tıklayınız...](#)

Figure 2.15 Mu la city's GARD program and the updated information and activities. See: <http://www.muglasm.gov.tr/gard/gardanasayfa.asp>

CHAPTER 3

RESEARCH MATERIALS, METHODOLOGY AND METHODS

3.1 DATA TYPES AND COLLECTION/MONITORING CRITERIA AND STAKEHOLDERS AND PLACES TO RETRIEVE DATA

3.1.1 Data Types and Data Retrieval Information

All the data used in this thesis work has been retrieved as being a member of the GARD in Mu la and as an authorized governmental official of Ministry of Environment with responsibilities and charges of a chemical engineer with the official correspondences/authority/permission.

3.1.1.1 Demographic and Geographic Data

Mu la's population is nearly 817.503 inhabitants including population in suburban areas, the details are given in Table 3.1 (Turkey's Statistical Yearbook, 2010).

Table 3.1 Mu la's population of counties and villages with 2010 data.

	Providence/City Center			Belde/Villages			Total		
	Total	Male	Female	Total	Male	Female	Total	Male	Female
Mu la									
Bodrum	33.258	17.357	15.901	91.562	47.205	44.357	124.820	64.562	60.258
Datça	10.450	5.408	5.042	6.025	3.043	2.982	16.475	8.451	8.024
Fethiye	77.237	38.788	38.449	111.022	56.305	54.717	188.259	95.093	93.166
Köyce iz	8.750	4.284	4.466	24.067	12.169	11.898	32.817	16.453	16.364
Marmaris	30.957	16.784	14.173	46.433	24.374	22.059	77.390	41.158	36.232
Milas	52.522	26.288	26.234	73.205	37.145	36.060	125.727	63.433	62.294
Merkez	60.066	30.334	29.732	34.894	17.367	17.527	94.960	47.701	47.259
Ula	5.683	2.850	2.833	18.066	9.062	9.004	23.749	11.912	11.837
Yata an	18.051	9.060	8.991	27.779	13.893	13.886	45.830	22.953	22.877
Dalaman	23.761	12.204	11.557	10.219	5.210	5.009	33.980	17.414	16.566
Ortaca	26.426	13.239	13.187	15.938	8.063	7.875	42.364	21.302	21.062
Kavakhdere	2.889	1.474	1.415	8.243	4.123	4.120	11.132	5.597	5.535
Total	350.050	178.070	171.980	467.453	237.959	229.494	817.503	416.029	401.474

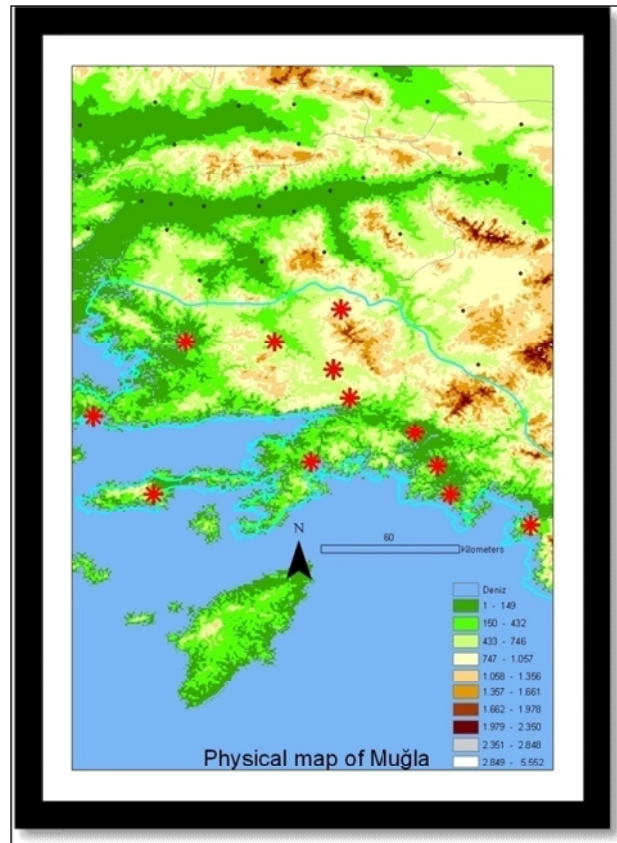


Figure 3.1 Physical map of Muğla showing the counties and elevations from sea level.

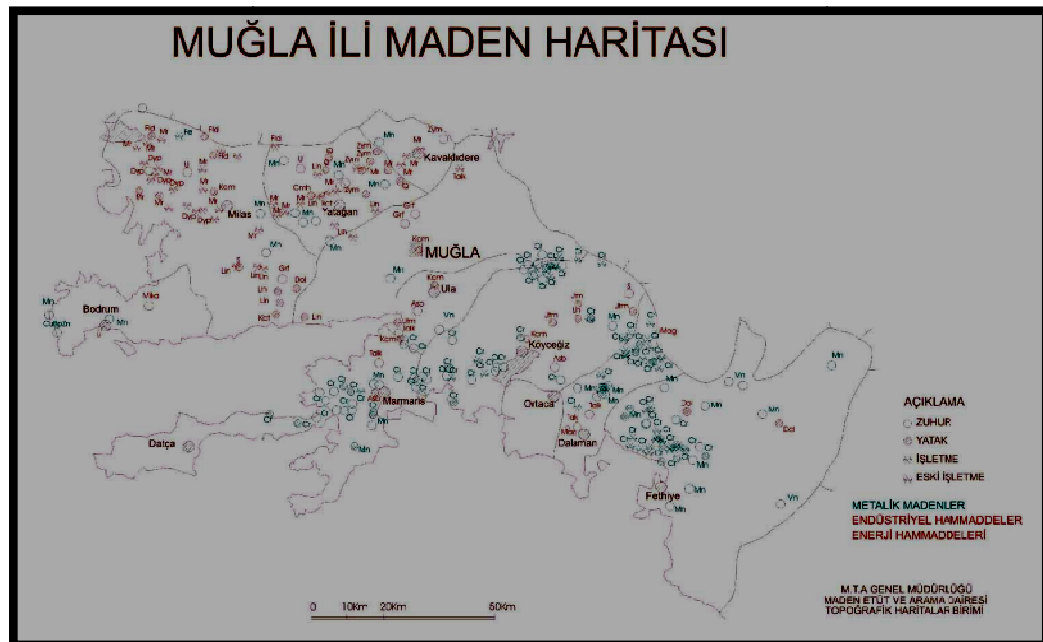


Figure 3.2 Natural ore reserves map of Muğla city. Wealth is an indication for the air pollution natural contributors.

3.1.1.2 Air Pollution Data

The air pollution data belongs to two criteria pollutants SO_2 and PM_{10} collected from two stations in Yata an and Mu la city center for three years (2008-2010). The photographs in Figures 3.3 and Figure 3.4 are during data controls at these two stations (Figures 3.5 and 3.6 respectively). Both SO_2 and PM_{10} values can be monitored from the internet and data can be retrieved from the address <http://www.havaizleme.gov.tr/Default.ltr.aspx> online.



Figure 3.3 SO_2 Monitoring Instrument (Brand: Horiba).



Figure 3.4 PM_{10} monitoring Instrument (Brand: MetOne BAM 1020).



Figure 3.5 Photograph from Mu la Air Quality Monitoring Network Center showing its location and neighbourhood.



Figure 3.6 Photographs from Yata an Air Quality Monitoring Network Center showing its location and neighbourhood.

Measurement data collected from stations established in computers belonging to the Ministry of Environment and Forestry, a private network (VPN) over the GSM Modems through of the Provincial Directorates and taken by the Ministry's Central Computer. Environmental Reference Laboratory for Air Quality Monitoring Network Center in the data center computer room is in the form of hourly averages. Data communication flow chart is given in Figure 3.7.

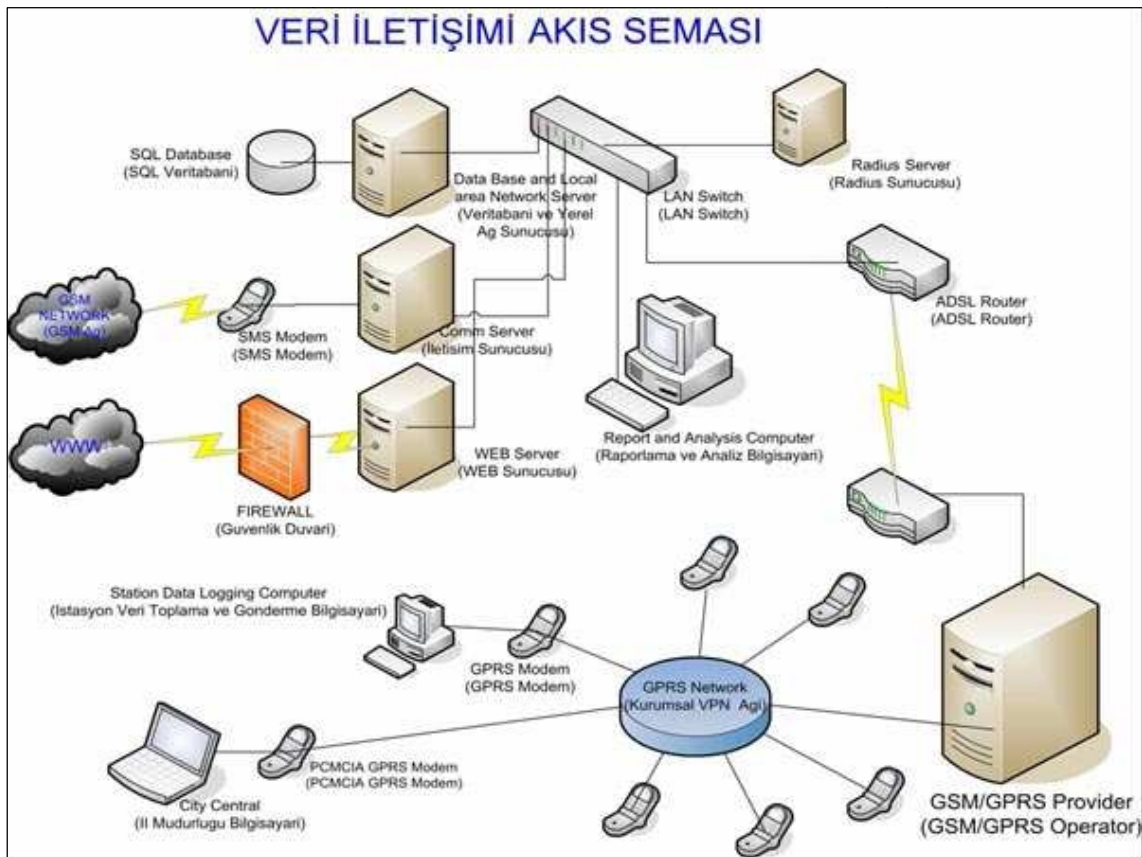


Figure 3.7 Data communication flow chart in the Air Quality Monitoring Network Centers in Mu la and Yata an.

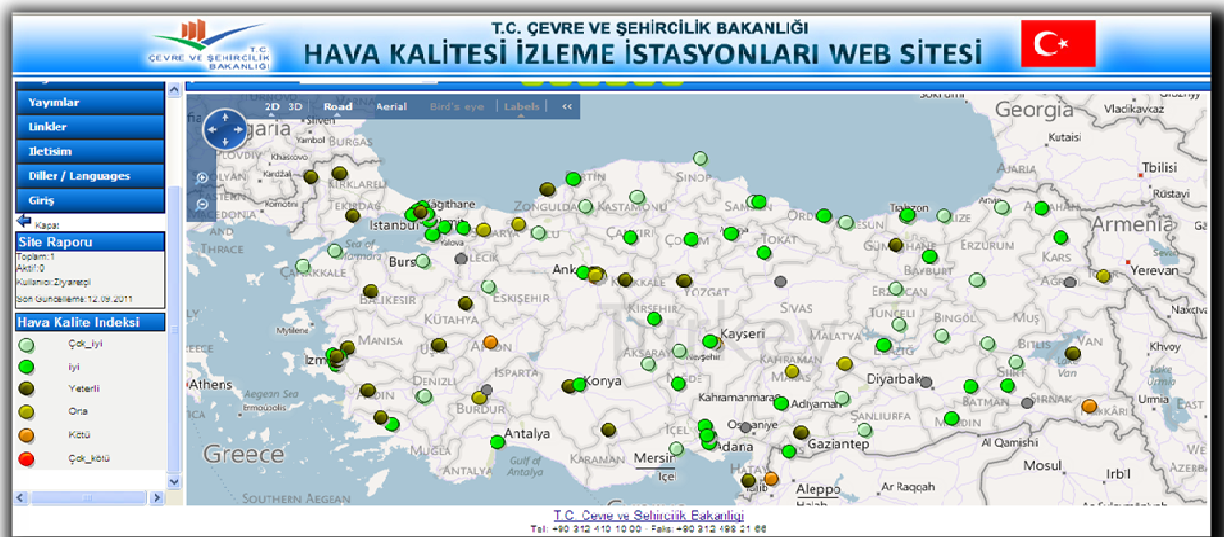


Figure 3.8 The on-line air quality and pollution data as transferred by the data communication system to follow the air quality for various parameters at different centers in Turkey via an interactive map. See: <http://www.havaizleme.gov.tr/Default.ltr.aspx>

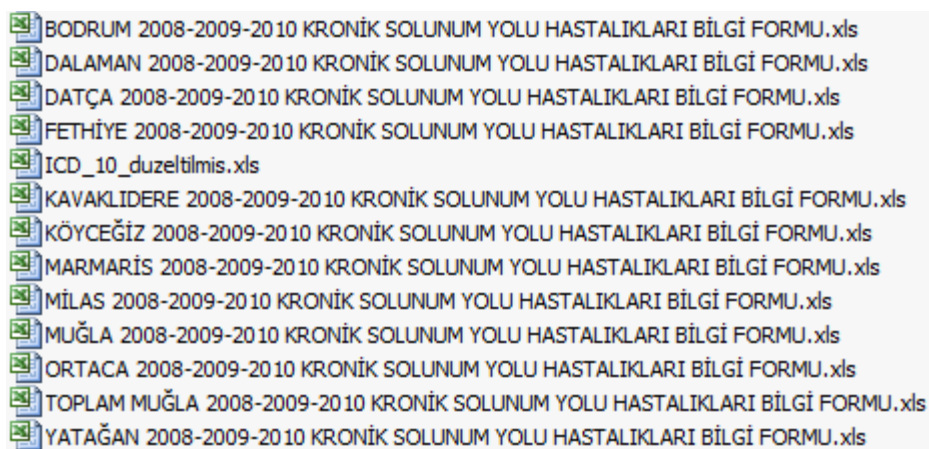
3.1.1.3 Sources of Air Pollution in Muğla City

Table 3.2 The number of mines in Muğla's counties.

Counties of Muğla	The number of mines producing air criteria pollutants especially PMs
Center of Muğla	46
Dalaman	5
Datça	2
Kavaklıdere	23
Köyceğiz	8
Ortaca	5
Ula	7
Yatağan	48
Milas	57
Fethiye	10
Bodrum	-
Marmaris	-

3.1.1.4 Chronical Disease Data in Muğla City

The disease raw data has been retrieved formally with the following documents and correspondence as excel files (Figure 3.9). The excel files are retrieved from Public hospitals' databases by formal communications given in Figures 3.10 and 3.11 (Private Hospital data is not available). Retrieved data contain the respiratory diseases sorted according to ICD-10 codes (given in part 2.2.3) where ICD-10 codes are retrieved from <http://www.muglasm.gov.tr/>. The raw patient data is given as excel format with the details of the patients given in Figure 3.9 b part.



a) The raw data retrieved as excel files.


1	Sıra No	D.Tarihi	Cinsiyet	Has.kodu	Açıklama(hastalık adı)
2	1	19.06.2000	KADIN	J45.1	Astım
3	2	07.12.2001	ERKEK	J45.1	Astım

1	SIRA	YAŞ		ICD-10 TANI KODU KHH ile ilişkili	AÇIKLAMA (HASTALIK)
2		(Doğum Tarihi)	CINSİYET		
3	1	1935	E	J-44	KOAH
4	2	1988	E	J-44	KOAH
5	3	1970	K	J-44	KOAH
6	4	1921	K	J-44	KOAH

28			ERKEK	J44	Kronik obstrüktif
27			ERKEK	J44	Kronik obstrüktif
28	84		ERKEK	J44	Kronik obstrüktif
29	54		ERKEK	J44	Kronik obstrüktif
1	SIRA NO	DOĞUM TARİHİ	CİNSİYET	ICD 10KODU	TANISI
2	1	01.02.1952	KADIN	J45	ASTİM
3	2	01.02.1934	ERKEK	J45	ASTİM
4	3	30.03.1925	KADIN	J45	ASTİM
5	4	01.06.1936	ERKEK	J44	KRONİK OBSTRÜKTİF AKCİĞER HASTALIĞI, DİĞER
6	5	12.08.1934	ERKEK	J44	KRONİK OBSTRÜKTİF AKCİĞER HASTALIĞI, DİĞER

b) The raw data retrieved from the state hospitals of each county with details.

Figure 3.9 The raw data retrieved as excel files (a) from the state hospitals of each county with details (b) in a non-standart format.



T.C.
MUĞLA VALİLİĞİ
İl Çevre ve Orman Müdürlüğü

20 Kasım 2011


Sayı : B.18.4.İÇÖ.48.02.00/125.02-703-1887
Konu: Bilgi Talebi

MUĞLA İL SAĞLIK MÜDÜRLÜĞÜNE
(Ruh Sağlığı ve Sosyal Hastalıklar Şubesi)

11.11.2010 tarih ve 602-24509 sayılı Valilik Oluru ile Müdürlüğümüzden Kimya Mühendisi Ekrem YAZICIOĞLU, GARD (Global Alliance Against Chronic Respiratory Diseases) il kurulunda görevlendirilmiştir.

Tarafınca yapılan "Hava Kirliliği ve Sağlık Bağlantısını Sorgulamada Muğla İli Kronik Hava Yolu Hastalıkları Örneği" başlıklı çalışma ile ilgili olarak, çalışmaya esasen Muğla iline ait Kronik Hava Yolu Hastalıklarının ilçeler bazında envanterine ihtiyaç olmuştur. İmkan ölçüsünde bu bilgilerin tarafımıza gönderilmesi gerekmektedir.

Bilgilerinizi ve gereğini arz ederim.



Mehmet SAHİN
İl Müdürü

Figure 3.10 Formal letters to retrieve data for chronic airway diseases from Mu la counties to be used in this thesis.



Figure 3.11 Formal letter of consent for the chonical airway diseases of Mu la data to be sent via e-mail to Ekrem Yazıcıo lu ekrem20@yahoo.com (me) to be used in this thesis.

3.1.2 Stakeholders

Stakeholders in GARD; their names, affiliations, addresses, web-pages and formal information necessary in the thesis given below.

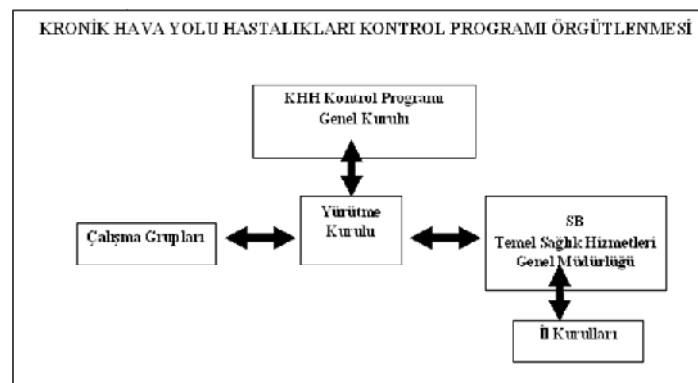


Figure 3.12 GARD organization scheme in Turkey and Mu la city.

According to the organization scheme in Figure 3.12 the below formal letters and documents (scanned) in Figure 3.13 are put in this materials and methods part to show the formal procedures to be assigned to my position in GARD Mu la.

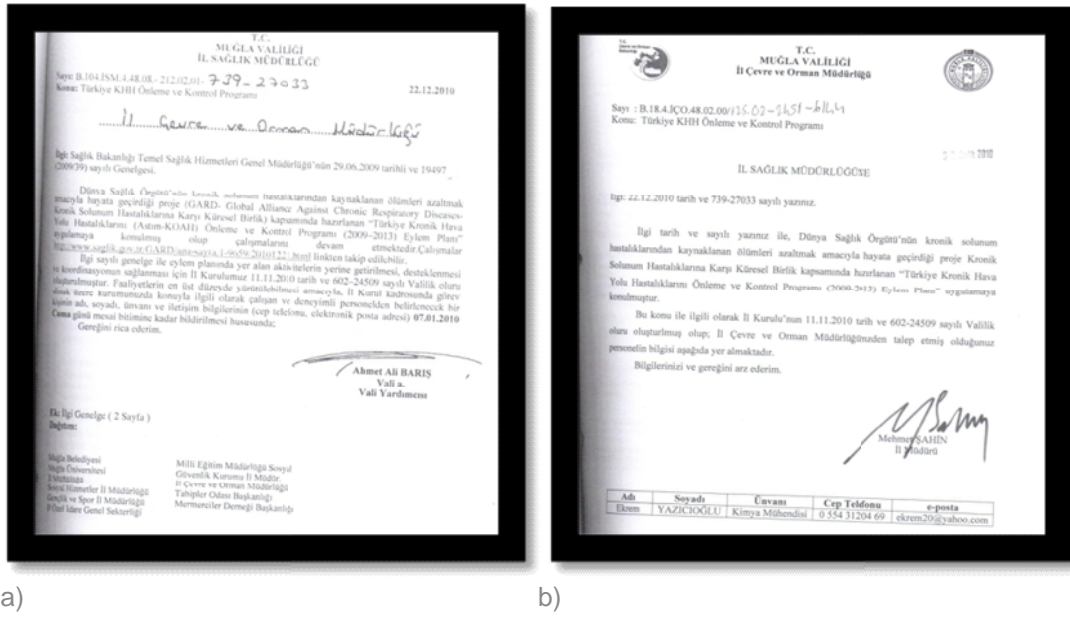


Figure 3.13 The scanned papers in (a) part is for the charging of a staff to be responsible as a qualified member of GARD in Mu la and in (b) part the formal reply of governmental authorities.

Assignment of Ekrem Yazıcıo lu (me) in GARD as a chemical engineer and the other stakeholders of the commissions that I have been selected to is given in Figures 3.14 and 3.15 with different information and contact details of the members.

TÜRKİYE KHH ÖNLEME VE KONTROL PROGRAMI İL YÜRÜTME KURULU						
SIRA	KURUMU	ADI-SOYADI	GOREVİ	İLETİŞİM BİLGİLERİ		İMZA
1	İL SAĞLIK MÜDÜRLÜĞÜ	DR. ENDER KASAP (BAŞKAN)	SAĞLIK MÜDÜR YARDIMCISI	0 532 253 96 47	dr.enderkasap@gmail.com	✓
2	İL SAĞLIK MÜDÜRLÜĞÜ	DR. ÖNER ONKAŞ	RUH SAĞ. VE SOS. HAST. ŞB. MD.	0 505 236 66 18	oneronkas@gmail.com	✓
3	İL SAĞLIK MÜDÜRLÜĞÜ	FİLİZ SALIŞ	SAĞLIK EĞİTİMİ ŞB. MD.	0 505 671 19 42	filizsalis@hotmail.com	✓
4	MUĞLA BELEDİYE BAŞKANLIĞI	DR. CAFER ŞAHİN	MUĞLA BELEDİYESİ SAĞLIK İŞLERİ MD.	0 533 594 99 46	dr_cafersahin@hotmail.com	✓
5	MUĞLA ÜNİVERSİTESİ	Yrd. Doç. Dr. METİN PİÇAKÇI EFE	HALK SAĞLIĞI UZMANI	0 536 256 34 49	mpicakcife@hotmail.com	✓
6	MİLLİ EĞİTİM MÜD.	GÜLİSTAN YILMAZOĞLU	SAĞLIK MESLEK LİSESİ MD.	0 532 407 40 00	gulyilmaz@hotmail.com	✓
7	İL ÇEVRE VE ÖRMAN MÜD.	EKREM YAZICIOĞLU	KİMYA MÜHENDİSİ	0 554 312 04 69	ekrem20@yahoo.com	✓
8	TABİPLER ODASI BAŞKANLIĞI	Uzm. Dr. SİBEL BARUT KESKİN	GOĞÜS HAST. UZMANI	0 535 278 93 89	drsibelkeskin@hotmail.com	✓
9	TÜRK TORAKS DERNEGİ MUĞLA TEMSİLCİSİ	Uzm. Dr. ALİ DİKER	GOĞÜS HAST. UZMANI	0 532 436 14 66	draldiker@hotmail.com	✓
10	FETHİYE DEVLET HASTANESİ	Uzm. Dr. MESUT KOSKU	GOĞÜS HAST. UZMANI	0 532 505 63 83	kosku@hotmail.com	✓

Figure 3.14 Member information and contact details in GARD Mu la.

BAŞKAN Dr. Ender KASAP II Sağlık Müdür Yardımcısı	Dr. V. Sumur GAZEZOĞLU II Sağlık Müdür Yardımcısı	Dr. Oner ONKAŞ Ruh Sağlığı ve Sosyal Hastalıklar Şube Müdürü
Dr. U Evrim KAZIL Gıda ve Çevre Kontrol Şube Müdürü	Filiz SALIŞ II Sağlık Müdürlüğü Eğitim Şube Müdürü	İncilay GEZGİN II Sağlık Müdürlüğü Bilgi İşlem ve İstatistik Şube Müdürü
Uzm. Dr. Sibel Barut KESKİN Muğla Tabip Odası Yürütme Kurulu Üyesi	Yrd. Doç. Dr. Metin PIÇAKÇIĞE Muğla Üniversitesi Tıp Fakültesi	Dr. Cafer ŞAHİN Muğla Belediyesi Sağlık İşleri Şube Müdürü
Uzm. Dr. Levent TETİK Muğla Devlet Hastanesi Göğüs Hastalıkları Uzmanı	Uzm. Dr. Ali DİKER Muğla Devlet Hastanesi Göğüs Hastalıkları Uzmanı	Uzm. Dr. Mesud KOSKU Fethiye Devlet Hastanesi Göğüs Hastalıkları Uzmanı
Uzm. Dr. Umit ÖZBEK Milas Devlet Hastanesi Göğüs Hastalıkları Uzmanı	Uzm. Dr. Mehmet OKUTUCU Muğla Verem Savaş Dispanseri Göğüs Hastalıkları Uzmanı	Faruk ÇELEBİ Millî Eğitim Müdürlüğü Eğitim Şube Müdürü
Ali KALAYCI II Emniyet Müdürlüğü	Kazım AŞLIK II Müftü Yardımcısı	Kim Müh. Ekrem VAZİCİOĞLU II Çevre ve Orman Müdürlüğü
Güven ÖZKAYA Memeciler Derneği Başkanı	Nilüfer KESER Sosyal Hizmetler II Müdürlüğü	Mehmet Yaşar USLU Gençlik ve Spor II Müdürlüğü
Zinnat ÇOBAN II Özel İdaresi Sağlık İşleri Müdürlüğü	Vet. Hek. Cuma KAHRAMANOĞLU II Tarım Müdürlüğü Hayvan Sağlığı Şube Müdürü	İsmail ÇOBANOĞLU Sosyal Güvenlik II Müdürlüğü

Figure 3.15 Member information and contact details in GARD Mu la.

As it is obvious in Figures 3.14 ve 3.15 there exists two different GARD commissions in Mu la city in both of which I have responsibilities. These two commissions are “Provincial Executive Committee and Provincial Executive Committee for the Prevention and Control Program”. Some details related with my responsibilities and fulfillments of these responsibilities are listed with details in Tables 3.4 and 3.5.

3.1.3 Data Providers of GARD

The data for GARD can be retrieved from the stakeholders given in Table 3.3 with thee columns to classify as the ones that is controlled under Ministry of Health, other governmental organizations and universities/non-governmental organizations.

Table 3.3 Data providers of GARD.

Republic of Turkey Ministry of Health	Other Governmental Organizations	Universities and Non-Governmental Organizations (NGOs)
Ministry of Health	Ministry of National Education	Turkish Thoracic Society
General Directorate of Primary Health Care Services	Ministry of Industry And Commerce	Turkish National Society Of Clinical
Department Of Non-Communicable Diseases And Chronic Conditions	Turkish Statistical Institute	Allergy And Immunology
Respiratory System Diseases Unit	Ministry of Interior	Turkish Pharmacists Association
Department of Health Promotion	Ministry of Labour and Social Security	The Society Of Public Health
General Directorate of Pharmaceuticals and Pharmacy	Social Security Institution	Turkish Society Of Family Practitioner
General Directorate of Curative Services	General Directorate For Occupational Health And Safety	The Society Of General Physicians
General Directorate of Health Education	World Health Organization (Regional Representative)	Medical Oncology Society
General Directorate of Maternal/Child Health and Family Planning	Ministry of Environment and Forestry	Turkish Medical Association
Personnel General Directorate	Ministry of Finance	Turkish Association Of Municipalities
Strategy Development Presidency	Ministry of Agriculture	
EU Coordination Department	General Directorate of Youth and Sport	
Information Processing Department	Tobacco, Tobacco Products and Alcoholic Beverages Regulatory Authority	
Cancer Control Department	Radio and Television Supreme Council	
Tuberculosis Control Department	General Directorate of Turkish Radio and Television Corporation	
Refik Saydam Hygiene Center Presidency	The Presidency of Religious Affairs	
School of Public Health	State Planning Organization	

3.1.3.1 Data Mining Tools

Excel filtering process has been used to help classifying the raw data for patients and the criteria pollutants.

3.1.3.2 Data Retrieval Addresses

The various data and information used in the thesis has been retrieved from the addresses in Table 3.4 freely without further permission unless otherwise mentioned.

Table 3.4 Data types and retrieval addresses.

Data types and descriptions	Reference addresses
PM ₁₀ -SO ₂ Values	http://www.havaizleme.gov.tr/
Patient Data	http://www.muglasm.gov.tr/
Enviromental Permits	http://eizin.cevreorman.gov.tr/Anasayfa.aspx?sflang=tr
Meteorology Data	http://dmi.gov.tr/tahmin/il-ve-ilceler.aspx?m=MUGLA
Provincial Environment and Forestry Directorate	http://mugla.cevreorman.gov.tr/Mugla/AnaSayfa.aspx?sflang=tr
Ministry of Environment and Forestry	http://www.ormansu.gov.tr/COB/AnaSayfa.aspx?sflang=tr
Provincial Healty Directorate	http://www.muglasm.gov.tr/pages.asp?Pagem=Kronik%20Hava%20Yolu%20Hastal%FDklar%FDn%FD%20%D6nl%eme%20ve%20Kontrol%20Program%FD&cat_id=4&cat2_id=12&wid=43
Mines in Mu la's counties	http://www.mutso.org.tr/default.asp (Mu la Chamber of Commerce and Industry) http://mitso.org.tr/default.asp (Milas Chamber of Commerce and Industry) http://www.fto.org.tr/index.aspx (Fethiye Chamber of Commerce and Industry) http://www.mto.org.tr/ (Marmaris Chamber of Commerce and Industry) http://www.bodto.org.tr/ (Bodrum Chamber of Commerce and Industry)

3.1.4 Conferences, Meetings, Briefings, Seminars Attended, Given and Followed

During and in preparation to this thesis work various important conferences, meetings, briefings and seminars have been attended, given and followed. Below in Table 3.5 is the list of most important ones that there exist a web address for or

published in a proceeding. Others are given as personel communication type of data or mentioned where needed throughout the thesis.

Table 3.5 Conferences, seminars given and attended and news followed with addresses.

Subject Title	Reference addresses
TRAF K KAYNAKLI EM SYON HESABINDA KAR ILA ILAN GÜÇLÜKLER, Ekrem YAZICIO LU, Ferhat KARACA, 01/10/2010 IV. HAVA K RL L VE KONTROLÜ SEMPOZYUMU, ODTÜ KKM – ANKARA, 25-27 EK M 2010	http://www.hkk2010.metu.edu.tr/
Seminar	http://www.mugla-bld.gov.tr/haberler/haberler2011expanded.html#242
Mu la GARD Commission	http://www.muglasm.gov.tr/pages.asp?Pagem=Kronik%20Hava%20Yolu%20Hastal%FDklar%FDn%FD%20%D6nleme%20ve%20Kontrol%20Program%FD&cat_id=4&cat2_id=12&wid=43
Control News in Mu la	http://www.bodrubaskisi.com/haber/index.php/hava-kirliligi-kontrol-altinda
Control News in Mu la	http://www.mugladevrim.com.tr/index.php?option=com_content&view=article&id=8962:kaloriferilere-eitim-semineri-&catid=1:son-haberler
Control News in Mu la	http://www.mugladevrim.com.tr/index.php?option=com_content&view=article&id=5992:mulada-kalorifer-daireleri-denetlendi
Mu la GARD Commission	http://www.muglasm.gov.tr/pages.asp?Pagem=Kronik%20Hava%20Yolu%20Hastal%FDklar%FDn%FD%20%D6nleme%20ve%20Kontrol%20Program%FD&cat_id=4&cat2_id=12&wid=43

3.1.5 Questions To Be Answered To Bridge The Gap From Sources To Health Outcomes [1].

1. How does our understanding of the health effects of air pollutants (singly or in mixtures) help identify pollutants that can be linked to sources the control of which would provide maximal health benefits? (overarching theme)
2. How reliable are methods (measurements and models) and approaches (epidemiological and toxicological) for studying and quantifying the links between air pollutants (species and or sources) and adverse health effects?
3. How do relevant pollutant properties vary in space and time from sources and in ambient air? What are the implications of these variations for population exposure?
4. What advances have been made in understanding the relationships between exposure, both spatially and temporally, and estimates of dose that tie to health outcomes?
5. Are patterns emerging that relate component(s) of air pollution and/or source types to mechanisms? What is the status of identifying and measuring biomarkers of exposure and/or adverse health effects from air pollution?
6. Who are the susceptible populations? What drives different susceptibilities to the same or different air pollutants? Are there susceptibility traits associated with specific health outcomes that are common among the subpopulations?
7. What roles do confounding or other factors have in increasing, decreasing, or obscuring attribution of the true health effects from ambient air pollutants?
8. Do actions taken to improve air quality result in reduced ambient concentrations of relevant pollutants, exposure, and health effects? Have we encountered unintended consequences?
9. What are the policy implications of our improved understanding of the source to health effect paradigm?

3.2 METHODS

3.2.1 GIS Evaluations of Various Health and Pollution Data Using ARCVIEW 10 Program

ArcView is a desktop GIS software that provides geographic data visualization, mapping, management, and analysis capabilities along with the ability to create and edit data.

3.2.2 Statistical and Graphical Evaluations of Various Health, Pollution and Epidemiological Data

Microsoft Excel basic tools for statistical calculations and graphical evaluations have been used to evaluate health data in calculating the prevalence and population-adjusted prevalence of the most frequent chronic airway diseases in Mu la city given in Table 4.3 and pollution data in calculating the means to show the cumulative values for SO₂ and PM₁₀ and the limit values and standards of EU, EPA and Turkish Ministry of Health.

3.2.3 Methods to Answer Some of the Questions in 3.1.5

1. How does our understanding of the health effects of air pollutants (singly or in mixtures) help identify pollutants that can be linked to sources the control of which would provide maximal health benefits? (overarching theme).
2. How reliable are methods (measurements and models) and approaches (epidemiological and toxicological) for studying and quantifying the links between air pollutants (species and or sources) and adverse health effects?
 - PM_{2.5} and less sized detection filters should be used (Figure: 2.5) [2, 7, 20].
 - Data collected should be in the same formats and units with the universal data [8, 9].
3. How do relevant pollutant properties vary in space and time from sources and in ambient air? What are the implications of these variations for population exposure?
4. What advances have been made in understanding the relationships between exposure, both spatially and temporally, and estimates of dose that tie to health outcomes?
5. Are patterns emerging that relate component(s) of air pollution and/or source types to mechanisms? What is the status of identifying and measuring biomarkers of exposure and/or adverse health effects from air pollution?

6. Who are the susceptible populations? What drives different susceptibilities to the same or different air pollutants? Are there susceptibility traits associated with specific health outcomes that are common among the subpopulations?
 - Susceptibility criteria should be evaluated for each patient according to medical history, anamnesis.
 - Detailed pedigrees should be drawn for each patient to assess genetic and environmental contribution to the disease.
 - Susceptibility genes such as GSTs, CYPs, NATs should be genotyped for personalized therapies [11, 21].
 - Drug-metabolizing enzyme polymorphisms; Phase I and Phase II drug metabolism; NAT2; G6PD; Exposure assessment; TPMT, CYP2D6, NQO1 and PON genes can be considered more specifically [11].
 - The patients' prognosis should be followed by routine and detailed progress and course reportings done by phone calls.

7. What roles do confounding or other factors have in increasing, decreasing, or obscuring attribution of the true health effects from ambient air pollutants?
 - Need for more demographic information to understand the real contribution of air pollutant exposure on the disease prevalence.

8. Do actions taken to improve air quality result in reduced ambient concentrations of relevant pollutants, exposure, and health effects? Have we encountered unintended consequences?

9. What are the policy implications of our improved understanding of the source to health effect paradigm?

CHAPTER 4

RESULTS AND DISCUSSIONS

4.1 ANALYSIS RESULTS AND DISCUSSION OF MU LA'S AIR POLLUTION DATA.

Mu la's air pollution data collected from Yata an and Mu la stations are retrieved, examined and filtered according to many confounding factors discussed under related titles are converted finally to the information available in Tables 4.1 and 4.2 and Figures 4.1 and 4.2 for the years 2008-2010. Many technical details are given as a summary in these tables and figures that can have an effect on our discussions. Numbers of measurements for both stations Yata an and Mu la and for both PM₁₀ and SO₂ in 2008 are very low, that is because the stations started to take data after that time on.

Table 4.1 PM₁₀ and SO₂ data statistics for 3 years (2008-2010) for Yata an Station.

YATA AN STATION

Year/Parameter	Measured Average Values		Number of measurements	
	PM ₁₀ 24H (µg/m ³)	SO ₂ 24H (µg/m ³)	PM ₁₀ 24H	SO ₂ 24H
2008	72	28	29	29
2009	59	19	333	333
2010	59	22	282	282

Table 4.2 PM₁₀ and SO₂ data statistics for 3 years (2008-2010) for Mu la Station.

MU LA STATION

Year/Parameter	Measured Average Values		Number of Measurements	
	SO ₂ 24H (µg/m ³)	PM ₁₀ 24H (µg/m ³)	SO ₂ 24H	PM ₁₀ 24H
2008	75	72	13	12
2009	42	69	255	255
2010	60	71	292	292

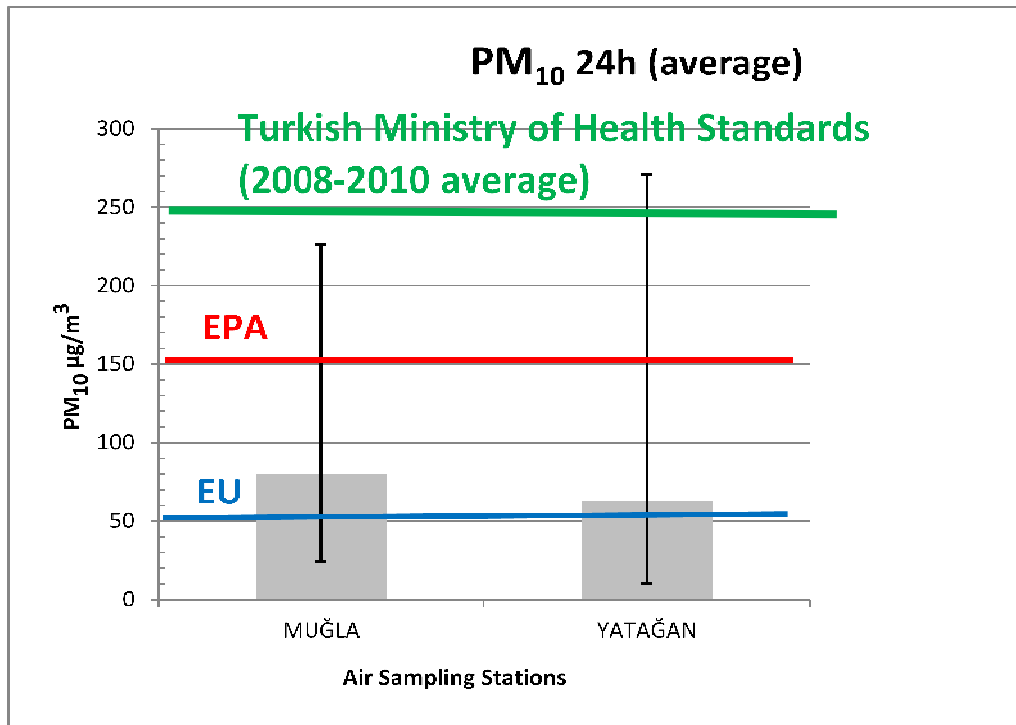


Figure 4.1 PM₁₀ 24h data averages of Mu la and Yata an air sampling stations for years 2008-2009-2010 showing the smallest and the highest values recorded for the two stations and the values that exceed EPA, EU and Turkish standards.

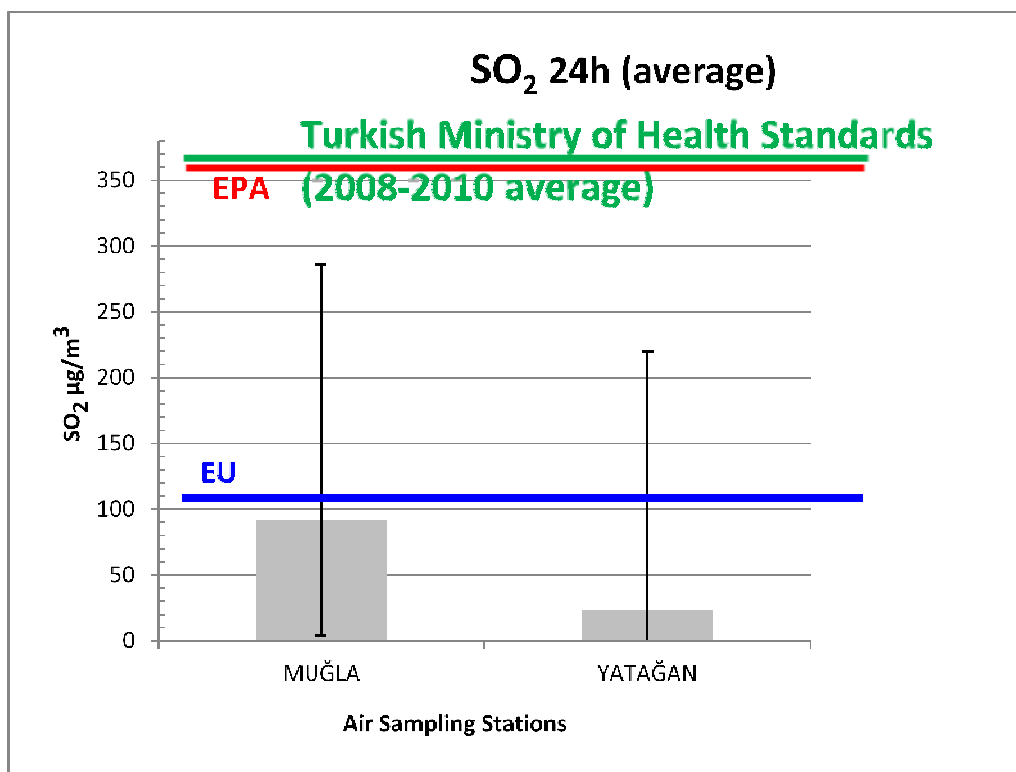


Figure 4.2 SO₂ 24h data averages of Mu la and Yata an air sampling stations for years 2008-2009-2010 showing the smallest and the highest values recorded for the two stations and the values that exceed EPA, EU and Turkish standards.

4.2 ANALYSIS RESULTS AND DISCUSSION OF MU LA'S CHRONICLE AIRWAY DISEASE DATA WITH GIS MAP EVALUATIONS AND THE CORRELATION OF CRITERIA POLLUTANTS' MEASUREMENTS AND CHRONICLE AIRWAY DISEASE DATA IN MU LA CITY.

Raw data (Figure 4.3) as retrieved from the hospitals are given first and the way it is analyzed and the discussions and evaluation are given in following Table 4.3, 4.4 and Figures 4.4 - 4.19.

İsim	Boyut	Paket
YATAĞAN 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	327.680	65.047
TOPLAM MUĞLA 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	4.430.848	676.028
ORTAÇA 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	234.496	35.367
MUĞLA 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	1.849.344	311.156
MİLAS 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	190.464	32.898
MARMARİS 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	1.706.496	304.628
KÖYCEĞİZ 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	338.432	59.064
KAVAKLIDERE 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	44.032	6.995
ICD_10_duzeltimis.xls	4.208.128	721.063
FETHİYE 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	862.208	111.126
DATÇA 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	513.024	90.596
DALAMAN 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	44.544	6.723
BODRUM 2008-2009-2010 KRONİK SOLUNUM YOLU HASTALIKLARI BİLGİ FORMU.xls	307.200	45.669

Figure 4.3 Raw hospital data excel files.

The limited epidemiological public hospital data were summarized with Tables 4.3 and 4.4 and Figures 3.9 a and b and 4.4.

Table 4.3 Mu la city and counties' total chronic airway diseases prevalence according to gender in years 2008-2009-2010.

Most Prevalent Chronic Airway Diseases in Mu la	Male	Female
Asthma	6532	9577
COPD	13722	9577
Emphysema	12	5
Bronchiectasis	161	164
Other Chronic Obstructive Pulmonary Disease	5	3
Simple and Mucopurulentchronic Bronchitis	27	14

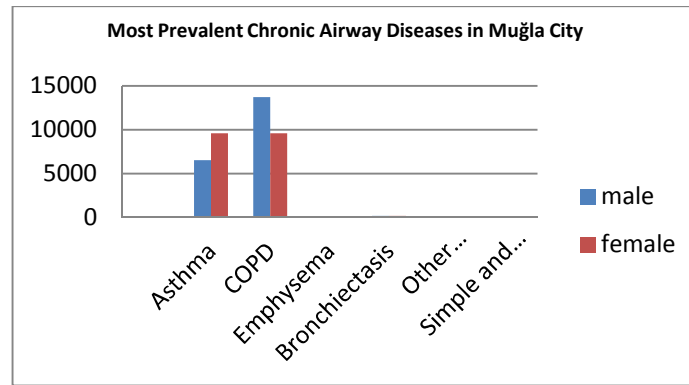


Figure 4.4 Most prevalent chronic airway diseases in Mu la city with gender differences (cumulative data for 2008-2010).

Table 4.4 Prevalance of Chronic Airway Diseases in Mu la City and Counties in years 2008-2009-2010 (numbers are cumulative for three years).

Location	Asthma	COPD	Emphysema	Bronchiectasis	Simple and Mucopurulentchronic Bronchitis	Unspecified Chronic bronchitis	Respiratory Failure	Bronchitis, not Specified as Acute or Chronic	Total Chronic Airway Diseases
Mu la	10257	6225		18		28		4003	20954
Bodrum	1910	1286		87					3283
Dalaman	24	18		6					48
Datça	1037	670	5	12	7	56	26		1813
Fethiye	5	8590			28	215	2		8840
Kavaklıdere		219			1	43			263
Köyce iz	702	244							946
Marmaris	2095	2738	12	40	3				4888
Milas	60	5							65
Ortaca	1090	743			2			1063	2898
Yata an	1095	1595		12	15	283		566	3566
Total	18275	22333	17	344	56	879	28	5632	

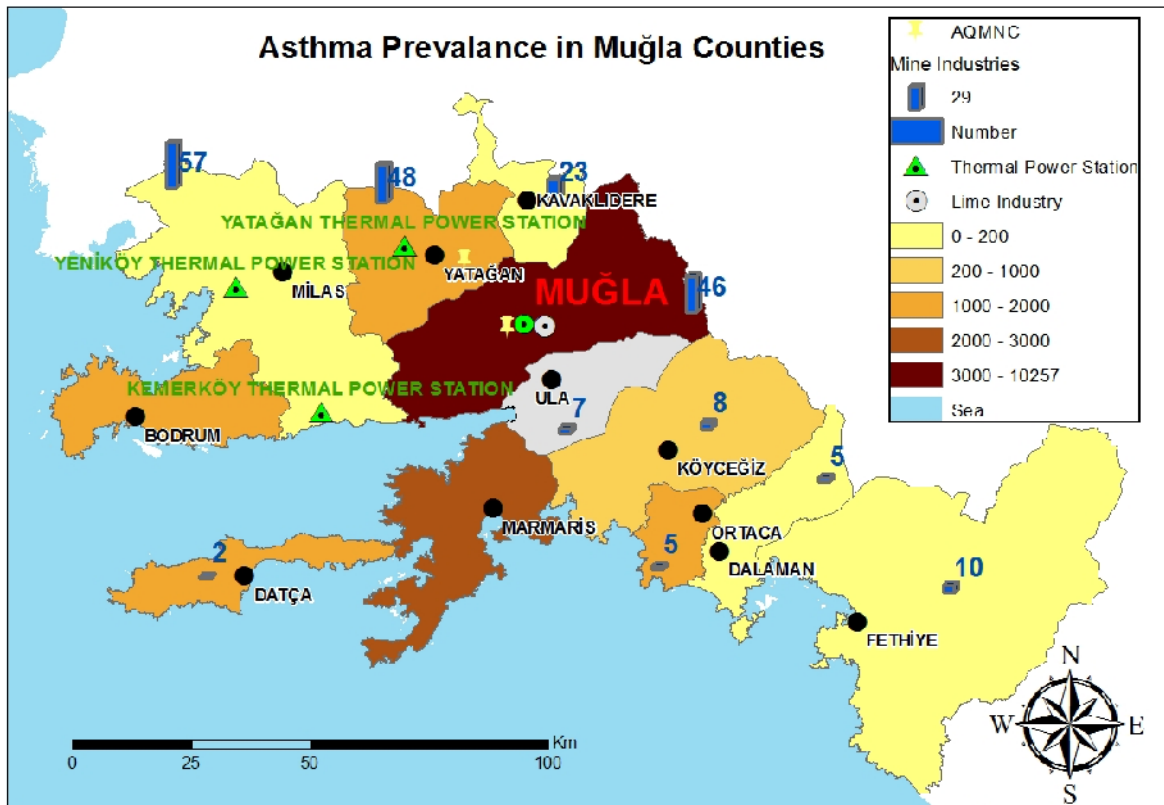


Figure 4.5 Asthma prevalence in Muğla counties (sum of 2008-2009-2010).

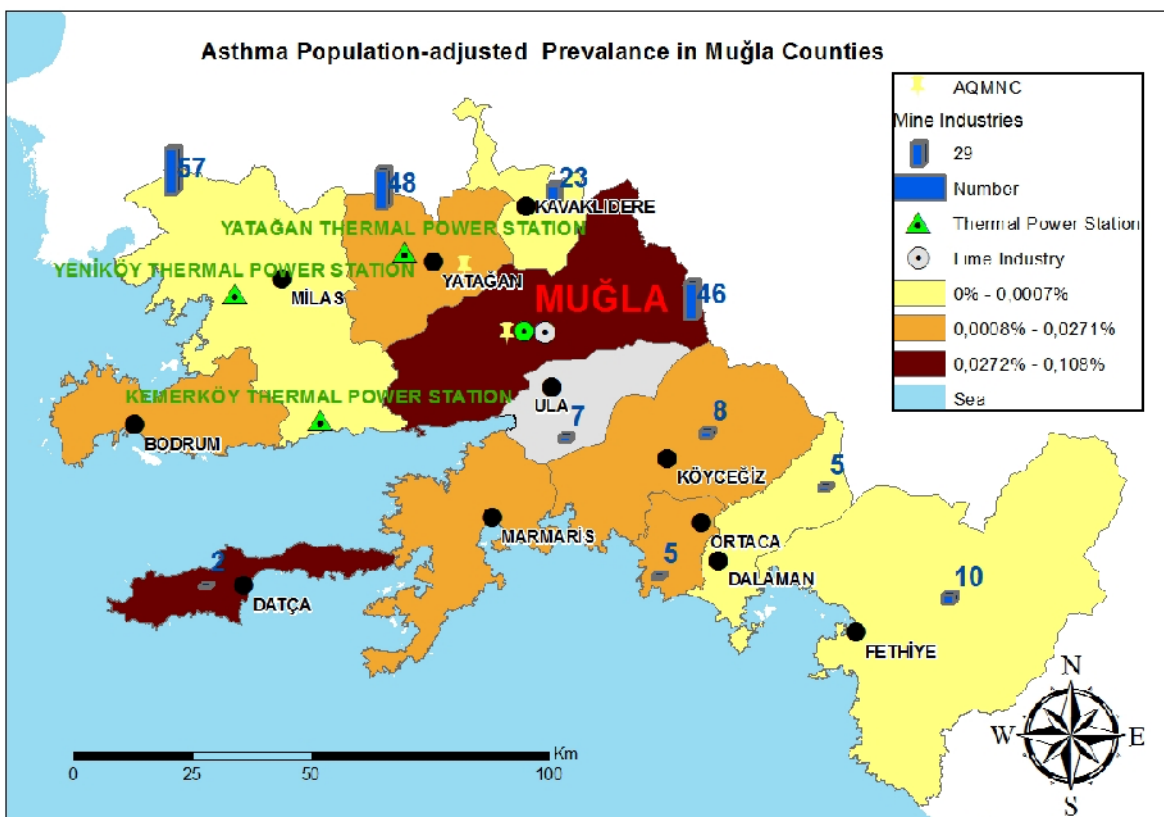


Figure 4.6 Population-adjusted prevalence of Asthma in Muğla counties (sum of 2008-2009-2010).

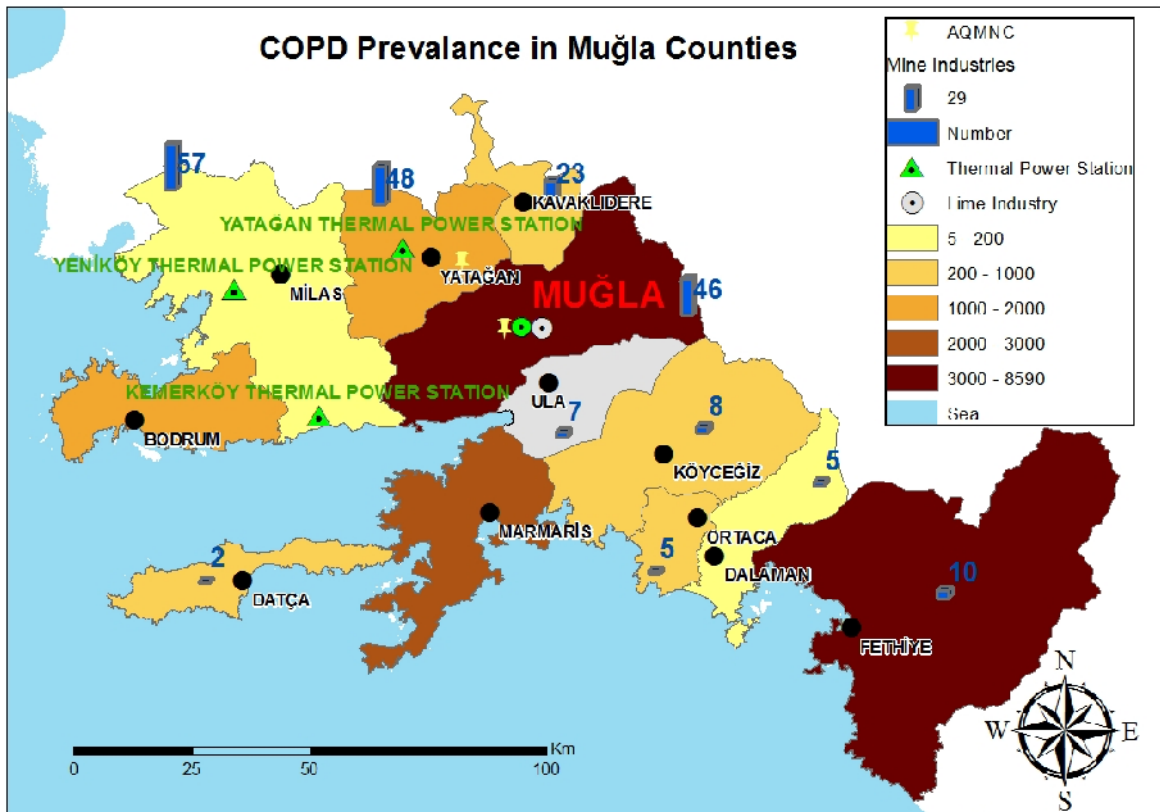


Figure 4.7 COPD prevalence in Muğla counties (sum of 2008-2009-2010).

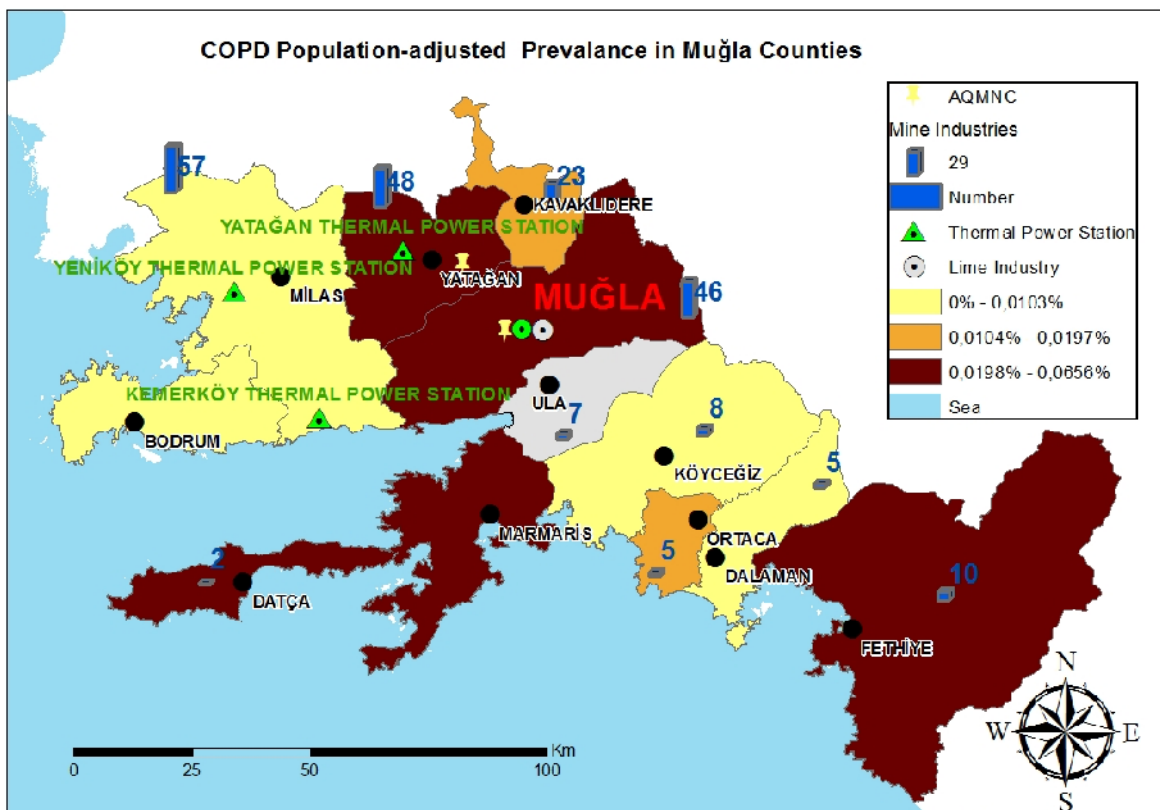


Figure 4.8 Population-adjusted prevalence of COPD in Muğla counties (sum of 2008-2009-2010).

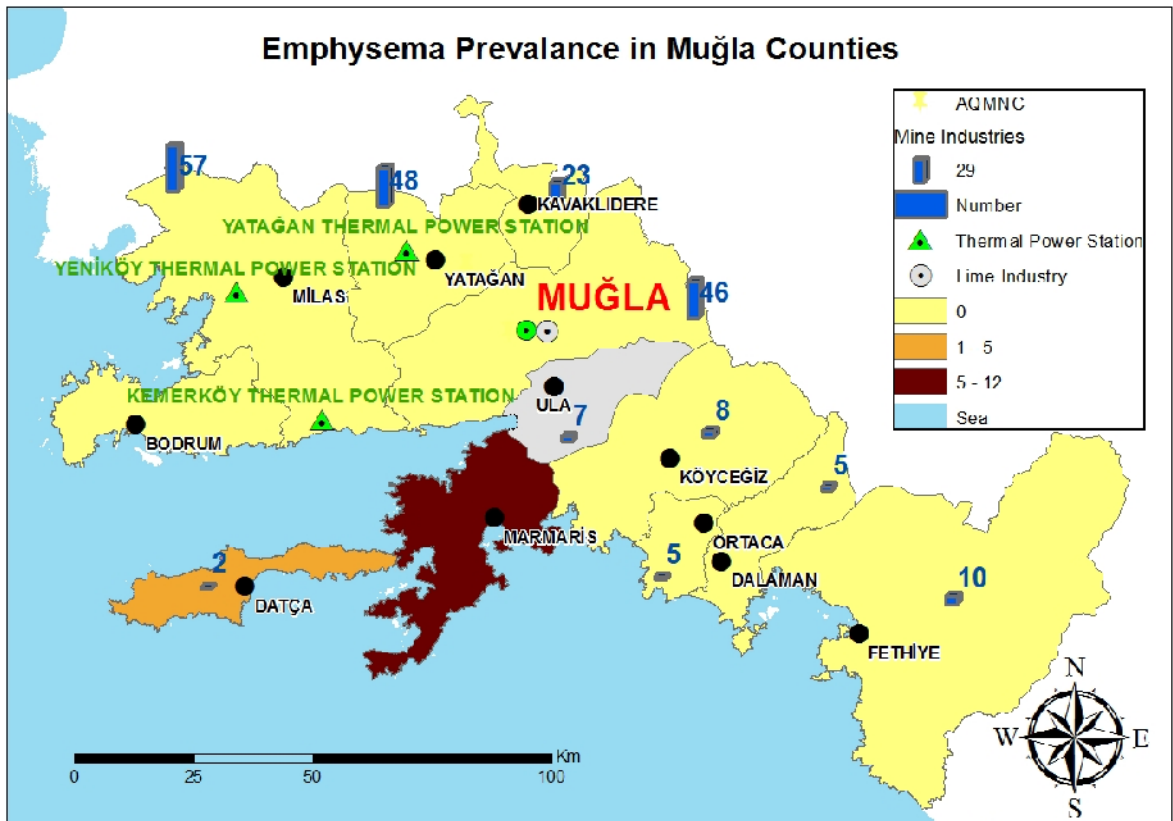


Figure 4.9 Emphysema prevalence in Muğla counties (sum of 2008-2009-2010).

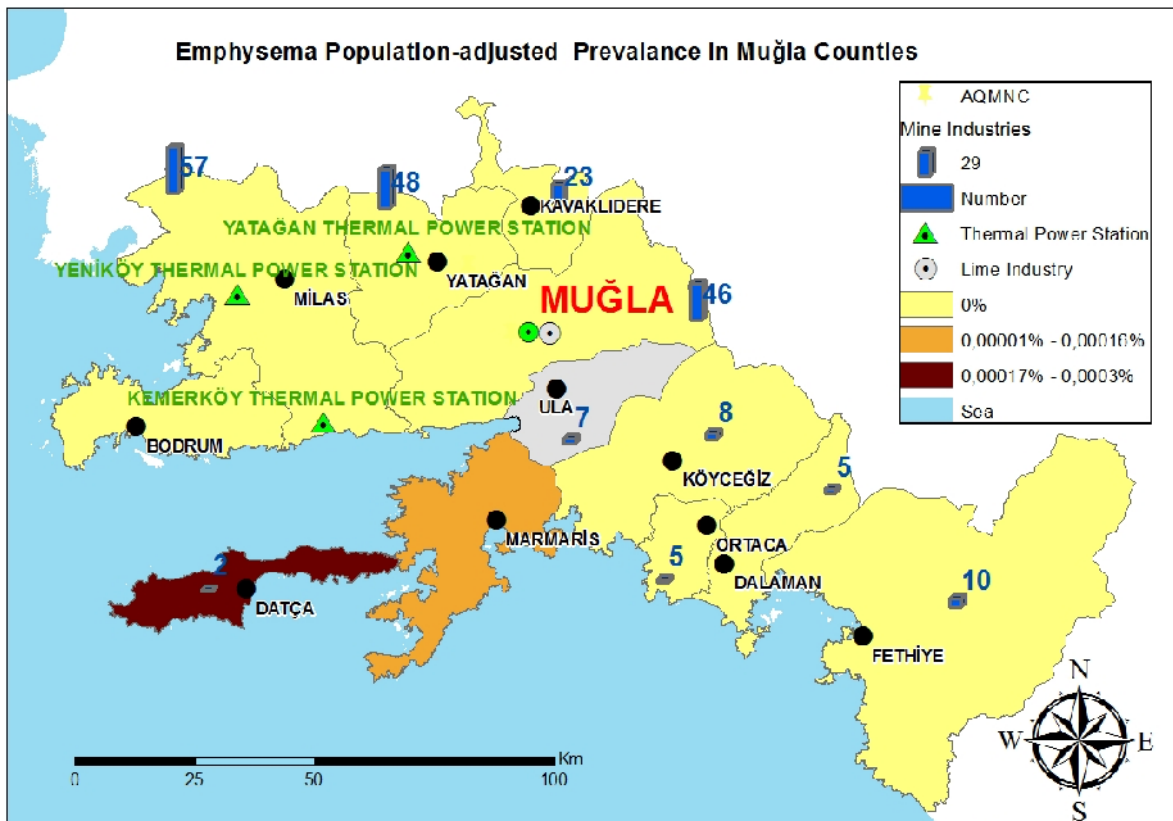


Figure 4.10 Population-adjusted prevalence of Emphysema in Muğla counties (sum of 2008-2009-2010).

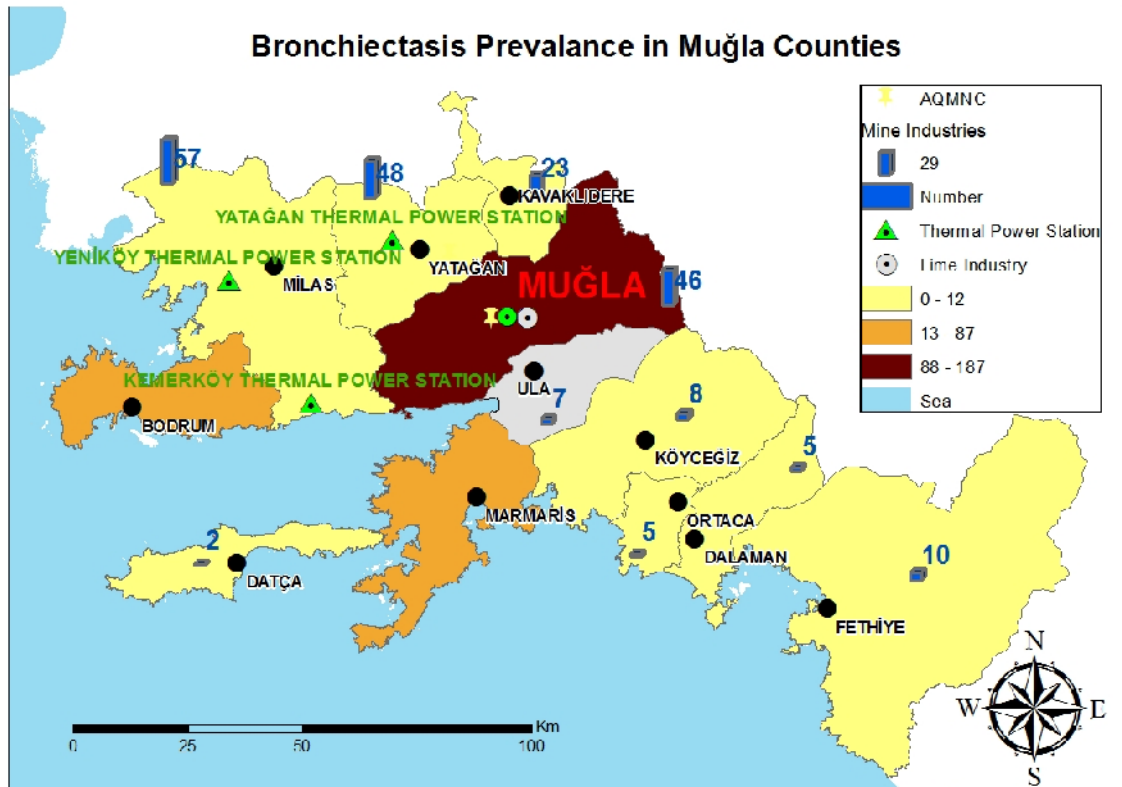


Figure 4.11 The Bronchiectasis prevalence in Mu la counties (sum of 2008-2009-2010).

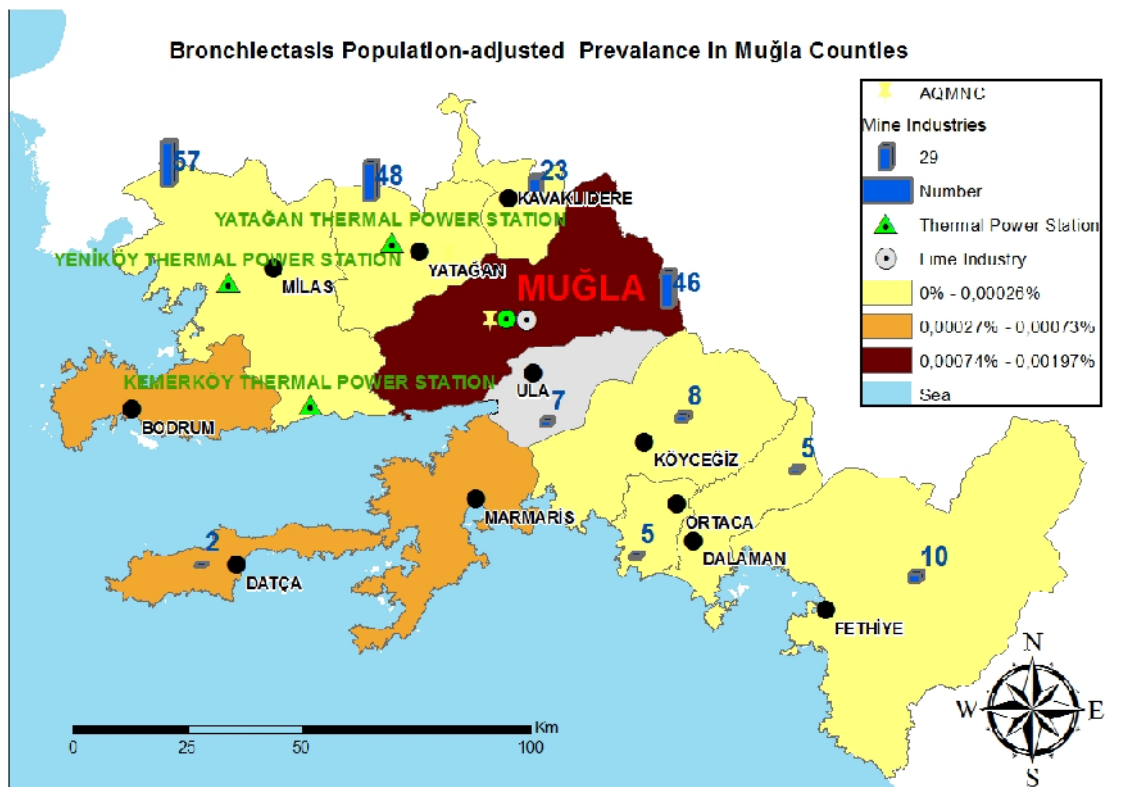


Figure 4.12 Population-adjusted prevalence of Bronchiectasis in Mu la counties (sum of 2008-2009-2010).

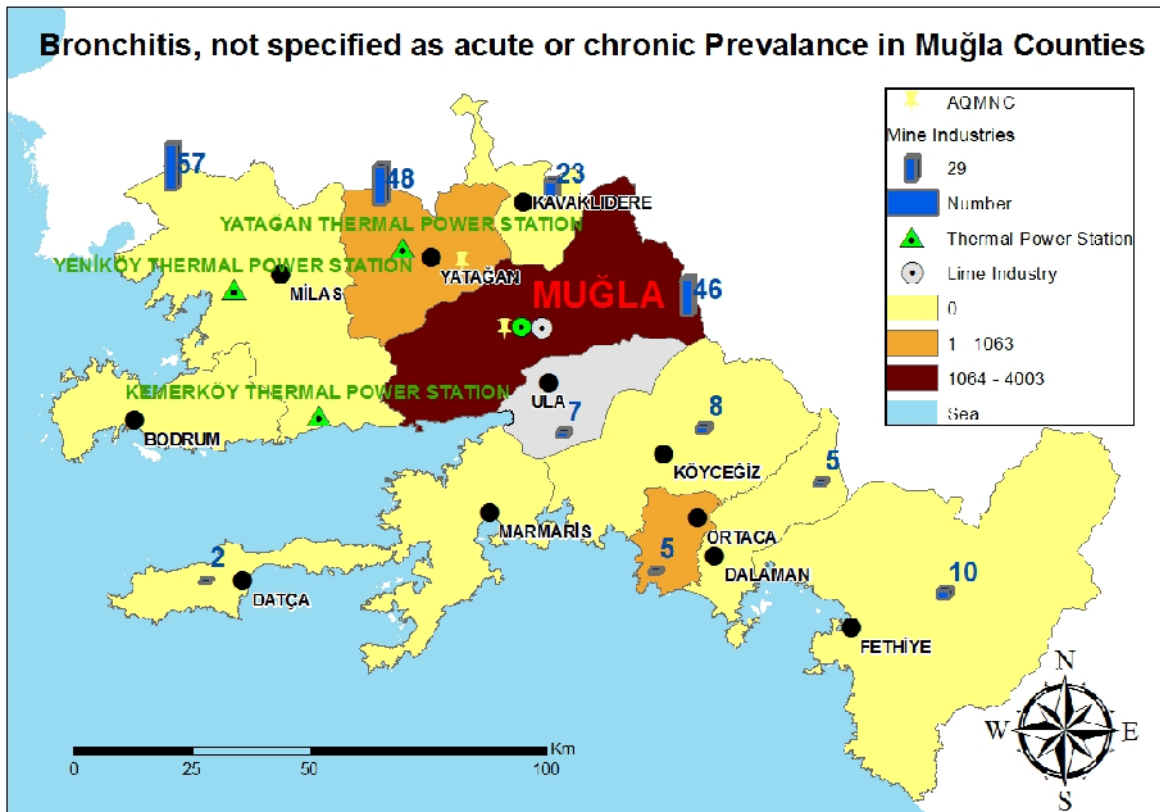


Figure 4.13 Bronchitis not specified as acute or chronic prevalence in Muğla counties (sum of 2008-2009-2010).

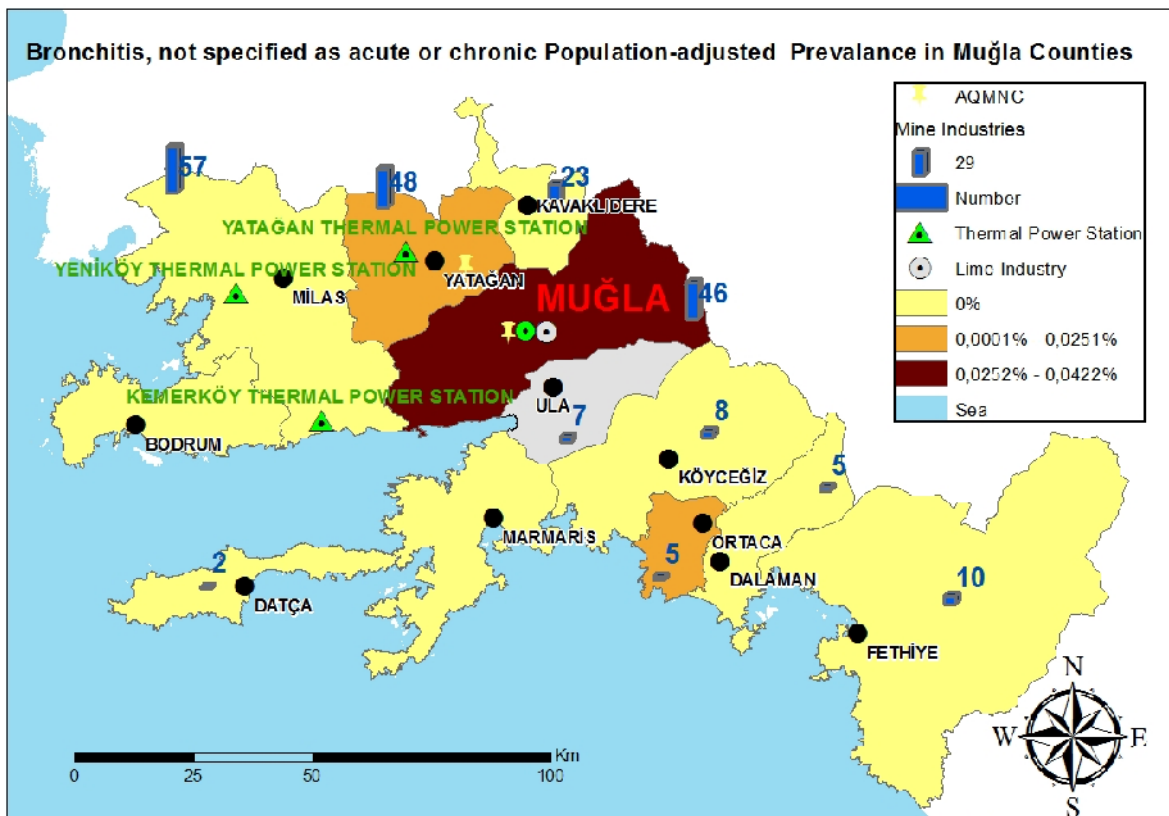


Figure 4.14 Population-adjusted prevalence of Bronchitis not specified as acute or chronic in Muğla counties (sum of 2008-2009-2010).

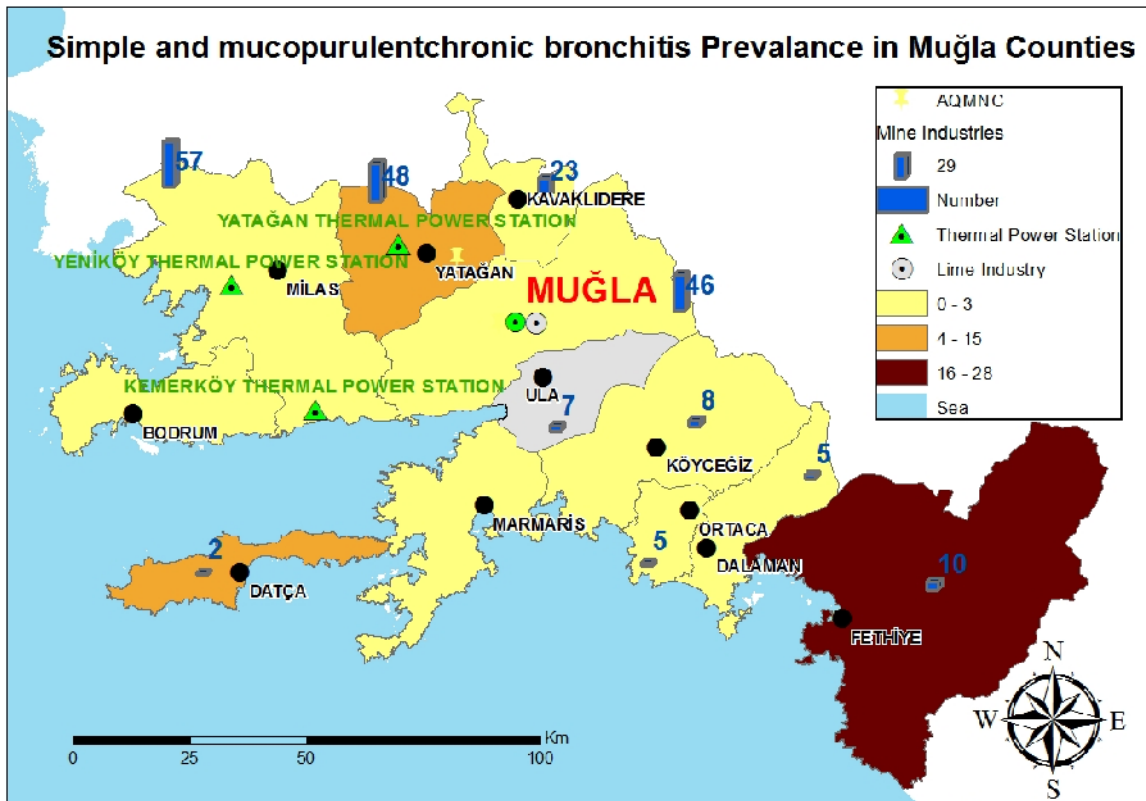


Figure 4.15 Mucopurulent Chronic Bronchitis prevalence in Muğla counties (sum of 2008-2009-2010).

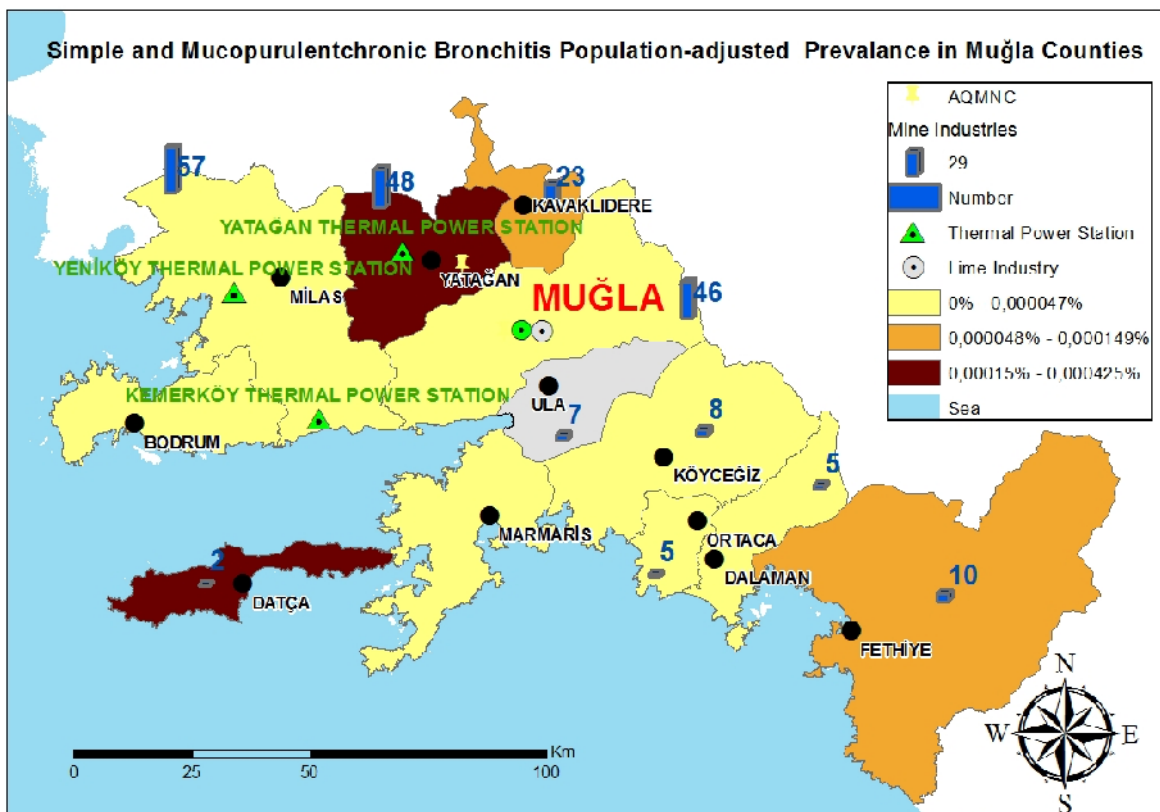


Figure 4.16 Population-adjusted prevalence of Mucopurulent Chronic Bronchitis in Muğla counties (sum of 2008-2009-2010).

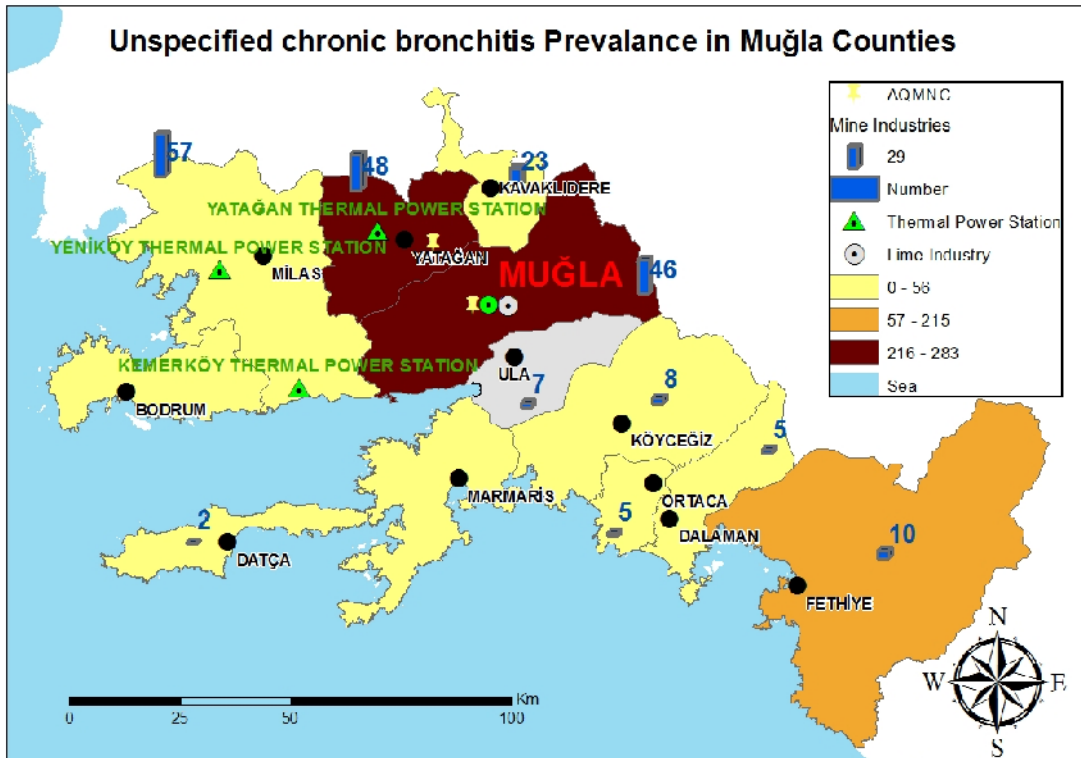


Figure 4.17 Unspecified Chronic Bronchitis prevalence in Mu la counties (sum of 2008-2009-2010).

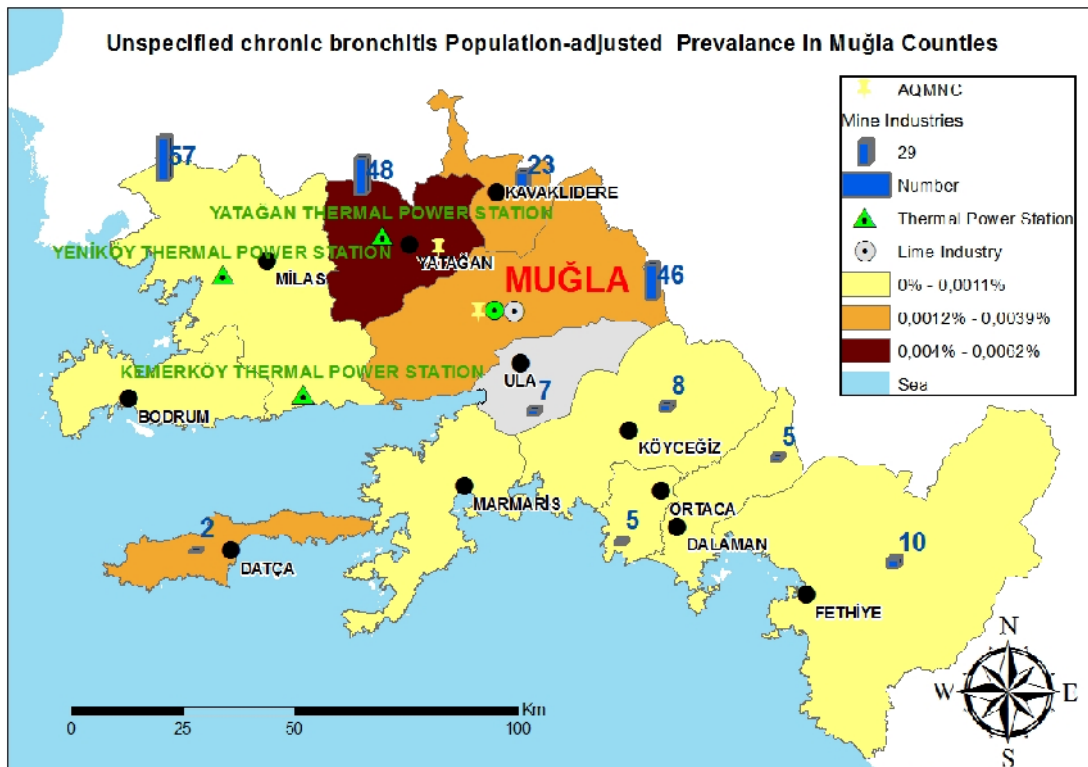


Figure 4.18 Population-adjusted prevalence of Unspecified Chronic Bronchitis in Mu la counties (sum of 2008-2009-2010).

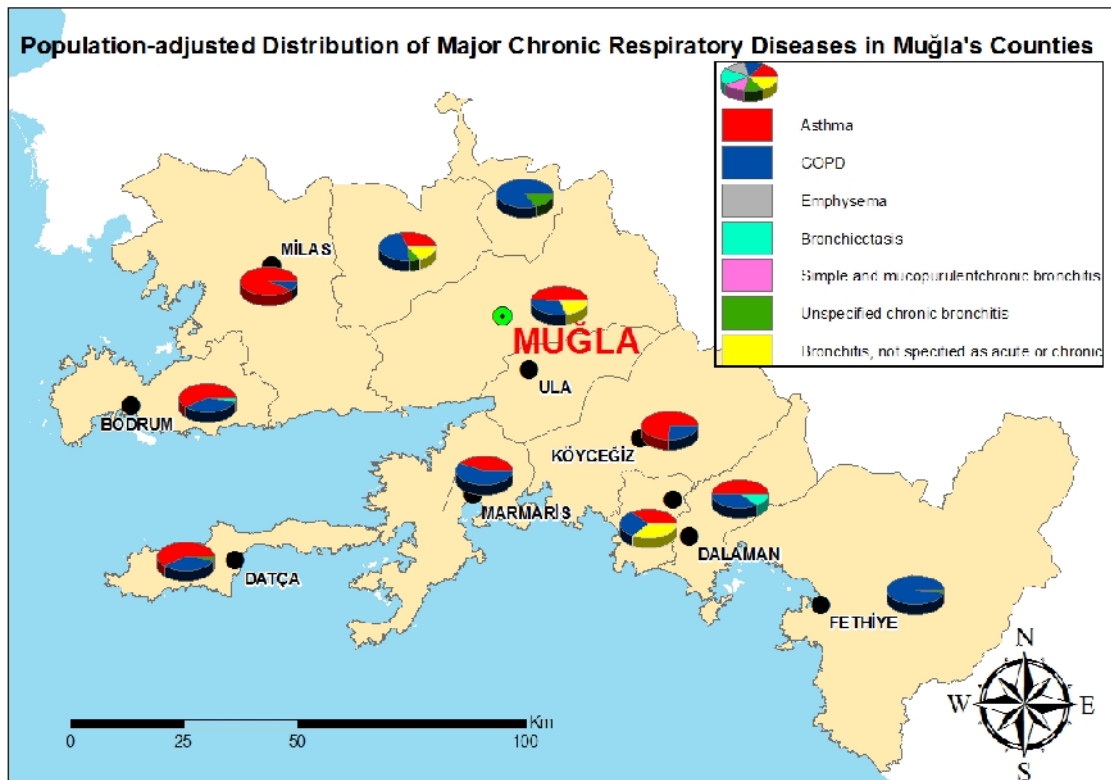


Figure 4.19 Population-adjusted distribution of major Chronic Respiratory Diseases in Muğla's counties.

4.2 ANSWERS OF QUESTIONS IN 3.1.4 AND RESULTS AND DISCUSSIONS OF LIMITATIONS AND POTENTIAL CONFOUNDING FACTORS IN THE WORK

The Muğla case work to bridge the gap between air criteria air pollutants and health effects had a goal to answer the below compromised questions to answer, unfortunately it was not possible to find out the exact answers to these questions in this thesis, but some answers and contributions are given extracted during this research work in Muğla GARD. The answers are usually for more than one question at the same time so they have been given and discussed separately, but not following the specific question [20, 22].

1. How does our understanding of the health effects of air pollutants (singly or in mixtures) help identify pollutants that can be linked to sources the control of which would provide maximal health benefits? (overarching theme)
2. How reliable are methods (measurements and models) and approaches (epidemiological and toxicological) for studying and quantifying the links between air pollutants (species and or sources) and adverse health effects?
3. How do relevant pollutant properties vary in space and time from sources and in ambient air? What are the implications of these variations for population exposure?

4. What advances have been made in understanding the relationships between exposure, both spatially and temporally, and estimates of dose that tie to health outcomes?
5. Are patterns emerging that relate component(s) of air pollution and/or source types to mechanisms? What is the status of identifying and measuring biomarkers of exposure and/or adverse health effects from air pollution?
6. Who are the susceptible populations? What drives different susceptibilities to the same or different air pollutants? Are there susceptibility traits associated with specific health outcomes that are common among the subpopulations?
 - a. Susceptibility criteria should be evaluated for each patient according to medical history, anamnesis.
 - b. Detailed pedigrees should be drawn for each patient to assess genetic and environmental contribution to the disease.
 - c. Susceptibility genes such as GSTs, CYPs, NATs should be genotyped for personalized therapies.
 - d. Drug-metabolizing enzyme polymorphisms; Phase I and Phase II drug metabolism; NAT2; G6PD; Exposure assessment; TPMT, CYP2D6, NQO1 and PON genes can be considered more specifically.
 - e. The patients' prognosis should be followed by routine and detailed progress and course reportings done by phone calls.
7. What roles do confounding or other factors have in increasing, decreasing, or obscuring attribution of the true health effects from ambient air pollutants?
8. Do actions taken to improve air quality result in reduced ambient concentrations of relevant pollutants, exposure, and health effects? Have we encountered unintended consequences?
9. What are the policy implications of our improved understanding of the source to health effect paradigm?

Confounding factors during the collection of pollutant data and evaluation of the data:

The air criteria pollutant data are collected from two stations and available freely in an easily downloadable format but still requires a deep evaluation from an experts point of view who is in charge of these measurements like me. I am in charge of every detail related to the collection of these data:

1. I was a member of GARD
2. I am in charge of these two stations' maintenance.
3. I am following the contributing factors (factories, facilities mentioned and drawn partly in many of the GIS maps.) to these pollutants.

The data obtained and the way they are obtained have many controversial discussions like [1, 2, 7, 20, 22]:

1. The data for Mu la's air pollution is only for two of the air criteria pollutants.

2. And only two stations collect this data.
3. Mu la station's place is not suitable for such data collection as centered in a sink place confusing the data collection and evaluations (Figure is for the photographs of the place).
4. The limit value evaluations and average value determinations are done from a 24 hourly basis which is confounding at the end, because the exposure and accumulation of the effects is not an average of events; if the organism is exposed to a deep pollution even once can be enough to induce many detrimental airway related health effects. So the evaluation of each fluctuation in these pollutants is very important and the awareness of each contributor for these fluctuations should be addressed and discussed as given in Figures below.
5. The pollution data to be addressed differently with an expert eye for summer and winter months to understand the confounding differences and variation in data see Figures 4.20 and 4.21.

Tarih	PM10 $\mu\text{g}/\text{m}^3$	SO2 $\mu\text{g}/\text{m}^3$
29.07.2011 00:00	83	1
29.07.2011 01:00	76	2
29.07.2011 02:00	63	2
29.07.2011 03:00	61	2
29.07.2011 04:00	57	2
29.07.2011 05:00	56	2
29.07.2011 06:00	64	2
29.07.2011 07:00	77	2
29.07.2011 08:00	79	2
29.07.2011 09:00	82	2
29.07.2011 10:00	88	2
29.07.2011 11:00	81	2
29.07.2011 12:00	74	2
29.07.2011 13:00	57	2
29.07.2011 14:00	48	10
29.07.2011 15:00	66	5
29.07.2011 16:00	70	3
29.07.2011 17:00	93	2
29.07.2011 18:00	80	2
29.07.2011 19:00	73	2
29.07.2011 20:00	88	2
29.07.2011 21:00	102	2
29.07.2011 22:00	103	2
29.07.2011 23:00	89	2

Figure 4.20 Yata an station summer month hourly taken PM₁₀ and SO₂ pollution data. Especially the SO₂ values are nearly 0 should be focused.

Tarih	PM10	SO2
	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$
30.11.2011 00:00	231	352
30.11.2011 01:00	228	266
30.11.2011 02:00	171	224
30.11.2011 03:00	98	166
30.11.2011 04:00	109	127
30.11.2011 05:00	89	168
30.11.2011 06:00	115	165
30.11.2011 07:00	107	251
30.11.2011 08:00	198	372
30.11.2011 09:00	197	277
30.11.2011 10:00	136	244
30.11.2011 11:00	86	253
30.11.2011 12:00	80	403
30.11.2011 13:00	77	140
30.11.2011 14:00	57	72
30.11.2011 15:00	58	63
30.11.2011 16:00	68	76
30.11.2011 17:00	103	108
30.11.2011 18:00	122	138
30.11.2011 19:00	122	340
30.11.2011 20:00	279	419
30.11.2011 21:00	307	470
30.11.2011 22:00	320	399
30.11.2011 23:00	298	281

Figure 4.21 Yata an station winter month hourly taken PM₁₀ and SO₂ pollution data. Especially the SO₂ values as compared to Figure 4.20 should be focused.

Tarih	PM10	SO2
	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$
01.07.2011 00:00	31	2
01.07.2011 01:00	27	5
01.07.2011 02:00	27	5
01.07.2011 03:00	27	5
01.07.2011 04:00	28	3
01.07.2011 05:00	30	6
01.07.2011 06:00	35	25
01.07.2011 07:00	42	17
01.07.2011 08:00	42	34
01.07.2011 09:00	48	45
01.07.2011 10:00	51	35
01.07.2011 11:00	56	16
01.07.2011 12:00	55	6
01.07.2011 13:00	45	7
01.07.2011 14:00	58	3
01.07.2011 15:00	55	3
01.07.2011 16:00	32	3
01.07.2011 17:00	31	6
01.07.2011 18:00	47	5
01.07.2011 19:00	56	6
01.07.2011 20:00	58	6
01.07.2011 21:00	61	5
01.07.2011 22:00	67	6
01.07.2011 23:00	43	4

Figure 4.22 Mu la station summer month hourly taken PM₁₀ and SO₂ pollution data. Especially the SO₂ values are much lower than winter data in Figure 4.23 should be focused.

Tarih	PM10	SO2
	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$
03.12.2011 00:00	278	191
03.12.2011 01:00	228	177
03.12.2011 02:00	219	127
03.12.2011 03:00	157	118
03.12.2011 04:00	143	99
03.12.2011 05:00	142	87
03.12.2011 06:00	143	113
03.12.2011 07:00	242	153
03.12.2011 08:00	237	160
03.12.2011 09:00	242	441
03.12.2011 10:00	345	408
03.12.2011 11:00	461	216
03.12.2011 12:00	185	87
03.12.2011 13:00	60	70
03.12.2011 14:00	52	63
03.12.2011 15:00	52	96
03.12.2011 16:00	61	207
03.12.2011 17:00	184	382
03.12.2011 18:00	479	313
03.12.2011 19:00	539	288
03.12.2011 20:00	576	211
03.12.2011 21:00	294	183
03.12.2011 22:00	219	124
03.12.2011 23:00	236	99

Figure 4.23 Mu la station winter month hourly taken PM₁₀ and SO₂ pollution data. The much higher PM₁₀ and SO₂ values as compared to Figure 4.22 should be focused.

Tarih	PM10	SO2
	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$
25.01.2011 00:00	30	23
25.01.2011 01:00	51	19
25.01.2011 02:00	15	17
25.01.2011 03:00	2	15
25.01.2011 04:00	2	13
25.01.2011 05:00	1	12
25.01.2011 06:00	1	12
25.01.2011 07:00	36	12
25.01.2011 08:00	48	12
25.01.2011 09:00	67	13
25.01.2011 10:00	32	14
25.01.2011 11:00	11	17
25.01.2011 12:00	5	29
25.01.2011 13:00	3	43
25.01.2011 14:00	5	25
25.01.2011 15:00	18	28
25.01.2011 16:00	8	17
25.01.2011 17:00	24	14
25.01.2011 18:00	35	13
25.01.2011 19:00	63	11
25.01.2011 20:00	34	11
25.01.2011 21:00	74	10
25.01.2011 22:00	44	10
25.01.2011 23:00	53	11

Figure 4.24 During snowy days the low PM₁₀ and SO₂ data are focused.

Tarih	PM10	SO2
	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$
01.06.2011 00:00	52	7
02.06.2011 00:00	54	4
03.06.2011 00:00	65	5
04.06.2011 00:00		
05.06.2011 00:00		
06.06.2011 00:00		
07.06.2011 00:00	65	14
08.06.2011 00:00	72	
09.06.2011 00:00	87	5
10.06.2011 00:00	72	22
11.06.2011 00:00	48	13
12.06.2011 00:00	41	25

Figure 4.25 Yata an station missing data type confounding factor example cases due to electricity blackout.

Table 4.5 Yata an station erranous data collection.

Collection Date	PM ₁₀	SO ₂
07.11.2010	40	78
08.11.2010	-4976	-5394
09.11.2010	-3733	-5366
10.11.2010	11	55
11.11.2010	7	43

Tarih	PM10	SO2	Hava Sýıcaklıđı	Ruzgar Yonu	Ruzgar Hızı
	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$	$^{\circ}\text{C}$	Derece	m/s
27.01.2011 00:00	92	93	13,6	26	0,8
27.01.2011 01:00	120	85	13,6	136	0,6
27.01.2011 02:00	93	76	13,5	126	0,6
27.01.2011 03:00	76	47	13,4	130	0,8
27.01.2011 04:00	26	29	13,3	123	1,3
27.01.2011 05:00	30	32	13,2	88	1,8
27.01.2011 06:00	30	156	13,2	128	1,9
27.01.2011 07:00	59	368	13,2	126	2,0
27.01.2011 08:00	113	194	13,2	131	2,0
27.01.2011 09:00	61	101	13,2	126	2,3
27.01.2011 10:00	56	231	13,3	95	3,5
27.01.2011 11:00	75	169	14,2	106	2,9

Figure 4.26 The effect of wind speed (when it is low) on PM₁₀ ve SO₂ hourly values.

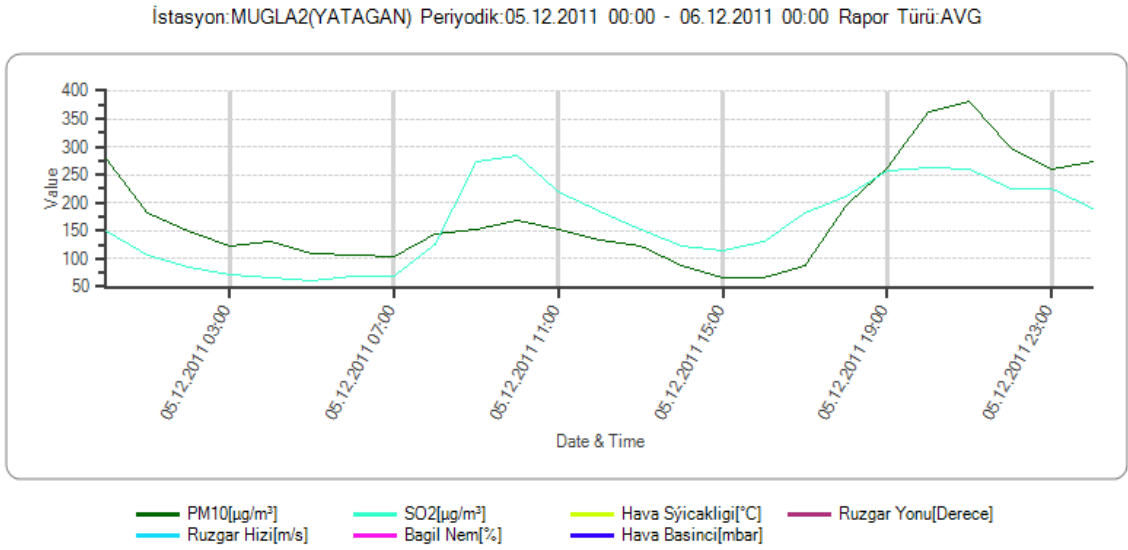


Figure 4.27 The effect of coal burning hours on PM_{10} ve SO_2 values.

T.C.
MUĞLA VALİLİĞİ
İL SAĞLIK MÜDÜRLÜĞÜ

Sayı :B.104.ISM.4.48.08.- 212.02.01- 473 -9277
Konu: Türkiye KMH Önleme ve Kontrol
Programı Hk. Bilgi Talebi

24.04.2011

ÇEVRE VE ORMAN İL MÜDÜRLÜĞÜ'NE

MUĞLA

İlgi: 20.04.2011 tarih ve 703-1887 sayılı yazınız.

İlgi sayılı yazınıza istinaden "Hava Kirliliği ve Sağlık Bağlantısını Sorgulamada Muğla İli Kronik Hava Yolu Hastalıkları Örneği" başlıklı çalışmanızla ilgili olarak, talep etmiş olduğunuz Muğla İline ait Kronik Hava Yolu Hastalıklarının ilçeler bazında envanterinin sayfa dökümünün çok uzun olması sebebi ile il GARD kurulu üyesi Kimya Müh.Ekrem Yazıcıoğlu'na elektronik ortamda ekrem20@yahoo.com adresine gönderilmesi uygun görülmüştür.

Bilgilerinize arz ederim.

Dr. İskender GENCER
Sağlık Müdürü

T.C.
MUĞLA VALİLİĞİ
İL SAĞLIK MÜDÜRLÜĞÜ

Sayı: B.104.İSM.4.48.00.08.- 212.02.01-13-25-23375
Konu: Türkiye KHH Önleme ve Kontrol Programı

03.11.2011


İL ÇEVRE VE ORMAN MÜDÜRLÜĞÜ'NE
MUĞLA

İlgi: Sağlık Bakanlığı Temel Sağlık Hizmetleri Genel Müdürlüğü'nün 29.06.2009 tarihli ve 19497 (2009/39) sayılı Genelgesi.

Dünya Sağlık Örgütü'nün kronik solunum hastalıklarından kaynaklanan ölümleri azaltmak amacıyla hayata geçirdiği proje (GARD- Global Alliance Against Chronic Respiratory Diseases-Kronik Solunum Hastalıklarına Karşı Küresel Birlik) kapsamında hazırlanan "Türkiye Kronik Hava Yolu Hastalıklarını (Astm-KOAH) Önleme ve Kontrol Programı (2009-2013) Eylem Planı" uygulamasına konulmuş olup çalışmalarına devam etmektedir.

İlgi sayılı genelge ile eylem planında yer alan aktivitelerin yerine getirilmesi, desteklenmesi ve koordinasyonun sağlanması için İl Kurulumuz 11.11.2010 tarih ve 602-24509 sayılı Valilik oluru ile oluşturulmuştur. Kurulumuzda halen görev yapmakta olan Kimya Mühendisi Ekrem Yazıcıoğlu'nun yerine GARD İl Kurul kadrosunda görev almak üzere kurumunuzda konuyla ilgili olarak çalışan ve deneyimli personelden belirlenecek bir kişinin adı, soyadı, ünvanı ve iletişim bilgilerinin (cep telefonu, elektronik posta adresi) 03.11.2011 pazartesi günü mesai bitimine kadar bildirilmesi hususunda;

Gereğini arz ederim.


Dr. İskender GENCER
İl Sağlık Müdürü

Ayrıntılı bilgi için: Dr. Ö. ONKAŞ Şb. Md
Elektronik Posta: muqlsah@gmail.com

Muğla İl Sağlık Müdürlüğü
Ruh Sağlığı ve Sosyal Hastalıklar Şubesi
Bulaşıcı Olmayan Hastalıklar ve Kronik Durumlar Birimi
Telefon: (0252) 214 1105/1128 Belge Geçer: (0252) 214 3685



T.C.
MUĞLA VALİLİĞİ
İL Çevre ve Orman Müdürlüğü



Sayı : B.18.0.İÇÖ.2.48.00.02/900-1915
Konu: Ekrem YAZICIOĞLU

İL SAĞLIK MÜDÜRLÜĞÜNE
MUĞLA

İl Müdürlüğümüz Çevre Yönetimi Şube Müdürlüğünde Kimya Mühendisi olarak görev yapmakta olan Ekrem YAZICIOĞLU'nun 21/11/2011 tarihli dilekçesi yazımız eki gönderilmektedir.

Bilgilerinizi ve gereğini arz ederim.


Mehmet ŞAHİN
İl Müdür V.

Eki: 1 Dilekçe (1 Sayfa)

İL ÇEVRE VE ORMAN MÜDÜRLÜĞÜ ÇEVRE YÖNETİMİ ŞUBE MÜDÜRLÜĞÜNE
MUĞLA İL SAĞLIK MÜDÜRLÜĞÜNE SUNULMAK ÜZERE

Valilik Makamının 01/03/2011 tarih ve 4378 sayılı olurları ile İlimiz GARD kurulu yürütme kuruluna görevlendirildim, ayrıca Valilik Makamının 13/04/2011 tarih ve 8208 sayılı olurları ile İlimiz GARD komisyonu İl Kuruluna görevlendirildim. Bu kapsamda düzenlenen toplantılara katıldım. Gerekli görüş ve önerilerimi kurulumuza sundum. Ayrıca Kurul Sekreteryası görevini yapan Dr.Öner ÖNKAŞ beye mail yolu ile ilettim.

İl Müdürlüğünüzün 03/11/2011 tarih ve 23375 sayılı İl Çevre ve Orman Müdürlüğüne yazılan yazısı ile benim yerime GARD İl Kurulunda görev almak üzere bu konuyla ilgili çalışan deneyimli personelin iletişim bilgileri istenmiş olup bu yazı üzerine İl Çevre ve Orman Müdürlüğümüze benim yerime başka personelin isim bilgileri Kurumunuza göndermiştir.

Söz konusu İl Kurulunda şahsımın hangi çalışmasından memnun olunmadığı ve deneyimsiz olduğum kanaatine varılarak görevimin değiştirilmesi hususunda yazı yazıldığının tarafıma Bilgi Edinme Kanununun kapsamında açıklanması hususunu;

Bilgilerinize arz ederim.

Ekrem YAZICIOĞLU
Kimya Mühendisi

T.C.
MUĞLA VALİLİĞİ
İL SAĞLIK MÜDÜRLÜĞÜ

Sayı : B.104.ISM.4.48.08 / 212.02.01 / 1457 - 25 326
Konu: Ekrem YAZICIOĞLU Hk.

02.12.2011

İL ÇEVRE VE ORMAN MÜDÜRLÜĞÜ'NE
MUĞLA

İlgi: 25.11.2011 tarih ve 1919-4882 sayılı yazınız.

İlgi tarihli yazı ile Müdürlüğümüze gönderilen dilekçede Kimya Mühendisi olarak görev yapan Ekrem YAZICIOĞLU GARD İl Kurulunda görev yapmakta iken kendi özel tezi için hasta bilgileri istemiştir. Kendisine hasta bilgileri gönderilmiştir. Ancak bununla yetinmeyip hasta bilgilerinin sınıflandırılması, ingilizce anlamları, hastalık tanımları gibi bilgiler istemeye devam etmiş olup gönderdiği mailler ekte sunulmuştur. Adı geçen şahıs bu konuda gerek mail yoluyla gerek telefonla uyarılmış, bu isteklere devam etmesi halinde GARD İl Kurulundan çıkarılacağı kendisine bildirilmiştir.


Şube Müdürü Doktor Öner ÖNKAŞ'ın, Ruh Sağlığı ve Sosyal Hastalıklar Şubesi'nin yanı sıra başka idari görevler de yaptığından dolayı kişisel taleplere zaman ayırması mümkün değildir.

Bu şartlarda Ekrem YAZICIOĞLU'nun GARD İl Kurulu çalışmalarında verimli olamayacağı anlaşılmış olup yerine GARD İl Kurulu kadrosunda görev almak üzere kurumunuzda konuyla ilgili olarak çalışan başka bir personel talebinde bulunulmuştur.

Bilgilerinize arz ederim.

Dr. İskender GENCER
Sağlık Müdürü

Ek: Yazı(4 Adet)

iyi günler-rica  1 Hide Details

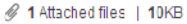
FROM: ekrem yazici oglu Tuesday, May 24, 2011 10:52 AM


TO: onerorkas@gmail.com ★

iyi günler Öner Bey,
öncelikle kolay gelsin bana gönderdiğiniz veriler için çok teşekkürler onlardan bazılarımda 2008-2009 yılları yok sanırım sizde de yok. size eğer şu şekilde daha basit bir taslak halinde ilçe bazlı hasta sayıları varsa onu listede halinde sadece sayıları oluşturduysanız gönderebilirsiniz çok sevinirim. Taslak örneği ekte gönderiyorum ben kolay gelsin iyi çalışmalar.

Ekrem Yazıcıoğlu

Saygılarımla.
Best Regards,
C*-----





Genel İlçe ba

[Download](#)

Fw: iyi günler Hide Details

FROM: ekrem yazici oglu Tuesday, May 31, 2011 9:40 AM

TO: onerorkas@gmail.com ★

Öner Bey merhabalar kolay gelsin,
size bazı sorularım olacaktı yardımcı olabilirsiniz çok sevinirim.
bildiğiniz üzere bir teze başladım bazı bilgilere ihtiyacım oluyor sizden ricam

size şu aşağıdaki hastalıkların İngilizce karşılıklarını ve klinik ve epidemiyolojik bilgilerin bana yollayabilirsiniz çok sevinirim. kolay gelsin iyi çalışmalar. verileri İngilizcede gönderebilirsiniz hocam tarketmez. tezimi İngilizce yazıyorum çünkü. İngilizce olması Türkçe olsa da olur. önemli olan literatüre koymam açısından sizde olabilecek kısa kısa en azından 1'er sayfalık veriler yeterli olacaktır. teşekkürler.

saygılarımla.

ASTIM,
KOAİ AMFİZEM ERONŞİEKTAZİ
İNTERSİTİSYEL AKCĞER HASTALIKLARI,
DİĞER KRONİK BRONŞİT,
BASİT VE MUKOPÜRÜLAN KRONİK BRONŞİT,
TANIMLANMAMIŞ SOLUNUM YETMEZLİĞİ HAVAYOLU HASTALIĞI
TANIMLANMIŞ ORGANİK TOZLARA BAĞLI SOLUNUM HASTALIKLARI SOLUNAN
KİMYASAL MADDE GAZ DUMAN VEYA BUHARLARA BAĞLI ASBESTOZİS BRONŞİT, AKUT VEYA
KRONİK OLARAK TANIMLANMAMIŞ

Ekrem Yazıcıoğlu

Saygılarımla.
Best Regards,
C*-----

Re: Fw: iyi günler Hide Details

FROM: ekrem.yazicioglu Wednesday, June 1, 2011 8:00 AM

TO: Öner Önkaş ★

Öner Bey kusura bakmayın ben sizde bunların hazır olabileceğini düşünmüştüm. en azından tıbbi terimler birimizin ilgili olduğu için. gene de teşekkürler. ben zaten hazırlamaya başladım güzel bazı tablolar yapıyorum. bunları sizlerlerde paylaşıp Muğlada kullanabiliriz diye düşünüyorum ben en azından o yüzden böyle bir konu seçmişim. bir işe yarasanı verimli bir şeyler yapabiliriz diye. çok teşekkürler ilginiz için. bana verdiğiniz veriler işime yarıyor zaten. bu literatür verilerini bende bulurum tabiki benimde iş yoğunluğum var da belki sizde vardır diye düşünmüştüm hepsi bu sağlık olsun kusura bakmayın. görüşmek üzere teşekkür ederim.

Ekrem Yazicioglu

Saygılar mla.
Best Regards,
C*-----

hayırlı ramazanlar-rica Hide Details

FROM: ekrem.yazicioglu Monday, August 0, 2011 8:00 AM

TO: Öner Önkaş ★

iyi günler kolay gelsin Öner Bey, nasılsınız? size bazı sorularım olacaktı.

1) aslında benim hocamın daha evvel yaptığı bir çalışma var malumunuz bu kronik hastalıkların bazı sıgı genlerindeki kaynakları olabilir. bu sebeple bana eğer dedi. bu hastaların bazısından 50-60 civarında kan alındığı zaman zaten bunlardan sanırım 10 ml alınsa 2 ml si EDTA'lı tüpe ayrılıp ben bunları üniversitede analiz yaptırma imkanım var ve burdan birşeyler çıkarabiliriz diyor. sizlerde eğer yapacağımız makalelere isminini mutlaka yazarsanız. etki izini GARLI kapsamında alınabilir mi? Bu hastaların nereden geldikleri, memeketten, cinsiyet ve yaşları, sigara kullanımı bilgileri ve hastalık teşhisi detayları önemli. 50-60 kadar hasta yeterli olur.

2) birde son 2 yıllık merkez hastaneye aylık olarak astım veya KOAH hastası başvurusu sayısını öğrenebilirsek yaklaşımda olsa olur tam rakamı bu amaçsak eğer bunu toz partikül kirlenme ile istatistiksel bir analize sokarak güzel bir veri oluşturabiliriz.

ben size bir ara tezimi göstereyim sizinde işinize yarayan yerler olur sanırım. sonuçta bu teze bu kuruda olduğum ve güzel sonuçlar elde edebilmek için başladım şimdi den yardımlarınız için çok teşekkürler.

sizinle müsait olduğunuzda tezimde gelip fikir alışverişinde bulunmak isterim.

YIL	ASTIM	KOAH	AMFİZEM	BRONŞİEKTAZİ	KRONİK BRONŞİT BASIL VE MUKOPÜRÜLAN	KRONİK BRONŞİT TANIMLANMAMIŞ	SOLUNUM YETMEZLİĞİ	BRONŞİT AKUT VEYAKRONİK OLARAK TANIMLANMAMIŞ
Merkez	10257	6225		187			282	4003
Bodrum	1910	1286		87				
Dalaman	24	18		6				
Datça	1097	670	5	12	7	56	26	
Fethiye	5	8590			23	215	2	
Kavaklıdere		219			1	43		
Köycegiz	702	244						
Marmaris	2065	2738	12	40	8			
Milas	60	5						
Ortaca	1090	743			2			1063
Yatağan	1065	1595		12	15	238		565
Total	18276	22833	17	344	56	879	28	5632

Figure 4.28 Confounding factor evidences mentioned in Part 1.1.

CHAPTER 5

CONCLUSION

Creating effective, evidence-based policy requires linking health and economic endpoints back to the sources, so that preventive policy options and recommendations can be tailored to address emissions' sources in order of their contribution to the air pollution problem. The research has five interconnected components:

- air quality, meteorology and emissions data analysis
- epidemiological studies of health effects in Mu la city and counties
- air pollution exposure assessment
- health impact assessment
- preventative policy discussion

This thesis presents the results of a pilot study covering Mu la city and its counties.

Those who suffer from a disease should become experts of their own disease. An association of patients could be very effective in lobbying government to put more energy and resources into research and education in health issues. The patient–doctor relationship should be a partnership. Thinking in terms of partnership means that the next step will be to engage the awareness of the patient who experiences a condition for 365 days a year, as opposed to the doctor who sees it for just a few hours a year. The doctor should serve as an adviser and researcher who should further evaluate the cues and hints given by the patients.

Air pollution regulation continues to have important implications for public health and environmental welfare, as well as the health of the economy. To be sure, these

critical policy decisions need to be made on a foundation of sound and innovative science.

The questions to be answered are: What is left to do regarding air pollution control, and can we further advance our understanding of air pollution risk and find ways to sustainably protect health and the environment as an environmental engineer working in the field? My position is as a civil servant working for the Ministry of Environment and City Planning at Mu la city. As a member of the GARD in Mu la I have had a chance to focus at the public health problems and underlying mechanisms related. To be a part of the scientific community as an MS thesis student at Fatih University Environmental Engineering department (under the supervision of a molecular biologist and geneticist Assist Prof. rem Uzonur from Biology Department with many health related projects) and working as a part of decision and policy making, regulatory position gave me the chance to further evaluate the existing gaps between science and governmental communities in the source to health paradigm that is the basic motivation of my thesis. In this thesis I have had the chance to disseminate the scientific facts in the policy making levels that can be furthered with such a preliminary work.

REFERENCES

1. Solomon, Paul A. 2011. "Air pollution and health: bridging the gap from sources to health outcomes." *Environmental health perspectives* 119(4): a156-7. Available at: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3080947&tool=pmcentrez&rendertype=abstract> [Accessed May 16, 2011].
2. Bell, Michelle L et al. 2011. "Environmental health indicators and a case study of air pollution in Latin American cities." *Environmental research* 111(1): 57-66. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/21075365> [Accessed January 30, 2011].
3. Cohen, Aaron J. 2000. "Outdoor Air Pollution and Lung Cancer Exposures to Carcinogens in Outdoor Air." *Environmental Health* 108 (September 1999): 743-750.
4. Perkins, H.C., Air Pollution, Mc Graw Hill, New York, 1974
5. Derwent, R.G. 1990 *Science of The Total Environment*, Volume 93, 255-262.
6. Nilgün Akbulut Çoban, Türkiye’de Kentlerde Ölçülen Partiküler Madde PM₁₀ Konsantrasyonlarının De erlendirilmesi, Yüksek Lisans Tezi, Hacettepe Üniversitesi, Çevre Mühendisli i Anabilim Dalı, 2009.
7. Tecer, L. H., Alagha, O., Karaca, F., Tuncel, G., and Nilufer Eldes. "Particulate matter (PM (2.5), PM (10-2.5), and PM (10)) and children’s hospital admissions for asthma and respiratory diseases: a bidirectional case-crossover study." *Journal of toxicology and environmental health. Part A* 71, no. 8 (January 2008): 512-20. <http://www.ncbi.nlm.nih.gov/pubmed/18338286>.
8. Khardori, Amit. 2000. "Health-Based Standards in the Clean Air Act The Controversy over Peace of Mind." *Clean Air* (May): 1-57.
9. Smith, K. R. 2002. "Indoor air pollution in developing countries: recommendations for research+." *Indoor Air* 12(3): 198-207. Available at: <http://www.blackwell-synergy.com/links/doi/10.1034%2Fj.1600-0668.2002.01137.x>.
10. Vanderroost, R, J Beyer, and N Vermeulen. 2003. "Fish bioaccumulation and biomarkers in environmental risk assessment: a review." *Environmental Toxicology and Pharmacology* 13(2): 57-149. Available at: <http://linkinghub.elsevier.com/retrieve/pii/S1382668902001266>.

11. Nebert, D W, and a L Roe. 2001. "Ethnic and genetic differences in metabolism genes and risk of toxicity and cancer." *The Science of the total environment* 274(1-3): 93-102. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/11453308>.
12. Global Alliance Against Chronic Respiratory Diseases (GARD): 5th general meeting report, 1-2 June 2010, Toronto, Canada.
13. Laurendi, Giovanna et al. 2011. "Global alliance against chronic respiratory diseases in Italy (GARD-Italy): Strategy and activities." *Respiratory medicine* (June): 1 - 2. Available at: <http://www.ncbi.nlm.nih.gov/pubmed/22024553>.
14. Global surveillance, prevention and control of Chronic Respiratory Diseases, A comprehensive approach.
15. Yoshida, T., and Rubin M. T. 2007. "Pathobiology of Cigarette Smoke-Induced Chronic Obstructive Pulmonary Disease." *North*: 1047-1082.
16. Tzortzaki, E. G., and Siafakas, N. M. "A hypothesis for the initiation of COPD." *The European respiratory journal : official journal of the European Society for Clinical Respiratory Physiology* 34, no. 2 (August 2009): 310-5. <http://www.ncbi.nlm.nih.gov/pubmed/19648516>.
17. Cohen, J. and Powderly, W. 2004. "Infectious Diseases. 2nd ed. Mosby (Elsevier),. Chapter 33: Bronchitis, Bronchiectasis, and Cystic Fibrosis.
18. Hueston, W. J. 1997. "Antibiotics: neither cost effective nor 'cough' effective". *The Journal of Family Practice*, 44 (3): 261–5. PMID 9071245.
19. Yorgancıo lu, A., N., Yardım, P., Ergün, C., Karlıkaya, A., Kocaba , D., Mungan, F Yıldız, et al. 2010 "Integration of GARD Turkey national program with other non-communicable diseases plans in Turkey." *Tüberküloz ve toraks* 58, no. 2: 213-28. <http://www.ncbi.nlm.nih.gov/pubmed/20865577>.
20. Corporan, E., Adam, Q., Matthew, DeW., Chun, L., Shannon, S., Yongxin, Z., Sung, L., et al. 2008 "Effect of Meteorological Parameters on Fine and Coarse Particulate Matter Mass Concentration in a Coal-Mining Area in Zonguldak, Turkey." *Journal of the Air & Waste Management Association* 58, no. 4: 543-552. <http://secure.awma.org/onlinelibrary/doihandler.aspx?doicode=10.3155-1047-3289.58.4.543>.
21. Ates, N. A., Yildirim, H., Ercan, B., Saritas, E., Ünlü, A. and Atik, U. "GSTP1 as increased risk factors for asthma." *Respirology* 493-498.
22. Karaca, F., Alagha, O., Ertürk, F., Yılmaz, Y. Z. and Özkara, Y. 2008 "Seasonal Variation of Source Contributions to Atmospheric Fine and Coarse Particles at Suburban Area in Istanbul, Turkey." *Environmental Engineering Science* 25, no. 5: 767-782. <http://www.liebertonline.com/doi/abs/10.1089/ees.2007.0170>.