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The Environmental and Health Impacts of Coal Thermoelectric Plants

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The Environmental and Health Impacts of Coal Thermoelectric Plants

Chapter 1 Assessment of health effects of the air pollution associated with emissions from Coal Thermoelectric plants

Chapter 2 Health Effects of Fine Particulate Matter and Contributions from Sources

Chapter 3 Evaluation of Recent Estimations of Impact of Air Pollution on Health in Europe.

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Executive Summary

Air pollution is a source of concern for individuals, scientists and regulators and has been a focus of considerable research activity in recent years. There are many sources which contribute to air pollution and some of these pollutants can negatively impact on human health. One of the contributors to pollution is the burning of coal, which is a widely available, abundant combustible and, consequently, widely used for a variety of purposes including electricity generation. Health concerns have been raised regarding the safety of several aspects of coal use and this Report attempted to respond to three major issues.

In **Chapter 1**, health effects related to pollution from coal-thermoelectric plants are reviewed. Overall, from the studies with reasonable methodology, no increased or decreased health risks were associated with coal plant pollution in either workers in such plants or residents in the vicinity of plants. There is currently no evidence of an increased risk of death or other health effects associated directly with pollution from coal power plants.

In **Chapter 2**, the effects of fine particle pollution to which coal emissions contribute are presented. Short term effects of exposure to fine particulates have been consistently reported in adults with increased risk of all-cause mortality, more specifically cardiovascular and respiratory mortality. Long-term exposure to fine particulates reveals consistently increased mortality rates associated with increasing levels of $PM_{2.5}$. In addition, decreases in air pollution levels have been associated with decreases in adverse health effects. The *Commercial, Institutional and Household* sector emissions represent 52% of $PM_{2.5}$ in EU-27. The *Energy Production and Distribution* sector is a major contributor of SO_x and it has shown the greatest decrease since the 1990s: emissions of $PM_{2.5}$ and PM_{10} have been reduced by a factor 3. In Italy, the *Energy Production and Distribution* sector is the eighth largest contributor to $PM_{2.5}$, and the eighth largest contributor to PM_{10} emissions, and emissions from these sources have dropped markedly over the past decades.

In **Chapter 3**, some recent estimations of the contribution of coal emissions to mortality from air pollution are discussed. Coal-fired power stations are emitting a certain quantity of $PM_{2.5}$ although these emissions are far lower than those resulting from other human activities such as household heating and road traffic and have been falling consistently and significantly in recent time. The *Stichting Onderzoek Multinationale Ondernemingen* (Centre for Research on Multinational Corporations; SOMO) has produced a Report which attempted to compute the contribution of ENEL's coal power plants to the global burden associated with $PM_{2.5}$ pollution in Italy. However, their report failed to a large extent to provide a robust, scientifically sound estimation.

To reach the European goals of air pollution limits for 2020, all sectors require to make efforts to reduce their emissions of atmospheric pollutants recognising that frequently the largest contribution to local pollution may be a variety of dispersed sources. However, specific actions must target the major sources of Particulate Matter emissions, namely Household Heating Sources and pollution from Road Traffic sources. Household use of coal, wood and biomass burning should be discouraged and solutions with low emission of $PM_{2.5}$ should be preferred.

To investigate and monitor the impact of air pollution on health, it is absolutely essential to prepare viable models that take into account all sources of pollution and not simply refer to a single, potential source. This is an absolute principle if the focus is, as it should be, on improving Public Health.

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The Environmental and Health Impacts of Coal Thermoelectric Plants

Chapter 1

Assessment of health effects of the air pollution associated with emissions from Coal Thermoelectric plants

List of abbreviations

95% CI: 95% confidence interval

ALRI: acute lower respiratory infections

BaP: benzo[a]pyrene

COPD: chronic obstructive pulmonary diseases

ESP: electrostatic precipitator

ETS: environmental tobacco smoke

FEV_1: forced expiratory volume in 1 second

$FEF_{25-75\%}$: forced expiratory flow et 25-75% of maximal lung volume

FVC: forced vital capacity

FGD: flue-gas desulphurisation

GIS: geographical information systems

HWE: healthy worker effect

IARC: International Agency for Research on Cancer

IGCC: integrated gasification combined cycle

NMSC: non-melanoma skin cancer

NS: not statistically significant

OR: odds ratio

OSHA: Occupational Safety and Health Administration

PAH: polycyclic aromatic hydrocarbon

PEF(R): peak expiratory flow (rate)

PFT: pulmonary function test

PM_x: particulate matter (diameter $\leq$ x μm)

RR: Relative risk

SCR: selective catalytic reduction

SMR/SIR: standardised mortality ratio/standardised incidence ratio

TSP: total suspended particles

WHO: World Health Organization

Chapter 1 Summary

Coal is a cheap and abundant combustible. It is widely used worldwide for electricity generation. However, health concerns have been raised regarding the safety of several aspects of coal use. Health effects of household use of coal and coal mining have been extensively studied in recent years. Health effects related to emissions from coal-fired power plants, with much lower exposure levels, has also been the subject of research, although no comprehensive review exists to date.

In order to conduct a qualitative and quantitative review about health effects related to pollution from coal-fired power plants, a literature search was carried out in the PUBMED database. Articles eligible had to report any health effect in humans related to any pollutant produced by coal-fired power plants or related to proximity of residence to coal power plant. For each study, exposure assessment method, definition and specificity of exposure, temporality, adjustment for confounders, strength of association and statistical power were evaluated. Main results of studies were then summarized.

In total, 39 relevant studies were identified, of which 6 were conducted among coal plant workers, 6 studied prenatal exposure to coal plant pollutants, 14 were conducted in children and 13 in adults. All-cause and cause-specific mortality, asthma and other respiratory diseases, lung function tests such as forced vital capacity, forced expiratory volume during first second and peak expiratory flow, were the main health outcomes studied. As health outcomes, assessment of exposure and measures of association were too heterogeneous to allow any meta-analysis to be conducted. In occupational studies, decreases in lung function were found in boilermakers although such workers were also likely to be exposed to other hazards, notably asbestos. Among studies investigating prenatal exposure to coal plant pollutants, none had an appropriate design to enable any conclusion to be drawn. Children were the most widely studied population. Some statistically significant associations between coal plant pollutant exposure and respiratory function were found in ecological studies but none could properly exclude the role of confounding factors such as tobacco smoking of parents or socio-economic status. Only one study in adults had a clear design which took into account potential biases and confounding factors, with in addition an individual exposure assessment. This case-control study showed no increased risk of lung cancer associated with pollution exposure from coal power plants.

The overall quality of the studies was low. Importantly, the commonest study design was ecological, where both outcome and exposure were measured independently. Second, exposure assessment was not evaluated properly in most studies because of the absence of individual data and the multiple sources of exposure. Third, the majority of studies could not adjust for the most influential confounding factors such as tobacco smoking, which makes results prone to confounding bias. Overall, from the studies with reasonable methodology, no increased or decreased health risks were associated with coal plant pollution. These studies suffer from low statistical power which limits conclusions on potential small health effects.

Because of the difficulty to properly define exposure at individual level and because of the multiple sources of exposure, epidemiological studies faced numerous limitations. There is currently no evidence of an increased risk of death or other health effects associated directly with pollution from coal power plants.

1. Assessment of health effects of the air pollution associated with emissions from Coal Thermoelectric plants

1.1. Introduction

Coal is the largest source of energy for the generation of electricity worldwide[1]. It is a highly variable fuel, depending on carbon content, volatile matter, moisture and calorific value. Anthracite, bituminous coal and lignite are examples of coal grades (Table 1.1).

Peat is sometimes considered as a coal, and sometimes as a coal precursor. Coal combustion produces inorganic compounds such as carbon monoxide, sulphur dioxide and nitrogen oxides, but also polycyclic aromatic hydrocarbons (PAHs), aldehydes, ketons, metals (including mercury, arsenic and lead), and other compounds. Moreover, coal smoke/dust contains various particles, in majority fine ($PM_{2.5}$) and ultrafine ($PM_{0.1}$) particulate matter (IARC 2010a; IARC 2012c).

1.1.1. Coal-fired power plants

A coal thermoelectric plant (frequently referred to as a coal-fired power plant and subsequently referred to as coal plant) is a power plant that burns coal, either solid or gaseous (the latter being very scarce (BINE Informationdienst, 2006)), to produce electricity. Various grades of coal are used for power production (lignite, bituminous coal, anthracite). Coal is first crushed into a fine powder which is then burned. The heat produced by coal combustion is used for boiling water. Then, water is transformed into steam which activates turbines that finally produce electricity. In coal plants, there are two main categories of boilers. The oldest type is known as "positive pressure boiler". In these boilers, pressure inside the boiler is higher than outside. Therefore, when leaks occur, gases and fly ash may escape and contaminate the work environment. Such leaks are not uncommon as the boiler keeps expanding and contracting due to changes in temperature. The other coal boiler is called "balanced draft" boiler, and is operated under negative pressure. Nowadays, the majority of coal-fired power plants use balanced draft boilers (Bird et al, 2004).

1.1.2. Potential exposures of coal-fired power plants workers

In order to define exposures related to coal plant emissions, exposures occurring in coal plant workers are very useful. Indeed, this population is by definition the most highly exposed to coal plant emissions.

[1] http://www.iea.org/stats/index.asp. Accessed 23/11/2012

A study conducted in five coal plants in the United States provides a good overview of exposure sources from coal plants (Bird et al, 2004). Air and noise samples were made in each power plant, using either personal samples or area samples. Within air samples, the authors focused on asbestos, arsenic and respirable dust. Heat stress was also assessed. Recorded levels of exposure were compared to current US norms. Among the 203 respirable dust samples, only 4 were above the limit of detection (0.13-0.37 mg/m^3), and one was above recommended limits (5.3 mg/m^3, whereas the limit is 2.4 mg/m^3). For arsenic, all 128 samples were below the limit of detection (0.37-0.72 µg/m^3) and below the recommended limits (10 µg/m^3). Concerning asbestos, 12 of the 61 samples were above the detection limit (0.003 fibre/cm^3), but ranged between 0.003 and 0.007 fibre/cm^3, which is below the recommendations (0.1 fibre/cm^3). The major concern for coal plants workers was noise: 55 of the 302 noise samples (i.e. 18%) were above the recommended limit of 85 dBA, and 12 were above 90 dBA. Finally, temperature in coal plants ranged from 26 to 64°C. Heat stress exceeded recommended limits in two of the five coal plants.

Potential exposures occurring in coal plants have been reported by Bridbord et al (1979) and Bird et al (2004). First of all, coal plant workers may be exposed to coal dust whenever handling coal (for example, coal storage and crushing). Health effects of coal dust are described elsewhere (cf. 1.5.3). Secondly, in coal plants using gaseous coal (IGCC power plants), workers are also exposed to the potential health effects of coal gasification (cf. 1.5.2). Third, potential exposures during boiler leaks include SO_2 and its reaction products, NO_x, CO, fly ash, unburned hydrocarbons, PAH and aldehydes. However, the levels of these pollutants in coal plants are not precisely known, as few studies were conducted on this issue (Bird et al, 2004).

Sulphur dioxide (SO_2) is an irritating agent which may decrease lung function, either temporarily (acute exposures) or permanently (chronic exposures), and cause various pulmonary symptoms such as chronic bronchitis. Moreover, SO_2 reaction products (sulphates, sulphites, sulphuric acid and so on) are more toxic than SO_2, because they can be transported into the lung (Bridbord et al, 1979; IARC 2012a; Guidotti 1979; SOMO, 2012). Nitrogen oxides (NO_x) have similar effects on health. In another report (SOMO, 2012), ozone (O_3) was also reported as a potential pollutant produced by coal plants. Health effects of ozone are mainly respiratory, like SO_2 and NO_x.

Carbon monoxide (CO) is formed during incomplete combustion of coal. This gas is highly toxic and can be deadly at high doses. Coal plant workers may be exposed to CO during boiler leaks, in particular during start-up and shut-down operations (Bridbord et al, 1979).

Fly ash is also produced during coal burning processes. The ashes are typically 0.5 to 10 µm in diameter ($PM_{0.5-10}$). The smallest particles of fly ash ($PM_{2.5}$) are respirable and can be deposited into the lung. Long-term exposure to fine particulate matter was shown to increase lung cancer and cardiopulmonary mortality (Pope et al, 2002; Englert, 2004). Composition of fly ash is variable and can include trace of toxic and/or carcinogenic elements like mercury, silica, beryllium, lead, cadmium, arsenic, selenium, thallium, antimony and vanadium (Bridbord et al, 1979; IARC 2012a; Ito et al, 2006; Bird et al, 2004). Current regulations in the USA, issued by the Occupational Safety and Health Administration (OSHA), impose to have less than 2.4 mg/m^3 for dust containing silica, less than 5 mg/m^3 for other dusts, and 10 $\mu g/m^3$ for arsenic (Bird et al, 2004).

Unburned hydrocarbons can be found in coal plants stack emissions. Among them, polycyclic aromatic hydrocarbons and benzo[a]pyrene have the most potential for harm. These compounds are Group 1 carcinogens for lung cancer (IARC 2010b; IARC 2012b).

Exposure to asbestos has also been reported (Bird et al, 2004; Bridbord et al, 1979). Asbestos is a well-established lung carcinogen (IARC, 2012a). It is used for thermal insulation in certain areas of coal plants (Bridbord et al, 1979). Asbestos was detected, but remained below authorized limits (measured: 0.003 $fibre/cm^3$; OSHA limit: 0.1 $fibre/cm^3$) in several coal plants in the USA as of 2001 (Bird et al, 2004).

Finally, noise and heat are two other health issues occurring in coal plants. Noise is a major problem during coal crushing operations, in turbine room and boiler bays, but can be prevented with use of personal protective equipment. Average noise levels can reach more than 85 dBa in some coal plants (Bird et al, 2004). Heat stress is an issue in the boiler bays (Bridbord et al, 1979).

In modern power plants, devices such as electrostatic precipitators (ESP), selective catalytic reduction (SCR) and flue-gas desulphurisation (FGD) are used (Ito et al, 2006; Penney et al, 2009). These equipments reduce the levels of PM (ESP) nitrogen oxides (SCR) and sulphur oxides (FGD) released in the atmosphere. ESPs are very common worldwide, but FGDs are less frequent in developing countries because of their high cost (Penney et al, 2009; Thanh and Lefevre, 2001).

In summary, the most reported exposures from coal plants are PM_x, SO_2 and NO_x. It should be noted that these substances are not only produced by coal plants, but also by traffic (road, rail and air), power production using other sources (gas, fuel, oil), and a wide variety of other industrial processes. Therefore, identifying the contribution of coal plant emissions to the totality of atmospheric pollution using regional-based air monitoring stations is greatly limited by the contributions from other sources.

1.1.3. Potential health outcomes

Suspected health outcomes related to the exposures defined previously are numerous, and were outlined in several reports (SOMO, 2012; EEA, 2011; Penney et al, 2009), literature reviews (Jedrychowski, 1999; Pope, 2000; Englert, 2004; Valenti et al, 2011) and original articles (Pope et al, 2002). These health effects may occur either after short-term exposure or after long-term exposure. Finally, several health outcomes were identified:

- Lung cancer (incidence and mortality; long-term effect) and other cancers;
- Other pulmonary diseases. This category is very broad and includes the following items: asthma, respiratory difficulties, irritation of the airways, chest tightness, chronic bronchitis;
- Respiratory performance test (FVC, FEV_1,);
- Prenatal outcomes;
- Children development;

1.1.4. Objectives

The objective of the present report is to provide an assessment of any adverse health effects of the air pollution associated directly with emissions of coal power plants (Chapter 1). The contribution of such emissions to total air pollution levels and its impact on health will be assessed separately (Chapter 2).

As no study reported an assessment of the contribution of coal power plant to the total air pollution, but rather focussed on specific pollutants as marker of pollution level (NO_x, SO_2, PM_{10}, $PM_{2.5}$), the two chapters have been merged.

In section 1.2, the methodological strategy used for this review is outlined. In section 1.3, health effects of air pollution associated with emissions of coal power plants are presented. In section 1.4, effects of coal plant air pollution on mortality are discussed. In this latter section, modelling studies, that estimated the specific contribution of coal power plants in the context of total air pollution, are reviewed. Section 1.5 reviews all other aspects associated with coal other than coal power plants (household use of coal, coal gasification, coal dust and coal mining).

1.2. Methodology of literature search

1.2.1 Literature search strategy

A literature search was conducted as of November 30th, 2012 in the PUBMED database[2]. The following request was searched:

coal-fired power plants OR coal-burning power plants OR coal-fuelled power plants OR (coal AND power plants) OR (coal AND thermoelectric)

1,029 articles were found. In order to be included in qualitative review, articles had to meet the following criteria:

[2] http://www.ncbi.nlm.nih.gov/pubmed Accessed 27/11/2012

(i) report any health effect related to air pollution from a coal-fired power plant;
(ii) study in humans;
(iii) to have an ecological, cross-sectional, case-control, cohort, or ecological design;
(iv) written in English.

There was no geographical or temporal restriction for studies. Reviews, case reports and editorials were not considered as eligible. Articles were first screened by title, then abstracts. When the abstract was relevant, full-text articles were retrieved. In total 39 relevant articles were identified from this search.

1.2.2 Qualitative assessment

All articles were fully read by two co-authors. For each article, a short summary of main results of the study, and its strength and limitations, was written. Quality of articles was evaluated using the following criteria:

- *Exposure assessment method*. This criterion was met when exposure assessment was done at individual level rather than for populations (for example, based on distance to coal plant);
- *Definition and specificity of exposure*. This criterion was met when pollutants from the coal plant were clearly defined and actually measured or modelled in the study area;
- *Temporality*. This criterion was met when the outcome was measured after the exposure occurred, and when the lag time between exposure and outcome was credible for the considered outcome;
- *Adjustment for confounders*. This criterion was met when basic confounding factors (smoking status, age, sex, socio-economic status....) were taken into account when measuring the association between exposure and outcome;
- If any association was found, *strength of this association* and its statistical power was evaluated;
- If any association was found, the presence of a *dose-response relationship* was assessed. A dose-response relationship is an indicator of causality.

1.2.3 Quantitative assessment

Observational studies were grouped according to the population in which they were conducted: occupational studies, prenatal exposure, studies in children and studies in adults. Studies with mortality as outcome were reviewed separately, regardless of the population under study. Modelling studies are also presented but no quantitative assessment is feasible on such studies.

For each population, a table summarising health outcomes, exposure assessment and measure of association was created. It had originally been envisaged to conduct meta-analysis for developing pooled estimates from studies with similar outcome (health condition) and exposure. A pooling of studies is feasible when a measure of association is reported (Odds Ratio, Relative Risk, Standardised Mortality Ratio, Hazard Ratio, or effect size). PRISMA guidelines (Moher et al, 2009) were followed to define methodology for any meta-analysis as well as the method developed by van Houwelingen (van Houwelingen et al, 2002) for the statistical analysis of data based on random-effects model.

1.3 Evaluation of health effects

In this section all articles studying health effects related to coal plants are reviewed. For each study, a short summary of results has been made. It is followed by a qualitative evaluation (italics and in parentheses). Quality assessment of articles is summed up in Table 1.10. In this table, a "X" means that the criterion is met, a "X-" means the criterion is partially met, and a blank means that the criterion is not met.

1.3.1 Occupational exposure

Three articles (Bencko et al, 1988; Hauser et al, 2001; Manna et al, 2003) studied coal plant workers. This population is exposed more directly to coal plant pollutants than the general population and at much higher levels. Therefore, if any health risk is present, it will be likely detected first in this population.

Bencko et al (1988) studied immunological profiles of coal plant workers (Bencko et al, 1988). In this study, 47 men working in a plant burning coal rich in arsenic (900 to 1,500g of arsenic per ton, dry weight) were compared to 27 men working in another coal plant, located in the same district, but burning coal 10 times less rich in arsenic. Groups were similar in terms of age, place of residence of type of work. Serum concentrations in immunoglobulin (IgG, IgA, IgM), α-1-antitrypsin, α-2-macroglobulin, transferrin, orosomucoid, ceruloplasmin and lysozyme were measured in the two groups. Tranferrin, ceruloplasmin and orosomucoid levels were significantly more elevated among workers of the arsenic-rich coal plant ($p<0.01$). Among factors associated with immunological profile, age of the workers was a significant determinant of IgA levels, α-2-macroglobulin and Transferrin. In the arsenic-rich coal plant, 21% of workers had all tests within normal limits; 28% had one abnormal test and 51% had 2 or more abnormal tests. In the other plant, percentages were 59%, 37% and 4%, which is expected in a "normal" population.
[In this study of Bencko et al, (1988), the association between duration of work in coal plant and protein level was in the same order of magnitude as the association between age and protein level. As no control is made to adjust for age or other factors such as smoking, the significant association described with exposure in coal plant could be due to confounding. This study also suffers from a small sample size and low statistical power to detect actual differences. The selection of workers included in the study is not detailed and could lead to selection bias.]

In a prospective study, Hauser et al (2001) examined decreases in lung function among boilermakers working in power plants. This population of workers is not specifically exposed in coal power plants and could work in other type of power plants (oil, gas, nuclear) and other industries (trash incinerator, paper mill). They are also exposed to several other components (cf 1.2.2). In this study, a cohort of 118 American boilermakers was followed for 2 years. At baseline (1997-1998), subjects underwent spirometry tests (forced vital capacity FVC and forced expiratory volume during the first second FEV_1) and completed a questionnaire on health condition. Subjects completed 2 additional spirometry tests and 3 detailed work history questionnaires during follow-up. The objective of this study was to analyse in linear regression models, taking into account auto-correlation, the associations between FEV_1 and occupational exposures. These models adjusted for confounders (age, tobacco smoking, years as a boilermaker). During the first year of follow-up, 65 individuals (66% of the cohort) worked in a coal plant, with a mean duration of 176 hours (range 10 to 782 hours). During the second year, 60 individuals worked in a coal plants and the mean duration was 297 hours (range 53-1090 hours). Most boilermakers worked in several types of power plants: 70% of them worked at 3 and more types of plants. After adjusting for age, baseline FEV_1 and smoking status, total hours worked regardless of power plant type were not associated with decreases in FEV_1 (-0.4 [-5.6; 4.8] mL/100 hours worked). Working in a coal plant was negatively associated with FEV_1, but this association was not statistically significant: -7.6 [-22.4; 7.2] mL/100 hours worked. In a further model (model 4) adjusted for ever worked on specific fuels, significant associations were found between ever working in a coal plant during follow-up and FEV_1: the decrease was -73.4 [-128.8; -18.0] mL for subjects having ever worked in a coal plant.
[This study of Hauser et al, (2001) has some methodological strengths: an individual assessment of exposure (before and after), an analysis taking into account auto-correlation and adjusted for potential confounders (including tobacco smoking). However, some limitations are present such as a potential selection bias since workers were selected among participants in a worker union, among which only 35% were contacted. The last model (model 4), which produced significant associations, also present apparent incoherent results as compared to other models (models 1 to 3). This last model was not based on mL/100 hours, but on ever worked in a power plant. Finally, the population studied is potentially exposed to several components in different industries, which limit the extrapolation of the results to coal plant only. The analysis also shows same outcome whatever the source of exposure (oil, gas, coal)]

In an ecological study conducted in India, Manna et al (2003) examined a group of 50 workers directly engaged in the process of coal handling at a coal plant for at least 5 years. General characteristics (age, sex, etc), smoking history, history of past diseases and clinical examinations (weight, height, eye-sight, hearing, pulse, blood pressure) were recorded. Mean age of participants was 34.8 years, with on average 7.9 years of job duration. Most workers (77%) smoked bidi, a sort of Indian cigarette. Respiratory morbidities were found in 72% (i.e. 36 out of 50) of workers. In particular, 60% (30 subjects) of workers had sore throat and 14% (7 subjects) had infective lung disease. No significant associations were found between use of personal protective equipment and respiratory morbidity; or between tobacco use and respiratory morbidity ($p=0.75$).

[The population of workers in this study of Manna et al (2003) is not compared to another reference group. The only useful result for risk assessment is the comparison of respiratory morbidity associated with use of protection methods. This study has a very small sample size. Health outcome is defined as "respiratory morbidity" with no further details].

Table 1.2 summarizes features of studies conducted in coal plant workers. Two studies were conducted on respiratory complaints, but outcomes were not homogeneous (FEV_1 and respiratory complaints and diseases for Hauser et al (2001) and Manna et al (2003) respectively). One study evaluated immunological profile of coal plant workers (Bencko et al, 1988). No meta-analysis was conducted on these data due to the large degree of heterogeneity in the exposures and the outcomes.

1.3.2 Environmental exposure

1.3.2.1 Studies of pregnancy outcomes/prenatal exposure

Two studies, one ecological and one cohort, were conducted in Croatia (Mohorovic, 2003; Mohorovic, 2004; Mohorovic et al, 2010) and in China (Perera et al, 2012; Tang et al, 2006; Tang et al, 2008) on pregnancy outcomes and prenatal exposure to coal plant pollutants.

The first study was conducted in Labin, Croatia, at the end of the 1980s (Mohorovic, 2003; Mohorovic, 2004; Mohorovic et al, 2010). Plomin 1 thermo-electrical coal plant was considered a major source of air pollution in the area. Every hour of operation, the plant was emitting about 8.5 tons (18,080 mg/m^3, or 6,900.8 ppm) of SO_2 in addition to NO_x, CO_2, CO, TSP, iron, titanium, vanadium, chromium, nickel, copper, zinc, selenium, lead, and other products of coal combustion. The coal used in this plant had very high sulphur content (9-11%) and high radioactivity (300 Bq/kg). The study design is ecological. It focuses on pregnancy outcomes and the prenatal health of the mother associated with the time when the coal plant was operating. In 1987, power plant Plomin 1 was operating almost throughout the year (4.772 work hours). In 1988 and 1989, it did not work at full capacity (2.754 and 579 work hours, respectively) (Mohorovic, 2004).The plant was closed from February to September 1989. These periods were used as a proxy of exposure by defining them as "clean period" (closed plant; April to July 1989) and the "dirty period" (December 1989 to March 1990) (Mohorovic, 2003).

Methaemoglobin and haemoglobin were assessed with blood and urine samples in 260 pregnant women: 138 during the "clean period" and 122 during the "dirty period" (Mohorovic, 2003; Mohorovic et al, 2010). There was a significant positive correlation ($r=0.72$) between daily SO_2 levels and methaemoglobin levels. Higher levels of methaemoglobin (>1.5g/L) were found in mothers of stillborn babies. The frequencies of miscarriages and stillbirths were significantly lower in the clean period. Methaemoglobin concentrations significantly changed with time: decreased during the "clean period" and increased in the "dirty period". The level of sulphates in urine samples significantly decreased during the clean period, whereas it increased in the dirty period, but not statistically significantly.

A second publication reported results based on a larger sample size: 704 women who gave birth to babies between 1987 and 1989 (Mohorovic, 2004). This study investigated the association between coal plant pollutants and preterm delivery (<37 weeks) and low birth weight (<2500 g). SO_2 emissions were significantly negatively correlated with the length of gestation (-0.0914 and-0.0806, for exposure during first month or second month of pregnancy respectively) and lower birthweight of newborns (-0.0807 and -0.0733, for exposure during first month or second month of pregnancy respectively). The risk of premature birth was increased in 1987, when the power plant was fully operating [RR = 1.76 (p=0.026)] as compared with 1988 and 1989 when the coal plant was partially operating (Mohorovic, 2004).
[This study (Mohorovic, 2003; Mohorovic, 2004; Mohorovic et al, 2010) has an ecological design, with exposure defined as period of operation of the local coal power plant. The seasonal variation in methaemoglobin concentration is considered as being due to air pollution for the coal plant, while several other seasonal factors could have played a role. No confounding factors were investigated in this study. Finally, there is no individual exposure assessment.]

The Chinese cohort study was conducted in Tongliang, a 810,000-inhabitants district of the Chongqing municipality (Central China). The cohort was constituted by 150 children born between 04/03/2002 and 16/06/2002 from non-smoking mothers living close to a coal power plant (within 2.5km). This power plant was the main source of air pollution in the city until 2004. No modern anti-pollutant technologies were used in this plant, so the emissions were very high in this area, in particular for $S0_2$. Domestic heating and cooking units were converted to natural gas as of 1995, hence there was no confounding with exposure from household coal emission.

For every child, PAH-DNA adducts were measured in cord blood and in also in mother's blood at delivery. Mothers were interviewed for socio-demographic variables, history of active and passive smoking, exposure to environmental tobacco smoke during pregnancy and other exposures. Children were followed from birth to 30 months for assessing the child development (height, weight, head circumference), until 2 years for developmental quotient (DQ) and until 5 years for intellectual quotient (IQ).

The first study with a follow-up from birth to 30 months showed that 80% of babies had a detectable level of PAH-DNA adducts (on average 0.33 ± 0.14 adducts/10^8 nucleotides)(Tang et al, 2006). This was higher than in mother's blood (0.29 ± 0.13/10^8 nucleotides) while cord blood levels were expected to be 10 times lower than maternal blood. High cord blood level was not associated with head circumference or height at all ages. But there was a significant lower weight associated with PAH-DNA adducts from age 18 months onward. The authors also investigated the role of duration of exposure: no association was found for weight and head circumference, but there was a significant decreased height.

The children's developmental quotient (DQ) at age 2 was evaluated and presented in a second report (Tang et al, 2008). After some loss to follow-up, 110 children of the cohort took part in this study (out of an initial 150 babies recruited). In addition to DNA-PAH adducts, the authors also investigated lead and mercury levels in cord blood. Mean lead and mercury concentrations were 3.6 ± 1.59 µg/dL and 7.0 ± 4.43 µg/L, respectively. The DQ test had 4 parts: motor behaviour, language behaviour, adaptive behaviour and personal/social behaviour. Overall, increased cord blood adducts were associated with lower scores in DQ, and in particular for motor and language domains. High cord blood lead levels were associated with decreased overall and social DQ. Finally, an increase in 0.1 adduct/10^8 nucleotides increased the risk of being developmentally delayed in motor area by 91% (multiple-adjusted OR=1.91 95%CI [1.22; 2.97]). An elevated lead level increased the probability of motor delay (multiple-adjusted OR=3.85 95%CI [1.04; 14.25]) and of social delay (multiple-adjusted OR=7.29 95%CI [1.35; 39.45]).

Children's intellectual quotient (IQ) was evaluated among the 100 children remaining in the study at 5 years of age (Perera et al, 2012). In this part, researchers studied in particular the relationship between environmental tobacco smoke (ETS) exposure during pregnancy and child's IQ. The IQ tests had a verbal scale, a performance scale and a full scale. Both ETS exposure and PAH-DNA adducts were associated with decreases in IQ, but these associations were not statistically significant. However, significant interactions between ETS and adducts were found. Among children with detectable adducts, a one hour/day increase in prenatal ETS corresponded to significant decreases of 10.07 (95%CI [17.29; 2.85]) points in the full IQ scale, 8.81 (95% CI [16.43; 1.19]) points in verbal IQ, and 9.79 (95%CI [18.19; 1.39]) points in performance IQ (Perera et al, 2012).
[This study (Tang et al, 2006; Tang et al, 2008; Perera et al, 2012) has an appropriate design, it is prospective, an individual measurement of PAH-DNA adducts is performed, and the children are followed from birth to the age of 5. Some association were found between PAH-DNA adducts and child development but shows an inconsistent pattern: e.g. association with PAH but not with duration for weight. The evaluation of PAH-DNA adducts is used as a proxy of exposure to coal power plant pollutants, but this is not specific of coal plan pollution and could be due to external causes. In the analysis of IQ, no association was found. In a stratified analysis, a decreased IQ was associated with PAH-DNA adducts, but this analysis is to be viewed with caution as it covers a subgroup of the population under study.]

The main characteristics of studies assessing health effects related to prenatal exposure to coal plants are displayed in Table 1.3. Two studies using the same data reported mother's methemoglobin levels, but no measure of association between exposure and pregnancy outcome were used (Mohorovic, 2003; Mohorovic et al, 2010). Birth weight was studied in two studies (Mohorovic, 2004; Tang et al, 2006), but there was neither common assessment of exposure nor common measure of association. Weights at other age, height, head circumference, developmental quotient at age two and intellectual quotient at age five were also evaluated. For these outcomes, only one study per outcome was available. As outcome or health effects were distinct between each study, it was not possible to perform any meta-analysis of these data.

1.3.2.2 Studies in children

Several studies have been conducted in children living near coal plants (Bencko and Symon, 1977; Goren et al, 1992; Peled et al, 2001; Peled et al, 2010; Goren and Goldsmith, 1986; Goren et al, 1988; Goren et al, 1997; Goren et al, 1991; Dubnov et al, 2007; Yogev-Baggio, 2010; Henry et al, 1991a; Henry et al, 1991b; Aekplakorn et al, 2003; Blanchard et al, 2011). These studies were conducted in the Czech Republic and Slovakia (Bencko and Symon, 1977), in Israel (Goren et al, 1992; Peled et al, 2001; Peled et al, 2010; Goren and Goldsmith 1986; Goren et al, 1988; Goren et al, 1997; Goren et al, 1991; Dubnov et al, 2007; Yogev-Baggio, 2010), in Australia (Henry et al, 1991a; Henry et al, 1991b), in Thailand (Aekplakorn et al, 2003) and in the United States (Blanchard et al, 2011).

In the seventies, a study was conducted in Slovakia (at that time part of Czechoslovakia) in an area close to a coal power plant burning a coal containing high dose of arsenic (900 to 1500g/tons of dry substance) (Bencko et al, 1977). The first part of the study investigated the arsenic contents in hair, urine and blood in 107 boys aged 9.5-11 years of age living in communities located 1.5-75 km from the coal plant (Bencko et al, 1977). A group of 44 boys living in an industrial city outside of the area of the power plant was used as control. Mean arsenic concentrations in children's hair ranged from 0.295 ± 0.413 to 3.793 ± 2.321 µg/g while this value was only 0.152 ± 0.279 µg/g for the control group. Urine arsenic concentrations were 0.0078 ± 0.011 to 0.0253 ± 0.025 mg/L on average and 0.0109 ± 0.016 in the control group. There was no association between proximity of residence and arsenic level in hair and urine. Blood levels were measured in some children from only two communities; levels were of 1.45 ± 23.1 and 4.53 ± 14.9 ppb in exposed children and 1.88 ± 21.7 ppb in control group.

A series of hearing test was conducted among 56 children (30 boys and 26 girls) living in the area close to the coal power plant, as well as in 51 control children (26 boys and 25 girls). For all frequencies and for air conduction and bone conduction, children living in the vicinity of the power plant had a significant decreased hearing threshold especially for low frequencies.
[In this study of Bencko et al (1977), blood levels were only measured in two communities and restricted to 10 children per group. This could constitute a strong selection bias. Children selected for hearing tests were not described. The arsenic measurements in hair and urine were not correlated with distance from coal power plant.]

Following the construction of a new coal power plant near Ashkelon in Israel in 1990, a study was conducted to follow population health for the residents living within 25 km of the power plant. Seven new air monitoring station were installed to specifically follow pollutants (SO_2, NO_x, hydrocarbons, O_3, CO, total suspended particulates) and climate data (wind speed and direction, temperature, relative humidity, barometric pressure, solar radiation and precipitation) in this area. Several health dimensions were followed-up: mortality, requests for health services, emergency room requests, and pulmonary conditions among school children. A further report was conducted on a selected population of 200 asthmatic children (Goren et al, 1992).

An initial report investigated pulmonary function, as measured by forced vital capacity (FVC) and forced expiratory volume during the 1st second (FEV_1), and proximity of residence. Children in 2nd, 5th and 8th grade of school were examined in 1990, 1994 and 1997 (2455, 1613 and 4346 children included respectively). Parents filled questionnaires about demographic variables (including tobacco smoking of the parents, parents' education level, crowding index, number of years of residence). SO_2 and NO_x were measured in 10 sites in the study area. Children resided in 5 cities: Ashkelon, Kiryat Gat, Sderot, Kiryat Malachi and Ashdod. The majority of variability across children (70%) was explained by sex, age, height and weight. Differences in FVC and FEV_1 were statistically significant between centres. FVC and FEV_1 values were higher in Ashkelon than in all other areas, after controlling for potential confounders. There was no clear association between air pollution (SO_2 and NO_x) and pulmonary function of children (Peled et al, 2001).
[This study of Goren et al (1992) describes a situation whereby a monitoring station was located in the city of Ashkelon where the coal power plant is active, but the pollution monitored was among the lowest as compared to another station located away from the power plant. Of note, another oil power plant is in activity in Ashdod in the northern part of the area. Hence, it is not possible to discriminate between the individual effects of the coal or oil power plant. In the Report of (Peled et al, 2001), age, height and weight of the children were similar in all centres; so were socio-demographic variables. However, there were important differences for the percentage of children born outside Israel, both between centres and between periods (1.5% in 1990; 39% in 1994). This was due to an important immigration trend during this period. This could create biases in the association between residence close to coal and pulmonary function.]

A subsequent study was conducted among 285 asthmatic children aged 10-12 living in the same area in 1999 (Peled et al, 2005). Children performed peak expiratory flow (PEF) tests twice daily for 5-6 weeks. Daily record of temperature, pressure, humidity, PM_{10} and $PM_{2.5}$ were retrieved from 6 monitoring stations. PEF was modelled with generalized linear models and generalized estimating equations. The objective was to evaluated effects of PM_{10} and $PM_{2.5}$ on PEF, controlling for demographic variables (age, sex, BMI), seasonal changes. PM_{10} concentrations were higher in Ashkelon, while $PM_{2.5}$ were higher in Sderot. Lung function of asthmatic children was negatively associated with air pollution by PM_{10} and $PM_{2.5}$ after controlling for meteorological, seasonal conditions, physical and clinical conditions and socio-demographic parameters (Peled et al, 2005).

[This study (Peled et al, 2001) suffers from a lack of specificity of exposure since two power plants are operating (in Ashkelon and Ashdod) and other sources of exposure are also present: traffic, desert dust, local industry. A difference in the highest values in PM_{10} and $PM_{2.5}$ is also problematic, if the highest exposure was suspected to be caused by the coal power plant of Ashkelon, the a large degree of consistency would have been expected. The modelling conducted seems rather problematic:

1. *tobacco from the parents is not selected in the final model while important differences exist between populations (e.g. women smoke twice more in Ashkelon than in Sderot).*
2. *the selection of variable is not described enough and vary from model to model, (e.g. age is included in the analysis of Ashkelon only, sex is only included in Ashdod, Sderot does not include either age or sex.)*
3. *several variables are likely collinear ($Cos(\omega_1 day)$ $Sin(\omega_2 day)$, T_{max}).*
4. *several interaction variables in the models are hardly interpretable (e.g. PM_{10max} X $Sin(\omega_2 day)$).]*

Goren and colleagues conducted a series of studies among children living in the vicinity of a coal plant located in Hadera, Israel (Dubnov et al, 2007; Goren and Goldsmith, 1986; Goren et al, 1993; Goren and Hellmann, 1997; Goren et al, 1991; Goren et al, 1992; Goren et al, 1995; Yogev-Baggio et al, 2010). The first study was conducted before the introduction in 1981 of the coal power plant, the second when two units of power plants were operating and subsequent reports refer to an era when all units were operating (since 1984). The most recent studies included a modelling of the pollution in the area based on air monitoring stations and GIS mapping. Children in second grade (7-8 years), fifth grade (10-11 years) living within 19 km of the coal plant were recruited and their pulmonary function was evaluated based on FVC, FEV_1, PEF tests. Respiratory symptoms and diseases were also recorded. Information on demography and lifestyle (including tobacco smoking) was also gathered from parents with questionnaire.

In the first study conducted before the starting of the power plant, analysis was restricted to the comparison of pulmonary function, symptoms and diseases between smoking and non-smoking parents (Goren and Goldsmith, 1986). No difference in respiratory symptoms was found between family with smokers and non smoking familial environments. Children in family with smokers had more ear infections and bronchitis, they also had lower respiratory performance tests. Children with pulmonary diseases in their families or with history of pneumonia had an increased risk of respiratory symptoms and diseases. Lower socio-economic status was associated with increased lung symptoms, but no clear association could be found for lung diseases.

[This study (Goren and Goldsmith, 1986) does not provide any information on risk associated with exposure to the coal plant. Reporting of respiratory symptoms and

diseases by the parents may be biased. No adjustment for confounders is performed.]

In the second report the health status of children was evaluated in 1983, the children were in fifth (10-11 years) and eighth grade (~13-14 years) (Goren et al, 1988). Prevalence of cough without cold (p=0.002) and measles (p=0.001) significantly increased in the younger cohort (fifth grade in 1983). Other respiratory symptoms and illnesses were more prevalent in 1983, but the increases were not statistically significant. In the older cohort (eighth grade in 1983), prevalence of respiratory symptoms/diseases were less prevalent in 1983 than in 1980, except for pneumonia (not significant increase), sputum without cold (p=0.036) and measles (p=0.017). Regarding change in FVC, FEV_1 and PEF between 1980 and 1983, there was an expected annual increase in FVC, FEV_1, PEF (normal development of children). The increases were smaller in communities labelled as having a "high pollution", the highest increases were found in "moderately polluted areas".
[This study (Goren et al, 1988) is a 'before-after' design. No specific information on the exposure to pollutant in children is available. For respiratory parameters, the differences between communities are statistically significant, but there is no clear trend.]

The third study was conducted in 1986 when the coal plant was fully operational. Second, fifth and eighth graders were included from three communities (Goren et al, 1991). SO_2, CO and NO_x measurements were also carried out from 01/1985 to 06/1986 in the 3 communities through a network of 12 automatic air monitoring stations within 25 km of the coal plant. This analysis reported higher increase in pulmonary functions in children living in areas labelled as "highly polluted". No clear association was found between pollution and lung disease and symptoms. This study also provided pollutants measurements by month for the three areas and there was no clear relation between the level of measured pollutants and the labelling of areas by the authors.
[This study (Goren et al, 1991) has similar limitations as previous study. Furthermore, the pollution evaluation from air monitoring stations shows that the labelling of highly, moderately, low pollution areas by the authors do not fit with actual measures.]

The fourth study published in 1997 investigates the pulmonary function, symptoms and disease of children from 1980 to 1989 (Goren and Hellman, 1997). An overall significant increase with time in asthma and asthmatic symptoms was described. Overall, respiratory performance test were lower in children with asthma.
[Again this study (Goren and Hellman, 1997) has same limitation as before and does not report health condition according to exposure to the coal power plant.]

In the same area, a further study was conducted in 1996-1999 in the same population source, i.e. children in 2nd, 5th, 8th grade (Dubnov et al, 1997; Yogev-Baggio et al, 2010). Respiratory performance tests were also performed (FVC, FEV_1). In this study, the pollution from the coal power plant was modelled from the 12 air station measurements (10 stationary and 2 mobile [Goren and Hellmann 1997]) located in the area. The modelling of air pollution was made employing a kriging method enabling an estimation of SO_2, NO_x pollution level for each place of residence. Overall a significant decrease of FVC and FEV_1 was described for exposure to NO_x and SO_2 after controlling for several confounders (including tobacco smoking, crowding index, parental education, and number of years of residence). The impact of NO_x and SO_2 on FVC and FEV_1 does not dependent on the child pulmonary health. *[The methodology of these two studies (Dubnov et al, 1997; Yogev-Baggio et al, 2010) is globally improved as compared to initial studies conducted in this population. For example, the new analysis included adjustment for confounding factors. This study also improved the assessment of exposure at an individual level by using modelling of pollution from air monitoring station. But this assessment is highly questionable and likely gives a false reassuring evaluation of pollution level:*

1. *Among the 12 air monitoring stations, only a are located in the area of residence of children.*
2. *There is a strong disagreement in the position of the air monitoring reported in two publications [Goren and Hellmann ,1997; Yogev-Baggio et al, 2010]. In all publication the authors report pollution evaluation from 12 stations of which only 2 station were mobile [Goren and Hellmann, 1997]. But when comparing maps of the publication of 1997 and 2010, stations numbered in 1997 "1","2","3","5","7" do not appear in the map in 2010, inversely 5 stations in 2010 do not appear in the maps of 1997.*
3. *The model with the krigging method is not sufficiently described and parameters of the krigging methods were not reported. No attempt was done to evaluate that the krigging extrapolation was accurate enough. This could however have been done for example by using 10 stations for krigging and 2 for validation.*
4. *The distribution of pollution from the model shows incoherence in the distribution of pollution with a white, unpolluted zone, in the vicinity of the coal power plant. This unlikely unpolluted area is immediately followed in the eastern part, by a peak in pollution 6-7 km from the power plant. This seems to be an artefact based on the position of air monitoring stations and from poor parameterisation of the krigging model.]*

Henry et al (1991a and 1991b) reported an ecological study in Australia comparing respiratory symptoms in schoolchildren living in two areas, one with coal power plants and the other without in 1986 and 1987. The "polluted" area is the region of Lake Munmorah where 2 coal plants are located within 5 km of the local school. The unpolluted area is the town of Nelson Bay (80km of Lake Munmorah) which has no major industry or pollution source nearby. In total, 201 children from Lake Munmorah and 401 from Nelson Bay participated in the study. Children were aged 8 to 9 years on average, the Nelson Bay children being slightly older (8.9 vs. 8.3 years, statistically significant). In Lake Munmorah, socioeconomic status of parents was lower than in Nelson Bay, parents were more likely to smoke, to use gas or wood for heating and not to use electricity for cooking. Parents of children filled questionnaires with basic socio-demographic data and data about their child's respiratory health and allergies. Children underwent spirometry testing, histamine challenge and allergen skin tests at baseline and after one year. At baseline and after one year, prevalence of asthma was more elevated in Lake Munmorah than in Nelson Bay, with statistically significant odds ratio ranging from 1.95 to 2.66 depending on the definition used for asthma. All odds ratio were statistically significantly increased, and did not change much with time. In multivariate analysis, the odds ratio remained in the same order of magnitude when adjusted for age, sex, smoking in home and dust mite. Other respiratory factors (dry cough without colds, chest colds and bronchiolitis) were significantly more prevalent in Lake Munmorah. Dry cough at night, bronchitis, eczema, hay fever and allergies were more prevalent in Lake Munmorah, but the associations were not statistically significant.

In a second publication, the investigators restricted the analysis to asthmatic children (Henry et al, 1991b): 49 from Lake Munmorah and 50 from Nelson Bay. From April to November 1987, SO_2 and NO_x concentrations were measured around-the-clock in Lake Munmorah and Nelson Bay schools. During follow-up, children of Lake Munmorah were more likely to be wheezing than those from Nelson Bay, but prevalence of events was low (10% for wheezing and 20% for all respiratory symptoms, weekly). Air quality was much higher in Nelson Bay: for example, the maximum hourly average of SO_2 and NO_2 were 43 µg/m^3 and 75 µg/m^3 in Nelson Bay; 139 µg/m^3 and 169µg/m^3 in Lake Munmorah. Air pollutant levels remained within recommended guidelines. In addition, there was no higher risk of wheezing on days with high SO_2/NO_2 concentrations compared with days with low pollution: the odds ratios were close to unity.
[This study (Henry et al, 1991a; Henry et al, 1991b) is an ecological study comparing symptoms in only two areas. Several factors external to coal power plant emission are influencing the outcome (asthma) investigated in these studies: tobacco, socio-economic status, use of gas or wood for heating, dust mite. Air quality measurements were not performed at an individual level.]

Aekplakorn et al (2003) studied changes in respiratory functions of children related to ambient air pollution in Thailand. Eighty eight asthmatic and 96 non-asthmatic children aged 6-14 were recruited in four villages of Mae Moh district, Thailand. The major source of pollution in the area was a lignite-fired power plant. Villages were located about 7 km south of the lignite plant. Children performed daily pulmonary function tests (FVC, FEV_1, PEFR, $FEF_{25\%-75\%}$) from 1/10/1997 to 30/11/1997 (61 days). During the same period, PM_{10} and SO_2, O_3 and NO_2 concentrations were measured in three villages. As O_3 and NO_2 were not detectable most of the days, they were not included in the statistical models. Temperature and humidity were also recorded. The association between air pollution and respiratory functions was analysed using general linear mixed models, controlling for gender, height (highly correlated with age and weight), village, temperature and weekday. During the study, SO_2 concentrations ranged from 0-4.92 to 99.01-128.01 µg/m^3, with a median of 4.58-17.29 µg/m^3 depending on the village. These values were relatively low as compared with Thai standards of 300µg/m^3. Likewise, PM_{10} levels ranged from 6.57-8.69 µg/m^3 to 60.39-92.02 µg/m^3 with a median of 22.12-24.66 µg/m^3. This was again lower than the Thai standards (120 μg/m^3). No clear correlation was found between PM_{10} and SO_2 levels, or between temperature and air pollution.

In asthmatic children, FVC, FEV_1 and PEFR had small significant declines associated with increases in SO_2 concentrations in univariate model. The associations were no longer significant after controlling for PM_{10}. No significant associations were found for healthy children. Regarding exposure to PM_{10}, significant decreases in FVC, FEV_1 and PEFR were found in asthmatic children: a 10 μg/m^3 increase in PM_{10} was associated with decreases in FVC of –0.33%, FEV_1 of –0.36%, PEFR of –0.42%, and $FEF_{25–75\%}$ of –0.17%, after controlling for SO_2, time and temperature. No significant effect was evidenced in non-asthmatic children.
[This study (Aekplakorn et al, 2003) is an ecological study. Pollution measurements were not conducted at individual levels but 3 villages out of 4 had a measurement done. No information was reported on exposure to tobacco smoking at home. No information was collected on socio-economic category.]

Blanchard et al (2011) conducted an ecological study about mercury concentration in ambient air and autism in two US counties: Bexar, Texas and Santa Clara, California. Only the county of Bexar has a coal power plant. The rate of autism was obtained from the Texas education association in 2007. Local mercury concentrations were extracted from ambient air estimation from 2002 data published by the US environmental protection agency. Overall, an increase in the prevalence of autism was observed for schools in districts with high concentration of mercury. No information by pollution source is reported
[This study (Blanchard et al, 2011) suffers from several limitations: first the design is ecological, there is no measurement of pollution (only estimate from US EPA), there is no control for confounders, there is neither measure of association nor uncertainty, data cannot discriminate the source of pollution. The definition of 'autism' has been subject to significant changes over the decades and between centres. It is also worth considering whether children with severe autism could require being educated in special school, the location of such schools is not randomly located but closer to city centre (where power plants are also likely located).]

Details of studies in children are summarised in Table 1.4. Respiratory function of children exposed to coal plant pollutants has been widely studied, using a variety of outcomes such as spirometric tests (FEV_1, FVC, PEF, $FEF_{25-75\%}$), asthma and other respiratory symptoms or diseases. FVC was a health outcome in 5 studies (Peled et al, 2001; Dubnov et al, 2007; Goren et al, 1988; Goren et al, 1991; Aekplakorn et al, 2003), each of them having a unique exposure assessment and measure of association. Similarly, FEV_1 was studied in 6 studies (Peled et al, 2001; Dubnov et al, 2007; Yogev-Baggio et al, 2010; Goren et al, 1988; Goren et al, 1991; Aekplakorn et al, 2003), PEF in 4 studies (Peled et al, 2005; Goren et al, 1988; Goren et al, 1991; Aekplakorn et al, 2003) and $FEF_{25-75\%}$ in one study (Aekplakorn et al, 2003). Studies having a common exposure assessment and measure of association were conducted in the same populations (e.g Goren et al (1988) and Goren et al (1991) conducted in the same schoolchildren), making a meta-analysis not relevant. Finally, hearing thresholds (Bencko and Symon, 1977), arsenic concentrations (Bencko and Symon, 1977) and autism (Blanchard et al, 2011) were evaluated although only one study was available for each outcome.

1.3.2.3 Studies in adults

There are remarkably few studies in adults which have evaluated health risks associated with exposure to pollutants from coal power plants. Only one study investigated risk of lung cancer (Pisani et al, 2006). Studies in Slovakia investigated risk of non melanoma skin cancer (NMSC) associated with arsenic from coal plants (Pesch et al, 2002; Ranft et al, 2003; Bencko et al, 2009). There have been three studies in Finland (Pershagen et al, 1986) and Turkey (Karavuş et al, 2002; Pala et al, 2012). One study in Israel analysed hospital visits according to pollution (Goren et al, 1995). And finally three studies reported modelling the risk lung cancer (Fábianová et al, 2000), health benefits from using modern pollution control (Thanh and Lefevre, 2001), and modelling of life expectancy and infant mortality (Gohlke et al, 2011). These last three studies do not rely on actual health assessment.

Risk of cancer

A case-control study of lung cancer was conducted in Lampang province, Thailand, between 1993 and 1995 (Pisani et al, 2006). A total of 211 lung cancer cases, 211 hospital and 202 population controls frequency matched to cases for age, sex were recruited. Hospital controls were matched to cases by place of residence (later not used in the assessment of risk by pollution factors). Participants were interviewed about socio-demographic variables, residential history, characteristics of household (including household fuels), occupational history, history of smoking (status, periods, amount smoked, type of tobacco) and alcohol drinking. Exposure to emissions from coal plants was modelled for each village around the power plant, taking into account distance to coal plant, year-specific SO_2, NO_2 and total suspended particulates (TSP) emissions, and wind characteristics (speed and direction) coming to the village. Based on residential history of subjects, an individual dose estimate was computed for each participant. No relationship was found between exposure to SO_2/NO_2 from the coal plant and the risk of lung cancer (highest vs. lowest exposure: OR=1.2, 95% CI [0.7; 2.0] adjusted for age, sex, tobacco smoking) or between exposure to TSP and lung cancer (highest vs. lowest OR=1.1, 95% CI [0.7; 1.8] adjusted to age, sex, tobacco smoking). In a subgroup analysis restricted to non-smokers, with 16 cases and 44 controls among which 3 cases and 18 controls were used as reference group, the risk remained non-significant (OR=3.4, 95% CI [0.7; 23.3]).
[This case-control study (Pisani et al, 2006) is well-conducted. The assessment of exposure was performed at individual level taking into account past history of residence. Estimation of risk of lung cancer associated with pollutants was adjusted for tobacco smoking. This study was conducted in an area covered by several coal

power plants (11 plants) although no information is provided on the distance of residence of an individual from such plants. Analysis on non-smokers is not informative as it is based on a very small sample size, with a reference category based on only 3 cases.]

Risk of non melanoma skin cancer

Three studies on non-melanoma skin cancer were conducted in Slovakia, in the Prievidza district which has a coal power plant burning coal rich in arsenic (Bencko et al, 2009; Pesch et al, 2002; Ranft et al, 2003) (previously described in section 1.3.2.1). In all studies, non melanoma skin cancer cases were selected from Bojnice Hospital data, this hospital serving as the "district reporting centre" for the National Cancer Institute of Slovakia.

The first study is a case control study 264 cases and 286 population controls frequency matched to cases on age and sex (Pesch et al, 2002). Assessment of environmental arsenic exposure was based on a subject's residential history, depending on distance from the coal plant (<5 km, 5–10 km, >10 km). There was no increased risk of NMSC associated with occupational exposure to arsenic: OR=0.75 (95%CI 0.41-1.37) for high exposure vs. none adjusted for age and sex. Some significant increased risks were found for subjects living close to power plants: OR=1.90 (95% CI 1.39-2.60) for highest (less than 5km from coal plant) vs. lowest (more than 10km from coal plant).

The previous case-control study was further analysed with urinary samples taken from some cases and controls (Ranft et al, 2003). Inorganic arsenic (As_{inorg}), monomethylarsonic acid (MMA), dimethylarsinic acid (DMA), and their sum (As_{sum}) were measured. Arsenic concentration in soils of gardens and dust in houses were also measured. Stepwise multiple linear regression was used to analyse determinants of urinary As. Differences in urinary arsenic were associated with sex, age, creatinine, but not with the distance to coal plant. Cases had around 10% more arsenic than controls.

The third ecological study was based on 1503 NMSC collected from 1977 to 1996 in an ad-hoc register set up for the study (Bencko et al, 2009). NMSC cases occurring within 7.5 km from the coal plants were compared to other NMSC registered in this area. Overall an increased risk of NMSC was evidenced in areas located closer to the coal power plant for the period 1977-1981. No significant association were reported for other periods.
[In all these reports (Pesch et al, 2002; Ranft et al, 2003; Bencko et al, 2009), exposure definition to arsenic is weak and based on distance to coal plant, while the distance is not associated with arsenic levels when measured in individuals. These studies also did not account for confounding factors such as sun exposure which is considered as the main risk factor for NMSC: 80-90% of NMSC being caused by sun exposure. In addition NMSC is the most difficult cancer to properly register (Parkin et

al, 2002). Therefore biases could be suspected in such study: an increased ascertainment could result from the suspicion of a link with coal power plant.]

Respiratory complaints

An ecological study of respiratory complaints and proximity of residence to coal plants was conducted in 1981 in Finland. A total of 12,000 subjects aged 15-64 living in 12 areas were included. Six areas were located near coal plants and they were matched with 6 others areas far from coal plants (Pershagen et al, 1986). A mailed questionnaire was sent to individuals. It contained general questions (age, sex, demographic variables, socio-economic status, etc), self declared respiratory health (bronchitis, asthma and allergies), annoyances caused by noise and air pollution, smoking habits, opinions/attitudes regarding environment and health, and finally questions about respiratory diseases of children living in the family. A subset of 171 subjects who reported respiratory problems were examined by doctors. Prevalence of respiratory diseases were sex-specific and age-standardised using the whole study population as reference. χ^2 tests were used for statistical analyses. For most symptoms, no difference was observed between exposed and non-exposed areas. Some symptoms (hawking, cough without phlegm) were significantly higher in some of the exposed areas as compared to their reference areas. Self declared annoyances (soot, dust or fly ash, odours) were also higher in some exposed areas.
[This ecological study (Pershagen et al, 1986) had a matched design (exposed areas matched to unexposed areas) but no information is available about the criteria used for matching areas (socio-economic, area population size). Although symptoms were self declared, medical examination of 171 subjects showed that these were real symptoms. No information on confounders was used as adjustment factor while individuals in areas close to power plant were more frequently current or former tobacco smokers.]

The study conducted in Hadera, Israel (section 1.3.2.2) was also focussing on adults (Goren et al, 1995). A publication reports ecological association between SO_2 and NO_x exposure and visits in clinics between 1982 and 1992. This analysis coincide with the progressive introduction of a new power plant in Hadera from 1981 (initial development) to 1984 (fully operating) and later. A seasonal variation of visit to clinics for respiratory disease was described. Overall, the major explanatory factor for a clinic visit was temperature. No increase in visits to clinics was evidenced during the study period.
[This study (Goren et al, 1995) has similar limitations as previously described, in particular an ecological design and a questionable modelling of SO_2 and NO_x pollution.]

In February 1999, Karavuş et al (2002) conducted an ecological study among 277 residents of villages within 5 km of the Seyitomer lignite plant in Turkey. The ash emissions of the coal power plant were 1830-2100 mg/m^3, well above the maximum allowed level of 750 mg/m^3. The SO_2 emissions were around 6000-6500 mg/m^3, whereas the maximum allowed level was 1000 mg/m^3. Only NO_x emissions were

below the maximum allowed levels. Residents were compared with 225 residents from other villagers living 30km away from the power plant. The 2 groups were comparable in terms of climate, culture and lifestyle. No subject worked in the coal plant. All subjects underwent spirometry tests (FVC; FEV_1; $FEF_{25-75\%}$) and were interviewed about basic characteristics and respiratory complaints ("chest tightness"; coughing; productive coughing). Chest tightness was significantly more prevalent in the study villages (46%) than in the controls (28%); the difference was significant for ages above 35 years. Repeated coughing attacks during the preceding year were more frequent among the study group, but the difference was significant only in one age group (35-54; 29% vs. 11.5%). No difference was found for productive coughing. Concerning spirometric parameters, FEV_1 and $FEF_{25-75\%}$ were significantly higher in the control group than in the study group, indicating a better respiratory health. Differences were more marked among non-smokers with significant differences favouring control group for FEV_1, FVC and $FEF_{25-75\%}$. No differences were found in current smokers of the two groups.
[This ecological study (Karavuş et al, 2002) was conducted in an area with extreme pollution from the local coal power plant. Several symptoms were described in the exposed area. Although the authors suggest that the reference group is comparable in term of culture, lifestyle, climate, major differences were existing for age (half of exposed individual aged 15-34, half of the unexposed individual aged more than 55 years). Definition of outcomes was not precise and prone to classification errors.]

In an ecological study, Pala et al (2012) evaluated respiratory functions (FVC, $FEV_{1,}$ FEV_1/FVC, $FEF_{25-75\%}$) of 2350 subjects aged over 15 years living close (1.5 to 12 km within the dispersion area predictable by prevailing winds) to the Orhaneli coal plant (Turkey). In addition, 469 subjects living in an area without coal plant were selected as control (Pala et al, 2012). The Orhaneli coal plant has been using a flue-gas desulphurisation filter since 1999. The study was conducted between September 2004 and October 2005. FVC and FEV_1 were significantly lower in the study group than in the control group, whereas FVC/FEV_1 was significantly higher in the study group, regardless of smoking status. There were no differences for $FEF_{25-75\%}$, except in ever smokers, where it was significantly higher in the study group.
[This study (Pala et al, 2012) is presented as a cross-sectional design, but because no individual exposure assessment is done, with exposure being only defined by proximity to the coal plant: however, this should be classified as an ecological study. A borderline non-significant imbalance of age exists between exposed villages and unexposed villages (p=0.053). A logistic regression between living near power plant and respiratory function was conducted but the dependent variable used was not the outcome (respiratory status) but the place of residence which rend this model non-interpretable. Participation rate varied strongly between areas: 66% in study group and only 15% in the control groups, hence a strong selection bias cannot be excluded.]

Summary of studies in adults is displayed in Table 1.5. Non-melanoma skin cancer incidence was the subject of two studies (Bencko et al, 2009; Pesch et al, 2002) conducted in the same place by the same team hence no pooling of estimate was feasible. Lung cancer incidence was also investigated, but only one article was available (Pisani et al, 2006). Respiratory symptoms and diseases were studied in 2 reports using a common exposure assessment (Pershagen et al, 1986; Karavuş et al, 2002), but measures of association were not suitable for meta-analysis (p-values of χ^2 tests and p-values of t-tests). Spirometric parameters (FVC, FEV_1, FEV_1/FVC, $FEF_{25-75\%}$) were evaluated in two studies using similar methodology (Karavuş et al, 2002; Pala et al, 2012), but measures of association, namely p-values of t-tests, were not usable in meta-analysis. Finally, one study evaluated number of visits to clinics (Goren et al, 1995).

1.4 Evaluation of impact on mortality

1.4.1 Observational studies

Risk among workers

Three articles (Bencko et al, 1980; Petrelli et al, 1989; Petrelli et al, 1994) evaluated the impact of coal power plant on mortality among workers.

Bencko et al (1980) studied mortality trends among coal plant workers. Deaths occurring in the previous 15-18 years were eligible. Death certificates were retrieved from 88 men working in a coal plant combusting coal rich in arsenic and 159 men working in 3 other coal plants where the arsenic content of the coal was lower. The distribution of causes of death (malignancy, cardiovascular, accidental and others) was compared between the two groups. This comparison was further stratified by age. There was no difference in the distribution of causes of death among the two groups. However, tumour deaths occurred at a significantly younger age in the exposed group.
[The sample size in this study Bencko et al, (1980) is small. The control group is made of men also working in power plant so likely exposed to several pollutants. No adjustment is made for confounders. The death certificates were obtained from the in-plant physicians. Therefore, deaths occurring in activity were complete, but not those of retired or workers on invalidity]

Petrelli and colleagues reported a retrospectively study on mortality in a cohort of Italian coal plant workers (Petrelli et al, 1989; Petrelli et al, 1994). From two coal plants, 1,307 men were followed from 1968 until 1984 (12,581 person-years). Outcomes were all-cause mortality; all malignant neoplasms; malignant neoplasms of digestive system; stomach neoplasm; respiratory system and lung cancer mortality. Mortality rates in the cohort were compared with those of all Italian men in 1951-1982. Forty one deaths occurred in the cohort with no significant difference from the rest of Italy (Petrelli et al, 1989). In a sensitivity analysis restricted to workers employed for more than 10 years, high SMRs were found for stomach cancer (SMR = 2.02, 95% CI = [0.24; 7.30]) although the confidence interval was large and included the value of unity (*ie* the risk of stomach cancer was not statistically significant).

In a subsequent report (Petrelli et al, 1994), workers of another coal plant were added to the initial cohort. The cohort of 1,772 men, with a mean employment of 9.5 years (22,000 persons-years), was retrospectively followed from 1968 to 1987 and their mortality rates compared to the Italian rates for 1968-1987. The age distribution of person-years was provided: more than 55% of the cohort was aged less than 35 years. 68 deaths occurred during follow-up, which was lower than expected (SMR = 0.79 [0.62; 1.01]) supporting the concept of a healthy-worker effect. The highest SMRs were found for larynx cancer (SMR = 3.36 [0.69; 9.83]) and digestive system excluding stomach and colon cancer (SMR = 4.00 [0.82; 11.69]) although neither was statistically significant. The authors mentioned that mortality was higher in one of the coal plants, but plant-specific results were not reported (Petrelli et al, 1994)
[Results of these studies (Petrelli et al, 1989; Petrelli et al, 1994) should be interpreted with caution because of methodological issues. First, there is likely to be a healthy worker effect in such population of coal plant workers. Second, in the first report, the reference population (1951-1982) does not correspond to the follow-up period (1968-1984). Third, the statistical power is limited with only 68 deaths from any cause. The young age of the cohort (55% aged less than 35) is also not adapted for studying cancer mortality.]

All-cause mortality and cancer mortality were assessed in these three articles (Bencko et al, 1980; Petrelli et al, 1989; Petrelli et al, 1994), but the exposure assessment and the measure of association were different. Two articles had common exposure assessment and measure of association, but they were two reports of the same cohort of Italian coal plant workers (Petrelli et al, 1989; Petrelli et al, 1994). Other causes of mortality were studied in a single study (Bencko et al, 1980). Despite a low statistical power in these studies, there is no evidence of an increase of death associated with occupational exposure to coal power plants.

Risk from environmental exposure

An ecological study on lung cancer mortality associated with residence in polluted areas was conducted in La Spezia, Italy in 1988-1996 (Parodi et al, 2004). The province is divided into 31 municipalities and 5 districts, with a population of 227,000 inhabitants. The most polluted areas were district 5 (D5) and Portovenere municipality. Population by area, gender and 5-year age group was obtained from the 1991 Italian National Census. Lung cancer deaths occurring in the period 1988-1996 were retrieved from the Liguria Mortality Registry. Urban-rural typology, educational level, unemployment, home ownership, housing conditions and family structure, were obtained from the 1991 Census. The risk of lung cancer mortality for D5 and Portovenere *vs.* the other urban or semi-urban areas were estimated via a Poisson regression adjusting for age and deprivation variables. In addition, a full Bayesian smoothing of SMR, estimated selecting the whole Province population as

standard, was obtained in order to evaluate the whole geographic pattern of risk in La Spezia Province. Among men, age-adjusted (on the Italian population of 1991) lung cancer mortality rates were 74.6 per 100,000 person-years in rural/semi-rural areas; 97.5/100,000 in urban/semi-urban areas; 106.2/100,000 in D5 and 93.3 in Portovenere. In women, mortality rates were of 9.3 lung cancer deaths per 100,000 person-years in the rural/semi-rural areas, 12.5/100,000 in urban/semi-urban areas, 22.5 per 100,000 in D5 and 29.5 per 100,000 in Portovenere. Adjusting for age, and socio-economic factors, the relative risk of lung cancer mortality was not increased in men in the most polluted areas as compared to urban/semi-urban areas. However, a significant increased risk was evidenced for women with a RR=1.54 (95% CI 1.01-2.36) for district 5 and a RR=2.14 (95%CI 1.09-4.20) for Portovenere. The Bayesian analysis highlighted a clear north to south increasing trend of risk for both sexes, which corresponds to a rural-urban gradient.
[This ecological study (Parodi et al, 2004) provides an attempt to adjust for at least socio-economic factors, but not for other important factors such as tobacco smoking. The results in women, although significant, are based on only 25 deaths in district 5 and 9 deaths in Portovenere. The important difference in reported risks between men and women limit the interpretation of the results in women as a marker of the role of pollution on lung cancer mortality. Finally, the sources of pollution were numerous (traffic, industries, shipyards, waste disposal, incinerators, lead processing plant), coal plant being only one of them.]

An ecological study on lung, larynx and bladder cancer mortality in the vicinity of combustion installations (coal, oil, natural gas) was carried out in Spain for the period 1994-2003 (García-Pérez et al, 2009). Deaths were retrieved from the National Statistics Institutes for the study period with information on the last place of residence. From the places of residence and location of power plants (57 power plant exceeding 50MW) obtained from EPER-Spain, towns were classified by proximity to power plants. The highly exposed group were towns located less than 5 km from a coal power plant. Relative risks for lung cancer death for people living in the highly exposed group were 1.13 (95% CI 1.05; 1.22) in men and 0.83 (95% CI 0.64; 1.06) for women. For laryngeal cancer the RR was 1.46 (95% CI 1.21; 1.77 in men (this site was not studied in women because of its scarcity). For bladder cancer, RRs were 1.22 (95% CI 1.03; 1.44) for men and 0.97 (95% CI 0.66; 1.42) for women. The increased risk of cancer was not specific to coal plant but also evidences in other areas (with plant using oil and natural gas).
[This ecological study (García-Pérez et al, 2009) has several limitations. Exposure of subjects is based on distance between the coal plant and the centroid of the last municipality of residence. There was no information on history of residence of subjects. There is no information on potential confounding factors such as smoking or socio-economic status, which play a major role in cancer mortality. There is no consistency in the results: increased risks were found only in men and not in women.]

Table 1.6 summarizes studies reporting death from any cause as health outcome. There were only two studies on lung cancer mortality using a common measure of association (Parodi et al, 2004; Garcia-Perez et al, 2006). However, exposure assessment was based on 5km distance to coal plant in the Spanish study while it was based on cluster of regions in Italy. Because these two studies were too different in term of design, and both based on ecological association, we did not provide a pooled estimation of risk from these two studies. Laryngeal and bladder cancer mortality were health outcomes considered in one single study (Garcia-Perez et al, 2009) and although both studies demonstrate evidence of increased risk of death from environmental exposure to pollutants from coal power plants, the evidence is weak and the study design is of an ecological nature.

1.4.2 Modelling studies

Three modelling studies investigated the role of pollution from coal plant (or coal consumption) on health in population. These studies are not based on health data from exposed populations. Hence, all the results are directly influenced by the assumptions of causality made by the authors. The first study from Fabiánová et al (2000) does not specifically address mortality, but because of the high fatality of lung cancer, this study was included in this section. Furthermore, because it follows a modelling approach, the same criticisms could be made for this study as for the two others. The modelling studies from Thanh et al (2001) and from Gohlke et al (2011) do provide estimation of mortality associated with exposure to coal plant pollutants.

Fabiánová et al (2000) modelled the risk of lung cancer related to arsenic exposure of the general and working population living and working near a coal plant burning arsenic-rich coal in Prievidza, Slovakia. The study period was 1953-1993. Arsenic concentration in air were measured for 1973-1993 and extrapolated for 1953-1972. Exposure to arsenic was modelled taking into account occupation, place of residence and years of exposure. Estimated arsenic concentrations were 1-2 µg/m^3 before the 1970s and 0.04-0.07 µg/m^3 after for the general population; 1-400µg/m^3 (before 1973) and 0.45-400µg/m^3 (after 1973) for the population working at the coal plant. Risk of lung cancer was taken from a World Health Organisation report (WHO, 1987) that produced risk of lung cancer per unit of arsenic exposure. In inhabitants, the lifetime risk of lung cancer attributed to arsenic inhalation varied from $5.2*10^{-5}$ to $2.53*10^{-3}$ depending on duration and period of exposure. Likewise, the risk for particular jobs was highly variable; for 40 years of exposure, it varied between $1.2*10^{-2}$ and $7.9*10^{-2}$.
[The unit risk used in this modelling (Fabiánová et al, 2000) was extracted from a WHO report published in 1987 which was based on studies in workers heavily exposed to arsenic (smelters).]

Thanh and Lefevre (2001) modelled the predicted decrease in health problems (mortality, respiratory diseases, hospital admissions) related to the potential introduction of new modern devices to limit emissions from the existing old coal power plant in the Mae Moh area (Thailand). The calculation was based on the

transfer of risk assessment in Bangkok from a report from Chestnut et al, published in 1998 (Chestnut et al, 1998). The impact of the change of air quality of the power plant of Mae Moh is performed for the whole of Thailand including areas more than 1000km from the power plant.
[Similarly as Fabiánová et al (2000) the transfer of a risk from one population to another as used in this study (Thanh and Lefevre, 2001) is questionable. The source of the risk used could not be evaluated. In this study, the scenario evaluate a change of 0.006 to 0.476µg/m^3 (in the mostly exposed area) which is a very small change when yearly average in modern cities are between 20-40 µg/m^3]

A world-scale ecological study on life expectancy and infant mortality associated with coal consumption and electricity was conducted for the period 1965-2005 (Gohlke et al, 2011). Data on life expectancy, infant mortality, population, electricity consumption and coal use were retrieved for 41 countries. Life expectancy and infant mortality were modelled using autoregressive equations taking into account electricity and coal consumption.

Modelled life expectancy and infant mortality were compared to actual data, and the fit was good for the most populous countries, for both life expectancy and infant mortality: R^2 ranged from 0.66 to 0.92. Moreover, a significant positive association was found between coal consumption and infant mortality in countries having low infant mortality and high life expectancy at baseline.
[This study is not specific to coal plants (Gohlke et al, 2011). The verification of the fit of the model was based on the same data that were used to generate the model and only a selection of countries, with high correlation, was presented in the results. Life expectancy and infant mortality rates were predicted in the model by coal consumption and electricity consumption only. No information on the health system (access to care, drug, coverage), poverty, risk factors, were available. This study has all the attributes of an ecological fallacy.]

The first two modelling studies suffer from the same limitation: the transfer of a risk associated with a pollutant from another situation of much higher exposure to the situation of environmental exposure to pollution with much lower, not comparable doses. In Fabiánová et al (2000), the risk is derived from studies of smelters, and this is transferred to outdoor air pollution. In Thanh and Lefevre (2001), the risk is transferred from a report (not available) with no information on how the risk is estimated.

Both modelling studies are all based on very poor assessment of air pollution. In Fabiánová et al (2000), some air measurements are extrapolated backward in time: arsenic air concentration measured for 1973-1993 are extrapolated for 1953-1972 while no information exist on air pollution for this period. In Thanh and Lefevre (2001), exposures are extracted from an unpublished report.

Finally the last modelling study is barely a modelling exercise as it consists of a raw ecological study where level of infant mortality are directly assumed as dependent of coal consumption and electricity consumption in each country. This hypothesis is neglecting all known factors associated with infant mortality, which are not related to pollution but to deprivation. The absence of adjustment for these factors is likely to produce a severe confounding bias.

In conclusion, all these modelling studies are based on poor data on exposure which is not specific to coal plants but to global air pollution. The health risks are not derived from studies on coal plant emission but on situations of much higher level of exposure (smelters or, in the case of Fabiánová et al (2000), study on arsenic). The dose-risk relation is then assumed as linear which is an assumption not supported by scientific evidence.

1.5. Studies on coal health effects not related to coal-plants

Coal can be used for the generation of electricity in power plants, or at individual level for cooking or heating. Concerns have been raised about cancer risk related to different aspects of coal production and use. The International Agency for Research on Cancer (IARC) has evaluated the risk of cancer related to household use of coal (IARC 2010a; IARC 2012c), coal gasification (IARC 2010b; IARC 2012b) and coal dust (IARC 1997; Bridbord 1979). Other health outcomes, in particular respiratory diseases, have also been studied. In this section an overview of health outcomes associated with these use of coal other than through coal power plants is presented bearing in mind that the exposure levels discussed in this section are much greater than those derived from coal plants.

1.5.1. Household use of coal

Coal is broadly used in developing countries for heating and/or cooking. In these countries, the number of people exposed to indoor coal emissions is very high and exposure takes place in rooms and houses in the absence of any form of air extraction (including windows and doors closed and sealed with no chimneys). The major health concern about these emissions is the risk of lung cancer. For example, it was estimated that about 40% of rural households in China (*i.e.* several hundreds of millions people) rely on coal for cooking and/or heating (National Bureau of Statistics, 2005) with coal frequently used in the absence of any form of ventilation.

Most epidemiological studies have been conducted in China. In those studies, measured pollutants levels were higher in rural households than in urban households, and reached extreme concentrations (several milligrams/grams per cubic metre, Table 1.7).

In total, 26 case-control studies (24 in China), 2 cohort studies (2 in China) and 2 ecological studies (outside China) assessing the risk of lung cancer in people exposed to indoor combustion of coal were reported by IARC (IARC, 2012c). Overall, several case-control studies found an increased risk of lung cancer associated with indoor coal emissions, taking into account possible confounding factors such as tobacco smoking. The increase in risk was more marked in women than in men, and a dose-response relationship was found. Typical odds ratios range from 1.5 to 10, depending on coal subtype, years of exposure, smoking status, sex, room ventilation, and so on. Studies about other cancer sites were inconclusive.

Based on this evidence, household use of coal was classified as carcinogenic to humans (Group 1) by IARC. Household coal use causes cancer of the lung (IARC 2010a; IARC 2012c).

Other health effects of household use of solid fuels[3] have been reported by the World Health Organization (Smith et al, 2004). Household use of coal has been linked with acute lower respiratory infections[4] (ALRI) in young children (younger than 5 years) and chronic obstructive pulmonary diseases[5] (COPD) (Smith et al, 2004). Other illnesses such as asthma, tuberculosis, visual impairments and birth defects (Li et al, 2011) may be related to household use of coal, but the evidence is scarce and inconsistent (Smith et al, 2004).

1.5.2. Coal gasification

Coal gasification is the process of reacting coal with oxygen, steam and carbon dioxide to form a gas containing hydrogen and carbon monoxide (IARC 2010b). The coal gas is much more efficient and convenient to transport (Guidotti 1979). The chemical and physical processes involved in coal combustion and coal gasification are similar, the difference being the nature of the final products. However, coal gasification has a better environmental performance than coal combustion. Applications of coal gasification include power production (IARC 2012b; BINE Informationdienst, 2006). The process used in gaseous coal-fired power plants is called integrated gasification combined cycle (IGCC). It is a relatively new technique, more efficient and less pollutant than "traditional" techniques (BINE, 2006).

Workers in the production of coal gasification are exposed to PAH and benzo[a]pyrene (BaP), and may be exposed to other substances such as asbestos, arsenic, cadmium and lead, among others (IARC 1984). Contrary to household coal use, the number of people exposed to coal gasification is limited because only occupational exposure occurs. Data about PAH and BaP concentrations in coal gasification plants are scarce (Gustavsson and Reuterwall, 1990; Lindstedt and Sollenberg, 1982), and are estimated to be several micrograms per cubic metre. These levels are much lower than household emissions of coal. There were 8 studies reporting cancer risk in cohorts of coal gasification workers, conducted in Europe and Asia. All studies reported increased lung cancer risk, with relative risks ranging

[3] Mainly coal and biomass (wood, dung, etc.)
[4] Examples: Acute Bronchitis, bronchiolitis, etc.
[5] Examples: Chronic bronchitis, emphysema, etc.

from 1.3 to 33.3, which is unlikely explained by smoking status of the workers. There were not enough data for evaluating other cancer sites.

Occupational exposures during coal gasification were classified as carcinogenic to humans (Group 1). Occupational exposures during coal gasification cause lung cancer (IARC 2010b; IARC 2012b).

1.5.3. Coal dust and coal mining

Coal dust is a complex and variable mixture, containing more than 50 elements. Coal dust content depends on the coal seam itself and the size of the particles. It contains not only coal, but also silicates (quartz), kaolin and mica (IARC, 1997). The coal dust particle size includes PM_{10} and $PM_{2.5}$. Coal dust exposure occurs mainly during coal mining operations, bulk loading and transfer, and at places were coal is stored, for example in power stations or for household use. Coal miners are particularly exposed to coal dust. That is why nearly all information about coal dust comes from studies conducted in mines. Before the seventies, coal dust concentrations in underground mining facilities were around 12 mg/m^3, or less, depending on workplace in the mine and the mine itself. More recently, regulations in some (developed) countries brought these levels to about 3 mg/m^3 or less (IARC, 1997; Bridbord et al, 1979). However, upper levels were also reported. In general, coal dust levels are higher in underground mines than in surface mines.

As of 1997, there had not been epidemiological studies about coal dust and cancer risk. However, a large number of studies assessed the risk of cancer in coal miners. Cancers of the lung and stomach were widely studied; other cancer sites such as urinary bladder were much less investigated. In total, 15 cohort studies (mainly in the United Kingdom) and 4 case-control studies were reported in IARC monograph on coal dust (IARC, 1997). Concerning lung cancer, results were inconsistent. Some studies reported risks below unity, while others reported higher risks in miners compared with non-miners. Eventually, the majority of risks reported in these studies (ranging from 0.5 to 2) were not statistically significant. Therefore, no conclusion could be drawn for lung cancer. For stomach cancer, results were more consistent, suggesting an increased risk among miners (RRs around 1.5-4), yet no dose-response relationship was found (IARC, 1997).

Finally, there is inadequate evidence available to classify exposure to coal dust as to its carcinogenicity to humans (IARC Group 3) (IARC, 1997).

Despite the inconclusive studies about cancer risks in coal miners, other respiratory diseases linked with coal dust exposure are present in coal miners, particularly those working underground. Pneumoconiosis[6], progressive fibrosis, chronic bronchitis and

[6] A set of lung diseases caused by inhalation and fixation of solid particles in the lung (examples: silicosis, asbestosis....). Some of them may cause lung fibrosis.

emphysema are examples of frequent respiratory diseases among underground coal miners (Bridbord et al, 1979; Guidotti 1979). Incidence, prevalence and mortality of such diseases are higher in underground and surface coal miners than in the general population. Surface coal miners, who are less exposed to coal dust, are affected by the same diseases as underground miners, but to a lesser extent. Health effects of coal mining that are not related to coal dust include work-related accidents, loss of hearing and cold/heat stress (only for surface miners). Use of diesel-powered equipment (a Group 1 carcinogen for lung cancer[7]) and longwall mining techniques[8] may also be associated with serious health effects (Bridbord et al, 1979).

1.6. Summary and Conclusions

The operation of power plants with coal as the main combustion source leads to the emission of CO_2 in important quantities and, although not critical for health aspect, these emissions are likely contributing to global warming. Several utilisations of coal have been classified as carcinogen to human by IARC such as household coal use (IARC 2010a; IARC 2012c) and coal gasification (IARC 2012b), but no evaluation was performed by IARC on coal plants (either through occupational or environmental exposure). The exposure levels on which the IARC classifications are based are much higher than any levels from coal plants which themselves have decreased markedly through time.

The production of energy in coal power plants is also associated with the release of several other components relevant to health, some being carcinogenic to humans. Three major pollutants from coal plants, SO_2, NO_x and fine particulates ($PM_{2.5}$), have been under scrutiny for their hypothesised health effects. Some coal could contain other compounds in important quantities, depending on the source of coal, like arsenic or radionuclides. These emissions have likely changed with time with modern power plants equipped with devices such as flue-gas desulphurisation or catalytic reduction. The workforce, in particular in developed countries, are more protected in recent decades. National regulations limit the emissions that a coal power plant is allowed to emit, and nearly all studies reviewed showed pollution values below emission limits.

As compared to some countries which have an important density of power plants like in Thailand or in the United States, Italy has a lower coal plant density, dispersed in the whole country.

Overall, few studies investigated associations between coal power plant emissions and health risk. Even fewer had an appropriate methodology which could provide good evidence for association. Indeed, most studies relied on an ecological design, i.e. where both outcome and exposure are measured independently with no

[7] http://www.iarc.fr/en/media-centre/iarcnews/2012/mono105-info.php. Accessed 26/11/2012
[8] This technique is more productive, but produces higher levels of coal dust, and is noisier than other techniques

individual exposure measurement. This design limitation is due to the nature of the subject, as a proper monitoring of individual exposure would require personalised dosimeter which is not adequate to investigate long-term effect of exposure to a factor. Some studies however used either an individualised pollution modelling, or used a biological test as a proxy to exposure (such as arsenic in urine). In the case of biological test, the concentration of pollutant measured is only a proxy of exposure to pollution from coal plants under the hypothesis that no other pollutants are involved. There was an important heterogeneity in the size of study areas, some limited to less than 5 km around power plants, other investigating health effects up to 80 km (Table 1.9). A modelling study in Thailand (Thanh and Lefevre, 2001) even estimated risk more than 1000 km away from the emission source.

A total of 39 studies were included in this literature review. Only qualitative assessment was presented. No meta-analysis was necessary or feasible because the definition of exposure greatly differed between studies, as well as health outcomes. The design of studies, mostly ecological, was a second limitation to perform meta-analysis.

We investigated studies on workers in coal power plants, because they could be potentially exposed to much higher pollutants level than general population, and if an important risk existed, we would have expected to observe it in these populations. Some difficulties of studying the health impact of coal plant pollution's exposure are specific to workers. First, a healthy worker effect (those working being selected as having a better health condition than the general population) could be playing a role and mask a real increased risk in these populations. Another difficulty is the multiple sources of exposure in workers in coal power plants. The best study conducted in workers came from investigation in boilermakers (Hauser et al, 2001) for which a decreased pulmonary function was evidenced. Because of the multiple sources of exposure of these workers, this decrease pulmonary function might result from exposure to other pollutants associated with their occupation. A study was conducted in Italy on total mortality and cancer mortality of workers in coal power plants (Petrelli et al, 1989, Petrelli et al, 1994) and showed no increased risk of death from any cause as compared to general population. This study was limited in sample size and this low power limits any conclusion for risk lower than 2.0.

Prenatal outcomes were investigated in few studies, but none of the studies had an appropriate design to draw any conclusion.

The development of children living in the vicinity of power plant has been the subject of several publications. Most studies investigated the pulmonary functions of children associated with coal plant pollutant's exposures, these being measured, modelled, or estimated by the distance to power plant used as proxy. Some significant associations were described but none could properly exclude the role of confounding factors such as tobacco smoking of parents. Most studies however showed that socio-economic status varied according to the proximity of residence to power plant, and point the likely problem of deprivation of population in the vicinity of coal power plant.

Only one study in adults had a clear design which attempted to account for the classical potential biases in observational studies. This case-control study was conducted in Lampang (Thailand) and show no increased risk of lung cancer associated with pollution exposure from coal power plants (Pisani et al, 2006). A study conducted in Italy (La Spezia), with an ecological design (Parodi et al, 2004) showed an increased risk of lung cancer in women but not in men in the areas highly exposed to pollutants. But this study had several limitations among which a poor design (ecological), a non-specific exposure (several pollutants activities other than coal plant are also present in the region) and a small sample size. All other studies had on average a poor design which limits the conclusions on the existence of risk to human health.

Because of the technological evolution of pollution control applied to coal power plants, and of more stringent regulation in recent years, studies performed on exposure before year 2000 are hardly relevant to the current situation of exposure to coal power plants. Among the 39 studies, only 6 were based on exposure to coal plant pollution performed after 2000. Only one study (Tang et al, 2008), on prenatal exposure and PAH-DNA adducts, was of good quality, even if the exposure definition was ecological.

Some studies reported pollution level measured in the vicinity of coal plants. We compared these measured to EU limit values for the protection of human health from the Directive 2008/50/EC of 21 May 2008.

For prenatal outcomes two studies reported air quality assessment. In the Croatian study in 1989, daily values of SO_2 varied from 34.1 to 252.9 µg/m^3 which are above EU limit (125 µg/m^3). In the study in China in the city of Tongliang in 2002, PAH values were not reported but described as above PAH measured in Krakow or New York City.

For studies in children, four studies reported air measurements. In the Israel study in the city of Askhelon, NO_x and SO_2 were reported but with no unit. However, the daily average of PM_{10} in 1999 was of 67.1 µg/m^3 which is above the EU limit (50 µg/m^3). In the Israel study in the city of Hadera, in 1985-86 only monthly average were reported: for the mostly exposed site, SO_2 ranged between 2.8 to 22.4µg/m^3 (not comparable to EU limits); NO_x and CO were also reported but in ppb. In the study in Australia in 1987, maximum hourly average of SO_2 and NO_2 for the most exposed site were of 139 µg/m^3 and 169 µg/m^3 respectively. These values are below EU limits (350µg/m^3 for SO_2 and 200 µg/m^3 for NO_2). In the study in Thailand in 1997, the maximum daily value of SO_2 for the most exposed sites was 128.01 µg/m^3 which is slightly above EU limits (125 µg/m^3) and PM_{10} was of 92.02 µg/m^3 which is well above EU limits (50 µg/m^3).

For studies in adults, three studies reported air quality measurement. The Lampang study in Thailand produced reconstructed individual exposure from 1978 to 1994 for SO_2 and NO_2, but the unit reported was not compatible with EU limits definition. The Israel study in the city of Hadera in 1981-1992 reported several (0 to 200 events) events per year, i.e. half-hour average exceeding limits of 183 µg/m^3 for SO_2 and 235 µg/m^3 for NO_x. These values are above EU limits for NO_x and likely higher for SO_2. In the study in Turkey in 1999, no information on air pollution in the city with coal power plant was reported, but information on the SO_2 emissions was of 6573 mg/m^3 for the highest unit of production. These values are higher than the limits defined by the Turkish regulation (limit of 1000mg/m^3).

In summary, for most studies reporting air quality assessment, average values of SO_2, NO_x or PM_{10} were higher than the EU limit values as defined by Directive 2008/50/EC/ on ambient air quality. All but one of these air quality measurement were performed outside Europe.

Most studies suffered from potential biases because of the difficulty to properly define exposure at the individual level and because of the multiple sources of exposures. With such limitations, there is no indication of an increased risk of death or other health effects associated directly with pollution from coal power plants. Future studies should be focussing on modelling accounting for differences in pollution sources (industries, road traffic, etc). To account for the dispersion of pollution which is influenced by wind speed and direction, these models should be conducted at a high resolution with information on population exposure to other compounds (tobacco smoking, household solid fuels, etc) and socio-economic deprivation.

The Environmental and Health Impacts of Coal Thermoelectric Plants

Chapter 2

Health Effects of Fine Particulate Matter and Contributions from Sources

List of abbreviations

2, 3, 7, 8-TCDD: 2, 3, 7, 8-tetrachlorodibenzodioxin
95% CI: 95% confidence interval
ACS: American Cancer Society
AHSMOG: Adventist smog and health
APHEA: Air Pollution and Health: A European Approach
APHENA: Air pollution and health: a combined European and Northern American Approach
BAT: Best available technologies
CASAC: Clean Air Scientific Advisory Committee
COMEAP: Committee on Medical Effects of Air Pollution
CPS: cancer prevention study
EEA: European Environment Agency
EMEP: European Monitoring and Evaluation Programme
EPA: Environmental Protection Agency
EpiAir: Air Pollution and Health: Epidemiological Surveillance and Primary Prevention
E-PRTR: European pollutant release and transfer register
EU/EU-27: European Union/European Union (27 members)
EUROCAT: European Network for the surveillance of congenital anomalies
HEI: Health Effects Institute
HR: Hazard Ratio
IARC: International Agency for Research on Cancer
IPPC: integrated pollution prevention control
IQR: interquartile range
IR: Incidence Rate
LRTAP: Long-range transboundary air pollution
NAAQS: national ambient air quality standards
NMMAPS: national morbidity and mortality air pollution study
NO_x: nitrogen oxides
OR: odds ratio
PAPA: Public Health and Air pollution in Asia
PM_x: particulate matter (diameter ≤ x μm)
RR: relative risk
SD: standard deviation
SE: standard error
SENTIERI: Studio epidemiologico nazionale territori e insidiamenti espositi a rischio da inquinamento (Epidemiological study of national territories and settlements exposed to risk from pollution)
SIR: standardised incidence ratio
SMR: standardised mortality ratio
SO_x: Sulphur oxides
SRR: summary relative risk
TSP: total suspended particles
WHO: World Health Organization

Chapter 2: Summary

Reducing levels of air pollution is an objective of all developed countries. In particular, most countries are engaged in efforts to reduce levels of $PM_{2.5}$. The European Union aims to have pollution limits of 20µg/m^3 of $PM_{2.5}$ by 2020 recognising that zero levels are not achievable. These declining norms are primarily motivated by the accumulating evidence of a detrimental effect of air pollution on health. Studies of outdoor air pollution have investigated several compounds such as NO_x, SO_2, and particulates PM_{10}, and $PM_{2.5}$. Even if fine particulates are a mixture of compounds and particulates sizes, $PM_{2.5}$ is now considered as a key marker of outdoor pollution and for which consistent results have been shown.

The *Energy Production and Distribution* sector is a minor source of $PM_{2.5}$ and PM_{10} pollution, and emissions of particulate matter have been decreasing steadily in this sector. The main sector emitting $PM_{2.5}$ in the EU is *Residential Stationary Plants* (in particular emissions from household firewood combustion).

Short term effects of exposure to fine particulates have been consistently reported in adults with increased risk of all-cause mortality, more specifically cardiovascular and respiratory mortality. Long-term effects of exposure to fine particulates generally have a less robust design than those on short-term effects as an ecological component is always present with place of residence being units of comparison. However, in several studies, with adequate accounting of several confounding factors, consistent increased mortality rates have been associated with increasing levels of $PM_{2.5}$. In addition, decreases in air pollution levels have been associated with decreases in adverse health effects. The role of air pollution on health has received a great deal of attention in Italy and several studies have been conducted in particular around large combustion plants.

Using the European Environment Agency's database, $PM_{2.5}$ emissions per capita are higher in Nordic European countries and central European countries. This reflects the role of household heating with coal, biomass or other fuel. The Commercial, Institutional and Household sector emissions represent 52% of $PM_{2.5}$ in EU-27. Transport is also an important contributor in particular for emission of NO_x. The Energy Production sector is a major contributor of SO_x and it has shown the greatest decrease since the 1990s: emissions of SO_x have been reduced by a factor 5, emissions of $PM_{2.5}$ and PM_{10} have been reduced by a factor 3. In Italy, the Energy Production and Distribution sector is the eighth largest contributor to $PM_{2.5}$, and the eighth largest contributor to PM_{10} emissions, and emissions from these sources have dropped markedly over the past decades.

To reach the European goals of air pollution limits for 2020, all sectors require to make efforts to reduce their emissions of atmospheric pollutants recognising that frequently the largest contribution to local pollution may be a variety of dispersed sources. However, specific actions must target the major sources of Particulate Matter emissions, namely Household Heating Sources and Traffic pollution. Household use of coal, wood and biomass burning should be discouraged and solutions with low emission of $PM_{2.5}$ should be preferred. Even if the relative risk for fine particulate matter pollution on health from road traffic sources may be lower than that of other sources, the attributable fraction will be higher because of the overall dominant contribution to this source of pollution.

2. Health Effects of Fine Particulate Matter and Contributions from Sources.

2.1 Introduction

The Earth's *Atmosphere* consists of a layer of gases surrounding the planet: the atmosphere protects life on Earth in a number of ways, for example by absorbing solar radiation, warming the surface through heat retention and reducing temperature extremes between day and night. *Air* is the name given to the atmosphere used in breathing and photosynthesis. The approximate contents of *Dry Air* are (by volume) 78% nitrogen, 21% oxygen, 0.9% argon, 0.04% carbon dioxide, and small amounts of other gases. Air also contains a variable amount of water vapour, on average around 1%.

Air is also polluted by a number of gases (O_3, CO, SO_x, NO_x etc), particles, bio-aerosols and toxic substances. Taken individually, some of these air pollutants are potentially related to specific adverse health effects. Pollutant concentrations are often highly correlated with each other, which makes it difficult to identify a specific health effect related to a given pollutant. It is a particular challenge since most of the studies are of an ecologic nature, arguably the weakest form of observational study, and it is also often very complex to identify the main source of a particular pollutant from such observational studies.

Airborne particulates are defined as all solid or liquid particles that can be carried by air, even for a very short period of time. There are two categories of airborne particulates according to their size: the grit fraction, and the fine fraction or particulate matter (PM). PM can penetrate into the lungs, while the grit fraction cannot. The limit between these two categories is an aerodynamic diameter of 10µm. PM can be further divided into 3 sub-categories: coarse particulates (diameter between 2.5 and 10µm or $PM_{2.5-10}$), fine particles (diameter less than 2.5µm or $PM_{2.5}$) and ultrafine particles (diameter < 0.1 µm of $PM_{0.1}$).

Before defining norms and enforcing new air quality regulations, it is fundamental to identify which air pollutants are the most dangerous for public health. Pollution reduction policies may be specifically directed to specific pollution sources having an important impact on population health. But, in order to identify these sources, health effects of total air pollution need to be assessed in a first step, and effects have to be decomposed and attributed to a specific pollutant. This is a highly complex task and requires state-of-the-art methodology and research.

In developed countries, current laws regulate a series of pollutants, the most common being particulate matter (PM), ozone (O_3), carbon monoxide (CO), nitrogen oxides (NO_x) and sulphur oxides (SO_x). It is now broadly accepted that each of these pollutants, taken individually, and at high concentration, could have substantial adverse effects on human health. However, it is very complex to separate health effects of each specific pollutant because of the intricate relationships between them. For example, SO_x and NO_x can react to form particulate matter, and NO_2 plays a role in the formation of O_3. Finally, identifying any effects of particulate matter is even more complicated than for other pollutants because it is a complex mixture having countless possible sources, either primary or secondary, some being from natural sources while others are a result of human activities, some arising from sources close to hand while other contributions have been transported over long distances.

Epidemiological studies showed that short-term exposure to PM and gaseous pollutants may contribute to mortality in the general population (Burnett et al, 2000). Advanced analysis of a large cohort study identified $PM_{2.5}$ and SO_2 as the main pollutants associated with increased mortality, the latter being more robust after controlling for confounding factors (Krewski et al, 2003; Krewski et al, 2004; Pope et al, 2002).

Given that a potential biological mechanism linking pollutant exposure and increased mortality exists for $PM_{2.5}$, but not for SO_2, sulphur dioxide may be a marker of another exposure, for example sulphate particles. Current evidence indicates that PM pollution is a major component of the total air pollution to which the general population is exposed (Greenbaum, 2003). In particular, $PM_{2.5}$ has been the centre of attention, because these particles can penetrate deep into the lung, and have been associated with increased mortality in a number of observational studies. A possible biological hypothesis is based on increased atherosclerosis, inducing cardiovascular and respiratory conditions.

Recent research has found that gaseous pollutants, especially O_3, may play a role in pollution-related morbidity and mortality. Nevertheless, using the current scientific literature available, it is not possible to separate the effects of PM and gas pollution on the global public health.

In recent years, $PM_{2.5}$ has increasingly been the focus of attention. $PM_{2.5}$ is particulate matter which passes through a size-selective inlet as defined in the reference method for the sampling and measurement of $PM_{2.5}$, EN 14907, with a 50% efficiency cut-off at 2.5µm aerodynamic diameter.

Technological differences in the measurement of $PM_{2.5}$ could lead to different estimates of the level of concentration. It is therefore important that levels of pollution measurements defined for exposures at population level are based on the same methods of defining $PM_{2.5}$ concentration as in studies of risks to health.

2.2 European Legislation and Norms

Such air pollution has been the subject of regulations at the European level (*Directive 2008/50/EC of the European Parliament and of the Council of 21 May*

2008 on ambient air quality and cleaner air for Europe). The text of this Directive is provided in Appendix 2. The rationale for such action was that fine particulate matter ($PM_{2.5}$) is responsible for significant negative impacts on human health. Further, there is as yet no identifiable threshold below which $PM_{2.5}$ would not pose a risk and it is acknowledged that zero levels in air are unobtainable. As such, this pollutant should not be regulated in the same way as other air pollutants. The approach should aim at a general reduction of concentrations in the urban background to ensure that large sections of the population benefit from improved air quality. However, to ensure a minimum degree of health protection everywhere, that approach should be combined with a limit value, which is to be preceded in a first stage by a target value.

The Directive outlined that Member States should take all necessary measures, not entailing disproportionate costs, to reduce exposure to $PM_{2.5}$ with a view to attaining the national exposure reduction target laid down in Section B of Annex XIV by the year specified therein. In 2013 the Commission plans to review the provisions related to $PM_{2.5}$ and, as appropriate, other pollutants, and shall present a proposal to the European Parliament and the Council.

As regards $PM_{2.5}$, the review shall be undertaken with a view to establishing a legally binding national exposure reduction obligation in order to replace the national exposure reduction target and to review the exposure concentration obligation laid down in Article 15, taking into account, *inter alia*, the following elements: the latest scientific information from the World Health Organisation (WHO) and other relevant organizations; air quality situations and reduction potential in the Member States; the revision of Directive 2001/81/EC; and progress made in implementing Community reduction measures for air pollutants. The Commission shall take into account the feasibility of adopting a more ambitious limit value for $PM_{2.5}$, shall review the indicative limit value of the second stage for $PM_{2.5}$ and consider confirming or altering that value.

The initial target of the Directive was established to reach 25µg/m^3 by 1st January 2015 and 20µg/m^3 by 1st January 2020. In comparison, the United States Environmental Protection Agency (US EPA) chose an annual standard of 15µg/m^3 for $PM_{2.5}$, averaged of 3 years, whereas US EPA's Clean Air Scientific Advisory Committee (CASAC) recommended a lower value.

Exposure levels have been recorded at much higher concentrations in China than in Europe or Northern America. In a study of fourteen large Chinese cities, $PM_{2.5}$ concentrations were on average 115 µg/m^3 over the year (Cao et al, 2012). The Chinese government issued national $PM_{2.5}$ standards requiring cities to have concentrations below 35 µg/m^3 on an annual average from 2016.

2.3 Methodology of Chapter 2

2.3.1 Emission data

Emission data were retrieved at the European level from the statistics reported by the European Environment Agency. Reporting emission of pollutants is compulsory in the European Union. Reporting of emissions is regulated by *Regulation (EC) No 166/2006 of the European parliament and of the council of 18 January 2006 concerning the establishment of a European pollutant release and transfer register and amending council directives 91/689/EEC and 96/61/EC*. Industrial facilities report their yearly pollutant emissions if they meet the following criteria:

- The facility exceeds at least one of the capacity thresholds defined in the *Regulation No 166/2006*
- The facility transfers off-site a certain amount of waste (exceeding defined threshold)
- The facility releases pollutants which exceed specific emission thresholds specified for each pollutant in the regulation.

Overall, more than 28,000 industrial facilities in Europe report their annual pollutant emissions into the European Pollutant Release Transfer Register (E-PRTR). These data are publicly available online through the European Environment Agency website.

For the purposes of this report, scientific articles describing the role of major emitters of pollutants were retrieved. In addition, articles showing evidence of significant decreases in pollution from intervention on emitter of pollutants were reviewed.

2.3.2 Search strategy for PM health effects

The review of studies on air pollution exposure and health was performed by reviewing scientific literature published in peer-reviewed biomedical research journals. Several thousand of articles have been published in *Pubmed* on the biomedical effects of air pollution: in *Pubmed*, as of 18th of March 2013, 39,393 articles on the MeSH (Medical Subject Headings) term "Air Pollution" have been referenced.

The annual number of publications since the last two decades have increased rapidly, especially since 2000 (Figure 2.1). Of all such publications found in *Pubmed*, one third was published before 1990 (13,838), while a similar number (13,344) were published between 2008 and 2012.

This bibliographic analysis shows the increasing interest being taken on the association between Air Pollution and its potential impact on health. It is impossible to make a full detailed overview of all literature published so far on this topic, and so focus has been restricted to reviewing publications providing the highest level of evidence from observational studies in humans.

For studies on short-term effects, focus was restricted to studies conducted on time-series. For studies on long-term effects, focus was restricted to publications reporting results from prospective (cohort) studies. For both short- and long-term effects, the mortality outcome was retained as the outcome with most convergent results.

Italy has received a specific interest in biomedical journals, and so there was a particular attempt to evaluate the situation in that country: studies performed in Italy were allowed to have broader inclusion criteria. In particular, the following series of studies were included:

- Several publications on the area of Brindisi where a major power plant is located;
- Publications on the EpiAir project;
- Some selected publications of importance performed in other areas of Italy;
- The Sentieri report.

The following MeSH terms were used in addition to the previously described selection criteria (in parenthesis year of introduction of the MeSH term):

- Particulate matter (2007)
- Air pollution (not reported)
- Air pollutants (1975)
- Particle size (1973)
- Cohort studies (1989)

2.4 Contribution of coal power plants to air quality

2.4.1. Particulate matter

Definition

Particulate matter (PM) consists of all particles, either solid or liquid, that can be carried by air, regardless of their composition. Particulate matter can be categorized according to the diameter of particles: PM_{10} (diameter < 10µm), $PM_{2.5}$ (diameter < 2.5µm), $PM_{0.1}$ (diameter < 0.1µm), and so on. Origins of such particulate are both natural and artificial; moreover there are primary sources and secondary PM sources (emitted from precursor substances such as SO_2 or NH_3).

Health effects

Health effects of PM have been widely studied and will be detailed in section 7. $PM_{2.5}$ can penetrate into the lung; the smallest particles ($PM_{0.1}$) can even be absorbed by blood. As the health effects associated with particulate matter have become better understood, a series of regulations have been established to limit their concentrations (cf European Legislation and Norms).

Emissions and Air Quality

PM emissions are described in another section (2.5.2.1). Concerning PM emissions from coal plants, European limits are 30mg/m^3 for 24 hours and 40mg/m^3 for half an hour. In case these limits are exceeded, sanctions are applied and the plant can be shut down (Eikmann et al, 2011). Electrostatic precipitators and fabric filters are used in coal plants for reducing PM emissions. Using these equipments in combination with other can reduce the PM emissions by 99%.

2.4.2. Sulphur dioxide

Definition

Sulphur dioxide (SO_2) is a gaseous molecule which is typically formed during combustion processes. However, the formation of SO_2 occurring during combustion is limited by the sulphur content of the fuel used. For example, the sulphur content of coal is highly variable depending on the coal subtype (anthracite, lignite etc) and the geographic origin of the coal. SO_2 is also produced and used in other industries such as chemical or pharmaceutical industries as a solvent, as an additive, or for bleaching papers. It is also an approved food additive (antioxidant E220).

Health effects

SO_2 is a highly toxic and corrosive gas that can cause irritation of the nose, throat, skin, and more severe outcomes at high doses such as pulmonary oedema and event death. WHO recommendations for short-term exposure is a limit of 500µg/m^3 for 10 minutes (WHO 2005) and 20µg/m^3 for 24 hours. There is no recommendation for longer exposure periods. According to IARC, SO_2 cannot be classified as to its carcinogenicity in humans (group 3 carcinogen) (IARC, 1992).

Emissions and Air Quality

Coal plants are significant SO_2 emitters, but emissions have decreased significantly in Europe during the past 3 decades (*cf* 2.5.2.4 Sulphur oxides (SO_x)). Emission limits for SO_2 and SO_3 from coal-fired power plants are the following (Eikmann et al, 2011):

- Daily average: 200 mg/m^3 and desulphurisation of 85% for low sulphur coal; 400 mg/m^3 and 95% desulphurisation for high sulphur coal;
- 30 minute average: 400 mg/m^3 and desulphurisation of 85% for low sulphur coal; 800 mg/m^3 and 95% desulphurisation for high sulphur coal.

Current SO_2 levels in many industrialised countries and regions of Europe are very low. For example, the highly industrialised Rhine-Ruhr region of Germany levels were 8µg/m^3 in 2007 (Eikmann et al, 2011). Flue-gas desulphurization is an efficient way to reduce SO_2 emissions from coal plants.

2.4.3. Nitrogen oxides

Definition

Nitrogen oxides are a wide family of gases composed of oxygen and nitrogen. However, only nitrogen monoxide (NO) and nitrogen dioxide (NO_2) are relevant to coal plants. These will be referred later as NO_x. NO_x emissions from coal plants are about 95% NO and 5% NO_2.

Health effects

Nitrogen oxides are irritant and toxic gases that can potentially harm the respiratory tract, and enhance respiratory problems in sensitive populations, for example asthmatics. Nitrogen oxides can also react with other substances and form particulate matter. NO_x have also effects on environment: it can harm the ozone layer and contributes to the formation of summer smog (Lindvall 1985). IARC evaluated the carcinogenicity of ingested nitrite and nitrate under conditions that result in endogenous nitrosation. In this particular context, nitrites and nitrates are probably carcinogenic to humans (IARC group 2A (IARC, 2010). This evaluation is not relevant for exposures related to coal plant emissions.

Emissions and Air Quality

Road traffic is the main source of NO_x emitions (cf 2.5.2.3 Nitrogen oxides (NO_x)), but coal plants also produce NO_x. There are also natural sources of NO_x, for example volcanic eruptions. The WHO recommendation is an annual limit value of 40µg/m^3 and an hourly limit of 200 µg/m^3 (WHO, 2005). Selective and non-selective catalytic reduction can be used to reduce NO_x emissions.

2.4.4. Carbon monoxide

Definition

Carbon monoxide (CO) is a colourless, odourless and highly toxic gas. It is formed when combustion processes are incomplete. It is also produced and used in the chemical and metal industry.

Health Effects

CO is a very toxic gas. Inhalation can lead to death at extreme doses (more than $1 g/m^3$). Biologically speaking, CO blocks the transport of oxygen in blood by binding to haemoglobin (instead of O_2). However, such doses are expected in case of accidents and bad ventilation; they are not found in nature and normal levels are so low that there is no associated health risk.

Emissions and Air Quality

Power plants contribute to about 4% of total CO emissions (Eikmann et al, 2011). Limit values for coal plants are 150 mg/m^3 for 50-100 MW plants and 200 mg/m^3 for >100 MW. Limit values in the atmosphere are 10 mg/m^3 for 8 hours. Actual levels are well below this limit (0.063-0.25 mg/m^3). Therefore, CO concentration is not a significant air quality concern. CO emissions in coal plants can be prevented with increased maintenance.

2.4.5. Carbon dioxide

Definition

Carbon dioxide (CO_2) is a colourless and odourless gas. It is a natural component of air accounting for about 0.04% (Eikmann et al, 2011). It is formed naturally, during volcanic eruptions or by breathing of animals and plants, but also artificially, for example during combustion processes. Moreover, CO_2 plays a major role in the carbon cycle, a bio-geological cycle in which carbon is exchanged between biosphere (plants and animals), pedosphere (soil), geosphere, hydrosphere (oceans) and atmosphere.

Health Effects

CO_2 is a natural component of air, therefore there are no health effects at low doses. Concentrations of 0.5% in air are considered as the maximal value for occupational exposure (8 hours a day). High CO_2 concentration can cause fatigue, dizziness, high blood pressure and a variety of ailments. Extreme concentrations (> 8%) are lethal within hours (Eikmann et al, 2011). However, such concentrations are not found in nature. Although CO_2 is not a health issue, it is a major greenhouse gas and contributes to global warming.

Emissions and Air Quality

CO_2 emissions by sector are available online for Europe[9]. In EU-27, about one third of CO_2 is emitted by the public electricity and heat production sector. Total CO_2 emissions have been stable or slightly decreasing between 1990 (4141×10^6 tonnes) and 2007 (4017×10^6 tonnes), with large increases recorded in the transport sector and decreases in other sectors (energy industries, industry, etc.). Reducing CO_2 emissions from coal plants is straightforward and can only b achieved by the introduction of high-efficiency coal plants.

2.4.6. Dioxins and furans

Definition

Dioxins and furans are broad families of chlorinated aromatic hydrocarbons. These molecules have 3 aromatic cycles and have similar properties. These substances are by-products of combustion processes. They are destroyed at high combustion temperature (900°C). The majority of dioxin and furans are produced artificially, yet there are some natural sources such as forest fires (Eikmann et al, 2011).

Health Effects

The best-known and best-studied dioxin is 2, 3, 7, 8-terachlorodibenzo-*para*-dioxin (2, 3, 7, 8 TCDD). Exposure to 2, 3, 7, 8 TCDD increases the risk of soft tissue sarcoma, non-Hodgkin's lymphoma and lung cancer, even at low doses. This is an IARC group 1 carcinogen (IARC 2012d). Similarly, 2, 3, 4, 7, 8-pentachlorodibenzofuran was also classified as group 1 carcinogen by IARC (IARC 2012d). Dioxins are particularly harmful because they can enter the body with food ingestion and are stored in the body fat. These molecules are capable of staying in the body for long periods of time as their half-life is between 7 and 20 years. Dioxins and furans can also cause immunological and reproductive disorders, and developmental disorders in young children. Dioxins are also toxic at high doses, and can cause skin damage. It has been recommended the daily intake of dioxins should not exceed 1-2 pg/kg in humans (Eikmann et al, 2011).

Emissions and Air Quality

To date, there is no European regulation for dioxin/furan emissions from coal plants (Eikmann et al, 2011). However, national regulations exist, eg $0.1 ng/m^3$ in Germany. Such emissions are not of concern in coal plants because formation of dioxin/furans from coal combustion is limited by the amount of chlorine contained in coal, which is usually very small (<0.5%).

[9] http://ec.europa.eu/energy/publications/doc/statistics/ext_co2_emissions_by_sector.pdf (accessed 15/03/2013)

The main emitter of dioxins and furans is waste incineration and the metal processing/extraction sectors. It is worth noting that total dioxin/furan emissions have decreased strongly with time.

2.4.7. Heavy metals

Definition

By definition, heavy metals are Antimony (Sb), Arsenic (As), Cadmium (Cd), Chromium (Cr), Cobalt (Co), Copper (Cu), Lead (Pb), Manganese (Mn), Nickel (Ni), Thallium (Tl), Tin (Sn) and Vanadium (V) (Eikmann et al, 2011). Mercury (Hg) is presented in more detail in the next section (Section 2.4.8).

Health Effects

Food is the main source of heavy metal exposure in humans. Effects caused by inhalation are very scarce. Health effects of heavy metals are heterogeneous. For example, copper and zinc are essential to humans as trace elements. On the other hand, Arsenic, cadmium, chromium and nickel have been established as carcinogenic to humans (IARC, 2012a). Moreover, lead is a known neurotoxic element (Murata et al, 2009) and heavy metals can cause renal damage (Sabath and Robles-Osorio, 2012).

Emissions and Air Quality

Heavy metal emissions from coal plants depend on the content of the coal. Coal power plants are mainly emitting Arsenic, Cadmium and Nickel. However, emissions have decreased greatly with time for all heavy metals (EEA 2012). Current emissions from power plants are less than 1µg/m^3. There is currently no European legislation specifying a maximum emission concentration for heavy metals. However, target values for ambient concentrations exist: 6ng/m^3 for arsenic, 5ng/m^3 for Cadmium and 20ng/m^3 for Nickel. Current ambient concentrations of heavy metals are well below these limits (Eikmann et al, 2011).

2.4.8. Mercury

Definition

Mercury is a liquid metal. Before it toxicity was known, it was widely used in several sectors. For example, it was used in thermometers, electrical switches, lamps, and even as disinfectant and drugs. Nowadays such uses are forbidden by law.

Health Effects

Mercury is a dangerous poison. It is mainly ingested from food sources, especially fish, as methyl-mercury (Me-Hg). Mercury exposure of young children and foetuses can lead to impaired neurological development, and is also a neurotoxic in adults (Fernandes Azevedo et al, 2012). It can also damage kidneys. Moreover, methyl-mercury is a possible human carcinogen (IARC classification 2A)(IARC 1993).

Emissions and Air Quality

Coal contains mercury as trace element; the exact proportion depends on the coal subtype. Mean mercury concentrations are between 0.04 mg/kg (Indonesian coal) to 0.35 mg/kg (Polish coal). Mercury is produced during coal combustion and is released as vapour (Hg^0) and as the oxidised form (Hg^{2+}). Most mercury emissions are produced by coal plants but these emissions can be greatly reduced using wet flue gas scrubbers. This equipment efficiently reduces Hg^{2+} emissions and is not relevant for Hg^0. In combination with other equipments (electrostatic precipitator, fabric filter....), mercury emissions can be reduced by up to 90%. Thanks to the introduction of these technologies, mercury emissions have sharply decreased over the past years (EEA, 2012). Ambient mercury concentrations are rarely measured by air monitoring stations.

2.4.9. Ammonia

Definition

Ammonia (NH_3) is a transparent, pungent-smelling gas. It is a basic chemical which is used mainly as a fertilizer in agriculture. Free ammonia is rare because it reacts quickly with other compounds.

Health Effects

NH_3 is a toxic and irritating gas. Inhalation of extreme doses can cause death. Such accidents are very rare because the distinctive smell of NH_3, even at low concentrations, acts as a warning signal.

Emissions and Air Quality

More than 95% of NH_3 is produced by the agricultural sector (EEA, 2012). NH_3 is not produced during coal combustion. However, it is used in coal plants for removing nitrogen. NH_3 is used for transforming NO_x into water and nitrogen. Some of the non-reacted NH_3 is emitted in the air. NH_3 emissions from coal plants are negligible. No specific regulations are applied to coal plants. Current annual NH_3 concentrations are between 3 and 18 µg/m^3, which is not considered as being dangerous for health (Eikmann et al, 2011).

2.4.10. Radioactive emissions

Definition

Radioactivity is the property of some elements to decay and emit radiation (α, β, γ rays for example) in the process. Sources of radioactivity can be natural as well as artificial (eg X rays).

Health Effects

Ionising radiation typically cause DNA damage, although not all radiation exposure leads to a biological effect because of DNA repairing systems. Radioactive emission is a well known recognised carcinogen to human (IARC classification group 1) (IARC, 2012c).

Emissions

There are radioactive elements in coal. However, there are no limit values for determining the amount of radioactive elements released by coal-fired power plants. Moreover, emissions from coal plants are negligible compared with natural emissions.

2.5. Total emissions of $PM_{2.5}$, PM_{10}, SO_x and NO_x in Europe

2.5.3. Data sources

Emission data are available online on the European Environmental agency website[10]. These data are based on National Emissions reported to the Convention on Long-range Trans-boundary Air Pollution (LRTAP Convention). Emissions from several air pollutants are estimated annually in European countries. Methods used for estimating pollutant emissions vary from country to country, but have to follow EMEP/EEA guidelines. Raw emission data were downloaded from EEA website. On the basis of considerations reported in Chapter 1 and in sub-chapter 2.4 (above), emissions of $PM_{2.5}$, PM_{10}, NO_x and SO_x were the focus of this Chapter.

Emissions sources were provided by activity sector, using EMEP/NFR 09 codes (EMEP/EEA 2009). These codes were grouped into aggregated sectors according to Appendix 5 of EEA Technical report No 8/2012 (EEA 2012). There are 11 aggregated sectors: Energy production and distribution, Energy use in industry, Non–road Transport, Road Transport, Commercial, Institutional & Households Energy Use, Industrial Processes, Solvent and Product Use, Agriculture, Waste, Natural Disasters

[10] http://www.eea.europa.eu/data-and-maps/data/national-emissions-reported-to-the-convention-on-long-range-transboundary-air-pollution-lrtap-convention-6 (29/01/2013)

and other sources. As an example, emissions from coal plants are part of the "Public Electricity and Heat Production" sector (NFR 09 code: 1 A 1 a), which is part of the "Energy Production & Distribution" EEA aggregated sector. Data availability by pollutant is described in Appendix 1. Most countries provide data from 1990 to 2010 for most aggregated sectors.

From raw data, emission estimates were grouped into aggregated sectors. Time trends of emissions were plotted for each aggregated sector. Finally, a histogram of emissions by aggregated sector was made for the last available year. All graphs were prepared with R software version 2.13.

2.5.4. Pollutant levels

For each pollutant ($PM_{2.5}$, PM_{10}, NO_x and SO_x), country-specific emission trends and pollution sources are presented in Appendix 1. For each country providing data and each pollutant, time trends for each aggregated sector as well as total emissions were plotted. For the latest available year, details of each aggregated sector were displayed. Data were aggregated for several countries having complete data for the period 1990-2010[11] for key sectors (Energy Production & Distribution, Energy Use in Industry, Non−road Transport, Road Transport, Commercial, Institutional & Households Energy Use, Industrial Processes, Agriculture and Waste). A short description of European and Italian trends are summarized below.

2.5.4.1. $PM_{2.5}$

Data for EU-27 $PM_{2.5}$ emissions exist since 2000. The main sector emitting $PM_{2.5}$ in the EU is Residential Stationary Plants, which are mainly products of household firewood combustion. Residential Stationary Plants emissions are the main source of $PM_{2.5}$ in its aggregated sector (Commercial, Institutional and Household Energy Use); this sector represented 52% of total EU-27 $PM_{2.5}$ emissions in 2010 (Figure 2.2). The second major contributor is Road Transport (16% of 2010 emissions), followed by Industrial Processes (11%) and Energy Use in Industry (7%). Energy Production and Distribution is the fifth largest contributor to $PM_{2.5}$, with 6% of all $PM_{2.5}$ emissions in 2010.

$PM_{2.5}$ emissions decreased by 15% in EU-27, from 1566 kT in 2000 to 1333 kT in 2010. Among the top five contributors, Public Electricity and Heat Production achieved a 41.5% reduction in $PM_{2.5}$ emissions over the period monitored. On the contrary, emissions from Residential Stationary Plants increased over the 2000-2010 period.

[11] Bulgaria, Croatia, Estonia, France, Ireland, Italy, the Netherlands, Portugal, Sweden, Switzerland and the United Kingdom

In Italy, data are available since 1990. As of 2010, the main sector contributing to $PM_{2.5}$ pollution is Commercial, Institutional and Energy Use (96.1 kT, or 50% of total emissions), followed by Road Transport (29.8 kT/16%), Non-road Transport (24.2kT/13%), Energy Use in Industry (13.3/7%) and Waste (10.0kT/5%). Energy Production and Distribution is the eighth largest contributor to $PM_{2.5}$ with 5.1 kT (2.6%). All sources involved in Public Energy and Heat Production released 1.6kT of $PM_{2.5}$ in Italy (0.8% of $PM_{2.5}$ emissions).

A slight decrease of total $PM_{2.5}$ emissions has been observed. However, some sectors increased their emissions whereas others decreased theirs. The main increase in $PM_{2.5}$ emissions was observed for Commercial, Institutional and Household Energy Use (driven by residential stationary plants: from ~45kT in 1990 to 96.1kT in 2010) and Non-road Transport (mainly international maritime navigation). Energy production and distribution emissions sharply decreased over the period, from more than 45 kT in 1990 (second largest contributor) to 5.1 kT in 2010 (eighth largest contributor). Other important decreases in $PM_{2.5}$ emissions were found for the sectors Road Transport and Energy Use in Industry.

2.5.4.2. PM_{10}

PM_{10} follows a similar pattern to $PM_{2.5}$. PM_{10} emissions are a bit higher than $PM_{2.5}$ emissions, which is expected as PM_{10} contains $PM_{2.5}$. Like for $PM_{2.5}$, the main contributor to PM_{10} emissions was Residential Stationary Plants, which accounted for 36% of total PM_{10} emissions in 2010. Other contributors were Road Transport (15%), Industrial Processes (15%), Agriculture (11%) and Energy Production and Distribution (7%) (Figure 2.3). Between 2000 and 2010, PM_{10} emissions in the EU-27 decreased by 14 %. PM_{10} emissions from Residential Stationary Plants increased between 2000 and 2010. Emissions from the Energy Production and Distribution sector decreased sharply over the study period (-42%); this decrease was even more marked for the Public Electricity and Heat Production sector (-49%). Other decreases were found for Road Transport, Energy Use in Industry and Industrial Processes.

In Italy, patterns were similar as in the rest of European Union. The main PM_{10} emitter in 2010 was the sector involved in Commercial, Institutional and Household Energy Use (96.8kT/44% of emissions), followed by road transport (34.0kT/15%), non-road transport (24.3kT/11%), agriculture (18.4kT/8%) and industrial processes (15.1kT/7%). Energy production and distribution was the eighth most important source of PM_{10} emissions (5.2kT/2%). 1.7 kT of PM_{10} were emitted by public electricity and heat production in 2010, representing 0.8% of total PM_{10} emissions.

Total PM_{10} emissions slightly decreased between 1990 and 2010, but patterns are heterogeneous. PM_{10} emissions from Residential Stationary Plants increased, whereas emissions from several sectors including Energy Production & Distribution, Road Transport and Energy Use in Industry sharply decreased.

2.5.4.3. Nitrogen oxides (NO_x)

Important emission sources of nitrogen oxides (NO_x) in Europe in 2010 are Road Transport (42% of emissions), Energy Production and Distribution (20%), Commercial, Institutional and Household Energy Use (14%), Energy Use in Industry (13%) and Non-road Transport (7%)(figure 2.4). Between 1990 and 2010, NO_x emissions decreased in the EU-27 by 47 %, mainly because of stricter regulations and emission standards, and a decline in the use of solid fuels. Large decreases in emissions were observed in road transport (e.g. passenger cars: -59.6% between 1990 and 2010). Reduced emissions from Road Transport are largely due to the introduction of three-way catalytic converters on cars and stricter regulation of emissions. Despite these technical and regulatory improvements, the Road Transport remains the largest source of NO_x emissions in 2010. In the Electricity/Energy Production sectors, large reductions were observed, as a result of a series of technical improvements (combustion modification technologies, flue-gas abatement techniques and fuel switching from coal to gas).

In Italy, NO_x emissions data are available from 1980 to 2010. As of 2010, energy production & distribution was the fifth most important source of NO_x emissions with 79.1 kT (7% of emissions), far below Road Transport (490.5 kT/43%), Non-road Transport (275.7 kT/24%), Commercial, Institutional and Household Energy Use (143.0 kT/13%) and Energy Use in Industry (131.0 kT/11%). Public Electricity and Heat Production emitted 40.9 kT in Italy in 2010 (3.6% of total emissions).

NO_x emissions increased until 1992, and decreased from 1992 onwards. Trends in emissions are not monotonous. Road Transport Emissions increased until 1992 and decreased after; Energy Production and Distribution increased until the mid eighties and decreased thereafter; Non-road Transport emissions decreased until the early nineties and increased after; finally Emissions from Energy Use in Industry continuously decreased over the study period.

2.5.4.4. Sulphur oxides (SO_x)

In EU-27 in 2010, the main source of SO_x emissions was from the Energy Production and Distribution sector (58% or total emissions), followed by Energy Use in Industry (20%), and Commercial, Institutional and Household Energy Use (13%) (Figure 2.5). Public Electricity and Heat Production is the most important key category for SO_x emissions, making up 47 % of total SO_x emissions. In Italy, SO_2 emissions from Public Electricity and Heat Production sources are much lower that the European average (9% in Italy compared to 47% in Europe).

Between 1990 and 2010, SO_x emissions decreased in the EU-27 by more than 80%. The biggest reductions in emissions between 1990 and 2010 were found in Stationary Combustion in Manufacturing Industries and Construction: Other (– 80.8%), Public Electricity and Heat Production (– 79.8%) and Residential: Stationary Plants (– 66.5%). For these main emitting sources, the reduction in emissions since

1990 is the result of a combination of measures, including switching fuels in energy sectors to low-sulphur fuels such as natural gas, the fitting of flue-gas desulphurisation abatement technology in industrial facilities, and the impact of European regulations relating to the sulphur content of certain liquid fuels.

In Italy, data are available from 1980 onwards. The situation is particular because emissions from volcanoes are by far the largest source of SO_x in the country. Volcanic emissions are highly variable with time; as an example, 2500 kT of SO_x were released by volcanoes in 2010. Excluding volcanoes, 341.8 kT of SO_x were emitted in 2010. The main contributors were Non-road Transport (153.9kT/45% of emissions), Energy Production and Distribution (107.2kT/31%) and Energy Use in Industry (47.4kT/14%). Concerning the Energy Production and Distribution sector, the main pollution source is the Petroleum Refining sector (40.9 kT/12%), followed by the sector involved in Public Electricity and Heat Production (31.1 kT/9%).

Sharp decreases in emissions were observed over the study period. Excluding volcanoes, the Energy Production & Distribution sector used to be the largest source of SO_x until the 2000s. SO_x emissions from this sector decreased dramatically from about 2000 kT in 1980 to 107.2 kT in 2010. Large decreases in emissions were also observed for energy use in industry.

SO_2 emissions

SO_2 emissions are mainly from power plants in general and coal-fired power plants in particular. However, because of the volatile nature of SO_2 and long lifetime, SO_2 emissions in one area will not necessarily affect the population nearby the power plant. As an example, in Toronto (Environment Canada 2003) although 70% of SO_2 emissions were identified as from power plant and metal smelters, and despite the presence of a small power plant in Toronto, most of the emissions were from external sources. Hence, control of SO_2 emission would require control of emission upwind of the city.

2.5.5. Energy production and distribution

The *Energy Production and Distribution* sector grouping comprises emissions from a number of activities involving fuel combustion for the production of electricity. The following activities are included in this sector:

- Public electricity and heat production
- Petroleum refining
- Manufacture of solid fuels and other energy industries
- Pipeline compressors
- Fugitive emission from solid fuels: Coal mining and handling
- Fugitive emission from solid fuels: Solid fuel transformation
- Other fugitive emissions from solid fuels

- Exploration, production, transport
- Refining/storage
- Distribution of oil products
- Natural gas
- Venting and flaring
- Other fugitive emissions from geothermal energy production, peat and other energy extraction

It is an important source of many pollutants, especially SO_x. Time trends in emissions from this activity sector are displayed in Figure 2.6. There were decreases in emissions for each of the pollutants ($PM_{2.5}$, PM_{10}, SO_x and NO_x). Despite huge past reductions (-81% between 1990 and 2010), the Energy Production and Distribution group still contributes 58 % of the total EU-27 SO_x emissions (omitting emissions from volcanoes). Within this sector, the Public Electricity and Heat Production sector and the Petroleum Refining sector are the main sources of SO_x. The Energy Production and Distribution sector is also an important source of NO_x, but far below emissions from Road Transport. Moreover NO_x emissions halved between 1990 and 2010. The sector is a minor source of $PM_{2.5}$ and PM_{10} pollution, and emissions of particulate matter have been decreasing steadily in this sector: for example a 43% reduction in PM_{10} emissions has occurred within this sector group since 2000 in the EU.

For each country, we computed the total emissions per capita (in kT per million) (Appendix 2). Populations were retrieved from the World Population Prospects, 2010 revision. Maps were made using R package rworldmap. Compared to other European countries, Italy has very low emission per capita for $PM_{2.5}$ (first quintile), PM_{10} (first quintile), SO_x (2nd quintile) and NO_x (first quintile). There is a clear spatial pattern in pollutant emissions: countries emitting low amount of pollutants per capita are mainly located in Western and Southern Europe, while higher emission levels per capita were observed in Central, Eastern and Northern Europe.

2.6. Sources and Factors influencing the pollution with $PM_{2.5}$

Among sources of $PM_{2.5}$, those primarily emitting $PM_{2.5}$ can be distinguished from secondary sources emitting precursor gas which will be later transformed in $PM_{2.5}$ in the atmosphere.

2.6.3. Weather

$PM_{2.5}$ particles have a very small weight and then a long lifetime. This favours the transportation over long distances, over 1000km (Brook et al, 1999; NARSTO 2003). Study of the source of ambient pollution is highly complex and requires the use of methods that enable the identification of the role of dissemination of pollutants.

In an analysis of $PM_{2.5}$ sources around the city of Toronto (Brook et al, 2007), it could be demonstrated that the major factor responsible for $PM_{2.5}$ episodes was the weather. Precipitation, wind, local or large-scale stagnation, vertical mixing could strongly influence the concentration of $PM_{2.5}$ particles. 55 to 70% of $PM_{2.5}$ in Toronto were not from local sources but transported into the area.

Similarly in Italy, in a study conducted in the Milan metropolitan area, in winter the conditions of atmospheric stability caused very high concentration of atmospheric pollutants at ground level (Ferrero et al, 2010).

2.6.4. Traffic

In a study conducted in Milan, a city where air quality limits are frequently exceeded, the source apportionment of PM showed that traffic was the strongest primary source of $PM_{2.5}$ (17-24%) (Perrone et al, 2012). Secondary sources were influenced by biomass burning including residential heating (1-30%). Several studies using chemical mass balance, i.e. a method used for the apportionment of source of emitters of particulate matters, described motor vehicles as a major source of emission of $PM_{2.5}$ and PM_{10}. Emissions from motorized vehicles depend on the type of vehicles, the engine and speed of vehicle. The type of fuel has also a role: diesel exhausts are major sources contributing to $PM_{2.5}$ and PM_{10} mass (Srimuruganandam and Shiva 2012). The particulates are primarily coming from exhaust pipes, but also come from break and tire wear (Saedler et al, 1996).

The EEA estimates that 70% of environment pollution and 40% of greenhouse gases are emitted by motorized transport in Europe (EEA 2010). In the United States, the EPA estimates that more than 50% of total PM emissions are coming from motorized vehicles (US Environmental Protection Agency 2004). Study in the Yampa valley (Colorado, USA), showed that around 46% of primary $PM_{2.5}$ were emitted by motor vehicles. In summer, 21% of $PM_{2.5}$ were emitted from natural dust sources and 11% from agricultural tilling (Watson et al, 2001).

2.6.5. Coal-fired power plants and impact of regulations

The body of scientific evidence demonstrates that coal plant emissions represent a small contribution to the total $PM_{2.5}$ and, consequently, only provide a small contribution to overall air pollution, as demonstrated by the lack of adverse impact on health from these sources reported from epidemiological studies considered in this document (see Chapter 1). Effective methods exist now to reduce emissions of SO_2, NO_x and fine particles from coal burning[12]. For example, electrostatic precipitators enable a removal of 99% to 99.99% of fine particles. Fabric filters also enable reducing particles of size between 0.01 to 100 µm ($PM_{2.5}$ including ultrafine particles) with a decrease of 99% to 99.9999%. Wet scrubbers for particulate

[12] http://www.iea-coal.org.uk/site/2010/database-section/clean-coal-technologies (accessed 19/03/2013)

control also allow a removal of 90% to 99.9% of fly ash in addition to sulphur dioxide.

These modern technologies for reducing emissions have been adopted in Europe, in particular in Italy, for complying with best available techniques (BAT) in energy production from coal burning. These were successively introduced through regulation. In 1988, the European Commission issued a directive limiting emissions from large combustion plants (*Council Directive 88/609/EEC of 24 November 1988 on the limitation of emissions of certain pollutants into the air from large combustion plants*). On the 24th of September 1996, the *Council Directive 96/61/EC concerning Integrated Pollution Prevention and Control (IPPC)* was introduced. A directive on *Large Combustion Plant* (*2001/80/EC*) set minimum requirements to be met by all installations in term of emissions limit values and monitoring requirements. The IPPC was revised in 2008 2008/01/EC aiming at high level of protection for the environment.

These changes of regulation had a clear effect on emissions from energy production sector (Figure 2.6).

2.6.6. Role of heating

Residential coal combustion for heating is also an important source of pollution as in Asia and still in North America. In the study in Yampa Valley (Colorado, USA), in winter months, the combustion of coal or wood was estimated to contribute to 11% of emissions of $PM_{2.5}$ (Watson et al, 2001).

In China, use of coal for heating is still widely used. In 2004, it was estimated that $PM_{2.5}$ emissions in Beijing were mainly coming from coal combustion. The study could not differentiate further the source of emission. Initiatives to reduce these emissions were: to reduce the use of coal-fired light-duty boilers used for winter heating, and to replace old coal-fired power plants with new technologies such as coal gasification technologies (Song et al, 2007).

While the use of coal for residential heating remains widely used worldwide, this source of heating has became marginal in Europe, in particular in Italy where coal is not used anymore for this purpose. Several European countries introduced legislation to discourage or even ban the use of coal in major cities for residential heating. Residential heating in western countries is now primarily based on biomass burning, wood being the main biomass source used. This shift to biomass burning for residential heating has increased with time and can be seen in trends of emission of pollutants in the appendix of the present report.

Whatever source is used for heating, residential heating contributes significantly to the global air pollution because of the absence of methods to reduce emissions of pollutants from burning of biomass.

2.6.7. Is there a bias in apportionment studies of particulate matters?

The placement of air quality monitoring stations respects European norms and includes urban and rural background stations so that the air quality studies can take into account possible hotspots and background concentrations. The studies described above are mainly conducted with $PM_{2.5}$ or PM_{10} assessment carried out in close proximity to roads with heavy traffic. Hence, it could be suspected that the contribution of motor vehicles might be overestimated. However, as mentioned earlier, the weather has an important role in the diffusion of PM, and particulates matters emitted by motorized vehicles will rapidly be dispersed by winds and not necessarily stay where the emission takes place. Furthermore, the measurement of interest for potential health effect should correspond to places where the population lives and has its major activities. Major roads, where most studies were conducted, are generally in areas also dense in residential areas, commercial areas, several educational institutes, hospitals, and suchlike. These studies could then be a good marker of exposure of populations to $PM_{2.5}$ and PM_{10}.

2.6.8. Reduction of PM_{10}/$PM_{2.5}$ emissions

Several studies provided information on the sources of pollution. To determine if an emitter has an important role and if real action could be taken, investigated was conducted into whether regulation of emissions was followed by measureable decreases in air pollution in studies adopting a scientific design. As a consequence, several attempts to reduce population exposure to this pollution have been set up. In most countries, regulations and intervention were conducted to reduce the concentration of PM, with quantifiable targets to meet in the next years (*cf* European Legislation and Norms).

Traffic control can effectively reduce air pollution. A study of the impact of reduction of speed limits on motorways in Amsterdam and Rotterdam (the Netherlands), showed that reducing from a speed limit of 100km/h to 80km/h had strong impact on the congestion of traffic (a drop from 42% to 14% in Amsterdam and from 40% to 20% in Rotterdam). This also resulted in reduction of NO_x emission in the range of 5-30% and reduction in PM_{10} emissions in the range of 5-25% (Keuken et al, 2010). Of note, this traffic control also has the potential impact of reducing traffic noise and accidents (Stoelhorst 2008).

In India, the PM_{10} concentration was evaluated and modelled in four cities. Overall an improvement of air quality was observed with an average decrease ranging between 2 to 5µg/m^3 per year. This decrease was mainly attributed to strong intervention on the vehicle sector and traffic management (Gupta et al, 2010).

It was also suggested that green space planning could help in reducing PM_{10} concentration (Tiwary et al, 2009). Some pine trees such as *Pseudotsuga menziesii* have an important capacity to intercept particulates from the atmosphere, and could therefore be used in green space planning in large cities.

2.7. Health Effects of PM Pollution

This section describes major epidemiological studies on air pollution and health effects. Studies were sometimes conducted in time and place of higher global air pollution as observable these days in Europe. The time and place of each study conducted is reported as well as levels of air pollution measured.

2.7.3. Short-term studies

Short-term studies are generally time-series that examine relationships between daily changes in air pollution and daily mortality counts within a given city or metropolitan area. Multivariate regression models adjusting for potential confounders such as weather characteristics (temperature, wind, humidity), seasonal variations, co-pollutants and so on are employed. Methodological strengths of such studies include large sample size (up to several years of daily data), large range of population demographics (several socio-economic statuses and baseline health) and "real world" exposure. Weaknesses include the difficulty to assess the actual exposure of subjects, potential exposure misclassification and difficulty to attribute health effects to a specific pollutant.

2.7.3.1. Short-term effects: all ages

Several multi-area studies and over 100 single-area studies examining daily PM pollution and daily mortality are available in the scientific literature. Most important studies will be summarised and their methods commented in this section.

North America

Samet et al, (2000) studied relationships between PM_{10}, O_3, NO_2, SO_2 and CO pollution and daily mortality in 20 cities of the United States, all of them having at least 1 million inhabitants in the 1990 census (Samet et al, 2000). Pollution and mortality data were collected for the 1987-1994 time period. Pollution data were extracted from a database maintained by Environmental Protection Agency (EPA). 24-hour averages were used. Daily mortality data were obtained from the National Center for Health Statistics. Mortality from external causes (accidents, suicides, homicides) was excluded. Analysis was carried out in two steps. In a first step, regressions of daily mortality on air pollution were run for each city. In a second step, estimates were combined for all cities.

Mean daily PM_{10} levels ranged from 20µg/m^3 to 50µg/m^3 with extreme values (10th and 90th percentiles) between 8.9 to 78.7µg/m^3. PM_{10} levels were positively and significantly correlated with NO_2, SO_2 and CO but not with O_3. At city level, PM_{10}

levels were generally positively associated with death rates for all causes and for cardiovascular and respiratory deaths. No age-associated trend was detected. Combined analysis for all cities confirmed the association between PM_{10} levels and all-cause mortality, cardiovascular and respiratory causes. The estimated relative rate for all-cause mortality was 1.0051 (95% CI (1.0007; 1.0093)) per 10 $\mu g/m^3$ increase in PM_{10}. Result for cardiovascular and respiratory mortality was 1.0068 (95% CI (1.0020; 1.0116)) per $10\mu g/m^3$ increase in PM_{10}. Adjustment for other pollutants did not change results. Results for ozone were null.

Europe

Studies which took place in Europe were combined in a meta-analysis by a World Health Organisation (WHO) task group (WHO Europe, 2004). The following outcomes were studied: all-cause mortality (excluding accidents), respiratory and cardiovascular hospital admissions, cough and medication use in people with respiratory symptoms. Summary relative risks were calculated for $10\mu g/m^3$ increase in pollutant concentration. Studies included in the meta-analysis had collected data mainly during the early nineties, but some studies used older data; the oldest studies was performed between 1975 and 1985 (Spix and Wichmann, 1996). Average PM_{10} levels were not specified.

For all-cause mortality, 33 independent estimates were retrieved and the SRR was 1.006 (95% CI (1.004; 1.008)) for a $10\mu g/m^3$ increase in PM_{10} concentration. There was no evidence of publication bias. Results for cardiovascular and respiratory mortality were 1.009 (95% CI (1.005; 1.013)) and 1.013 (95% CI (1.005; 1.020)) based on 17 and 18 estimates, respectively. There was some evidence of publication bias for respiratory mortality and strong evidence of publication bias for cardiovascular mortality: studies having higher relative risks were more likely to be published.

This report also studied relationships between $PM_{2.5}$ and mortality. In this part, as only 3 studies conducted in Europe were available, studies conducted in the whole world are included. SRR associated with a $10\mu g/m^3$ increase in $PM_{2.5}$ levels were 1.009 (95% CI (1.006; 1.013)) for all-cause mortality (23 estimates); 1.013 (95% CI (1.005; 1.022)) for cardiovascular causes (8 estimates) and 1.011 (95% CI (1.002, 1.020)) for respiratory mortality (8 estimates).
[Publication bias was evaluated graphically with funnel plots, and tests were performed, but p-values were not provided.]

Most estimates used in the WHO meta-analysis come from the *Air Pollution and Health: a European Approach* 2 study (APHEA 2 study) (Katsouyanni et al, 2001; Katsouyanni et al, 2003; Katsouyanni et al, 1997). This project used daily data of PM_{10} and black smoke levels in 29 cities in 15 European countries. Data were collected between 1990 and 1997. Like in other studies, deaths from external causes were excluded. PM_{10} pollution data were 24-hour averages. Median PM_{10} and black smoke levels ranged between 14 and 166 $\mu g/m^3$ and between 9 and $64\mu g/m^3$, respectively. Several confounders (weather, meteo, day of the week....) were taken into account in the models. City-specific models were computed and then pooled. Analyses were restricted to days with concentrations below 150µg/m^3.

For all-cause mortality, estimates increases per 10µg/m^3 increase in PM_{10} ranged from 0.994 to 1.015. The pooled estimate for all cities was 1.0062 (95% CI, (1.004, 1.008)) for all ages and 1.007 (95% CI, (1.005; 1.010)) for people aged over 65. Adjustment for NO_2 decreased the pooled all-ages estimate to 1.0041 (95% CI (1.002; 1.007)). O_3 was also a confounder as adjustment increased the estimate to 1.0073 (95% CI (1.005; 1.009)).

Similar results were found for black smoke: values ranged between 0.998 and 1.016, and the pooled estimate was 1.0058 (95% CI (1.003; 1.008)) for all ages and 1.007 (95% CI (1.004; 1.009)). Adjusting for NO_2 decreased the summary estimate for all ages to 1.0026 (95% CI (1.000; 1.006)); adjustment for O_3 increased the estimate to 1.0088 (95% CI (1.005; 1.013)).

Asia

Lee et al (2000) reported associations between air pollution and mortality in seven south Korean cities accounting for half of the south Korean population (Le Tertre et al, 2002). Daily concentrations of total suspended particles (TSP), SO_2, NO_2 and CO were measured in several monitoring stations in each city, for the 1991-1997 time period. Mortality data were obtained for the same period from the National Statistics office of Korea. Deaths due to accidents were excluded. Data were analysed in two steps: city-specific models were run, and estimates were pooled in a second step.

Mean TSP levels ranged from 60 to 90µg/m^3, with extreme values (5^{th} and 95^{th} percentile) between 25.9 and 167µg/m^3. TSP levels were strongly and positively correlated with SO_2, NO_2 and CO levels, but not with O_3. A 100µg/m^3 increase in TSP concentration lead to statistically significant mortality rate ratios in 2 of the 7 cities. When estimates from the 7 cities were pooled, the mortality rate ratio was 1.028 [1.013; 1.044] after adjustment for weather and O_3, and 1.009 [0.998; 1.021] after further adjustment for SO_2. When estimates were obtained from a meta-analysis, the SRR was 1.022 (95% CI (1.012; 1.030)).
[Contrary to other studies, no PM_{10} or $PM_{2.5}$ data were available; total suspended particles data were used instead. Therefore this study is not directly comparable with others.]

The *Public Health and Air* population in Asia (PAPA) study was conducted in four Asian cities between 1996 and 2004 (Wong et al, 2008). Cities included were Bangkok (Thailand), Hong Kong, Shanghai and Wuhan (China). Daily mortality data for all natural causes were retrieved in each city. Pollution data were 24-hour averages for NO_2, SO_2 and PM_{10}; 8-hour averages for O_3 in several monitoring stations in each city. Both city-specific and pooled estimates were calculated.

Mean PM_{10} levels ranged from 51.6µg/m^3 (Hong Kong) to 141.8µg/m^3 (Wuhan), which is much higher than levels observed in Northern America and Western Europe. Overall, PM_{10} levels ranged from 13.7µg/m^3 (Hong Kong) to 566.8µg/m^3 in Shanghai. Relative risk for all natural causes mortality associated with a 10µg/m^3 increase of PM_{10} were statistically significant in each city; the pooled relative risk was 1.0055 (95% CI (1.0026; 1.0085)) for the 4 cities and 1.0037 (95% CI (1.0021; 1.0054)) for the 3 Chinese cities. Mortality risk was higher and less precise in Bangkok than in the other cities. Relative risks for cardiovascular mortality were 1.0058 (95% CI (1.0022, 1.0093)) for all cities and 1.0044 (95%CI (1.0019; 1.0068)) in China. Results for respiratory mortality were 1.0062 (95% CI (1.0022; 1.0102)) in all cities and 1.0060 (95% CI (1.0016; 1.0104)) in China. Relative risks increased with age in all cities. Sensitivity analyses showed that, in all cities, effect estimates were sensitive to exclusion of the highest concentrations. Concentration-response relationships were linear for the inter-quartile range of concentrations.
[*Note: this study was funded by HEI and a very detailed report is available (Health Effects Institute 2010).*]

Multicentric studies

European and Northern American data were used for the APHENA study (Air Pollution and Health: a combined European and Northern American approach) (Samoli et al, 2008). Daily PM_{10} and mortality data (excluding external causes) were collected in 90 United States cities (National Morbidity and Mortality Air Pollution Study or NMMAPS project), 22 European cities (APHEA project) and 12 Canadian cities. The analysis was restricted to days with PM_{10} concentration < 150 µg/m^3, with represented more than 98% of data in all but three cities. Analyses were conducted first at city-level, then at centre level (Europe, USA, Canada) and at global level. Models controlled for seasonal effects, weather and other potential confounders.

Relative risks in daily mortality associated with an increase of 10µg/m^3 of PM_{10} were 1.0076 (95% CI (1.0020, 1.0130)) in Canada, 1.0032 (95% CI (1.0021; 1.0042)) in the USA and 1.0024 (95% CI (1.0008; 1.0041)) in Europe, after adjusting for O_3 levels. Effects were more pronounced in the 75+ age group than in the younger age group. Air pollution risk estimates in Canada were more than double those in Europe and the USA; they were as well much less precise because of smaller sample size.

Stieb et al, (2002) conducted a meta-analysis of daily time-series studies conducted around the world (Stieb et al, 2002). 109 individual studies were included in the meta-analysis. Studies had to evaluate the relationship between daily pollution level (PM_{10}, CO, NO_2, SO_2, O_3) and daily mortality in general population. The summary relative risk for all-cause mortality corresponding to an increase of 31.3µg/m^3 of PM_{10} was 1.020 (95% CI (1.015, 1.024)). A larger SRR was found for respiratory mortality. There was a large amount of heterogeneity among studies, which was mainly explained by variability of pollutant concentrations.

Results of short-term studies are very consistent and suggest that short-term exposure to particulate matter is associated with small but statistically significant increases in daily mortality. Results are quite similar between countries. One hypothesis is that short-term exposures may affect primarily frail individuals with one or more prevalent co-morbidity (Dominici et al, 2003; Zanobetti and Schwartz, 2002; Zanobetti et al, 2000; Schwartz, 2000; Fung et al, 2005; Forastiere et al, 2007).

2.7.3.2. Short-term effects: children

Several studies have assessed the relationship between daily PM levels and childhood mortality (e.g. children aged less than 5). The main difficulty in these studies is to separate the effects of air pollution from other factors such as pre-existing illnesses, deprivation, exposure patterns or other aspects of lifestyle. There are few studies focussing on children, and all of them were conducted in middle income countries (Brazil, Mexico, Thailand).

Three studies assessing the relationship between daily respiratory mortality in young children (less than 5 years old) and pollution were conducted in São Paulo, Brazil (Coneçião et al, 2001; Gouveia and Fletcher, 2000; Saldiva et al, 1994).

Saldiva et al (1994) studied daily child mortality between May 1990 and April 1991 (Saldiva et al, 1994). Daily pollution data for SO_2, CO, PM_{10}, O_3 and NO_x were obtained retrieved from São Paulo state air pollution controlling agency. Daily mortality data were provided by the municipal mortality information improvement program. Respiratory mortality in children aged less than 5 was studied. Data were analysed with Poisson regression. It is important to note that this study uses data which represent the situation twenty years ago.

During the study period, the average concentration of PM_{10} was 82.38 µg/m^3 with a standard deviation of 38.82 µg/m^3. PM_{10} levels were strongly correlated with CO (r = 0.54), SO_2 (r = 0.67) and NO_x (r = 0.68) levels, but not with O_3 (r = 0.00). PM_{10} levels were not significantly associated with respiratory deaths in children: the regression coefficient was -0.603 (SE: 0.707; p-value 0.394) for a 100 µg/m^3 difference in PM_{10}. This corresponds to a RR of 0.54 (95% CI (0.14, 2.19)).

Gouveia and Fletcher (2000) used time-series to analyse air pollution and mortality at different ages in São Paulo, Brazil (Gouveia and Fletcher, 2000). The study took place in 1991-1993, again some years ago. In particular, they studied respiratory and pneumonia mortality among children under 5 years of age. Daily mortality data were provided by the municipal mortality information improvement programme. Socioeconomic status was evaluated based on district of residence. Pollution data for SO_2, CO, PM_{10} and O_3 were retrieved from São Paulo state air pollution controlling agency. Pollution levels were either 24-hour means (SO_2, PM_{10}), or 24-hour peak (O_3, NO_2) or greater 8-hour moving average (CO).

During the study period, pollution levels were relatively high and most pollutants exceeding recommended concentrations. Mean PM_{10} level was 64.3 (SD = 27.6) μg/m^3 and ranged between 19.6 and 184.5 μg/m^3. Relative risks were computed in relation to a change from the 10th to the 90th percentile in pollutant level. For PM_{10}, this corresponded to a change from 36.8μg/m^3 to 101.1 μg/m^3. The RR for respiratory death in children was 0.994 (95% CI (0.810, 1.098)); for pneumonia, the result was 1.041 (95% CI (0.900; 1.196)).

Conceição et al, (2001) studied relationships between child respiratory mortality and air pollution in 1994-1997 (Conceição et al, 2001). Daily mortality data were provided by the municipal mortality information improvement programme for children under 5 years. Pollution data for SO_2, CO, PM_{10} and O_3 were retrieved from São Paulo state air pollution controlling agency. Pollution levels were either 24-hour means (SO_2, PM_{10}), or 24-hour peak (O_3) or greater 8-hour moving average (CO). NO_2 was not studied because of technical problems. Weather data were collected from the Institute of Astronomy and Geophysics of the University of São Paulo. Daily mortality was modelled with Poisson regression adjusting for seasonality, temperature, humidity and non-respiratory deaths.

During the study period, the average PM_{10} level was 66.2 (SD = 31.2) μg/m^3. PM_{10}, SO_2 and CO levels were significantly correlated with each other (r from 0.44 to 0.60); O_3 levels were not correlated with other pollutants. Unadjusted coefficient of Poisson regression was 0.0043 (SD: 0.0006) for PM_{10}; this was statistically significant and corresponds to a RR of 1.0043 (95% CI: (1.0031; 1.0055)). The fully adjusted coefficient was 0.0016 (SD: 0.009); this was not significant and corresponds to a RR of 1.0016 (95% CI (0.9841; 1.0194)). The fully-adjusted relative risk corresponding to the highest vs. lowest quintile of PM_{10} was about 1.21 (95% CI (1.08, 1.35)) based on a figure.
[Mortality decreased strongly in the study period. It is unclear what the regression coefficient represents.]

Loomis et al (1999) studied infant mortality in Mexico City, Mexico between 1993 and 1995. Daily pollution data ($PM_{2.5}$, O_3, NO, NO_2, NO_x and SO_2) were measured. Mortality data excluding external causes were provided by the Instituto Nacional de Estadistica, Geografia e Informatica. Infant mortality was defined as death of children aged less than 1 year of age. Weather data (temperature and humidity) were retrieved from *Observatorio Meteorologico del Colegio de Geografia* (*Universidad Nacional Autonoma de México*). Daily infant mortality was modelled with Poisson regression.

During the study period, average $PM_{2.5}$ levels were 27.4μg/m^3 (SD: 10.5 μg/m^3; range 4-85 μg/m^3). 2798 infant deaths occurred over the whole period. Infant mortality was not associated with $PM_{2.5}$ levels the same day (RR = 0.9864, 95% CI (0.9449, 1.028)) or the previous 2 days (one day before: RR = 0.9905 95% CI (0.9490, 1.0320); two days before RR = 1.0278, 95% CI (0.9867, 1.0689)). However, a 10μg/m^3 increment in $PM_{2.5}$ 3 days before death was associated with a

RR of 1.042 (95% CI (1.0097, 1.0861)), and 1.048 (95% CI (1.0037, 1.0793)) for 4 days.

Ostro et al (1999) studied relationships between daily mortality and PM_{10} levels in Bangkok, Thailand between 1992 and 1995. Daily PM_{10} was measured by the Pollution Control Department (Thai Ministry of Science, Technology and Environment). Mortality data for all natural causes were provided by the Central Registrar office of Bangkok. Ostro et al (1999) studied mortality for all ages, and particularly children under 6 years. PM_{10} concentrations were between 64.4μg/m^3 and 73.5μg/m^3 on average (range: 21-227μg/m^3), depending on the method of measurement. There was an association between PM_{10} concentration and daily mortality in children aged less than 6. The RR (all natural cause mortality) for a 10μg/m^3 PM_{10} increase was 1.0180 (95% CI (1.0023, 1.0337)).

In conclusion, results for children were more conflicting than for results for persons of all ages. The relative risks for child mortality related to air pollution exposure are much less consistent. Moreover, very few studies were conducted specifically in young children. However, the results suggest a small increase in daily mortality.

2.7.4. Long-term effects

Time-series studies are useful to evaluate the effects of short-term exposure to ambient air pollution on mortality. However, such studies are not designed to detect effects with a lag period longer than a few weeks (Dominici et al, 2000). Effects of long-term exposure to particulate matter can be identified with cohort mortality studies.

Long-term studies are generally large cohort studies, of which the majority has been conducted in the USA. Such studies use individual data (e.g. baseline questionnaire about demographic characteristics, lifestyle, socio-economic status, etc.) in order to adjust for confounding factors. Models estimate the relationship between long-term PM averages and several causes of mortality, for example lung cancer and cardiopulmonary mortality. Several limitations occur in these studies. First, it is hard to detect brief life-shortening in a long-term study. Second, the studies take place when participants are adults, therefore exposure during childhood is not taken into account. Third, no within-city differences are included in the models. This last point is likely to produce null results.

Two long-term cohort studies conducted in the USA were carried out and have been used for the development of annual air quality standards: the Harvard Six Cities study (Dockery et al, 1993) and the Cancer Prevention Cohort II (CPS II) study of the American Cancer Society (ACS) (Pope et al, 2002).

The Harvard Six Cities study was the first large prospective study assessing relationships between long-term exposure to outdoor air pollution and mortality. This study was followed more than 8,000 subjects for 14 to 16 years (Dockery et al, 1993). Data collection started in 1974. Subjects were recruited in the mid seventies in 6 US cities (Watertown – MA, Harriman – TN, Portage – WI, St Louis – MO, Topeka – KS and Steubenville – OH). Fine particles ($PM_{2.5}$) concentration ranged between 11 and 30 μg/m^3. Relative risk for all-cause mortality was 1.26 (95% CI (1.08, 1.47)) in the most polluted city compared to the less polluted city. The RR for lung cancer mortality was 1.37 (95% CI (0.81, 2.31)). RR for cardio-pulmonary mortality was 1.37 (95% CI (1.11, 1.68)).

The CPS II cohort was set up in the early eighties. It included more the half a million adults living in all 50 US states, who were followed for 16 years. Specific outcomes of interest were all-cause mortality, lung cancer mortality and cardiopulmonary mortality (Pope et al, 2002; Pope et al, 1995). Air pollution data were collected in the late seventies and early eighties. RRs associated with a 10μg/m^3 increase in $PM_{2.5}$ were: 1.06 (95% CI 1.02, 1.11) for all-cause mortality, 1.09 (95% CI (1.03, 1.16)) for cardiopulmonary mortality, 1.14 (95% CI (1.04, 1.23)) for lung cancer mortality and 1.01 (95% CI (0.95, 1.06)) for all other causes mortality after adjusting for age, sex, race, smoking, education, marital status, body mass, alcohol intake, occupational exposures and diet.

These two studies were re-analysed by the Health Effects Institute (HEI) after requests from Industry and Congress (Kreski et al, 2000; Pope et al, 2002). Results of the re-analysis confirmed those of the original studies. After a first follow-up between 1974 and 1989, the Harvard Six Cities cohort was followed from 1990 until 1998 (Laden et al, 2006). For the whole period (1974-1998), relative risks associated with 10μg/m^3 increase in $PM_{2.5}$ were 1.16 (95% CI (1.07, 1.26)) for all-cause mortality; 1.28 (95% CI (1.13, 1.44)) for cardiovascular mortality; 1.08 (95% CI (0.79, 1.49)) for respiratory mortality; 1.27 (95% CI (0.96, 1.69)) for lung cancer mortality and 1.02 (95% CI (0.90, 1.17)) for mortality from all other causes. A 10 μg/m^3 reduction in $PM_{2.5}$ in the late period (1990-1998) compared to the early period (1974-1989) was associated with a reduction in total mortality (RR = 0.73, 95% CI (0.57, 0.95)). This reduction was observed for cardiovascular and respiratory mortality, but not for lung cancer. These results suggest that reduction of $PM_{2.5}$ levels has health benefits.

When the ACS cohort was followed until 1998 (Pope et al, 2004), results were again confirmed and showed that long-term exposure to particulate matter were more strongly associated with ischemic heart disease mortality (RR = 1.18, 95% CI (1.14, 1.23) for 10μg/m^3 increase in $PM_{2.5}$) and dysrhythmias/heart failure/cardiac arrest mortality (RR = 1.13, 95% CI (1.05, 1.21) for 10μg/m^3 increase in $PM_{2.5}$). Associations with respiratory mortality were weaker and non-significant. A longer follow-up until year 2000 did not change results. A possibility for explaining the increased cardiovascular mortality is that $PM_{2.5}$ might lead to accelerated atherosclerosis (Pope et al, 2004).

Besides these two studies, several long-term studies were conducted in the USA and in Europe. These studies were often involved specific populations, for example veterans or subjects diagnosed with a given disease. Although they have a smaller sample size than the Harvard Six Cities or the CPS II cohort, these studies are of interest and are described below.

A sub-cohort of the Cancer Prevention Study I was followed from 1973 to 2002 in California (Enstrom, 2005). Results indicated a statistically significant association between $PM_{2.5}$ and all-cause mortality in the first years of follow-up (1973-1982), but not in the second part of the study (1983-2002), when $PM_{2.5}$ concentration had decreased over the area under study. $PM_{2.5}$ levels used in this study were county averages; this likely leads to exposure misclassification because Californian counties are often very large and topographically variable.

The Adventist Health and Smog (AHSMOG) study followed more than 6,000 non-smoking Californian 7th day Adventists from 1977 to 1987. The follow-up was extended to 1992 (Abbey et al, 1999). The goal was to evaluate relationships between mortality and long-term exposure to PM_{10} and other pollutants. Relative risks were computed for a difference of inter-quartile range of PM_{10} across the population, i.e. an increase of 24.08 μg/m^3. Lung cancer mortality RR was 3.36 (95% CI (1.57, 7.19)) in men and 1.33 (95% CI (0.60, 2.96)) in women. RR for other respiratory mortality were 1.23 (95% CI (0.94, 1. 61)) for men and 1.10 (95% CI (0.86, 1.40)) for women. Follow-up was then extended until year 2000, with $PM_{2.5}$ and PM_{10} data (Chen et al, 2005). RRs for coronary heart disease mortality were significantly associated with PM_{10} and $PM_{2.5}$ levels, but only in women. For example, an increment of 10μg/m^3 for $PM_{2.5}$ was associated with a RR of 1.42 (95% CI (1.06, 1.90)) in women and 0.90 (95% CI (0.76, 1.05)) in men.

The EPRI–Washington University Veterans' Cohort Mortality Study used a prospective cohort of around 50,000 middle-aged men (51 ± 12 years at baseline), who were diagnosed with hypertension in the mid seventies (Lipert et al, 2000, Lipert et al, 2006). The cohort was followed for about 21 years, until the mid nineties. No relationship was found between exposure to particulate matter and mortality. However, only all-cause mortality results were provided, precluding conclusion for cardiovascular and respiratory causes. Another limitation of this study is the statistical modelling: some models included more than 200 terms and the effects of active smoking on mortality were much smaller than in other cohorts.

Goss et al (2004) followed 11,000 patients with cystic fibrosis in the USA in 1999-2000 (Goss et al, 2004). $PM_{2.5}$ levels were 13.7μg/m^3 on average. The main goal of the study was to evaluate relationships between air pollution and pulmonary exacerbations, but data for mortality were available. As only 200 deaths occurred during the follow-up, the statistically power of this study was low; the all-cause mortality RR was 1.32 (95% CI (0.91, 1.93)) for an increment in $PM_{2.5}$ of 10μg/m^3.

Tonne and Wilkinson (2013) studied a cohort of 154,000 patients admitted as hospital for acute coronary heart syndrome in England and Wales between 2004 and 2007 (Tonne and Wilkinson 2013). Patients were followed until 2010, for 3.7 years on average. The outcome of interest was all-cause mortality. For each subject, annual pollution data (NO_2, NO_x, PM_{10} and $PM_{2.5}$) at place of residence were obtained from the Department for Environment Food and Rural Affairs. Several Cox proportional hazards models adjusting for demographic and clinical variables were computed.

Average $PM_{2.5}$ concentrations during follow-up were 11.0 (SD = 1.9) µg/m^3 in England and 9.1 (SD = 1.3) µg/m^3 in Wales. All-cause mortality hazards ratio associated with a 10µg/m^3 increment of $PM_{2.5}$ was 1.44 (95% CI (1.29, 1.60)) after adjustment for age, sex and time. Further adjustments for region, smoking status, drugs taken and other co-morbidities did not alter the result (HR = 1.40; 95% CI (1.22, 1.39)). However, further adjustment for income played a very important role as the hazard ratio was reduced to 1.20 (95% CI (1.04, 1.38)). Association with other pollutants became non-significant when income was included in the models. This study suggests that mortality is more strongly associated with $PM_{2.5}$ and not with other pollutants. Moreover, socio-economic status is a major confounding factor.

Other long-term studies were conducted in Europe, namely in Sweden (Rosenlund et al, 2006) and Germany (Gehring et al, 2006). Results were similar to the US studies and suggest that findings from the USA may be applicable to European populations.

These long-term cohort studies showed consistent associations between mortality and exposure to ambient air pollution. Such studies played a major role for implementing air quality targets in the USA (Greenbaum et al, 2001). In 2002, the World Health Organization (WHO) identified ambient air pollution as high priority for the Global Burden of Disease initiative. Following the ACS/CPS II study results, WHO adopted a relative risk for all-cause mortality of 1.06 (95% CI (1.02, 1.11)) per 10µg/m^3 of $PM_{2.5}$ as international standard. There is evidence that in studies assessing long-term exposure to PM, higher relative risks are found when the study area is smaller than an urban area (Hoek et al, 2002; Jerrett et al, 2003a; Jerrett et al, 2003b). Moreover, relative risks found in the Six cities studies (Dockery et al, 1993) were higher than those of the CPS II (RR = 1.09). Eventually, the standard adopted by World Health Organisation seems a good choice for estimating the mortality hazards associated with long-term exposure to $PM_{2.5}$.

In conclusion, both short-term and long-term studies show that exposure to ambient particulate matter is related to increases in mortality in the general population. Socio-economic status is an important confounding factor that should be taken into account in studies.

2.7.5. Studies in Italy

The role of air pollution on health has received a lot of attention in Italy and several studies have been conducted in particular around the province of Brindisi.

2.7.5.1. Studies in the region of Brindisi

The Province of Brindisi covers a population of 400,000 inhabitants and has one major city (Brindisi) which is characterised by an important economic and industrial activity resulting in considerable industrial emissions. This area has been considered in the 1980s by the Italian government as an area of high environmental risk. The major potential sources of emissions are: maritime ports, an airport, a highway, petrochemical plants, several industries (pharmaceutical, metallurgical, manufacturing, and cement production), a large steel production industry, and a large coal power plant. Since the 1980s, some measures for abatement of air pollution have been taken and a significant decline in SO_2 concentration has been reported between 1992 and 2007 (Mangia et al, 2011).

A case control study (Belli et al, 2004) was conducted on deaths occurring in 1996-1997 around the petrochemical plant of Brindisi which is located in the same area as two important power plants. Deaths from lung cancer, pleural neoplasm, bladder cancer and lymphomas were considered. Controls were deaths from any other causes in individuals living in the same area. Overall, 95 lung cancers, 5 pleural cancers, 13 bladder cancers and 31 lymphomas were included as cases, and 170 deaths from other causes were included as controls. Adjusting for age, sex, smoking, and education reported by next of kin, a non significant increase in risk of lung cancer was reported: OR = 3.1 (95%CI (0.83, 12)) for living less than 2km from the source as compared to those living more than 5km from the source.
[The design of this study suffers from several weaknesses. One of which is the comparison of deaths from different causes. This comparison is likely susceptible to several bias as these deaths might originate from different causes. The authors attempted to adjust for some confounding factors by interviewing next of kin but this approach is also susceptible to declaration bias. The results do not allow firm conclusion as there is no trend in increasing risk. While an increased risk was found for residence less than 2km compared to more than 5km, in between categories also show contradictory patterns (decreasing risk) that are more likely indicating different socio-economic conditions. Overall, the sample size of this study is too small to draw conclusions for a risk in the order of magnitude described earlier on from studies conducted with a more rigorous methodology.]

An ecological study was conducted on deaths occurring in the province of Brindisi between 1981 and 2001 (Gianicolo et al, 2008). This study compared the occurrence of deaths for major causes of deaths for the municipality of Brindisi as compared to the whole Puglia region (the broader region which includes Brindisi). Overall, an increased risk of all cause of death was reported for residents of the province of Brindisi: SMR=105.3 for 1981-1990 and 103.9 for 1991-2001 in men, and

SMR=104.0 for 1981-1990 and 100.6 for 1991-2001 in women. Analysis by commune of the province of Brindisi (table 4 of the article) shows inconsistencies with greatest risk reported for areas not in the area of high emission of pollutants.
[This study has the usual limitation of ecological studies, mainly the impossibility to adjust for the basic confounding factors for each cause of death. While authors could adjust for age, and stratify for gender and study period, there is no possibility to take into account socio-economic characteristics, smoking and drinking habits, access to health care, etc. Furthermore, the results are inconsistent: they vary strongly by gender and period, they are not specific to a cause of death. For example, for men the greatest risk are observed for injury and poisoning which could hardly be associated with air pollution but reflect more the divergence in populations.]

A time-series analysis was performed on deaths and hospital admissions among residents of Brindisi between 2003 and 2006 (Serinelli et al, 2010). The authors conducted a modelling approach taking some meteorological elements (temperature, humidity). They analysed the role of PM_{10}, NO_2 and CO daily concentration and computed the hazard rate for different cumulative lags. An increased risk of death (RR=1.10 95%CI 1.02-1.20 at lag 0-1) and of cardiovascular mortality (RR=1.14 95%CI 1.02-1.28 at lag 1) was observed for PM_{10}. An increased risk of hospital admission was observed with PM_{10} for women at lag 4 and for elderly at lag 4.
[The modelling approach did not include the role of wind in the dispersion of pollution while this factor was considered as major in most other well conducted studies. The analysis is conducted on a relatively small population with 1792 deaths and 6925 hospital admissions. With such a small sample size, the multiplication of analysis: for several lag time, for each gender, and for each pollutants, increase the risk of false positive results (the probability of a statistically significant test occurring by chance alone).]

An ecological study investigated the incidence of congenital anomalies in newborns between 2001 and 2010 in the city of Brindisi (Gianicolo et al, 2012). Overall 194 subjects with congenital anomalies were included in this study. When compared to the average rate of congenital anomalies observed in the European Network for the Surveillance of Congenital Anomalies (EUROCAT), an increased risk was observed for the city of Brindisi: RR=1.17 95%CI (1.02-1.35). In an analysis within the province of Brindisi, adjusting or socio-economic deprivation index (at municipality level) and maternal age, a non significant increased risk of congenital anomalies was observed for women who lived in the city of Brindisi during pregnancy as compared to women who lived in areas outlying Brindisi: OR=1.13 95%CI(0.94-1.35). The risk was significant in a sub-analysis restricting to congenital heart disease: OR=1.85 95%CI (1.36-2.50).
[This study has an ecological approach for the comparison to EUROCAT data. This analysis can only be indicative. The second analysis within the province of Brindisi also has a strong ecological approach. Even though the authors managed to adjust for maternal age at delivery or socio-economic deprivation index, they could not account for other factors such as tobacco smoking, alcohol etc. A higher diagnostic

rate in excellence centres in the city cannot be excluded, being able to diagnose more frequently congenital anomalies than delivery centres in rural area and thus creating artificially an apparent increase of incidence of anomalies.]

A case cross-over study was conducted on the association between daily PM_{10} and NO_2 and hospital admission between 2001 and 2007 in Brindisi (Gianicolo et al, 2013). Over 108,372 hospital admissions recorded, 12,806 were unplanned events and thus kept in the analysis. As for the study of Serinelli et al, 2010, several lag times were considered. Contrary to the study of Serinelli et al, 2010, this study included information on wind direction. The study reported an increased risk of hospital admission associated with PM_{10} at lag 0 for men (IR=7.5% 95%CI 2.5-12.8) but not for women (IR=-0.4% 95%CI -5.7-5.2). When restricted to wind blowing from the port or the industrial site the risk of hospital admission was not significantly increased.
[This study has the same limitations as Serinelli study (Samoli et al, 2008). There is a discrepancy in the number of hospital admissions considered in the present study and in Serinelli study while we could have expected the same order of magnitude as study place and period overlap: study in Brindisi for both studies, 2003-2006 for Serinelli and 2001-2007 for Gianicolo et al. Because of this overlap, these two studies cannot be considered as independent. The association observed in Gianicolo et al, study are not homogeneous and could reflect potential biases: results are clearly different between men and women, table 4 show inconsistent results: significant increased risk for cardiac causes, but also significant decreased risk for respiratory causes.]

Overall, these studies around Brindisi area have a poorer methodology or several limitations as compared to other international studies performed on the air pollution and health. Studies on time-series, properly accounting for the wind direction reported inconsistent results and no association when limiting the analysis to exposure when wind was blowing from the harbour or from industrial plants.

2.7.5.2. EpiAir project

The EpiAir project is a case cross-over study conducted initially on 9 large Italian large cities: Bologna, Florence, Mestre-Venice, Milan, Palermo, Pisa, Rome, Taranto, Turin (Colais et al, 2009). The city of Cagliari was included in some further publications (Faustini et al, 2011). This study investigated hospital admissions and mortality between 2001 to 2005 in these cities in relation to daily air pollution (PM_{10}, NO_2 and ozone) measured by city-specific air monitoring stations. Between one (Bologna) and five (Milan) air monitoring stations were available per city. As for studies reported above, the analysis of case cross-over studies involved the use of several scenarios of lag-time to assess health risks.

The analysis of hospitalisation data was based on 701,902 hospital admissions in the 9 Italian cities (Colais et al, 2009). An increased risk of hospital admission for cardiovascular and respiratory diseases associated with daily pollution from PM_{10} and

NO_2 was detected. Slight heterogeneity was present depending on the analysis (according to the lag-time considered). No association was in evidence for cerebrovascular diseases or death from accidental causes. This latest observation is a good argument in favour of the plausibility of observed association with cardiac and respiratory diseases. A further analysis was conducted to identify susceptible factors associated with these increasing risks (Colais et al, 2012). The risk was different between genders for some specific diseases. The risk was also higher for older populations. No specific chronic condition was found as associated with the relation between air pollution and health risk.

The analysis on mortality data focussed on deaths occurring after the age of 35 years (Chiusolo et al, 2011; Faustini et al, 2011). Overall, 276,205 deaths occurring between 2001 and 2005 in 10 Italian cities were included in the analysis. An increased risk of death from any cause was associated with increasing of NO_2 concentration: for 10 $\mu g/m^3$ increase in NO_2 the mortality increase by RR=1.021 for lag 0-5 days 95%CI (1.009-1.032). The risk was higher for cardiovascular deaths (RR=1.026 95%CI 1.015-1.037) and for respiratory mortality (RR=1.035 95%CI 1.008-1.063). The risk was also described as higher for older population (older than 85), for lower socio-economic status, for association with 3 or more chronic conditions and for months of April to September. This last observation could be either due to more exposure of populations during summer, or through synergic effect with temperature, or more likely with a decrease risk of death in general during summer which could emphasis the observed association between pollution and mortality.

These analyses from EpiAir project are in line with observations performed in other larger international studies (*cf* section 2.7.1.1).

2.7.5.3. Other studies conducted in Italy

Borgia et al (1994) investigated mortality in a cohort of taxi drivers in Rome, Italy. 2,311 taxi drivers were followed between 1965 and 1988. Taxi drivers had to be registered as taxi driver between 1950 and 1975. All-cause and cause-specific mortality rates in the cohort were compared to mortality rates in the Latium province with standardised mortality ratios. No measurement of air pollution was performed.

All-cause mortality among taxi drivers was lower than in the general population: SMR = 0.89 (95%CI (0.82, 0.96)). All-cancer mortality was the same as in the general population (SMR = 0.99, 95% CI (0.86, 1.13)), but mortality for some cancers was lower than expected (digestive organs), and higher than expected for other sites (respiratory system, prostate). The SMR for lung cancer was 1.23 (95% CI (0.97, 1.54)). Sensitivity analyses were conducted and the excess risk was restricted to drivers starting in the most recent period (1965-1975) and for deaths occurring at young age (<65). Taxi drivers were more likely to die from diabetes than the general population (SMR = 1.73, 95% CI (1.25, 2.34)), but less likely to die

from circulatory system diseases (SMR = 0.78, 95% CI = (0.69, 0.88)) and accidents (SMR = 0.61, 94% CI (0.38, 0.93)).
[This study compares taxi drivers to the general population. Healthy worker effect is likely to occur. Moreover, confounding factors such as smoking status are not accounted for. Finally, the cohort is small and therefore the power of this study is low.]

Rossi et al (1999) studied daily mortality in Milano from 1980 to 1989. Mortality data were provided by the vital statistics department of the municipality of Milano. Mortality from natural causes occurring between 1980 and 1989 was considered. The Interdistrict Defense Network for Health and Protection of Milano Province provided air pollution data (SO_2, NO_2 and TSP) for the 1980-1989 period.

During the study period, TSP concentrations ranged between 4 and 529 µg/m^3, with a mean of 142 (SD=81) µg/m^3. Relative risks associated with a 100µg/m^3 increase in TSP were 1.033 (95% CI (1.024, 1.043)) for all-cause mortality, 1.11 (95% CI (1.05, 1.17)) for respiratory infections mortality, 1.07 (5% CI (1.03, 1.11)) for heart failure deaths, 1.10 (95% CI (1.03, 1.18)) for myocardial infarction and 1.12 (95% CI (1.06, 1.17)) for COPD mortality.
[In this study, PM_{10} levels are not available and TSPs are used instead, therefore results are not comparable with other studies.]

Air pollution and daily mortality in Rome were studied between 1992 and 1995 (Michelozzi et al, 1998). Pollution data (SO_2, NO_2, CO, O_3, TSP) were recorded by the Regional Department of Environment in 5 fixed monitoring stations. Daily mortality data was retrieved from the Regional Register of Deaths. Deaths due to accidents and deaths occurring outside Rome were excluded. Poisson models were computed for the whole Rome area and for the central area, based on place of residence of subjects. Models were controlled for temperature, humidity, day of the week and holidays.

Average level of particles was 84.2 (SD = 26.1); IQR = 65.7-100.3 µg/m^3. In single pollutant models, an increment of 10µg/m^3 of TSP was associated with a all-cause mortality RR of 1.0038 (95% CI (1.0009, 1.0068)) in the whole metropolitan area and 1.0066 (95% CI (1.0030, 1.0120)) in the central area. There were marked seasonal differences: in the whole area, RR for all-cause mortality was 1.0002 (95% CI (0.9967, 1.0037)) in the cold season and 1.0096 (95% CI (1.0049, 1.0144)) in the warm season. In the two-pollutant model (TSP and NO_2), RRs for all-cause mortality were 1.0029 (0.9997, 1.0061) in the whole area and 1.0058 (95% CI (1.0018, 1.0098)) in the central area for 10µg/m^3 increase in TSP.
[PM_{10} were not available and TSPs were used. Moreover, only 3 years of data are available, therefore the power of this study is low.]

Forastiere et al (2007) analysed daily mortality and PM_{10} pollution in Rome, taking into account socioeconomic status and income (Forastiere et al, 2007). All natural deaths occurring in Rome among city residents aged over 35 between 1998 and

2001 were identified with the Regional Registry of Causes of Death. Chronic conditions (neoplasms, diabetes, hypertension, cardiac conditions, cerebrovascular diseases and COPD) in the 2 years preceding death were identified via record linkage with the regional hospital discharge registry. Socioeconomic status of subjects was derived from the 1991 census data (census block). Income was estimated at census block level using 1998 Italian Tax register data. Daily PM_{10} concentrations were provided by the regional agency for environmental protection.

From 1998 to 2001, the daily average PM_{10} level in Rome was 51.0 (SE = 21.0; IQR = 36.1-63.0) µg/m^3. There was a clear socioeconomic pattern: residents with higher income and socio-economic status were more likely to live in the city centre than others. Moreover, PM_{10} emissions from traffic in the city centre were more elevated than in the city outskirts. Finally, people with lower socioeconomic condition were more likely to be diagnosed with one or several co-morbidities the 2 years preceding death. The overall effect of a 10µg/m^3 increase in PM_{10} on natural mortality was RR=1.011 (95% CI (1.007, 1.016)). However, the RR was not the same for all socioeconomic statuses. The RRs were significantly elevated for low, mid-low and mid-high incomes and socioeconomic status, whereas the RR for high income and high socio-economic status were perfectly null (RR=1.000).
[RR by income and RR by socioeconomic status are provided in graphs only therefore they are not very precise]

Daily mortality in Rome was studied again in 2001-2004 (Mallone et al, 2011). Study subjects were 80,423 Roman residents aged over 35 years who died within the city from natural causes between 2001 and 2004. Mortality data were obtained from the Regional Register of Causes of Deaths. Pollution data (PM_{10}, $PM_{2.5}$, $PM_{2.5-10}$, NO_2, O_3) were retrieved from Lazio Environmental Protection Agency and Italian National Institute of Heath. Weather data were provided by the Italian Air Force Meteorological service. Saharan dust events were also recorded. Models adjusted for weather, flu epidemics, time trends, seasonality and changes in the size of the population at risk. RRs for mortality are expressed for an increment of an inter-quartile range in each pollutant.

Saharan dust events occurred in 18.6% of days during the study period, with high seasonal variations. $PM_{2.5}$ average concentrations were 23.4 (SD = 12.5; Range 2.4-91.7) µg/m^3 on Saharan-dust-free days and 25.6 (SD = 9.8; range 5.6-86.0) µg/m^3 on Saharan-dust-affected days. PM_{10} average levels were 38.4 (SD = 17.0; range 6.7-115.2) µg/m^3 on Saharan-dust-free days and 47.2 (SD = 18.8; range 19.0-156.9) µg/m^3 on Saharan-dust-affected days. RRs associated with a 12.8µg/m^3 increase in $PM_{2.5}$ were 1.0124 (95% CI (0.9935; 1.0317)) for all-cause mortality, 1.0138 (95% CI (0.9832, 1.0455)) for cardiac mortality, 0.9968 (95% CI (0.9574, 1.0378)) for cerebrovascular mortality, 1.0123 (95% CI (0.9858, 1.0395)) for circulatory system disease mortality and 1.0025 (95% CI (0.9010, 1.1154)) for respiratory system disease mortality. RRs associated with a 19.8µg/m^3 increase in PM_{10} were 1.0304 (95% CI (1.0153, 1.0456)) for all-cause mortality, 1.0404 (95% CI (1.0149, 1.0665)) for cardiac mortality, 1.0264 (95% CI (0.9874, 1.0668)) for

cerebrovascular mortality, 1.0299 (95% CI (1.0082, 1.0519)) for circulatory system disease mortality and 1.0497 (95% CI (0.9782, 1.1263)) for respiratory system disease mortality.
[Relative risks are given for one IQR increase in pollutant concentration, so results are not comparable with other studies. Both PM_{10} and $PM_{2.5}$ data are available.]

Daily mortality was also studied in Emilia-Romagna between 2002 and 2006 (Zauli et al, 2011). Mortality data (all cause, cardiovascular and respiratory causes) were retrieved for the 6 main towns of Emilia-Romagna province for the 2002-2006 period from Regional Register of Causes of Deaths. PM_{10}, NO_2 and O_3 levels were provided by several fixed monitoring stations in each town. Models took into account Saharan dust outbreaks and controlled for temperature, holidays, summer population decrease, flu epidemics and heat waves. Results were expressed for an increase of 10 μg/m^3 of PM_{10} and were stratified by age (less than 75; more than 75 years old). Saharan dust events accounted for 16% of the total study period and were more frequent during the warm season. Average PM_{10} concentration was 41 (SD = 23; range = 7-164) μg/m^3 for the whole year; 28 (SD = 11; range = 7-66) μg/m^3 in the hot season and 73 (SD = 50; range = 9-164) μg/m^3 in the cold season. For people aged over 75, RR for all-cause mortality was 1.008 (95% CI (1.000; 1.016)); for cardiovascular mortality 1.002 (95% CI (0.991; 1.014)) and for respiratory mortality 1.015 (95% CI (0.991; 1.040)). Corresponding RRs for people aged less than 75 were 1.009 (95% CI (0.995; 1.022)); 1.014 (95% CI (0.988; 1.040)) and 0.990 (95% CI (0.926; 1.059)). RR did not differ with season.

2.7.5.4. Sentieri

The "Studio Epidemiologico Nazionale Territori e Insediamenti Esposti a Rischio da Inquinamento" (SENTIERI) project is study conducted in Italy on the evaluation of mortality for residents located in the vicinity of polluted sites. The study was conducted in two steps:

- First a review of literature by an Italian working group which decided which exposure and disease to be considered as relevant for an association. The working group classified evidence from literature as Sufficient, Limited or Inadequate.

- Second, for each site considered as high risk for health, the mortality in residents living close to polluted areas was compared to regional average. An indirect standardisation was used to report increase of decrease in risks for residents of polluted area.

The study covered the region of Brindisi, where an important coal power plant is operating as well as other sources of pollution including traffic, several industries etc. (*cf* section 2.7.3.1) Overall, no increase in mortality was observed for residents of Brindisi. For men, the SMR corrected for deprivation index was SMR = 99 (95%CI

95-102) while for women the SMR was 90 (95%CI 87-93). No other increase was reported for other causes.

[The evaluation for the Italian working group can be questioned especially when more authoritative sources exist to define which association can be considered as causal or not. For example in the field of cancer, the International Agency for Research on Cancer already publishes regularly evaluations from international working groups. The inclusion of these evaluations from large international scientific groups would result in less subjective judgement. This can be especially suspected as several authors of the SENTIERI report were also involved in several studies on environmental exposure and health. An example of the divergence between the SENTIERI working group and international group can be illustrated for association between tobacco smoking and leukaemia and lymphoma which was considered as sufficient by SENTIERI working group while IARC does not consider that sufficient evidence have been accrued yet (IARC 2012c).

Overall, the computational aspect of this project relies on an ecological analysis of mortality data. The analysis enables to account for a deprivation index, but still suffers from the general potential limitation from ecological approach namely the lack of adjustment for confounding factors which could result in the reporting of biased associations.

The results of this study diverge from results from other studies which reported increasing risk associated with global air pollution. This shows the limitation of such approach based on just a global ecological analysis with no data on exposure of populations. Furthermore, some paradoxical results are found, in particular when adjusting for deprivation index, such as an important significant decrease of global mortality (SMR=90) in women associated with residence in Brindisi area.]

2.7.6. PM source apportionment and health effects

Fine particles are a mixture of several chemical compounds. It can be predicted that even if health risks were identified for a global exposure to $PM_{2.5}$, not all mixtures might have the same effect. It is however highly difficult to apportion $PM_{2.5}$ by emission sources at present: this should be a priority area for further research. Some studies have analysed the components of $PM_{2.5}$ and used some trace elements as markers of source to attempt an allocation of source to $PM_{2.5}$. Several receptor models have been developed to identify the contribution of each source from source contribution and source profile matrix (Hopke et al, 2006).

From these apportionments some studies focussed on the health risk from $PM_{2.5}$ according to different sources. A European study, the *Air Pollution and Health: A European Approach 2* (APHEA2) *project* investigated daily mortality for increases of 10µg/m^3 in PM_{10} in 29 European cities over 5 years (Katsouyanni et al, 2001). The study covered a population of more than 43 million inhabitants. Overall, the relative risk for daily mortality associated with PM_{10} for a 10µg/m^3 increment was RR=1.006 (95% CI (1.004, 1.008), this risk being higher for the elderly. An important effect modifier was identified based on NO_2 emissions: in cities with low NO_2 averages the relative risk per 10µg/m^3 increment of PM_{10} was RR=1.0019 (95% CI (1.0000, 1.0041)) but was higher for cities with higher concentration of NO_2 with risk of RR=1.0080 (95% CI (1.0067, 1.0093)) per 10µg/m^3 increment of PM_{10}. The risk was therefore increased in cities with higher pollution of NO_2 which is a clear marker of traffic.

Similarly, in the Harvard Six Cities study investigating impact of $PM_{2.5}$, particles from vehicle exhausts were found as having much stronger association with daily mortality (Laden et al, 2000). The dose-response relation between $PM_{2.5}$ and daily mortality was higher: RR=1.034 (95%CI (1.017, 1.052) per increment of 10µg/m^3 of $PM_{2.5}$ for mobile sources, while the RR was 1.011 (95%CI (1.003, 1.020)) for coal combustion sources. Of note, this study was conducted on samples performed at the end of the 1970s to the 80s in United States where there was a dense concentration of coal combustion sources and when modern methods of reduction of emission from coal power plants were not available.

Studies on animals also suggested this observed increased risk for traffic-derived particles. The risk of cardiovascular response in dogs exposed to air particles was higher for traffic-derived particles than from sulphate/coal particles (Godleski et al, 2000).

2.8. Health risks decline following decreasing levels of air pollution

In both developing and developed countries, overall pollution levels (PM, NO_x, SO_x, O_3....) have been decreasing for the last decades. This led to reduced exposure to ambient air pollution, and this is expected to reduce related health risks. However, it is difficult to prove simultaneous reductions in pollution and mortality in the general population. Indeed, pollution reductions are moderate in developed countries, i.e. where long-term exposure data has been available for a long period of time, and attributable health risks related to air pollution are small.

Short-term studies in developed countries demonstrate significant relationships between air pollution and morbidity/mortality (Katsouyanni et al, 2009, Tonne and Wilkinson 2013) even at low concentrations of PM. These studies on short-term effects indicate that decrease of levels of air pollution ($PM_{2.5}$ and PM_{10}) could have an impact on mortality. Indeed, as it evaluates peak in morbidity and mortality in days of high pollution, it implies that days with lower levels of pollution are associated with decreasing health events.

Data on the effect of declining air pollution levels on long-term health events are scarcer. Only data from the Harvard Six Cities study (Dockery et al, 1993; Villeneuve et al, 2002) and from the ACS cohort study (Pope et al, 2002; Pope et al, 2004; Pope et al, 1995) are available. In the Harvard Six Cities study, general pollution levels declined during the follow-up period in all cities. No relationship was found between RRs for all-cause mortality and period of exposure to $PM_{2.5}$ (Villeneuve et al, 2002). A possible explanation is that the decline in pollutant levels was relatively small. On the other hand, results from the ACS study (Pope et al, 1995) indicate a decrease in the mortality RR related to decreasing levels of $PM_{2.5}$. Nevertheless this decrease may be related to increased exposure misclassification because of increases in population mobility (within and between cities) during the follow-up. If the population at risk remains stable, a real decrease of the mortality RR would lead to a decreased population attributable risk.

Given the economic costs of measures for improving air quality, finding evidence of related public health improvements is an important task. The Health Effects Institute investigated this issue in a monograph: *Assessing Health Impact of Air Quality Regulations Concepts and Methods for Accountability Research* (Health Effects Institute 2003a). The main conclusion was that such evidence is still not present and proposes new methods for achieving this.

Assessing heath impacts of new air quality regulations is difficult. Multiple approaches at multiple levels (local, regional, national) are necessary and several time frames have to be considered. It is very hard to identify a specific health effect related to a specific air quality improvement measure. Country level regulations, for example the *USA Clean Air Act*, may require extended observation periods before reductions in mortality and morbidity are observed. The problem is that when the latency between new regulation implementation and observed health effects is long, it may be biased by changing behaviours in the population, by other changes in environment and by development of secondary and tertiary prevention (screening and new treatments).

Results of epidemiological studies are used by governments to create new air quality guidelines and regulations (U.S. Environmental Protection Agency 1997, .S. Environmental Protection Agency 1999, Canada Wide Standards Development Committee for Particulate Matter (PM) and Ozone 1999). Reduced health impacts are then expressed as reduced costs based on an economic valuation from the available literature. Several air quality scenarios can then be compared to find optimal solution for reducing health impacts from air pollution.

US EPA based its analysis of the USA Clean Air Act on the ACS cohort study (Pope et al, 1995). Results showed that health-related benefits ($22,171 billion) greatly exceeded costs ($523 billion) for the 1970-1990 period. Above 80% of benefits were linked to avoided deaths ($4.6 million per life). These avoided deaths were attributed to reductions in PM pollution and suppression of lead in gasoline. Between 1990 and 2010, health-related benefits also exceeded costs, but to a lesser extent.

Another simulation was carried out in the United Kingdom by the Committee on Medical Effects of Air Pollution (COMEAP). The goal was to estimate the gain in life years in the 2001 general population of England and Wales if the $PM_{2.5}$ levels decreased by 1$\mu g/m^3$ compared to the pollution occurring in 2000. Several scenarios were used and the results range from 0.2 to 4.1 million life-years gained, depending on the reduction in mortality rates used in the models (UK Department of Health 2001). The most conservative scenario was based on the HEI report and 0.2 to 0.5 million life years were expected to be gained. The most optimistic scenario was based on the ACS study and 1.8 to 4.1 million life years were expected to be gained.

Several studies conducted all over the world evaluated health benefits related to radical reductions in pollutant emissions. For example, changes in air quality regulations have led to extreme decreases in air pollution in a very short period of time in Dublin (Clancy et al, 2002) and Hong Kong (Hedley et al, 2002). Other studies investigated decreases in pollution from a single source (Pope, 1989; Pope 1996).

Household use of coal was banned in Dublin from 1st of September 1990 (Clancy et al, 2002). This resulted in a quick and permanent decrease in air pollution. Following the ban, black smoke concentration decreased from 50.2µg/m^3 in 1984-1990 to 14.6µg/m^3 in 1990-1996 (-35.6%). The decrease was more pronounced in winter (85.4 to 21.5µg/m^3, or a decrease of 63.8%). Similarly, SO_2 levels decreased from 33.4 to 22.1µg/m^3 (-11.3%) for the same periods. Additionally, age-standardised all (natural) cause mortality rates significantly decreased from 9.41 deaths per 1,000 person-years (1984-1990) to 8.65 deaths per 1,000 person-years (1990-1996). Mortality decrease was more marked in winter. Moreover, decreases in mortality rates were more pronounced among younger people (less than 60 years), and for respiratory and cardiovascular mortality, after adjustment for weather, flu epidemics and secular changes in whole-country mortality. The observed effect on mortality is somewhat greater than predicted from other studies conducted in Europe (Katsouyanni et al, 1997). Because of the ecological nature of the analysis of health impact in this study, the magnitude of the reduction should be viewed with caution.

In Hong Kong, the sulphur content of oil used for power generation and road transport was reduced to 0.5% in weight in 1990. This change was implemented over a single week-end (Hedley et al, 2002; Peters et al, 1996). Ambient SO_2 and SO_4 levels were recorded before and after the change in two areas, one being highly polluted and the other being less polluted. Respiratory health of children residing in those two areas was also monitored. SO_2 and SO_4 levels decreased by 80% and 38% in the most polluted area. There was a simultaneous decrease in respiratory symptoms among primary school children living in the most polluted area. Such decreases were not observed in the less polluted area (Peters et al, 1996). Sulphur reduction in fuels was also associated in declines of all natural cause, cardiovascular and respiratory diseases mortality; these reductions were bigger in the most polluted areas. Gains in life expectancy per year of exposure after the regulation were estimated to 20 days for women and 41 days for men (Hedley et al, 2002).

The steel mill of Provo, Utah (USA) closed in August 1986 and re-opened in September 1987. Morbidity and mortality was studied before, during and after this period (Pope 1989; Pope 1996). PM_{10} concentrations were also monitored for the same period. The steel mill was producing about half of the respirable PM of the

Utah Valley. For example, PM_{10} levels were on average 120-125µg/m^3 in January 1986 (mill operating) and 60µg/m^3 in January 1987 (mill closed) (Pope, 1989). High PM_{10} levels were associated with increased hospital admissions for respiratory symptoms and diseases. This was particularly marked among children: hospitalizations for respiratory problems were 2 to 3 times lower when the mill was closed. Extracts collected from PM monitoring sites in the Utah Valley were analysed. The toxicologic analyses showed that extracts caused airways inflammation and other symptoms of lung injury. Effects were more pronounced in extracts gathered when the steel mill was operating. Nevertheless, these results should be interpreted with caution because confounding is likely to occur. Moreover, the reductions in PM_{10} pollution were so extreme that results are not easily transferable to other situations which would result from voluntary air quality regulations (Pope 1996).

A recent study in Australia (Johnston et al, 2013), conducted in residents of Launceston, Tasmania, demonstrated the impact of reduction of pollution from residential heating. Following an educational programme, enforcement of environmental regulation and replacement of wood heaters in 2001, daily wintertime concentration of PM_{10} felt from 44µg/m^3 in 1994-2000 to 27µg/m^3 in 2001-2007. This decrease was followed by a reduction of annual mortality in men (-11.4%, $p=0.01$), cardiovascular and respiratory mortality (-17.9% and -22.8% respectively). No decrease was observed for women. However, when combining men and women, a borderline significant decrease was observed for cardiovascular and respiratory mortality ($p=0.06$ and $p=0.07$ respectively).

Although these examples can all be criticised on the ecological approach they all have adopted, it is important to note that measuring the impact on health following a firm action on regulation could also be viewed as quasi-experimental situations. In all these examples reported in this section, actions for air quality control were very strong, followed by decrease in pollution measured by monitoring stations, and rapidly followed by a drop in health events (in particular specific to air pollution, *ie* cardiovascular and respiratory mortality).

2.9. Conclusion

Reducing levels of outdoor pollution is an objective of all developed countries. Most studies are engaged in reducing levels of $PM_{2.5}$ such as Europe which aims to have pollution limits for 2020 of 20µg/m^3 of $PM_{2.5}$. (Directive 2008/50/EC of the European Parliament and of the Council of 21 May 2008 on ambient air quality and cleaner air for Europe).

These declining norms are primarily motivated by the accumulating evidence of detrimental effects of air pollution on health. Studies on outdoor pollution investigated several compounds such as NO_x, SO_2, fine particulates PM_{10}, and $PM_{2.5}$. Even if fine particulates are a mixture of compounds and particulates sizes, $PM_{2.5}$ is now considered as the key marker of outdoor air pollution and for which consistent results have been shown. The small size of such particles is likely to allow their deposit deep into the lung.

Pollution reduction policies may be specifically directed to specific pollution sources having an important impact on population health. But, in order to identify these sources, health effects of total air pollution need to be assessed in a first step, and effects have to be decomposed and attributed to a specific pollutant. This is a highly complex task and requires state-of-the-art methodology and research. For this analysis, $PM_{2.5}$, PM_{10}, NO_X and SO_X were studied because of they are produced by coal plants.

The first step was to the evaluate of contribution of coal power plants to air quality in terms of total emissions of $PM_{2.5}$, PM_{10}, SO_X and NO_X in Europe and in Italy (emission data are available online on the European Environmental Agency website). The *Energy Production and Distribution* sector is a minor source of $PM_{2.5}$ and PM_{10} pollution, and emissions of particulate matter have been decreasing steadily in this sector (for example a 43% reduction in PM_{10} emissions has occurred within this sector group since 2000 in the EU). The main sector emitting $PM_{2.5}$ in the EU is *Residential Stationary Plants* (in particular emissions from household firewood combustion) with 52% of total EU-27 $PM_{2.5}$ emissions in 2010. *Energy Production and Distribution* is the fifth largest contributor to $PM_{2.5}$, with 6% of all $PM_{2.5}$ emissions in 2010.

In Italy, *Energy Production and Distribution* is the eighth largest contributor to $PM_{2.5}$ with 5.1 kt (2,6%). All sources involved in *Public Energy and Heat Production* released 1,6 kt of $PM_{2.5}$ in Italy (0.8% of $PM_{2.5}$ emissions). Among sources of $PM_{2.5}$ we can distinguish those primarily emitting $PM_{2.5}$ and secondary sources emitting precursor gas which will be later transformed in $PM_{2.5}$ in the atmosphere (in particular SO_X and NO_X).

In Europe, the most important key category for SO_X emissions is *Public Energy and Heat Production* (47% of total emissions in 2010) but more less in Italy (only 9% of total emissions of SO_X). When reported per million inhabitants, $PM_{2.5}$ emissions are higher in Nordic and Central European countries; this likely highlights the role of household heating with coal, biomass or other fuel.

A second aim was to investigate the health effects of air pollution exposure. Short-term effects of exposure to fine particulates have been consistently reported in adults with increasing risk of all-cause mortality, more specifically cardiovascular and respiratory mortality. Mortality increases by an estimated 1.006 per $10\mu g/m^3$ increment in PM_{10}. Studies in children also showed a small increase in daily mortality when the level of fine particulates increase, but these studies were less consistent. Long-term effects of exposure to fine particulates have been investigated in several studies. These studies have a less robust design than those on short-term effects as an ecological component is always present with place of residence being units of comparison. However, in several studies, with adequate accounting of several confounding factors, consistently increased mortality rates have been associated with increasing levels of $PM_{2.5}$. The ACS/CPS II study has been the largest study and adopted by WHO for their modelling of burden of disease. The all-cause mortality is increase by 1.06 per 10µg/m^3 increment of $PM_{2.5}$.

The role of air pollution on health has received a lot of attention in Italy and several studies have been conducted in particular around large combustion plants. The studies around the coal power plant of Brindisi have a poorer methodology or several limitations as compared to other international studies performed on air pollution and health.

Air pollution with $PM_{2.5}$ is influenced strongly by meteorological features. With a very small weight, wind conditions can transport fine particulates over hundreds of kilometres. Combustion of solid fuels for residential heating (mainly coal and biomass) are the main emitters of fine particulates. Other sources are contributing to air pollution by releasing fine particulates, such as motorized vehicles, not only from exhaust pipes, but also from friction wear of breaks and tyres, coal-fired electric power plants, and also some natural emissions such as volcanoes, forest fires, or sea spray.

Effective methods to reduce emissions of SO_2, NO_X and fine particles from coal burning are already available. For example, electrostatic precipitators enable a removal of 99% to 99.99% of fine particles. Fabric filters also enable reducing particles of size between 0.01 to 100 um ($PM_{2.5}$ including ultrafine particles) with a decrease of 99% to 99.9999%. Wet scrubbers for particulate control also allow a removal of 90% to 99.9% of fly ash in addition to sulphur dioxide. With not negligible investments, these modern technologies for reducing emissions have been adopted in Europe, in particular in Italy, for complying with Best Available Techniques (BAT) in energy production from coal burning.

To reach the European goals of air pollution limits for 2020, all sectors are required to make efforts to reduce their emissions of atmospheric pollutants. However, significant actions should target the major sources of Particulate Matter emissions, namely Household Heating Sources and Traffic pollution. Household use of coal, wood and biomass burning should be discouraged and solutions with low emission of $PM_{2.5}$ should be preferred. As traffic remains an important factor in all European countries, it is important to take action. Some methods focussing on avoiding congestion have shown promising results such as speed limitations. Further technological changes in the sector of motorized vehicles are still required to reduce significantly the emissions of $PM_{2.5}$.

International Prevention Research Institute

The Environmental and Health Impacts of Coal Thermoelectric Plants

Chapter 3

Evaluation of Recent Estimations of Impact of Air Pollution on Health in Europe.

List of abbreviations

95% CI:	95% confidence interval
CAFE:	Clean Air For Europe
CBA:	Cost-Benefit Analysis
EEA:	European Environmental Agency
EMEP:	European Monitoring and Evaluation Programme
E-PRTR:	European Pollutant Release and Transfer Register
EU/EU-27:	European Union/European Union (27 members)
ExternE:	External Costs of Energy
GW:	Gigawatt
OR:	Odds Ratio
NO_x:	Nitrogen oxides
PM_x:	Particulate Matter (diameter $\leq$ x μm)
RR:	Relative Risk
SO_2:	Sulphur dioxide
SOMO:	Stichting Onderzoek Multinationale Ondernemingen (Centre for Research on Multinational Corporations)
SOMO35:	Sum of means over 35 parts per billion (for ozone)
TSP:	Total Suspended Particles
UN:	United Nations
VOLY:	Value of a life year
VSL:	Value of a statistical life
WHO:	World Health Organization

Chapter 3: Summary

There are clear needs for finding solutions to air pollution due to $PM_{2.5}$, assessing adverse health impacts associated with such pollution and for taking steps to minimise any potential health impact. Coal-fired power stations are emitting a certain quantity of $PM_{2.5}$ and these emissions contribute to global air pollution but are only one of the sources of this pollution in Europe. These emissions are far lower than those resulting from other human activities such as household heating and road traffic and have been falling consistently and significantly in recent time. If legitimate, the estimation of the role of these emissions should be based on adequate modelling.

The *Stichting Onderzoek Multinationale Ondernemingen* (Centre for Research on Multinational Corporations; SOMO) has produced a Report which attempted to compute the contribution of ENEL's coal power plants to the global burden associated with $PM_{2.5}$ pollution in Italy. It is not clear why a Report on Public Health ignored the impact of pollution from other Italian coal plants[13] and other sources of $PM_{2.5}$ pollution. Although a first attempt which raised further the levels of public awareness, this report failed to a large extent to provide a robust scientifically sound estimation.

Major approximations were necessary in the evaluation carried out by SOMO. The computation required several steps, each of which could result in severe uncertainties in the estimation of the impact of air pollution. The most problematic of all is the transfer of emission from power plants in Italy without apparently accounting for the geographical location of the power plants. This transfer of emission values considered, for example, that pollution emitted in Brindisi (south east) could be applied to the whole of Italy. The assumption in the calculation is even more inappropriate as the scaling factor used by SOMO was derived from a European Environmental Agency (EEA) report which was based on all industries in Italy (mainly located in the North West of the country) and pollution from other European countries (north).

The estimation of adverse health effects from air pollution resulting from coal-fired power plants performed by SOMO, although a first initiative, cannot be considered as valid or useful. The present report outlines why this is the case and provides elements for conducting a better estimation based on the most valid methods currently available. The SOMO Report fails to contribute to Public Health by concentrating on a single source of air pollution.

Much work remains to be done with the needs for better monitoring of pollutants, measuring individual exposures and collecting detailed health outcomes essential. Research is urgently needed to discover the sources of $PM_{2.5}$ pollution in each area. As regards methodology, in general terms the major need is to present findings and calculations in terms of absolute risk (and attributable fraction) rather than as a relative risk (or odds or hazard ratio). One particular source of $PM_{2.5}$ pollution may be associated with a higher relative risk but a more common source may be associated with a lower relative risk and yet have a higher absolute risk (and a higher attributable fraction).

It is absolutely essential to prepare viable models that look at all sources of pollution and not simply refer to a single, potential source. This is an absolute principle if the focus is, as it should be, on improving Public Health.

[13] ENEL owns 8 of the 13 coal plants located in Italy. http://www.assocarboni.it/index.php/en/the-coal/coal-plants-in-italy (accessed on 25/03/2013)

3. Evaluation of Recent Estimations of Impact of Air Pollution on Health in Europe.

3.1. Introduction

Particulate matter pollution is a public health issue. Short-term exposure to particulate matter (PM_{10}, $PM_{2.5}$) is associated with a slight but significant increased risk of non-accidental mortality. Several cohort studies demonstrated a statistically significant relationship between long-term exposure to $PM_{2.5}$ and mortality, in particular cardio-pulmonary mortality and lung cancer mortality (Chapter 2).

In Europe, PM_{10} pollution is very heterogeneous: the most polluted areas are mainly located in Central Europe, but Northern Italy is also amongst the most PM_{10}-polluted areas in Europe (Figure 3.1). Sources of PM_{10} and $PM_{2.5}$ include household combustion of solid fuels (in Europe, mainly biomass), road transport, industrial processes, agriculture and energy production (Chapter 2).

Assessing the pollution-related health impact of economic activities has been the subject of a great deal of recent research. Recently, the *Stichting Onderzoek Multinationale Ondernemingen* (SOMO/Centre for Research on Multinational Corporations) attempted to evaluate health impacts from some coal-fired power plants in Italy (SOMO, 2012). This (SOMO) report does not take into account all coal plants in Italy, but only those owned by ENEL[14]. ENEL is a multinational energy production and distribution company based in Italy. It currently produces the majority of energy in Italy. More than 37,000 people in Italy and around 41,000 people outside Italy (mainly Europe, South America and Russia) are employed by ENEL. As of 2011, energy generated by ENEL in Italy was 40% derived from coal-fired power plants, 26% from hydroelectric power stations, 22% from natural gas-fired power plants, 12% from other sources (ENEL 2011). An important proportion of electricity produced by ENEL is produced in 8 coal-fired power plants located all over Italy (Figure 3.2). As these coal-fired power plants emit $PM_{2.5}$, and because both short-term and long-term exposure to $PM_{2.5}$ are associated with increased mortality, it is relevant to try and assess the health effects associated with $PM_{2.5}$ pollution from such power plants although, from the public health perspective, the health impact of all emissions of $PM_{2.5}$ or those from all coal power plants is the key issue.

The SOMO Report serves to focus attention once again on the important issue of pollution and mortality in Europe. However, it also serves as a first attempt at quantification of the potential impact of pollution on mortality. The main focus of this chapter is to evaluate the methodology employed in the SOMO Report and its impact on the conclusions of the report. In a second part, alternative methodologies will be

[14] There are in total 13 coal-fired power plants in Italy, of which 8 are owned by ENEL and 5 are owned by other companies (A2A: 2 plants; E.ON, Tirreno Power, Edipower: one plant each). Source: http://www.assocarboni.it/index.php/en/the-coal/coal-plants-in-italy (accessed on 25/03/2013)

proposed which would improve the accuracy of the estimates and, hopefully, highlight priority areas where improvements in air pollution could be made optimally for the impact on reducing the impact on human health.

3.2. Presentation of SOMO report

3.2.1. Origin of the SOMO report

In May 2012, SOMO published a report entitled "*ENEL Today and Tomorrow: Hidden costs of the path of coal and carbon versus possibilities for a cleaner and brighter future*" (SOMO, 2012). This report, commissioned by Greenpeace Italy, aimed "*to raise public awareness about some of the hidden costs and benefits of ENEL's electricity generation activities in order to contribute to an informed and open public debate about national and international energy strategies*". The objective of this report was to support a communication campaign from Greenpeace promoting sustainable energy systems in Italy and other parts of Europe.

The Report is partially based on an unpublished Bachelor's Thesis of an Open University (Heerlen, the Netherlands) from Peeter Saaman (one of the authors of the report) although it is authored by employees of SOMO. The authors acknowledge the contribution of Lauri Myllyvirta (Greenpeace International), who "*contributed significantly to the description of the methodology in section 2.2. Heartfelt thanks also goes to Andrea Boraschi and Giussepe Onufrio (Greenpeace Italy) for their constructive comments and suggestions on various drafts of the report*".

As mentioned in the acknowledgements, the role of the funder was crucial for the choice of the technical methodology, in particular in developing alternative scenarios with renewable energies. Greenpeace was also implicated in reviewing the document. The influence of the funder is such that the report should hardly be considered as an independent assessment. Greenpeace Italy commissioned the SOMO report, contributed significantly to its development then employed this "independent" report to make statements about ENEL and its Coal Thermoelectric Plants.

3.2.2. Rationale of the SOMO report

The objective of the SOMO report was to compute the "*hidden costs*" from energy production with coal power plants. The main hidden costs were considered to be those from adverse health outcomes. Such outcomes are of potential importance in any circumstance. It has been demonstrated earlier in this Report (Chapter 1) that there is no specific health hazard associated with working in Coal Thermoelectric plants or living in the proximity to one.

There is evidence from other studies that exposure to fine particles ($PM_{2.5}$) poses a health hazard: these effects are summarised and discussed in Chapter 2. Coal-powered electricity generating plants are susceptible to emitting pollutants in the air in particular $PM_{2.5}$ which seems clearly related to a risk of increased mortality (see Chapter 2). The contribution of the energy production sector is minor as an emitter of $PM_{2.5}$ as compared to residential heating and road traffic sources (Chapter 2). Consequently, coal-powered plants are likely to be a minor contributor to adverse health effects from air pollution. The evaluation of the impact of this energy production is then important for evaluating different strategies of energy production.

However, such a rationale would be defensible if a similar assessment was conducted for each energy production method. As such, the use of scenarios of costs from Greenpeace for the costs of alternative, mainly green, energy sources cannot be compared to the evaluation performed on Coal Thermoelectric plants.

3.3. Analysis of the methodology of SOMO report

3.3.1. Calculation

Conclusions of the SOMO report are based on computations from Table 3.1 and results are presented in Tables 3.2 and 3.3. These tables use information from Tables 3.4, 3.5 and 3.6. Derivation of these figures is presented in section 2.1 of the SOMO report. This is a very helpful section describing calculations made for obtaining estimates laid out in the report.

For a given health outcome, the basic calculation requires an estimate of the risk associated with a unit change in $PM_{2.5}$ exposure. The risk is then multiplied by the excess exposure associated with ENEL's emissions. Adverse health effects are then expressed as costs using values using values estimated in another report (Holland et al, 2005). These values are displayed in Table 3.4.

3.3.2. Risk

The entries in Table 3.4 represent the estimated health effects of $PM_{2.5}$ for various outcomes. Going back to the original tables in Holland et al, (Holland et al, 2005), it can be seen that they are the product of the 5 following factors:

- **Pollutant factor 1 for $PM_{2.5}$:**

This factor is equal to 1 when the original function is expressed in terms of $PM_{2.5}$ and to 1.54 when it is expressed in terms of PM_{10}.

- **Pollutant factor 2:**

The factor is equal to 1 when the original function is expressed per 1µg/m^3 and to 0.1 when it is expressed per 10µg/m^3.

- **Population factor 1:**

This factor accounts for most functions applicable to a given part of the population. For example, the chronic mortality function (deaths) is applicable only to those aged over 30, which accounts for 62.8% of the population in the modelled domain.

- **Population factor 2:**

This factor accounts for some functions being expressed per thousand or per hundred thousand of population.

- **Incidence rate, response functions, and valuation data:**

These are all given in Volume 2 of the CAFE CBA methodology report (Hurley et al, 2005).

The figure of 6.07×10^{-5} in table 3.4 is calculated as the product of:

- Pollutant factor 1 = 1
  as the original response function is expressed in terms of $PM_{2.5}$
- Pollutant factor 2 = 0.1
  which scales the response function which was originally for 10µg/m^3 to µg/m^3
- Population factor 1 = 0.628
  which corresponds to the proportion of population older than 30 in Europe*
- Population factor 2 = 1
  as the incidence rate is expressed as deaths per 100
- Incidence rate = 0.0161
  which is in fact the mortality rate for all Europe: 1.61%**
- Response function = 0.06
  which is the relative risk (1.06) of long-term mortality associated with an increase of 10µg/m^3 of average daily exposure to PM_{10} from Pope et al, (Pope et al, 2002). The RR is expressed as percentage change, that is 0.06.

**** Which is younger than for the population of Italy where 70.5% of the population is 30 or older***
***** The actual death rate for Italy is much lower: 0.993%***
Source for both is www.istat.it

There are two major issues with this calculation which can be identified which could lead to considerable imprecision in the results.

First of all, the scale of this figure (6.07×10^{-5}) in the SOMO report is cases per µg/m^3 per person per year exposure. Working through the units of all the measurements in Table 3.4 it would appear that 6.07×10^{-5} is the *per capita* risk of dying associated with an increase in **average daily** $PM_{2.5}$ of 1 µg/m^3. The relative risk derived from Pope et al, is clearly associated with average daily exposure to $PM_{2.5}$ over a period of a number of years (Pope et al, 2002). It is not clear whether the figure in the SOMO report makes any adjustment for total exposure over a year or average daily exposure over the year. This is important when this figure is combined in Table 3.1.

Secondly, the apparent use of European mortality and population data rather than the (easily available) Italy-specific data contributes considerable imprecision to a calculation specific to Italy. Simply using the Italian data rather than the European data reduces the risk calculation to 3.93×10^{-5}, a reduction of 35%.

A minor issue is the correction for the risk associated with 10 µg/m^3 to 1 µg/m^3 by dividing by 10. The percentage change (relative risk) is measured in a statistical model with a log link and the appropriate correction is to divide by 10 on the logarithmic scale and then to exponentiate. The differences are not great but demonstrate some lack of insight into the methodological issues being employed.

The main focus of the SOMO report is the number of premature deaths among people aged over 30 years so little attention has been paid to the other health endpoints. It should be noted that mortality is also measured as years-of-life-lost and so cannot be interpreted as cases per µg/m^3 per person per year exposure. Furthermore for Infant Mortality, the mortality rate appears to be per 1,000 live births as a mortality rate of 0.19% (Table 3.4). This is not consistent with a birth rate in Italy of 0.9% population. According to Eurostat[15], correct figures for Italy are 0.90% for the birth rate and 0.32% for the infant mortality rate.

3.3.3. Exposure

In this subsection, the computations described in section 2.1 of the SOMO report will be carefully reviewed and critiqued. The same calculations were carried out for each coal plant. Table 3.1 presents details of computations relative to the Federico II coal-fired power plant. It is directly reproduced from the SOMO report.

[15] http://epp.eurostat.ec.europa.eu/portal/page/portal/statistics/search_database (accessed on 26/03/2013)

Table 3.1: Exposure Calculation from SOMO Report (from SOMO 2012)

	PM_{10}	NO_x	SO_2
Emissions, tonnes per year	473	7,300	6,540
		TIMES	
PM_{10} to $PM_{2.5}$ conversion factor	0.649	1	1
		TIMES	
Emissions-to-concentration factors for Italy	703.69	156.66	153.84
		TIMES	
Power sector adjustment factors	0.5	0.78	0.87
		EQUALS	
Increase in population-weighted concentrations, $\mu g/m^3$/person		1,875,407	
		TIMES	
Risk factor for chronic premature deaths		6.0665×10^{-5}	
		EQUALS	
Amount of premature deaths caused per year		113.77	
		TIMES	
Value of statistical life, M€		2.00	
		EQUALS	
Economic losses due to premature deaths, M€		227.54	

The tonnes of emission for PM_{10}, NO_x and SO_2 in row 1 of Table 3.1 were extracted from the E-PRTR database, which is mandatory to be completed by industries whose emissions contribute potentially to air pollution. A problem is that only $PM_{2.5}$ data (or PM_{10}) would need to be included. These values are total yearly emissions whereas the relative risks are based on daily average exposure to $PM_{2.5}$.

The conversion factors from PM_{10} to $PM_{2.5}$ in row 2 of Table 3.1 appear to be correct.

A major issue occurs in the third row of Table 3.1 which displays the *Emissions to Concentration Factors* for all of Italy. This is the crucial conversion factor in the report as it seems to transform tonnes per year to $\mu g/m^3$. This conversion factor is not clearly described in the SOMO report and its derivation is obscure. It seems to be an attempt to transform yearly emissions of pollutants to a daily average for the population, though this is not clear. The conversion factors come from unpublished data obtained from authors of EEA (2011) report.

The major problem is that no modelling appears to be applied for each power plant: a global dispersion factor is applied for each plant while this dispersion factor seemed to be initially computed for all industrial facilities in Europe. Details in the EEA (2011) report suggest that the conversion factors are derived from fitting a dispersion-type model to recorded $PM_{2.5}$ based upon emissions from industrial plants in Europe. The effect of one tonne of emissions of each of the pollutants is obtained by decreasing the emissions in each plant by 15% and comparing the predictions of the model with those predictions obtained from the model fitted to the data.

Consequently they appear to be the factors for 1 tonne of pollutant from all sources over all of Italy rather than from specific industrial plants.

The power sector adjustment factors are presented in the fourth row of Table 3.1. References regarding these adjustment factors are hard to find. The SOMO report refers to an annex of an EEA technical report (EEA, 2011), which itself refers to the Eurodelta II study (Thunis et al, 2008). These factors are an attempt to down weight the emissions from power stations relative to other types of industries. They take into account the role of specific characteristics of emission sectors (eg stack height) on dispersion of emissions from a point source. These factors are specific to pollutants ($PM_{2.5}$, SO_2 and NO_x) were estimated from data collected in France, Germany, Spain and the United Kingdom (Thunis et al, 2008). The average value was selected and used in the SOMO report, and no sensitivity analysis was carried out.

The values which appear in the first four rows of Table 3.1 are multiplied together and then summed to give 1,875,407 in row 5. This is labelled the increase in population-weighted concentrations in units of $\mu g/m^3$/person. Again this metric is not detailed in the SOMO report. It is the sum of PM_{10}, NO_x, SO_2 converted to $PM_{2.5}$ converted to concentration expressed as $\mu g/m^3$ and multiplied over the whole population of Italy. The unit of measurement is not clear; it cannot be daily average of $\mu g/m^3$/person, unless the conversion factors in row 3 make this adjustment. It might be the total exposure to $PM_{2.5}$ by the whole population of Italy over a year. This requires clarification.

The product of the increase in population-weighted concentrations by the risk factor for chronic premature deaths gives the estimate of the deaths associated with the emissions i.e. 114 deaths.

Finally, chronic premature deaths are expressed as costs using the value of a statistical life (VSL). Multiplying by the value of a statistical life is an error. Here, the report uses the "price" of a full statistical life, whereas the risks from epidemiological studies correspond to years of life lost. These are unknown but are less than a full life. Furthermore the strongest associations are from causes of death like respiratory which occur mainly in the elderly. The economic loss is here likely largely overestimated. It would have been more adequate to express health damages as life year lost, and costs in terms of value of a life year (VOLY).

3.3.4. Mapping of power plant location and Italian population covered

The computation methodology adopted was based on an application of pollution from each power plant to the whole population of Italy with an emission-to-concentration factor extracted from EEA 2011 report (EEA, 2011). This emission-to-concentration factor was initially computed for all facilities in Europe with coverage far different from the location of coal power plants in Italy. The location of ENEL's power plants investigated in by SOMO is plotted in Figure 3.2.

The EMEP50 grid[16], which was used initially in the EEA 2011 report (EEA, 2011), was employed. Conversion factors were issued from this report, based on EMEP50 grid. This grid could be associated with population data; 2000 United Nations (UN) estimates were used. From the geographical location of power plants and population density, we computed the population at risk in the vicinity of coal-power plants. Individuals living within 50km of a coal power plant were considered at risk. Alternative scenarios were also computed with distance to power plants up to 75km, 100km, 150km and 200km.

Figure 3.3 presents the density of population in Italy based on EMEP grid (resolution 50kmx50km).

The map of population living within 50km of coal plants are displayed in Figure 3.4. 8.1 million inhabitants live within 50km of ENEL's coal plants, which represents 14.3% of the whole Italian population. This percentage gives a broad idea of the factor by which could be multiplied SOMO's estimate to avoid the bias of using a global conversion factor from EEA for the entire Italian population.

As sensitivity analysis, more extreme scenarios with population at risk up to 100km, 150km and 200km of coal power plants were also computed (Figure 3.5).

Even with the worst-case hypothesis of diffusion of pollution with population at risk when living up to 200 km, this would cover 76.6% of the Italian population (Table 3.7). This shows that even with unrealistic assumptions, the SOMO's estimates would be reduced by 25% by better accounting for the places of emission from Coal power plants.

3.4. Discussion

Overall, even if the rationale of SOMO report is sound, this report can hardly be considered as a regular scientific evaluation. The style of writing is not scientific but rather in a style usually associated by activists and this is an indication of the subjectivity present in the report.

The references used in the report were identified from searches in *Bloomberg* and *LexisNexis* databases. These databases are designed for mass media, contain legal documents and are mainly used by journalists. The authors did not mention the use of biomedical databases such as *Pubmed*. Qualitative or quantitative assessments of risks for health require a literature search strategy using several biomedical

[16] www.emep.int (accessed on 27/03/2013)

resources[17]. As a contribution to public health, it is lacking. Concentration on all sources of $PM_{2.5}$ or all coal power stations in Italy would have been of some significance.

3.4.1. Need for computation accounting for place of pollution emission

The major limitation of the computation used in SOMO report is the use of a common factor for Italy applied for each power plant, whatever the geographical localisation of the plant. As an example, for the power-plant of Brindisi, it corresponds to taking emission data from this south east location, and distributing it from an average central point in Italy based on external emissions and industries in Italy likely being in the North.

In section 3, it was demonstrated that even when accounting for an important coverage of pollution from coal power plants, only a small proportion of the Italian population is at-risk.

The factors used in the SOMO report came initially from the outcome of an analysis performed in all Europe modelling the impact of changing global emissions from all major industrial sites by 15%. This is then neither representative of the location of coal plants, nor of the Italian situation as it reflects the impact of change for all Europe at once. Hence, the factor for Italy is made of potential changes from both pollution emitters abroad impacting Italy and the global Italian industries. In section 3, we described the geographical location of power plants: they are mainly close to the coast, in lightly populated areas. From the introduction, it was also evident that the most polluted areas in Italy are the Lombardy area (North) which is also the most populated.

3.4.2. Pollutant assessment and mixing pollutants for health effects

In the SOMO report, the authors included PM_{10}, NO_x and SO_2 emissions and later converted these particles to $PM_{2.5}$. The estimation of $PM_{2.5}$ was later based on a modelling from EMEP modelling of particulates diffusion in Europe. However, transferring a risk from an epidemiological study to an exposure performed in population for computation of health burden assumes that the method used for exposure ascertainment is the same for risk assessment and population exposure. Here, the epidemiological studies were initially conducted with a direct assessment of $PM_{2.5}$ levels from monitoring stations providing ground measurements.

Furthermore, it is unlikely that all the pollution effect of NO_x and SO_2 could be converted to $PM_{2.5}$. If such a transfer was correct, then epidemiological studies on NO_x or on SO_2 would have retrieved similar relative risks for NO_x and SO_2 per $10µg/m^3$ as for $PM_{2.5}$. But this is not the case in the scientific literature we evaluated

[17] http://www.nlm.nih.gov/services/research_report_guide.html (accessed on 12/03/2013)

(Chapter 2) and this is the reason why NO_x and SO_2 were not considered as primary risk indicators.

3.4.3. Inclusion of CO_2 in health-related costs

The authors of the SOMO report included in the computation of the role of CO_2 in the financial burden associated with coal power plants emissions. This emission factor has been included in total contradiction with the initial stated objective of the SOMO evaluation which was on "hidden" costs, i.e. indirect costs not reflected on the economy of coal plant activities. Indeed, the market of CO_2 is well established, as mentioned by the SOMO authors emitters of are subject to buy shares on a specific CO_2 market in proportion of their emissions. This system was set up following the Kyoto Protocol to the United Nations Framework Convention on Climate Change signed in 1997 and effective since 2005. In other words, ENEL has to trade its emissions to the benefit of low emitters. This requirement will be reflected in the price of energy from exploitation of coal power-plant and cannot be considered as a "hidden" cost.

3.4.4. Misuse of health risk

Computational issue

Epidemiological studies provided a relative risk per increment of 10µg/m^3 of $PM_{2.5}$, extracted from the study of Pope et al (2002). These risks were computed in the initial study within a statistical model which implies a log link between exposure values and the risk. This relative risk cannot be directly applied to exposure values by a product but must account for the log-link relationship in the initial model. Because the values of risk are low, the deviation due to this error in computation is marginal.

No threshold assumption issue

The studies on all-cause mortality, including Pope et al (2002), did not show a threshold under which no risk could be identified. The relative risk from epidemiological studies could then theoretically be used, as in the computation from SOMO, under the assumption of a reference pollution of 0 µg/m^3 of $PM_{2.5}$.

However, several limitations exist for such an assumption. First, such a value of 0 µg/m^3, which is used as counterfactual scenario, corresponds to a situation with not a single fine particle in the air. Such a situation does not, and can not, exist on earth, not only because of human activity but also because of natural $PM_{2.5}$ emissions. Secondly, most studies on the health effects of $PM_{2.5}$ compared pollution levels between cities; cities with the lowest pollution provided a background level of risk associated with low air pollution. No studies could ever investigate risks at lower levels than in cities with the lowest pollution levels. This has led several institutions, such as the World Health Organisation, to consider a baseline concentration of

7µg/m^3 when computing the burden of disease associated with air pollution. This low value of $PM_{2.5}$ corresponds then to the counterfactual scenario which should be used in computations.

Transfer of risk from United States to European situation

The risk derived from the study of Pope et al, (Pope et al, 2002) was computed from values obtained from United States cities. Transferring this risk to Italy could pose a number of difficulties and is associated with the assumption that the situation of risk and the exposure pattern are similar between the United States and Italy.

For the background risk, these two populations are clearly different because of different histories of exposure to other major risk factors (tobacco, alcohol,…), different socio-economic status, different health care systems, different life-expectancy, not to mention different genetic background. All these could have impacted the estimation of risk if conducted on the Italian population. Due to the complexity of factors influencing global mortality, the direction of the change in the risk estimate is not predictable.

Regarding exposure pattern, the concentration of fine particles ($PM_{2.5}$) in the United States originating from Coal plants is likely to be much higher than Italy. When weighting by population, there is 8.9 times more energy (GW) produced from Coal plants in the United States as compared to Italy (318 GW[18] in United States for 315 Million inhabitants vs. 6.8 GW[19] in Italy for 60 Million inhabitants). The analysis of the literature does not seem to indicate that pollution from coal plants produces a higher risk than other emitters (see Chapter 2). If $PM_{2.5}$ emitted from coal power plants produced a higher risk than produced by other emitters, the risks estimated in the United States from Pope et al (2002) of 1.06 per 10µg/m^3 would reflect this higher proportion of particulates from coal plants. And for 10µg/m^3 in countries with less coal emissions such as in Europe, the risk would be less than 1.06. Hence, applying the US risk of 1.06 to the European situation would considerably overestimate the attributable deaths from this pollution.

3.4.5. A need for sensitivity analysis

Deriving such an estimation of the health burden associated with exposure to a risk requires the definition of several assumptions. The methodology from SOMO was based on several calculation steps (see section 3.3). Each step was performed under a series of assumptions which are inherent to the methodological limitations of such an exercise. Each calculation could potentially introduce biases and thus would require a minimum discussion and several sensitivity analyses. The SOMO report

[18] www.eia.gov/electricity/capacity (accessed on 02/04/2013)
[19] Table 11 (p. 27) of SOMO report (SOMO 2012)

presents only one estimation of burden without any single sensitivity analysis or discussion on validity of results.

Several sensitivity analyses with different scenarios would have improved significantly the quality of the SOMO report and could have revealed potential erroneous conclusions based on a single computation.

3.4.6. Need for a global assessment of pollutant emitters

Finally, the SOMO report initially stated that the computation of the costs associated with exploitation of coal power plants was made in order to allow further comparison with other energy production methods. But the SOMO report does not produce any similar analysis of costs associated with energy production based on similar methodology. It would be important to have these data available.

More importantly is the estimation of the role of the major emitters of pollution. As reported in Chapter 2, energy production has only a marginal effect on the global $PM_{2.5}/PM_{10}$ emissions as compared to household heating and road traffic. A proper evaluation of the assessment of pollution should preferably integrate all aspects of the problem of pollution, and include at least the most important sectors that contribute to air pollution. For example, to decrease the pollution from household heating and traffic, some solutions rely on use of electricity-powered heating systems and electricity-powered cars. Broadly used, these solutions could potentially decrease the overall air pollution but would require further energy production. The balance of gain and losses from changing the current system should be weighted to find the most appropriate solution to decreasing air pollution and cannot be based on a single evaluation of a source of pollution.

3.5. Picture of an alternative (more valid) method for computation of health impact of pollution in particular from Coal power plants

Methodological limitations of the SOMO report are outlined in section 3.4. In this section, we describe an alternative method which could be used for computation of health impacts from natural and anthropogenic sources of pollution.

3.5.1. Need for computation accounting for place of pollution emission

The estimation of health impact of pollution, in particular from coal plants in Italy should be based upon the most appropriate models for the dispersion of the pollutants from a point source. This would take into account information about the power plant, such as the stack height and diameter, the location of the plant and the amount of pollutants emitted, as well as dispersion modelling which will also take into account the prevailing climate (wind, temperature....), topography, natural sources and background pollution. Such models exist for Europe.

For instance, EcoSenseWeb is an atmospheric dispersion and exposure assessment model which was used with thin the ExternE project. It was designed specifically for the analysis of a single point source but can be used for multiple emission sources in a single region. The calculations in this model are based upon the Impact Pathway Approach which begins with emissions from a single point source and then models the dispersion of this pollutant into the environment and its chemical transformation. There are also defined conversion factors which relate the expose to its impact on human health, among others, and costs of these impacts.

An example of what can be achieved with the EcoSenseWeb model is presented in the EcoSenseWeb website[20]. This is based upon a (fictitious) power plant located in France on the border with Luxembourg. The background ozone without this power plant is displayed in Figure 3.6 and the change in the concentration due to the power plant is shown in Figure 3.7. The map of the changes clearly shows that although the power station is located in France the dispersion is over only a small part of France. The dispersion of the pollutant is predominantly local but does have a small amount dispersed over an extremely wide area covering much of central Europe.

EcoSenseWeb could be able to model the impact of a single point location, or a group of sources in a specified region, and to show its relative contribution; so it seems likely that alternative estimates of the impact of industrial plants could be obtained from this source. These would likely be more realistic than those in the SOMO report as they would measure the impact not just in Italy but in other parts of Europe. Furthermore, more weight would be placed on the greater local pollution as opposed to attributing the pollution over the whole of Italy.

The EcoSenseWeb model is based upon the EMEP model which is available online[21]. This model can also be used to assess the impact of individual power stations or other industrial facilities in Italy. However, we cannot remove the limitations of predictive dispersion models (even those site specific) related to the sum of many associated uncertainties. Such modelling would then require sensitivity analysis and comparison to real data for validation (see below).

3.5.2. Assessment of all pollutant emitters

We propose that the contribution of coal plant emissions to pollution's associated health burden should be evaluated jointly with all other sources of emissions. This would improve the completeness of the analysis.

[20] http://ecosenseweb.ier.uni-stuttgart.de/concentration.html (accessed on 02/04/2013)
[21] https://wiki.met.no/emep/page1/unimodopensource2011 (accessed on 04/02/2013)

First, all coal plants located in Italy could be assessed: five additional coal power plants located all over Italy could be taken into account. Second, impact of transborder pollution could also be modelled. For example, all coal plants located within 50 km (or 100, 200, etc) from Italy could be included in the study, and effects of pollution from Italian coal plants on adverse health outcomes in neighbouring countries could be evaluated. Third, the same methodology could be used to determine the role of other sectors such as industry, household heating or road traffic. This would allow a comparison of the impact of each sector on public health. Fourth, health impacts of alternative energy scenarios could also be investigated.

3.5.3. Use direct measurements

Measuring pollution nearby coal plants would generate less uncertainty than modelling pollutant concentrations from global emissions. Data from automatic pollutant monitoring stations located around coal plants could be used (use of real data to validate the models). With such measurements, several steps in the computations would be avoided and potential uncertainties would be reduced. This would allow a finer description of the issues related to air pollution. Furthermore such a metric would better fit to the original exposure definition that is used for the computation of risks, and this would limit several sources of biases.

3.5.4. Sources of mortality data

Since location of coal plants can be taken into account. It would be interesting to use local or regional statistics when assessing health impacts of pollution from coal plants. In Italy, regional mortality data are available on the website of the Italian Institute of Statistics (Instituto Nazionale di Statistica)[22]. Detailed mortality data are available by age and sex at National, Areas (North-West, North-East, Centre, South, Islands), Regional (20 regions) and Provincial (110 provinces) levels. Data for regional and provincial capitals are also available. Using of regional data is even more relevant for Italy as a very strong South to North gradient exists for major diseases.

Regional data of neighbouring countries could be used when evaluating the impact of Italian coal plants in these areas.

3.5.5. Sensitivity analysis

The computation of the impact of coal plant emissions requires a series of steps to get to the final result. Assumptions and hypotheses are made at each step. Several scenarios need to be developed, for example a "best-case scenario", a "worst-case scenario" and a "median scenario". This sensitivity analysis would account for the

[22] http://sitis.istat.it/sitis/html/indexEng.htm (accessed on 19/04/2013)

variability of risk estimates (using confidence intervals limits of risk estimates), and the uncertainties in pollutant emissions. Different lag times between exposure and health outcomes (occurrence of disease, mortality) would be used. These lag times would differ between health outcomes.

These sensitivity analyses would be helpful when evaluating the robustness of results.

3.5.6. Diffusion of results

The evaluation of adverse health effects related to pollution from coal-fired power plants, as described above, should be carried out by an independent and multidisciplinary group. Epidemiologists, biostatisticians and specialists of pollutant diffusion should take part in the design, data selection and analyses. Results of the evaluation should be published in a peer-reviewed journal as it is the only recognised way to disseminate results of scientific studies and to have them validated by the scientific community.

3.6. Conclusion

There is a clear need for assessment of solutions for decreasing air pollution by $PM_{2.5}$. Coal-fired power stations are emitting a certain quantity of $PM_{2.5}$ and these emissions contribute to global air pollution. However, these emissions are far lower than those resulting from other human activities such as household heating or road traffic and have been falling significantly. If legitimate, the estimation of the role of these emissions should be based on adequate modelling. The SOMO report attempted to compute the contribution of ENEL's coal power plants to the global burden associated with $PM_{2.5}$ pollution ignoring other coal power plants in Italy and other sources of $PM_{2.5}$ pollution. This report failed to a large extent to provide a robust scientifically sounded estimation.

Major approximations were performed in the evaluation done by SOMO. The computation required several steps, each of which could result in severe deviation in the estimation of impact of air pollution. The most emblematic of all is the transfer of emission from power plants to Italy without accounting for the geographical location of the power plants. This transfer of emission values considered for example that pollution emitted in Brindisi (south east) could be applied to the whole of Italy. The assumption in the calculation is even more inappropriate as the scaling factor used by SOMO was derived from an EEA report which was based on all industries in Italy (mainly North West) and pollution from other European countries (north).

In conclusion, the estimation of health effect from air pollution resulting from coal fired power plant performed by SOMO, although a first initiative, cannot be considered as valid. The present report provides elements for conducting an estimation based on the most valid methods currently available. Moreover, it is absolutely essential to prepare models that look at all sources of pollution and not simply refer to a single marginal source. With the inherent limitation of such modelling (even those site-specific), sensitivity analysis should be conducted and modelling should be compared to actual measurements.

This is an absolute principle if the focus is, as it should be, on improving Public Health.

Tables and Figures

Table 1.1: Classification of coal according to rank [a] (from IARC 1997)

Class	Group	Limits of fixed carbon or Btu, mineral-matter-free basis	Requisite physical properties
I. Anthracite	1. Meta-anthracite	Dry FC, ≥98% (dry VM, ≤2%)	
	2. Anthracite	Dry FC, 92-98% (dry VM, 2-8%)	
	3. Semi-anthracite	Dry FC, 80-92% (dry VM, 8-14%)	Non-agglomerating [b]
II. Bituminous	1. Low-volatile bituminous coal	Dry FC, 78-86% (dry VM, 14-22%)	
	2. Medium-volatile bituminous coal	Dry FC, 69-78% (dry VM, 22-31%)	
	3. High-volatile A bituminous coal	Dry FC, <69% (dry VM, >31%); and moist Btu, ≥14,000 [c, d]	
	4. High-volatile B bituminous coal	Moist Btu, 13,000-14,000 [e]	
	5. High-volatile C bituminous coal [f]	Moist Btu, 11,000-13,000 [e]	
III. Sub-bituminous	1. Sub-bituminous A coal	Moist Btu, 11,000-13,000 [e]	Both weathering and non-agglomerating
	2. Sub-bituminous B coal	Moist Btu, 9,500-11,000 [e]	
	3. Sub-bituminous C coal	Moist Btu, 8,300-9,500 [e]	
IV. Lignite	1. Lignite	Moist Btu, <8,300	Consolidated
	2. Brown coal	Moist Btu, <8,300	Unconsolidated

FC: fixed carbon; VM: volatile matter; Btu: British thermal units

[a]: This classification does not include a few coals that have unusual physical and chemical properties and that come within the limits of fixed carbon or Btu of the high-volatile bituminous and sub-bituminous ranks. All these coals contain less than 48% dry, mineral-matter-free fixed carbon or have more than 15,500 moist, mineral-matter-free Btu

[b]: If agglomerating, classified in low-volatile group of the bituminous class

[c]: it is recognized that there may be non-caking varieties in each group of the bituminous class

[d]: "Moist Btu" refers to coal containing its natural bed moisture but not including visible water on its surface

[e]: Coals having ≥ 69% fixed carbon on the dry, mineral-matter-free basis shall be classified according to fixed carbon regardless of Btu

[f]: There are three varieties of coal in the high-volatile C bituminous coal group: variety 1, agglomerating and non-weathering; variety 2: agglomerating and weathering; variety 3: non-agglomerating and non-weathering

Table 1.2: Summary table - Occupational studies

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/ Weaknesses	Ref
Respiratory function	FEV_1	Working as a boilermaker in a coal plant vs. working in non-coal power plants Date of exposure: 1997-1998	Coefficients and p-values of linear regressions	-7.6 [-22.4; 7.2] mL/100 hours worked NS	Exposure among highly exposed individuals; individual exposure assessment / selection bias; exposure not specific (several sources)	Hauser et al, 2001
	Respiratory morbidity Respiratory diseases	Working in coal plant, directly engaged in coal handling Date of exposure: (2003 publication date)	None	Not applicable	Highly exposed population / no control group; small sample size	Manna et al, 2003
Immunological profile	Blood serum IgG, IgA, IgM, α-1-antitrypsin, α-2-macroglobulin, transferrin, orosomucoid, ceruloplasmin and lysozyme	Working in a coal plant burning coal rich in arsenic vs. working in other coal plant Date of exposure: (1988 publication date)	p-values of F-test and t-test	Tranferrin, ceruloplasmin and orosomucoid levels significantly higher among workers of the arsenic-rich coal plant ($p<0.01$).	Highly exposed population / no adjustment for confounders; small sample size	Bencko et al, 1988

Table 1.3: Summary table - Prenatal exposure

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Methaemoglobin concentration	in mother's blood	Pregnancy during "clean period" vs. "dirty period" Date of exposure: 1989-1990	Correlations (r) between pregnancy variables and SO_2 exposure	significant positive correlation (r=0.72) between daily SO_2 levels and methaemoglobin levels	No strength / ecological design; confounding factors very likely	Mohorovic 2003; Mohorovic et al, 2010
Weight	At birth	Cord blood PAH-DNA adducts Date of exposure: 12/2001-05/2002	Coefficients and p-values of linear regressions	β = -0.007 (NS)	Prospective design; individual assessment of exposure; adjustment for confounders / PAH-DNA not specific to coal plant; no dose-response	Tang et al, 2006
		Pregnancy during "clean period" vs. "dirty period" Date of exposure: 1989-1990	Correlations (r) between pregnancy variables and SO_2 exposure	r=-0.0807 and r=-0.0733, for exposure during first month or second month of pregnancy respectively (significant); other NS	No strength / ecological design; confounding factors very likely	Mohorovic 2004
	at 18, 24 and 30 months	Cord blood PAH-DNA adducts Date of exposure: 12/2001-05/2002	Coefficients and p-values of linear regressions	β=-0.048, β=-0.041 and β=-0.040 at 18, 24 and 30 months, resp. (all $p < 0.05$)	Prospective design; individual assessment of exposure; adjustment for confounders / PAH-DNA not specific to coal plant; no dose-response	Tang et al, 2006

(Table 1.3 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Height	At birth and at 18, 24 and 30 months	Cord blood PAH-DNA adducts Date of exposure: 12/2001-05/2002	Coefficients and p-values of linear regressions	β=-0.001, β=-0.005, β=-0.007 and β=-0.006 at birth, 18, 24 and 30 months, resp. (NS)	Prospective design; individual assessment of exposure; adjustment for confounders/ PAH-DNA not specific to coal plant; no dose-response	Tang et al, 2006
Head circumference	At birth and at 18, 24 and 30 months	Cord blood PAH-DNA adducts Date of exposure: 12/2001-05/2002	Coefficients and p-values of linear regressions	β=-0.011, β=-0.012 and β=-0.006 and β=-0.005 at birth, 18, 24 and 30 months, resp. (NS)	Prospective design; individual assessment of exposure; adjustment for confounders / PAH-DNA not specific to coal plant; no dose-response	Tang et al, 2006
Developmental quotient	At age 2	Cord blood PAH-DNA adducts; lead and mercury Date of exposure: 12/2001-05/2002	Coefficients and p-values of linear regressions Odds ratios	an increase in 0.1 adduct/10^8 nucleotides increased the risk of being developmentally delayed in motor area by OR=1.91 [1.22; 2.97]. An elevated lead level increased the probability of motor delay (OR=3.85 [1.04; 14.25]) and of social delay (OR=7.29 [1.35; 39.45]). Other NS	Prospective design; individual assessment of exposure, adjustment for confounders; dose-response / PAH-DNA not specific to coal plant	Tang et al, 2008

(Table 1.3 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Intellectual quotient	At age 5	Cord blood PAH-DNA adducts, environmental tobacco smoke exposure Date of exposure: 12/2001-05/2002	Coefficients and p-values of linear regressions	ETS exposure and PAH-DNA adducts were associated with decreases in IQ, but NS. Significant interactions between ETS and adducts.	Prospective design; individual assessment of exposure, adjustment for confounders / PAH-DNA not specific to coal plant; no dose-response; no significant association	Perera et al, 2012
Gestation length		Pregnancy during "clean period" vs. "dirty period" Date of exposure: 1989-1990	Correlations (r) between pregnancy variables and SO_2 exposure	r=-0.0914 and r=-0.0806, for exposure during first month or second month of pregnancy respectively (significant). Other NS	No strength / ecological design; confounding factors very likely	Mohorovic 2004

Table 1.4: Summary table - Studies in children

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Respiratory function	FVC	Living in Askhelon (coal plant) vs. living in other communities (Peled et al, 2001) Date of exposure: 1991-1997	Coefficients and p-values of regressions	70% of variability across children explained by sex, age, height and weight. FVC significantly higher in Ashkelon than in all other areas. No association between air pollution (SO_2 and NO_x) and FVC (Peled et al, 2001)	No strength / ecological design; no adjustment; strong immigration during study period likely to create bias.	Peled et al, 2001; Dubnov et al, 2007
		Exposure to SO_2 and NO_x (Dubnov et al, 2007) Date of exposure: 1996-1999		Significant decrease of FVC for exposure to NO_x and SO_2 (all children, Dubnov et al, 2007).	Exposure assessed by modelling; adjustment for confounders / Strong suspicion on the modelling and location of air monitoring station	
		Annual increases between 1980 (before coal plant), 1983 (coal plant partially operating) and 1986 (coal plant fully operating) Date of exposure: 1981-1986	p-values of Anova	The FVC increases were smaller in communities labelled as having a "high pollution", the highest increases were found in "moderately polluted areas". (Goren et al, 1988) Significantly higher increase FVC in children living in area labelled as "highly polluted" (Goren et al, 1991).	No clear strength / ecological design; no adjustment for confounding factors. In the Goren study (1991), the categories of high, moderate and low exposure do not fit with actual measurements	Goren et al, 1988; Goren et al, 1991

(Table 1.4 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Respiratory function	FVC (cont.)	Exposure to PM_{10} and SO_2 in 3 villages near coal plant Date of exposure: 1997	Mean changes in pulmonary function in relation to 10µg/m^3 increase of SO_2/PM_{10}	Healthy children: NS. Asthmatic children: NS declines associated with increases in SO_2. A 10 µg/m^3 increase in PM_{10} was associated with significant decreases in FVC of –0.33%.	No strength / ecological design, no information on confounding factors.	Aekplakorn et al, 2003

(Table 1.4 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Respiratory function	FEV_1	Living in Askhelon (coal plant) vs. living in other communities Date of exposure: 1990-1997	Coefficients of and p-values linear regressions	70% of variability across children explained by sex, age, height and weight. FEV_1 significantly higher in Ashkelon than in all other areas. No association between air pollution (SO_2 and NO_x) and FEV_1	No strength / ecological design; no adjustment; strong immigration during study period likely to create bias.	Peled et al, 2001
		Exposure to SO_2 and NO_x Date of exposure: 1996-1999	Coefficients and p-values of regressions	Significant decrease of FEV_1 for exposure to NO_x and SO_2 (Dubnov et al, 2007; Yogev-Baggio et al, 2010).	Exposure assessed by modelling; adjustment for confounders / Strong suspicion on the modelling and location of air monitoring station	Dubvnov et al, 2007; Yogev-Baggio et al, 2010

(Table 1.4 cont.)

Health outcome	**Health outcome details**	**Exposure assessment**	**Measure of association**	**Main results**	**Strengths/Weaknesses**	**Ref**
Respiratory function	FEV_1 (cont.)	Annual increases between 1980 (before coal plant), 1983 (coal plant partially operating) and 1986 (coal plant fully operating) Date of exposure: 1981-1986	p-values of anova	The increases in FEV_1 were smaller in communities labelled as having a "high pollution", the highest increases were found in "moderately polluted areas". (Goren et al, 1988) Significantly higher increase FEV_1 in children living in area labelled as "highly polluted" (Goren et al, 1991).	No clear strength / ecological design; no adjustment for confounding factors. In the Goren et al, study (1991), the categories of high, moderate and low exposure do not fit with actual measurements	Goren et al, 1988; Goren et al, 1991
		Exposure to PM_{10} and SO_2 in 3 villages near coal plant Date of exposure: 1997	Mean changes in pulmonary function in relation to 10µg/m^3 increase of SO_2/PM_{10}	Healthy children: NS. Asthmatic children: NS declines associated with increases in SO_2. A 10 µg/m^3 increase in PM_{10} was associated with significant decreases in FEV_1 of –0.36%.	No strength / ecological design; no information on confounding factors.	Aekplakorn et al, 2003

(Table 1.4 cont.)

Health outcome	**Health outcome details**	**Exposure assessment**	**Measure of association**	**Main results**	**Strengths/ Weaknesses**	**Ref**
Respiratory function	PEF(R)	Asthmatic children living in several communities Date of exposure: 1999	Coefficients and p-values of regression models	Lung function of asthmatic children was significantly negatively associated with air pollution by PM_{10} and $PM_{2.5}$.	Adjustment for confounders / ecological design; statistical modelling problematic (variable selection, colinearity)	Peled et al, 2005
		Annual increases between 1980 (before coal plant), 1983 (coal plant partially operating) and 1986 (coal plant fully operating) Date of exposure: 1981-1986	p-values of anova	The increases in PEF were smaller in communities labelled as having a "high pollution", the highest increases were found in "moderately polluted areas" (Goren et al, 1988). NS higher increase PEF in children living in area labelled as "highly polluted" (Goren et al, 1991).	No clear strength / ecological design; no adjustment for confounding factors. In the Goren study (1991), the categories of high, moderate and low exposure do not fit with actual measurements	Goren et al, 1988; Goren et al, 1991

(Table 1.4 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Respiratory function	PEF(R) (cont.)	Exposure to PM_{10} and SO_2 in 3 villages near coal plant Date of exposure: 1997	Mean changes in pulmonary function in relation to 10µg/m^3 increase of SO_2/PM_{10}	Healthy children: NS. Asthmatic children: NS declines associated with increases in SO_2. A10 µg/m^3 increase in PM_{10} was associated with significant decreases in PEFR of –0.42%	No strength / ecological design; no information on confounding factors.	Aekplakorn et al, 2003

(Table 1.4 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Respiratory function	Asthma	5th grade children in 1980 (no coal plant) vs. 5th grade children in 1983 (partially operating), 1986 and 1989 (fully operating) (Goren and Hellmann 1997) Date of exposure: 1981-1989	Odds ratios	Significant increase with time in prevalence of asthma: OR=1.01 [0.64; 1.60] in 1983; 1.54 [1.00; 2.35] in 1986; 1.79 [1.16; 2.74] in 1989 vs. 1980 (Goren and Hellmann 1997)	No clear strength / ecological design; no adjustment for confounding factors.	Goren and Hellmann 1997; Henry et al, 1991a
		Living in a city close to coal plants vs. city far from coal plant (Henry et al, 1991a) Date of exposure: 1986-1987		Prevalence of asthma was significantly more elevated in study area than in control area (ORs from 1.95 to 2.66 depending on the definition of asthma, all statistically significant). (Henry et al, 1991a)	No strength / ecological design; no adjustment for confounders; no individual assessment	

(Table 1.4 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths / Weaknesses	Ref
Respiratory function	Wheezing	5th grade children in 1980 (no coal plant) vs. 5th grade children in 1983 (partially operating), 1986 and 1989 (fully operating) (Goren and Hellmann 1997) Date of exposure: 1980-1989	Odds ratios	Significant increase with time in prevalence of wheezing: OR=1.07 [0.74; 1.55] in 1983; 1.30 [0.91; 1.84] in 1986; 1.59 [1.11; 2.28] in 1989 vs. 1980 (Goren and Hellmann 1997)	No clear strength / ecological design; no adjustment for confounding factors	Goren and Hellmann 1997; Henry et al, 1991b
		High, medium SO_2/NO_x vs. low SO_2, NO_x Date of exposure: 1986-1987 (Henry et al, 1991b)		Children of study area were more likely to be wheezing than those from control area (prevalence ~10%). NS risk of wheezing on days with high SO_2/NO_2 concentrations compared with days with low pollution. (Henry et al, 1991b)	No strength / ecological design; no adjustment for confounders; no individual assessment	

(Table 1.4 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Respiratory function	Other symptoms and diseases	Annual increases between 1980 (before coal plant), 1983 (coal plant partially operating) and 1986 (coal plant fully operating) (Goren et al, 1988; Goren et al, 1991) Date of exposure: 1981-1986	p-values of χ^2 tests	Prevalence of some symptoms significantly increased in the younger cohort. Other respiratory symptoms and illnesses were more prevalent in 1983, but NS. In the older cohort, prevalence of respiratory symptoms/diseases were generally less prevalent in 1983 than in 1980 (Goren et al, 1988) No clear association was found between pollution and lung disease and symptoms (Goren et al, 1991).	No clear strength / ecological design; no adjustment for confounding factors. In Goren et al, 1991, the categories of high, moderate and low exposure do not fit with actual measurements	Goren et al, 1988; Goren et al, 1991

(Table 1.4 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/ Weaknesses	Ref
Respiratory funciton	Other symptoms and diseases	Non-asthma symptoms: Living in a city close to coal plants vs. city far from coal plant (Henry et al, 1991a) Date of exposure: 1986-1987	Odds ratios	Some symptoms significantly more prevalent in study area; some NS (Henry et al, 1991a)	No strength / ecological design; no adjustment for confounders; no individual assessment	Henry et al, 1991a; Henry et al, 1991b
		Any symptom: High, medium SO_2/NO_x vs. low SO_2, NO_x (Henry et al, 1991b) Date of exposure: 1986-1987		Children of study area were more likely to be have respiratory symptoms than those from control area (prevalence ~20%). NS risk of respiratory symptoms on days with high SO_2/NO_2 concentrations compared with days with low pollution (Henry et al, 1991b)	No strength / ecological design; no adjustment for confounders; no individual assessment	
		Living in 3 communities with different pollution levels in 1996-1999 Date of exposure: 1996-1999	Changes 1996-1999	The impact of NO_x and SO_2 on FVC and FEV_1 does not dependent on the child pulmonary health.	Exposure assessed by modelling; adjustment for confounders / Strong suspicion on the modelling and location of air monitoring station	Yogev-Baggio et al, 2010

(Table 1.4 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Hearing thresholds	Air and bone conduction	Living 1.5-75 km of coal plant burning arsenic-rich coal vs. control group Date of exposure: (publication date 1977)	p-values of χ^2 tests	Significant decreased hearing threshold in children living near the plant for all frequencies (air and bone conduction)	No strength / ecological design; selection bias; very small sample size	Bencko & Symon 1977
Arsenic concentrations	In hair and in urine	Living 1.5-75 km of coal plant burning arsenic-rich coal vs. control group Date of exposure: (publication date 1977)	None	Not applicable	No strength / ecological design, selection bias, very small sample size	Bencko & Symon 1977
Autism		Prevalence of autism in 2 counties sub-areas Date of exposure: 2002	None	Not applicable	No strength / ecological design, selection bias likely	Blanchard et al, 2011

Table 1.5: Summary table - Studies in adults

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Incidence	Lung cancer	Pollution level (SO_2, NO_2, suspended particulates) of residence Date of exposure: 1978-1994	Odds ratios	Highest vs. lowest 20% increased risk, but NS	Case-control design; highly exposed population; modelling of air pollution from coal plant including wind dispersion; adjustment for confounders / too small sample size for stratified analysis	Pisani et al, 2006
	Non-melanoma skin cancer	Distance residence/coal plant Date of exposure: at least 1 year; 1953-1999	Odds ratios	Highest vs lowest: 90% significantly increased risk: other results NS	Case-control design/ exposure assessed by distance to coal plant (ecological component); no adjustment for sun exposure.	Pesch et al, 2002
		Residence < 7.5 km of coal plant vs. >7.5km in 4 time periods Date of exposure: 1977-1996 (recruitment of cases)	Relative risks	102% increased risk (men) in 1977-1981; other periods NS; 125% increased risk (women) in 1977-1981; other periods NS	No strength/ ecological design, exposure assessed by distance to coal plant (ecological component); no adjustment for sun exposure.	Bencko et al, 2009

(Table 1.5 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
Respiratory function	Symptoms and diseases	Residence near or far from coal plant Date of exposure: 1981 (Pershagen et al, 1986); 1999 (Karavuş et al, 2002)	p-values of χ^2 tests	Most symptoms NS; some (hawking, cough without phlegm) significantly higher in some of the exposed areas	Matched study design / ecological design, self-declared symptoms, no adjustment performed, matching criteria not reported	Pershagen et al, 1986
			p-values of t-tests	Chest tightness significantly more prevalent in the study villages (46%) than in the controls (28%). Repeated coughing attacks during the preceding year more frequent among the study group. Other NS	Extremely exposed population / ecological design; definition of outcome not clear ("Chest tightness")	Karavuş et al, 2002

(Table 1.5 cont.)

Health outcome	Health outcome details	Exposure assessment	Measure of association	Main results	Strengths / Weaknesses	Ref
Respiratory function	FVC FEV_1 FEV_1/FVC $FEF_{25-75\%}$	Residence near or far from coal plant Date of exposure: 1999 (Karavuş et al, 2002); 2004-2005 (Pala et al, 2012)	p-values of t-tests	FEV_1 and $FEF_{25-75\%}$ significantly higher in the control group; differences were more marked among non-smokers (Karavuş et al, 2002). FVC and FEV_1 were significantly lower in the study group; FVC/FEV_1 and $FEF_{25-75\%}$ were significantly higher in the study group (in ever smokers only for $FEF_{25-75\%}$) (Pala et al, 2012)	(Karavuş et al, 2002) Extremely exposed population / ecological design; definition of outcome not clear ("Chest tightness") (Pala et al, 2012) No strength / ecological design; imbalance in average age between exposed and unexposed villages; selection bias	Karavuş et al, 2002; Pala et al, 2012
Visits to clinics	Total visits Respiratory diseases visits	NO_x and SO_2 concentration; flu epidemics Date of exposure: 1982-1990	Coefficients and p-values of regressions	Main explanatory factor: temperature	No clear strength / ecological design; no adjustment for confounding factors	Goren et al, 1995

Table 1.6: Summary table – Studies on mortality associated with emissions from coal power plants

Cause of death	Exposure assessment	Measure of association	Main results	Strengths/Weaknesses	Ref
	Occupational exposure				
All-cause	Working in a coal plant burning coal rich in arsenic vs. working in other coal plant Date of exposure: 1960-1978	Distribution of mortality cause by age	NS	Highly exposed population / control group also likely exposed; small sample size	Bencko et al, 1980
	Working in coal plant vs. whole Italian population Date of exposure: 1968-1987	Standardised mortality ratio (SMR)	NS	Highly exposed population / healthy worker effect likely; no corresponding period between exposed and reference; small sample size	Petrelli et al, 1989; Petrelli et al, 1994
Cancer	Working in a coal plant burning coal rich in arsenic vs. working in other coal plant Date of exposure: 1960-1978	Distribution of mortality cause by age	Tumour deaths occurred at significantly younger ages in the exposed group.	Highly exposed population / control group also likely exposed; small sample size	Bencko et al, 1980
	Working in coal plant vs. whole Italian population Date of exposure: 1968-1987	Standardised mortality ratio (SMR)	NS	Highly exposed population / healthy worker effect likely; no corresponding period between exposed and reference; small sample size	Petrelli et al, 1989; Petrelli et al, 1994
Cardiovascular Accidental Other	Working in a coal plant burning coal rich in arsenic vs. working in other coal plant Date of exposure: 1960-1978	Distribution of mortality cause by age	NS	Highly exposed population / control group also likely exposed; small sample size	Bencko et al, 1980

(Table 1.6 cont.)

Cause of death	Exposure assessment	Measure of association	Main results	Strengths / Weaknesses	Ref
Environmental exposure					
Lung cancer	Residence in several more or less polluted areas (Parodi et al, 2004) Date of exposure: 1988-1996[23]	Relative risks	NS for men; increased 54-114% risk in the most polluted areas for women (Parodi et al, 2004)	Adjustment for socio-economic factors / ecological design; no adjustment for tobacco smoking; no specific exposure source	Parodi et al, 2004; Garcia-Perez et al, 2009
	Place of death <5km of "isolated" coal plant vs. >5km (Garcia-Perez et al, 2009) Date of exposure; 1994-2003[24]		13% increased risk for men; NS for women (Garcia-Perez et al, 2009)	No strength / ecological design; exposure based on distance to coal plant; no adjustment; no consistent results	
Laryngeal cancer Bladder cancer	Place of death <5km of "isolated" coal plant vs. >5km Date of exposure; 1994-2003	Relative risks	46% (laryngeal) and 22% (bladder) increased risk for men; NS for women	No strength / ecological design; exposure based on distance to coal plant; no adjustment; no consistent results	Garcia-Perez et al, 2009

[23] Study conducted on 1988-1996 deaths
[24] Study conducted on 1994-2003 deaths

Table 1.7: Level of indoor air pollutants from household coal emissions in China (from Sinton et al, 1995)

Pollutant	Urban (mg/m^3)	Rural (mg/m^3)
TSP	0.21–2.8	0.01–20
PM_{10}	0.16–2.7	0.12–26
CO	0.58–97	0.7–87
SO_2	0.01–5.8	0.01–23
NO_x	0.01–1.8	0.01–1.7
B[*a*]P	0.3–190	5.3–19000

Table 1.8: Basic characteristics of studies included in the qualitative review

Reference	Design	Country	Year of recruitment	Population	Outcome
			Occupational		
Bencko et al, 1988	Ecological	Former Czechoslovakia (probably)	NA (probably 1980s)	47 men working in a coal plant burning high arsenic coal + 27 men working in another coal plant burning "regular" coal	Blood serum IgG, IgA, IgM, α-1-antitrypsin, α-2-macroglobulin, transferrin, orosomucoid, ceruloplasmin and lysozyme
Hauser et al, 2001	Cohort	USA	1997-1998	118 boilermakers	FCV; FEV_1
Manna et al, 2003	Ecological	India	Not specified	50 workers directly implicated in the process of coal handling for at least 5 years	Respiratory morbidity

(Table 1.8 cont.)

Reference	Design	Country	Year of recruitment	Population	Outcome
			Prenatal		
Mohorovic 2003	Cohort	Croatia	1989-1990	138 pregnant women living near Plomin 1 coal plant (Larbin/Croatia)	Methemoglobin concentration in mother's blood; pregnancy losses
Mohorovic 2004	Cohort	Croatia	1987-1989	704 pregnant women living near Plomin 1 coal plant (Larbin/Croatia)	Preterm birth; low birthweight related to pollutants
Mohorovic et al, 2010	Cohort	Croatia	1989-1990	138 pregnant women living near Plomin 1 coal plant (Larbin/Croatia)	Methemoglobin concentration in mother's blood; pregnancy losses
Tang et al, 2006	Cohort	China	2002	150 children born of non-smoking mothers between 4/3/2002 and 19/6/2002; living within 2.5 km of coal plant (seasonally operating until 12/2004)	Cord blood PAH-DNA adducts; weight; height and head circumference at birth; 18 months; 24 months and 30 months
Tang et al, 2008	Cohort	China	2002	150 children born of non-smoking mothers between 4/3/2002 and 19/6/2002; living within 2.5 km of coal plant (seasonally operating until 12/2004)	Cord blood PAH-DNA adducts; Developmental quotient at age 2
Perera et al, 2012	Cohort	China	2002	122 mother-child pairs; children born within 2.5km of Tongliang coal plant (seasonally operating until 12/2004) between 4/3/2002 and 19/6/2002	Child IQ at age 5; PAH/DNA adducts in cord blood

(Table 1.8 cont.)

Reference	Design	Country	Year of recruitment	Population	Outcome
			Children		
Bencko et al, 1977	Ecological	Former Czechoslovakia	NA (probably 1970s)	107 boys aged 9.5-11 years living near coal plant	Arsenic in hair; arsenic in urine; hearing threshold
Goren et al, 1992	Description of cohort	Israel	1990	2nd, 5th and 8th grade schoolchildren living within 25 km of Rotenberg coal plant + 200 children with asthma	FCV; FEV_1; PEF; $FEF_{50\%}$; $FEF_{25\%}$
Peled et al, 2001	Ecological	Israel	1990; 1994; 1997	2455; 1613; 4346 children living near 2 power plant (1 coal; 1 oil)	FVC; FEV_1
Peled et al, 2005	Ecological	Israel	1999	285 children with asthma living near 2 power plants (1 coal; 1 oil)	PEF
Goren et al, 1986	Cohort	Israel	1980	~1,000 schoolchildren (2nd and 5th grade) living near coal plant in Hadera	FVC; FEV_1; FVC/FEV_1; PEF; respiratory symptoms
Goren et al, 1988	Cohort	Israel	1980; 1983	~1,000 schoolchildren (2nd and 5th grade) living near coal plant in Hadera	FVC; FEV_1; FVC/FEV_1; PEF; respiratory symptoms
Goren et al, 1991	Cohort	Israel	1980; 1983; 1986	~3,000 school children (2nd, 5th and 8th grade) living near coal plant in Hadera	FVC; FEV_1; FVC/FEV_1; PEF; respiratory symptoms
Goren et al, 1997	Cohort	Israel	1980; 1983; 1986; 1989	~3,000 school children (2nd, 5th and 8th grade) living near coal plant in Hadera	FVC; FEV_1; FVC/FEV_1; PEF; respiratory symptoms
Dubnov et al, 2007	Cohort	Israel	1996-1999	1,492 school children aged 7-14 living near coal plant of Hadera	FVC; FEV_1;

(Table 1.8 cont.)

Reference	Design	Country	Year of recruitment	Population	Outcome
				Children cont.	
Yogev-Baggio et al, 2010	Cohort	Israel	1996-1999	1,181 school children (2nd and 5th grade) living near Hadera	FVC; FEV_1; Chest symptoms; pulmonary diseases
Henry et al1991a	Ecological	Australia	1986-1987	201 schoolchildren aged 8-9 living near coal plant + 401 living far from coal plant	Asthma prevalence; respiratory symptoms; bronchial hyperreactivity
Henry et al, 1991b	Ecological	Australia	1986-1987	49 asthmatic schoolchildren aged 8-9 living near coal plant + 51 far from coal plant	Time trends of wheezing and other respiratory symptoms; correlation with pollutant levels
Aekplakorn et al, 2003	Ecological	Thailand	1997	88 asthmatic and 96 non-asthmatic children aged 6-14 living near a coal plant	FVC; FEV_1; PEFR
Blanchard et al, 2011	Ecological	USA	NA	Residents of 2 US counties	Prevalence of autism

(Table 1.8 cont.)

Reference	Design	Country	Year of recruitment	Population	Outcome
Adults					
Pisani et al, 2006	Case-control	Thailand	1993-1995	211 lung cancer cases + 211 hospital controls + 202 population controls	Lung cancer
Pesch et al, 2002	Case-control	Slovakia	1996-1999	264 NMSC cases aged less than 80 at diagnosis + 286 controls frequency matched on age and sex living near Prievidza coal plant	Risk of NMSC related to arsenic exposure from coal plant
Ranft et al, 2003	Ecological	Slovakia	1999-2000	411 subjects living near Prievidza coal plant	Urinary arsenic concentration; arsenic in house dust and soil
Bencko et al, 2009	Ecological	Slovakia	1977-1996	1,503 NMSC cases living near Prievidza coal plant	Correlation between arsenic exposure and NMSC incidence
Pershagen et al, 1986	Ecological	Finland	1981	~12,000 subjects aged 15-64 living in 6 areas near coal plants and in 6 reference areas without coal plants	Prevalence of hawking, cough, acute dyspnoea in the 2 populations
Goren et al, 1995	Ecological	Israel	1982-1990	Population residing near a coal plant	Visits in clinics (total and respiratory diseases) in children and adults (time trends) correlated with pollutant levels
Karavuş et al, 2002	Ecological	Turkey	1999	277 villagers living near Seyitomer coal plant + 225 living far from coal plant	Chest tightness; repeated coughing; FEV_1; FVC; FEV_1/FCV; $FEF_{25-75\%}$
Pala et al, 2012	Ecological	Turkey	2004-2005	2,350 residents aged 15+ living near Orhaneli coal plant + 469 other residents living far from coal plant	FCV; FEV_1; $FEF_{25-75\%;}$ FEV_1/FCV

(Table 1.8 cont.)

Reference	Design	Country	Year of recruitment	Population	Outcome
			Adults		
Fabiánová et al, 2000	Modelling study	Slovakia	1953-1993	Population living near Prievidza coal power plant	Lifetime risk of lung cancer
Thanh and Lefevreet al, 2001	Modelling study	Thailand	NA	Thailand population	---
Gohlke et al, 2011	Modelling study	World	1965-2005	Population of 41 countries in the world	Evolution of life expectancy and infant mortality

(Table 1.8 cont.)

Reference	Design	Country	Year of recruitment	Population	Outcome
			Mortality		
Bencko et al, 1980	Observational	Former Czechoslovakia	1960-1978	88 + 159 male deaths among coal plant workers	Distribution of causes of death
Petrelli et al, 1989	Cohort	Italy	1968	1,307 men working in Porto Marghera and Fusina coal plants	Mortality of all causes; all cancers; digestive system cancer (+ subcategories); respiratory system cancer; Lung cancer
Petrelli et al, 1994	Cohort	Italy	1968	1,772 men working in Porto Marghera, Fusina and Monfalcone coal plants	Mortality of all causes; all cancers; digestive cancer (and subcategories); respiratory system cancer (+ subcategories); and other sites
Parodi et al, 2004	Ecological	Italy	1988-1996	24 subjects died of lung cancer in La Spezia + 1,318 subjects dead of lung cancer in the Liguria	Comparison of lung cancer mortality in two areas
García-Pérez et al, 2009	Ecological	Spain	1994-2003	Residents of Spanish towns living near coal plants/far from coal plants (5 exposure groups)	Mortality of: - Lung cancer - Bladder cancer - Laryngeal cancer

Table 1.9: Distances between coal plant and study site

Reference	Distance to coal plant (km)
	Occupational
Bencko et al, 1988	0
Hauser et al, 2001	0
Manna et al, 2003	0
Bird et al, 2004	0
	Prenatal exposure
Mohorovic 2003	3.5-12.5
Mohorovic 2004	3.5-12.5
Mohorovic et al, 2010	3.5-12.5
Tang et al, 2006	2.5
Tang et al, 2008	2.5
Perera et al, 2012	2.5

(Table 1.9 cont.)

Reference	Distance to coal plant (km)
	Children
Bencko and Symon 1977	1.5-75
Goren et al, 1992	<25
Peled et al, 2001	<25
Peled et al, 2005	<25
Goren et al, 1986	<19
Goren et al, 1988	<19
Goren et al, 1991	<19
Goren et al, 1997	<19
Dubnov et al, 2007	Up to ~10
Yogev-Baggio et al, 2010	<19
Henry et al, 1991a	4.5 (study group)/80(control group)
Henry et al, 1991b	4.5 (study group)/80(control group)
Aekplakorn et al, 2003	~7
Blanchard et al, 2011	Bexar county (3256 km^2); Santa Clara county (3377km^2)

(Table 1.9 cont.)

Reference	Distance to coal plant (km)
	Adults
Pisani et al, 2006	Not specified
Pesch et al, 2002	3 categories: <5; 6-10; >10
Ranft et al, 2003	3 categories: <5; 6-10; >10
Bencko et al, 2009	7.5
Pershagen et al, 1986	Depending on coal plant; <5 for plant A; <3 for plant D; not specified for others
Goren et al, 1995	<19
Karavuş et al, 2002	5 (study group)/30 (control group)
Pala et al, 2012	1.5-12 (study group)/50-60 (control group)
Fabiánová et al, 2000	Area = 70km²
Thanh et al, 2001	Thailand (whole country)
Gohlke et al, 2011	World

(Table 1.9 cont.)

Reference	Distance to coal plant (km)
	Mortality
Bencko et al, 1980	0 (occupational)
Petrelli et al, 1989	0 (occupational)
Petrelli et al, 1994	0 (occupational)
Parodi et al, 2004	La Spezia county (51 km^2)
García-Pérez et al, 2009	<5 to 50

Table 1.10: Quality evaluation of individual studies

Reference	Individual exposure assessment	Specificity of exposure	Temporality	Adjustment for main confounding factors	Strength of association/power	Dose-response
Occupational studies						
Bencko et al, 1988		X				
Hauser et al, 2001	X		X	X	X	
Manna et al, 2003						
Prenatal exposure and pregnancy outcomes						
Mohorovic, 2003						
Mohorovic, 2004						
Mohorovic et al, 2010						
Tang et al, 2006	X		X	X		
Tang et al, 2008	X		X	X	X	X
Perera et al, 2012	X		X	X		

(Table 1.10 cont.)

Reference	Individual exposure assessment	Specificity of exposure	Temporality	Adjustment for main confounding factors	Strength of association/power	Dose-response
Children						
Bencko et al, 1977						
Goren et al, 1992						
Peled et al, 2001				X		
Peled et al, 2005				X		
Goren et al, 1986						
Goren et al, 1988			X			
Goren et al, 1991						
Goren et al, 1997						
Dubnov et al, 2007	X-			X	X	
Yogev-Baggio et al, 2010	X-			X	X	
Henry et al, 1991a				X	X	
Henry et al, 1991b						
Aekplakorn et al, 2003				X-		
Blanchard et al, 2011						

(Table 1.10 cont.)

Reference	Individual exposure assessment	Specificity of exposure	Temporality	Adjustment for main confounding factors	Strength of association/power	Dose-response
Adults						
Pisani et al, 2006	X	X	X	X		
Pesch et al, 2002				X-		
Ranft et al, 2003	X	X				
Bencko et al, 2009						
Pershagen et al, 1986						
Goren et al, 1995		X				
Karavuş et al, 2002						
Pala et al, 2012						
Mortality						
Bencko et al, 1980						
Petrelli et al, 1994			X			
Petrelli et al, 1989			X			
Parodi et al, 2004				X-		
García-Pérez et al, 2009						

Table 3.1: Exposure Calculation from SOMO Report (from SOMO 2012)

	PM_{10}	NO_x	SO_2
Emissions, tonnes per year	473	7,300	6,540
		TIMES	
PM_{10} to $PM_{2.5}$ conversion factor	0.649	1	1
		TIMES	
Emissions-to-concentration factors for Italy	703.69	156.66	153.84
		TIMES	
Power sector adjustment factors	0.5	0.78	0.87
		EQUALS	
Increase in population-weighted concentrations, µg/m^3/person		1,875,407	
		TIMES	
Risk factor for chronic premature deaths		6.0665 x 10^{-5}	
		EQUALS	
Amount of premature deaths caused per year		113.77	
		TIMES	
Value of statistical life, M€		2.00	
		EQUALS	
Economic losses due to premature deaths, M€		227.54	

Table 3.2: Emissions from Enel's fossil fuel-based power plants in Italy and the associated health and financial impacts, 2009 (from SOMO 2012)

Enel Facility	Emissions to Air (T)				Exposure		Health and financial impacts associated with Enel emissions				
	PM_{10}	NO_x	SO_x	CO_2	$PM_{2.5}$	SOMO35[25] (ozone)	Premature deaths	(€) Crop damages	Costs air pollution (€)	Costs CO_2 (€)	Total costs (€)
Coal-fired plants											
Genova		3320	4910	1670000	1062840	99641016	66	575313	152761077	56112000	208873077
Federico II (Brindisi Sud)	473	7300	6540	13000000	1875407	254252853	119	1372376	269785802	436800000	706585802
Eugenio Montale (la Spezia)	106	1790	1870	2340000	493228	60143627	31	329794	70924771	78624000	149548771
Torrevaladiga Nord (Civitavecchia)		835	769	2860000	204956	28909416	13	156449	29508784	96096000	125604784
Sulcis	93	15	3030	2240000	607538	37468468	38	235241	87185030	75264000	162449030
Fusina	104	2500	2010	4300000	598268	88970832	38	475788	86121948	144480000	230601948
Porto Marghera		380	284	315000	84445	13701359	5	72863	12167398	10584000	22751398
Pietro Vannucci (Bastardo)	112	2220	4620	1010000	915199	55583071	57	350969	131341715	33936000	165277715
TOTAL COAL	**888**	**19825**	**24033**	**27735000**	**5841881**	**638670642**	**366**	**3568792**	**839796526**	**93189600**	**1771692526**
Other (non-coal) fossil fuel-based power plants											
Piombino		322	671	475000	129153	8054681	8	50884	18541876	15960000	34501876
Porto Corsini		360		990000	43991	15203078	3	75816	6380618	33264000	39644618
Augusta		516	1440	306000	255781	9894171	16	72338	36689614	10281600	46971214
Porto Empedocle		369	329	262000	89123	12865042	6	69411	12833187	8803200	21636387
Porto Tolle		127	250	208000	48979	3297873	3	20439	7032895	6988800	14021695
Rossano		197		268000	24073	8319462	2	41488	3491616	9004800	12496416
Livorno		301	794	240000	143049	6151640	9	43358	20522395	8064000	28586395
Priolo Gargallo		529		1600000	64642	22340079	4	111407	9375964	53760000	63135964
La Casella		452		1360000	55233	19088309	4	95191	8011221	45696000	53707221
Leri		207		148000	25295	8741770	2	43594	3668856	4972800	8641656
Montalto Di Castro		998	1210	2650000	283897	32149604	18	179651	40833154	89040000	129873154

[25] for ozone, the sum of means over 35 ppb (daily maximum 8-hour) (source http://glossary.eea.europa.eu/terminology/concept_html?term=SOMO35, accessed on 24/04/2013)

Termini Imerese		950		2020000	116087	40119234	8	200070	16837743	67872000	84709743
Assemini		671		130000	81994	28336848	6	141313	11892764	4368000	16260764
Maddaloni		210			25661	8868462	2	44226	3722027	0	3722027
Portoscuso		157		123000	19185	6630231	1	33064	2782659	4132800	6915459
Bari				128000	0	0	0	0	0	4300800	4300800
Pietrafitta		131		275000	16008	5532231	1	27589	2321836	9240000	11561836
Santa Barbara		176		442000	21507	7432616	1	37066	3119413	14851200	17970613
TOTAL NON-COAL FOSSIL FUEL		**6673**	**4694**	**11625000**	**1443658**	**243025331**	**94**	**1286905**	**208057838**	**390600000**	**598657838**
TOTAL ALL FOSSIL FUEL	**888**	**26498**	**28727**	**39360000**	**7285539**	**881695973**	**460**	**4855697**	**1047854364**	**1322496000**	**2370350364**
Coal's percentage of the total (%)	**100**	**75**	**84**	**70**	**80**	**72**	**80**	**73**	**80**	**70**	**75**

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Table 3.3: Emissions to air from Enel's coal-fired power plants in Europe (excluding Italy) and the associated health and financial impacts, 2009 (from SOMO 2012)

Enel Facility	Emissions to Air (T)				Exposure		Health and financial impacts associated with Enel emissions				
	PM_{10}	NO_x	SO_x	CO_2	$PM_{2.5}$	SOMO35 (ozone)	Premature deaths	(€) Crop damages	Costs air pollution (€)	Costs CO_2 (€)	Total costs (€)
TETs "Enel Maritsa iztok 3" (Bulgaria)		3870	14900	4950000	1335058	269494425	86	728267	193505377	166320000	359825377
Central Termoelétrica de Pego (Portugal)	60	2210	1340	2830000	127925	4256949	8	243166	18625213	95088000	113713213
Central Termica de andorra (Teruel) (Spain)		10000	11170	2610000	1474364	353397657	96	2061735	214847958	87696000	302543958
UPT Compostilla (Spain)	390	8420	3770	2640000	749296	354979616	52	1921160	110518231	88704000	199222231
Unidad de produccion termica as pontes (Spain)	264	7460	4990	5220000	800393	298929636	54	1651883	117439796	175392000	292831796
Central Térmica litoral de Almeria (Spain)	564	9740	14000	5090000	1737725	319621228	111	1928832	252284081	171024000	423308081
Slovenské elektrarme a.s. – Elektrarna Vojany zavod (Slovakia)		1390	446	898000	263353	115708980	18	385748	38927755	30172800	69100555
Slovenké elektrarne a.s. – Elektrarne Novaky, zavod (Slovakia)		3820	32400	2450000	4922819	174158416	302	818824	698085984	82320000	780405984
Total EU excluding Italy	1278	46910	83546	26688000	11410932	1890546908	727	9739613	1644234395	896716800	2540951195

Table 3.4: Health Effects of $PM_{2.5}$, taken from Table 1 of SOMO Report (SOMO 2012) and table 4 from Holland et al, 2005

Health end-point	Pollutant factor 1	Pollutant factor 2	Population factor 1	Population factor 2	Incidence rate	Response functions	Valuation (€)	Cases per $\mu g/m^3$/person/year exposure
Chronic effect on mortality (premature deaths)	1	0.1	0.628	1	0.0161	0.06	2000000	6.07×10^{-5}
Chronic effect on mortality (life years lost)	1	1	1	0.00001	1	65.1	52000	6.51×10^{-4}
Infant mortality (1-11 months)	1.54	0.1	0.009	1	0.0019	0.04	1500000	1.05×10^{-7}
Chronic bronchitis, population aged over 27 years	1.54	0.1	0.7	1	0.00378	0.07	190000	2.85×10^{-5}
Respiratory hospital admissions, all ages	1.54	0.1	1	0.00001	617	0.0114	2000	1.08×10^{-5}
Cardiac hospital admissions, all ages	1.54	0.1	1	0.00001	723	0.006	2000	6.68×10^{-6}
Restricted activity days (RADs) working age population	1	1	0.672	1	19	0.00475	82	6.06×10^{-2}
Respiratory medication use by adults	1.54	0.1	0.817	0.001	0.045	908	1	5.14×10^{-3}
Respiratory medication use by children	1.54	0.1	0.112	0.001	0.2	180	1	6.21×10^{-4}
Lower respiratory syndromes (LRS), including cough, among adults with chronic symptoms	1.54	0.1	0.817	1	0.3	1.3	38	4.91×10^{-2}
LRS (including cough) among children	1.54	0.1	0.112	1	1	1.85	38	3.19×10^{-2}
Consultations for asthma, ages 0-14	1.54	0.1	0.170	0.001	47.1	0.025	53	3.08×10^{-5}

Consultations for asthma, ages 15-65	1.54	0.1	0.672	0.001	16.5	0.031	53	5.29×10^{-5}
Consultations for asthma, ages over 65	1.54	0.1	0.158	0.001	15.1	0.063	53	2.31×10^{-5}
Consultations for upper respiratory symptoms (excluding allergic rhinitis) ages 0-14	1.54	0.1	0.170	0.001	574	0.007	53	1.05×10^{-4}
Consultations for upper respiratory symptoms (excluding allergic rhinitis) ages 15-64	1.54	0.1	0.672	0.001	180	0.018	53	3.35×10^{-4}
Consultations for upper respiratory symptoms (excluding allergic rhinitis) ages over 65	1.54	0.1	0.158	0.001	141	0.033	53	1.13×10^{-4}
Restricted activity days, non-working age population	1	1	0.328	1	19	0.00475	69	2.96×10^{-2}

Table 3.5: Effect of emission of 1 t of each pollutant from each country on population weighted concentration of $PM_{2.5}$ across Europe (from SOMO 2012)

Country	$PM_{2.5}$	NO_X	SO_2
Albania	3.9151E+02	5.8082E+01	8.2488E+01
Austria	5.8772E+02	2.2591E+02	1.8662E+02
Bosnia and Herzegovina	3.5199E+02	1.1425E+02	9.9051E+01
Belgium	8.5339E+02	1.6853E+02	2.1207E+02
Bulgaria	3.8085E+02	1.0154E+02	7.9346E+01
Belarus	2.2580E+02	9.7429E+01	1.1698E+02
Switzerland	7.3239E+02	3.5889E+02	2.6298E+02
Cyprus	2.5548E+02	9.8007E+00	2.7207E+01
Czech Republic	4.1200E+02	1.6107E+02	1.6297E+02
Germany	8.8171E+02	2.6561E+02	2.3820E+02
Denmark	2.1593E+02	7.1436E+01	9.0450E+01
Estonia	1.4089E+02	3.2905E+01	8.2895E+01
Spain	3.8324E+02	5.5669E+01	1.0219E+02
Finland	1.4099E+02	2.4983E+01	5.7584E+01
France	6.0059E+02	1.8773E+02	1.8401E+02
United Kingdom	4.8684E+02	1.0308E+02	1.5082E+02
Greece	3.5998E+02	2.2957E+01	5.8540E+01
Croatia	5.3045E+02	1.5928E+02	1.3954E+02
Hungary	5.8052E+02	2.1239E+02	1.5878E+02
Ireland	3.0100E+02	7.1488E+01	1.1304E+02
Italy	7.0369E+02	1.5666E+02	1.5384E+02
Lithuania	1.9183E+02	8.1914E+01	9.7355E+01
Luxembourg	6.3598E+02	2.3283E+02	1.9175E+02
Latvia	1.9150E+02	5.2563E+01	8.7021E+01
Moldova	4.2904E+02	1.3034E+02	1.2050E+02
Macedonia	2.3254E+02	5.9562E+01	6.3020E+01
Malta	3.1282E+02	7.0875E+00	
Netherlands	7.8787E+02	1.5920E+02	2.4723E+02
Norway	1.5740E+02	3.2299E+01	4.7047E+01
Poland	4.0410E+02	1.2328E+02	1.4186E+02
Portugal	4.7380E+02	2.2621E+01	6.8326E+01
Romania	4.1235E+02	1.6450E+02	1.1866E+02
Sweden	2.2151E+02	3.9651E+01	6.0636E+01
Slovenia	4.3189E+02	1.8557E+02	1.5780E+02
Slovakia	4.0689E+02	1.8749E+02	1.5482E+02
Turkey	3.7775E+02	2.7340E+01	5.9347E+01
Ukraine	4.1453E+02	1.0216E+02	1.3098E+02

Table 3.6: Effect of emission of 1 t of each pollutant from each country on population weighted SOMO 35 concentration across Europe (from SOMO 2012)

Country	NO_x	SO_2
Albania	8.8167E+04	-3.4164E+03
Austria	7.8029E+04	-1.2443E+04
Bosnia and Herzegovina	1.0376E+05	-3.5514E+03
Belgium	-6.6578E+04	-5.6995E+03
Bulgaria	9.2314E+04	-7.0708E+02
Belarus	4.8965E+04	-4.4867E+03
Switzerland	9.0287E+04	-1.8906E+04
Cyprus	5.5981E+04	-1.4830E+03
Czech Republic	5.5582E+04	-7.9477E+03
Germany	3.4242E+04	-1.2346E+04
Denmark	6.9511E+03	-1.0464E+04
Estonia	3.0064E+04	-4.2567E+03
Spain	5.9470E+04	-1.0853E+04
Finland	1.7214E+04	-6.2519E+03
France	9.2518E+04	-1.1662E+04
United Kingdom	-3.4462E+04	-9.9640E+03
Greece	4.3628E+04	-1.6494E+03
Croatia	1.1231E+05	-7.5827E+03
Hungary	1.1316E+05	-4.4170E+03
Ireland	4.8069E+04	-1.5386E+04
Italy	5.4142E+04	-9.4963E+03
Lithuania	6.6340E+04	-6.2456E+03
Luxembourg	1.5503E+04	-9.0853E+03
Latvia	4.5998E+04	-5.8752E+03
Moldova	9.0858E+04	-3.1587E+03
Macedonia	6.4754E+04	-2.0287E+03
Malta	1.2860E+04	
Netherlands	-7.0151E+04	-5.4803E+03
Norway	5.2553E+04	-1.4571E+04
Poland	3.9462E+04	-3.6812E+03
Portugal	6.3186E+03	-5.6914E+03
Romania	1.0215E+05	-2.0537E+03
Sweden	3.1619E+04	-1.1205E+04
Slovenia	8.6406E+04	-1.1863E+04
Slovakia	1.0862E+05	-5.3033E+03
Turkey	6.1512E+04	-8.7392E+02
Ukraine	4.5440E+04	-3.0269E+03

Table 3.7: Population of Italy living within different distances to Coal power plants

Distance to power plants	Diameter around power plants	Population covered	Percentage of the Italian population*
50 km	100km	8,154,802	14.3%
75 km	150km	13,068,772	22.9%
100 km	200km	21,141,548	37.1%
150 km	300km	37,485,971	65.8%
200 km	400km	43,658,237	76.6%

* out of 56,995,742 inhabitants in 2000 (from United nations population prospects[26]

[26] http://www.un.org/esa/population/unpop.htm (accessed on 12/03/2013)

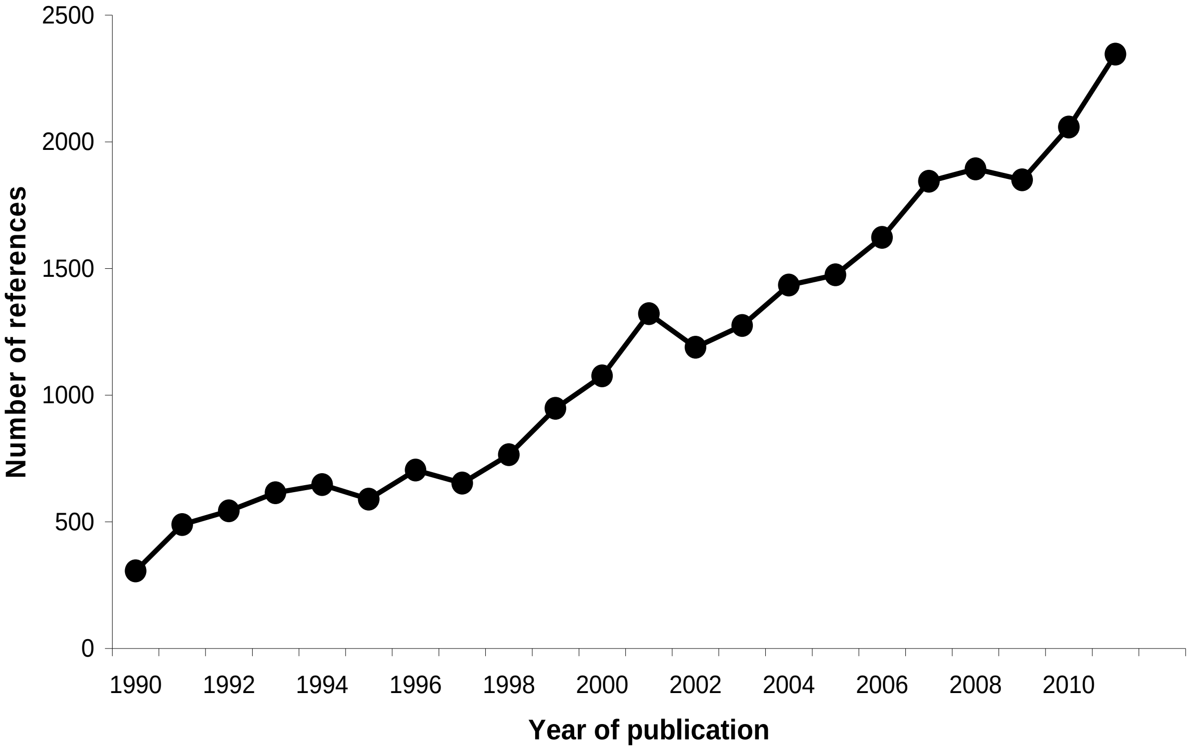


Figure 2.1: Number of publications per year on “Air Pollution” (MeSH term) referenced in *PubMed* since 1990.

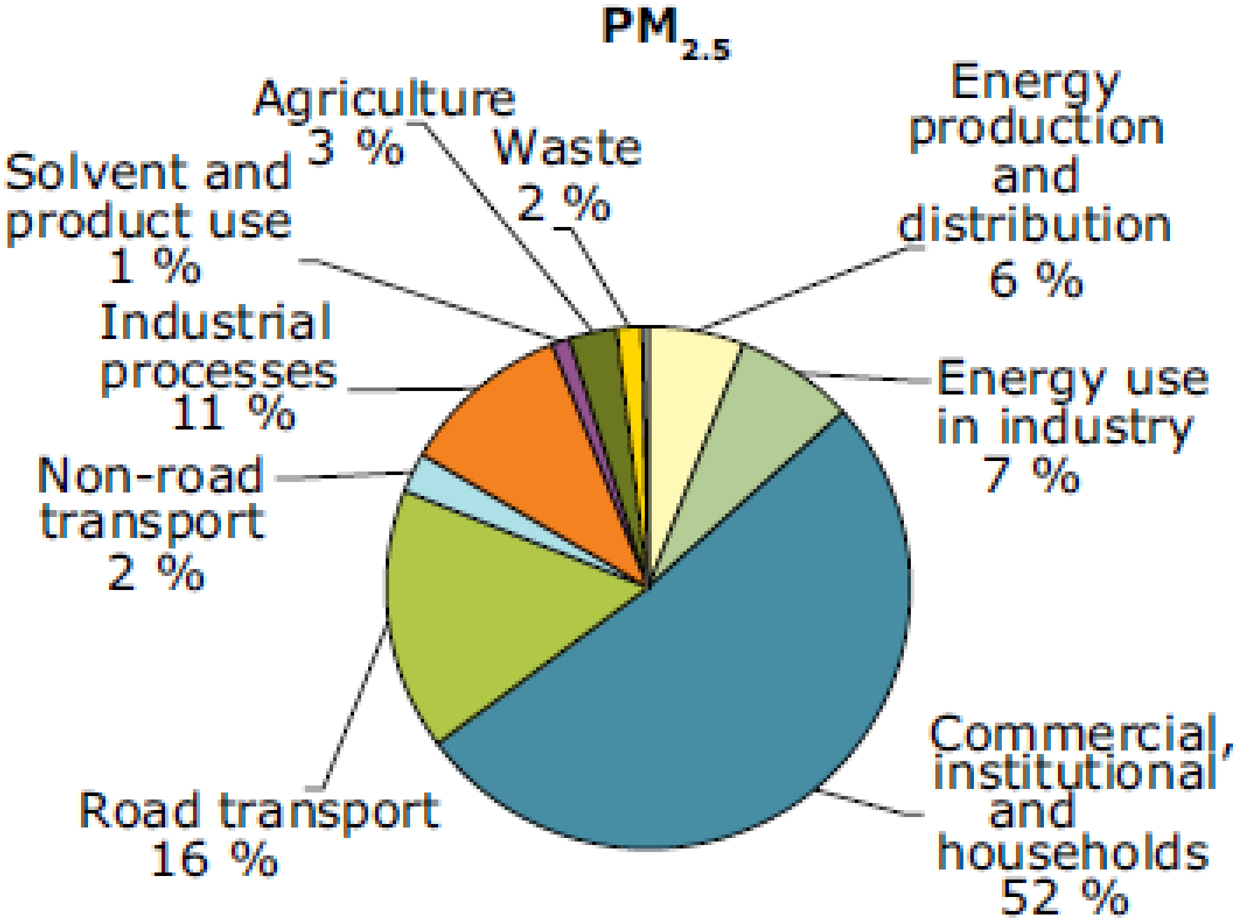


Figure 2.2: share of EU-27 emissions of $PM_{2.5}$ by sector group, 2010 (from EMEP/EEA, 2009)

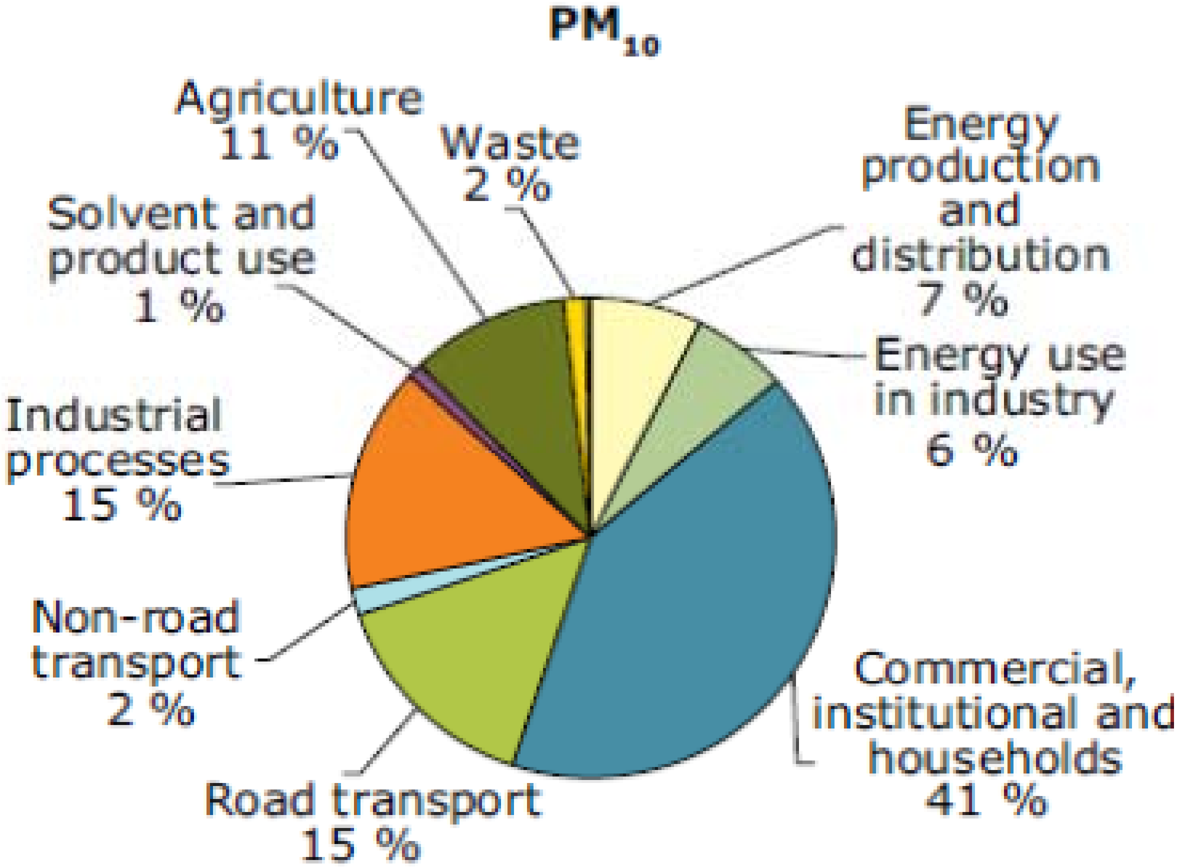


Figure 2.3: share of EU-27 emissions of PM_{10} by sector group, 2010 (from EMEP/EEA, 2009)

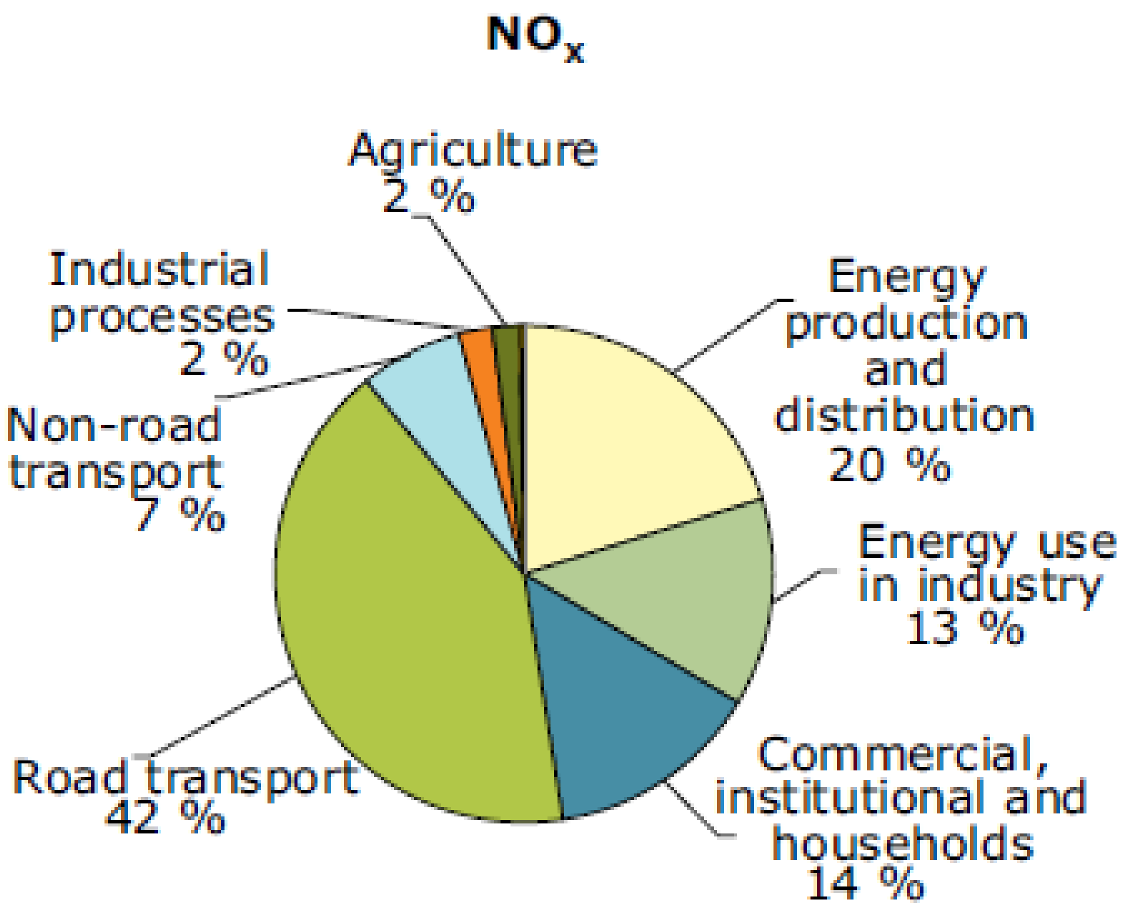


Figure 2.4: share of EU-27 emissions of NO_x by sector group, 2010 (from EMEP/EEA, 2009)

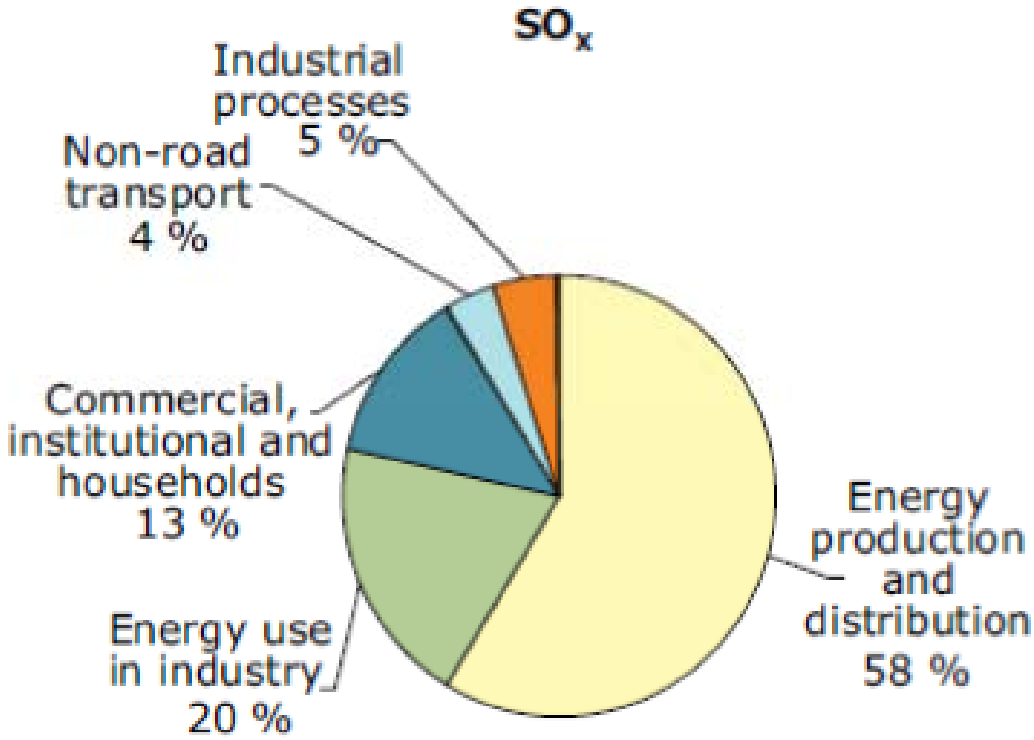
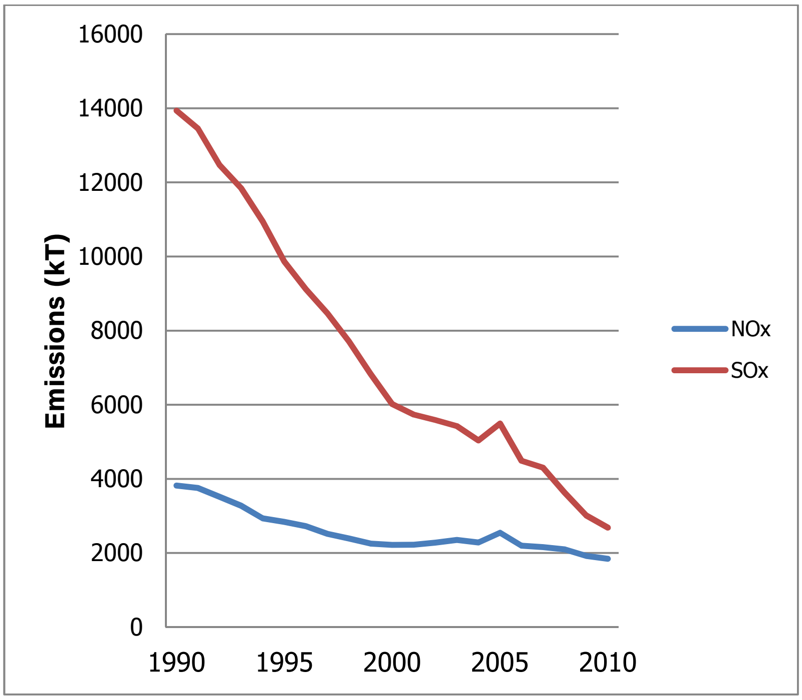


Figure 2.5: share of EU-27 emissions of SO_x by sector group, 2010 (from EMEP/EEA, 2009)



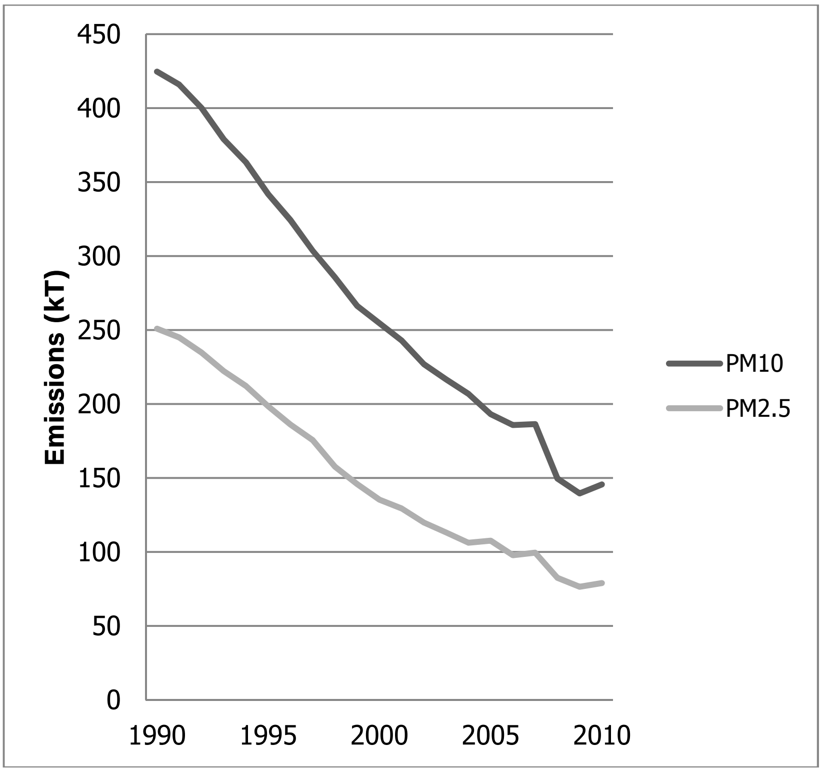


Figure 2.6: EU-27 emissions (kT) from Energy production and distribution sector for NO_x, SO_x, $PM_{2.5}$ and PM_{10}, 1990-2010[27]

[27] Data retrieved from http://www.eea.europa.eu/data-and-maps/data/data-viewers/air-emissions-viewer-lrtap (accessed 28/02/2013)

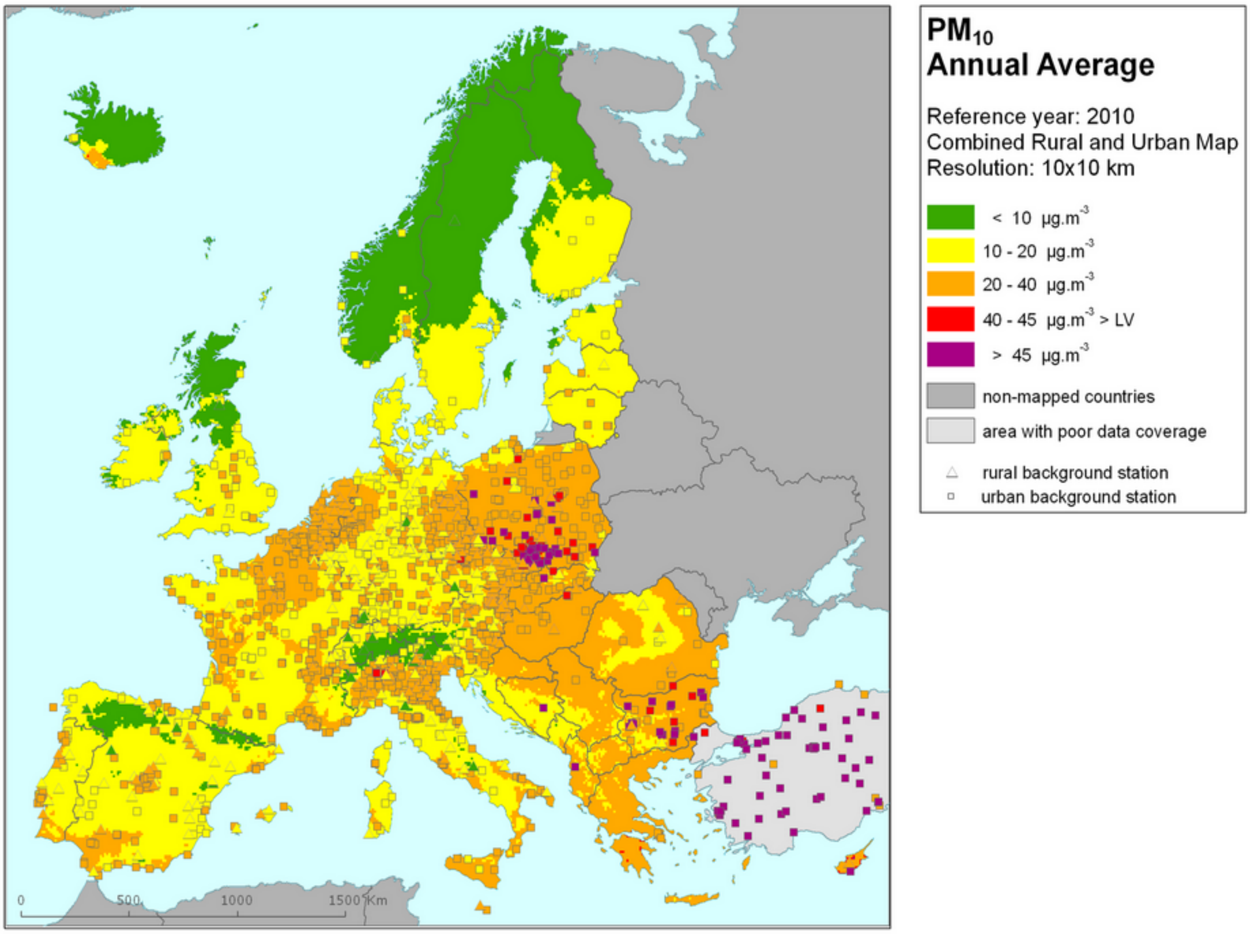


Figure 3.1: Air pollution with PM_{10} in Europe, 2010 annual average. Source: http://www.eea.europa.eu/data-and-maps/figures/pm10-annual-average-2010 (accessed on 17/04/2013)

Figure 3.2: Map of Italy with location of ENEL's coal-fired power plants included in SOMO report (note: there are actually two coal-fired power plants at the north-easternmost point). Each grey dot represents the centroids of geographical units of EMEP grid.

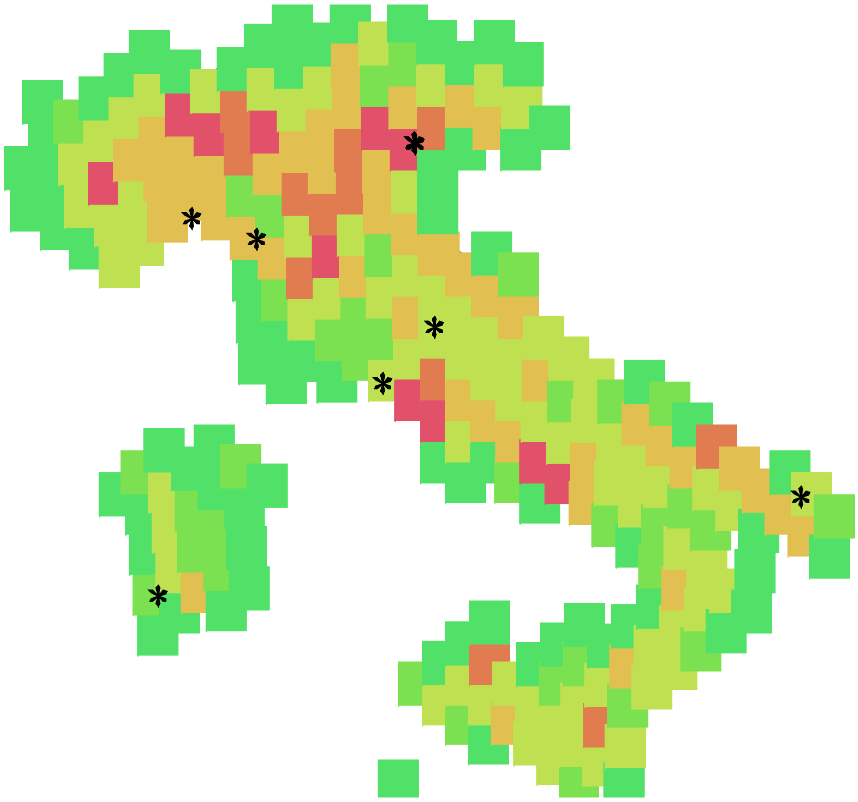


Figure 3.3: Map of population density from EMEP grid

Colour scale: from dark green (less than 50,000 inhabitants), green (50,000 to 100,000 inhabitants), light green (100,000 to 250,000 inhabitants), light orange (250,000 to 500,000 inhabitants), orange (500,000 to 800,000 inhabitants) to red (more than 800,000 inhabitants). Stars represent coal-fired power plants investigated in the SOMO report.

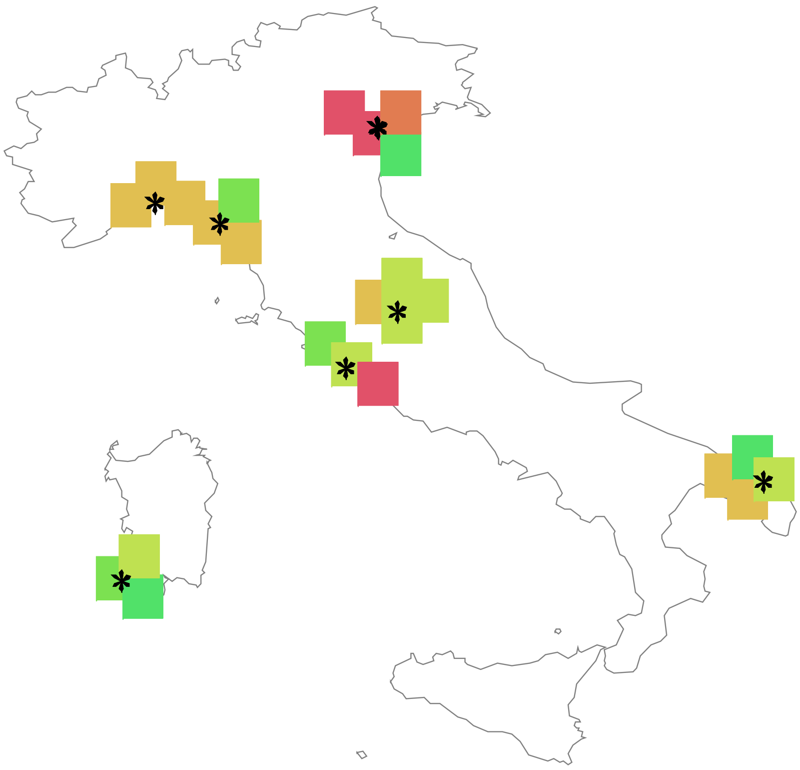


Figure 3.4: Map of population density living within 50km of a coal power plant* from EMEP grid.

Colour scale: from dark green (less than 50,000 inhabitants), green (50,000 to 100,000 inhabitants), light green (100,000 to 250,000 inhabitants), light orange (250,000 to 500,000 inhabitants), orange (500,000 to 800,000 inhabitants) to red (more than 800,000 inhabitants).

* investigated in SOMO report

a/

b/

c/

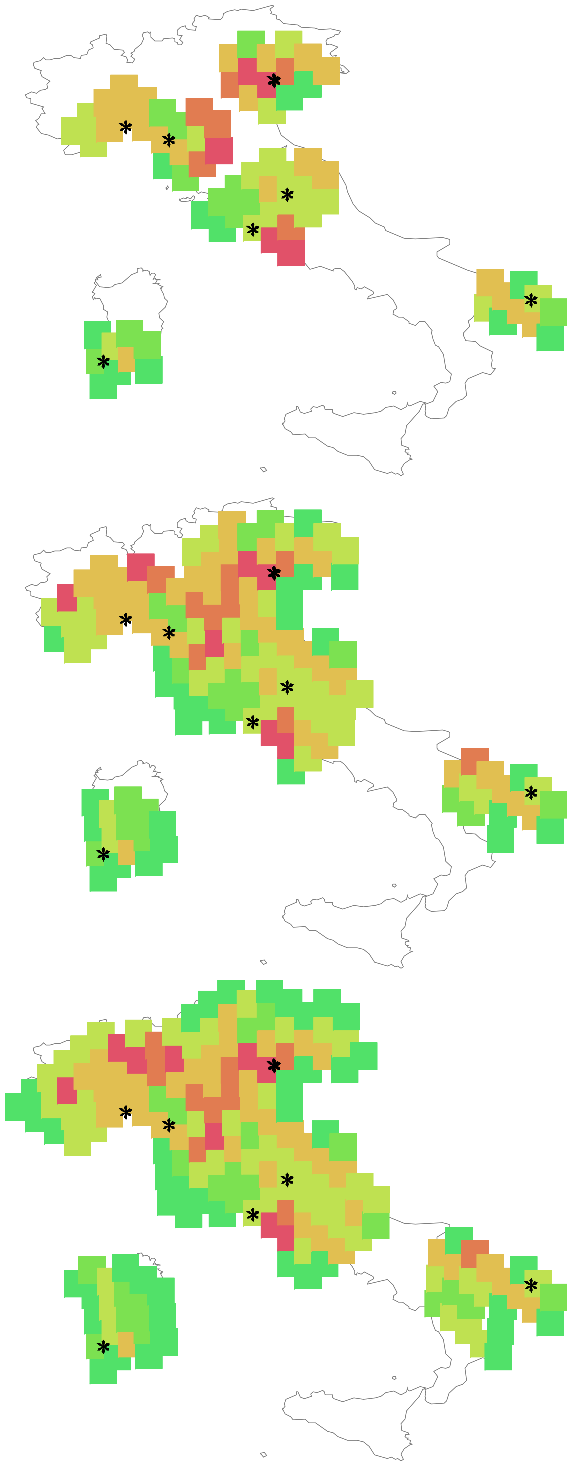


Figure 3.5: Maps of population density living within 100km(a), 150km(b), and 200km(c) of a coal power plant* from EMEP grid.

Colour scale: from dark green (less than 50,000 inhabitants), green (50,000 to 100,000 inhabitants), light green (100,000 to 250,000 inhabitants), light orange (250,000 to 500,000 inhabitants), orange (500,000 to 800,000 inhabitants) to red (more than 800,000 inhabitants) * investigated in SOMO report

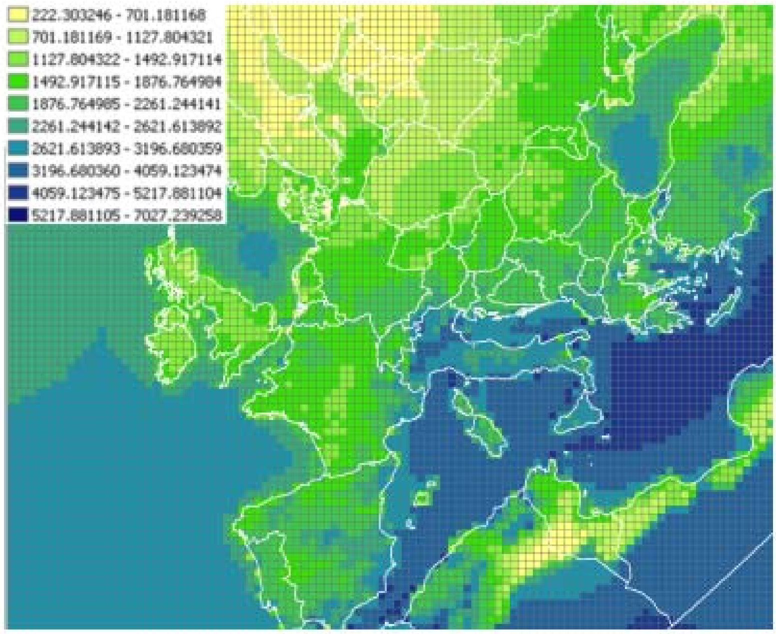


Figure 3.6: Map of the concentration of ozone over all Europe expressed by SOMO35 (http://ecosenseweb.ier.uni-stuttgart.de/concentration.html, accessed on 17/04/2013)

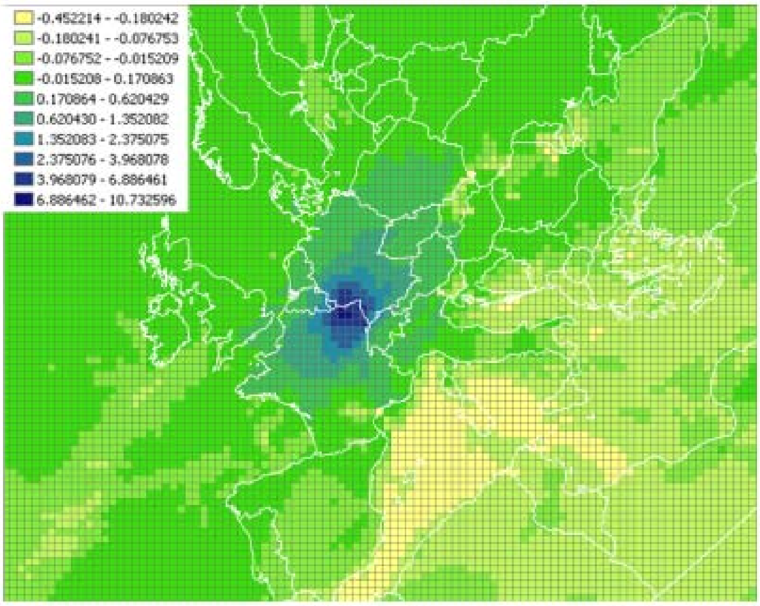


Figure 3.7: Map of the change in concentration of SOMO35 over Europe associated with a power station in France on the border with Luxembourg (http://ecosenseweb.ier.uni-stuttgart.de/concentration.html accessed on 17/04/2013)

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I. Appendix 1: DIRECTIVE 2008/50/EC OF THE EUROPEAN PARLIAMENT AND OF THE COUNCIL of 21 May 2008 on ambient air quality and cleaner air for Europe

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THE EUROPEAN PARLIAMENT AND THE COUNCIL OF THE EUROPEAN UNION,

Having regard to the Treaty establishing the European Community, and in particular Article 175 thereof,

Having regard to the proposal from the Commission,
Having regard to the opinion of the European Economic and Social Committee [1],
Having regard to the opinion of the Committee of the Regions [2],
Acting in accordance with the procedure laid down in Article 251 of the Treaty [3],

Whereas:

(1) The Sixth Community Environment Action Programme adopted by Decision No 1600/2002/EC of the European Parliament and of the Council of 22 July 2002 [4] establishes the need to reduce pollution to levels which minimise harmful effects on human health, paying particular attention to sensitive populations, and the environment as a whole, to improve the monitoring and assessment of air quality including the deposition of pollutants and to provide information to the public.
(2) In order to protect human health and the environment as a whole, it is particularly important to combat emissions of pollutants at source and to identify and implement the most effective emission reduction measures at local, national and Community level. Therefore, emissions of harmful air pollutants should be avoided, prevented or reduced and appropriate objectives set for ambient air quality taking into account relevant World Health Organisation standards, guidelines and programmes.
(3) Council Directive 96/62/EC of 27 September 1996 on ambient air quality assessment and management [5], Council Directive 1999/30/EC of 22 April 1999 relating to limit values for sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter and lead in ambient air [6], Directive 2000/69/EC of the European Parliament and of the Council of 16 November 2000 relating to limit values for benzene and carbon monoxide in ambient air [7], Directive 2002/3/EC of the European Parliament and of the Council of 12 February 2002 relating to ozone in ambient air [8] and Council Decision 97/101/EC of 27 January 1997 establishing a reciprocal exchange of information and data from networks and individual stations measuring ambient air pollution within the Member States [9] need to be substantially revised in order to incorporate the latest health and scientific developments and the experience of the Member States. In the interests of clarity, simplification and administrative efficiency it is therefore appropriate that those five acts be replaced by a single Directive and, where appropriate, by implementing measures.
(4) Once sufficient experience has been gained in relation to the implementation of Directive 2004/107/EC of the European Parliament and of the Council of 15 December 2004 relating to arsenic, cadmium, mercury, nickel and polycyclic aromatic hydrocarbons in ambient air

[10] consideration may be given to the possibility of merging its provisions with those of this Directive.

(5) A common approach to the assessment of ambient air quality should be followed according to common assessment criteria. When assessing ambient air quality, account should be taken of the size of populations and ecosystems exposed to air pollution. It is therefore appropriate to classify the territory of each Member State into zones or agglomerations reflecting the population density.

(6) Where possible modelling techniques should be applied to enable point data to be interpreted in terms of geographical distribution of concentration. This could serve as a basis for calculating the collective exposure of the population living in the area.

(7) In order to ensure that the information collected on air pollution is sufficiently representative and comparable across the Community, it is important that standardised measurement techniques and common criteria for the number and location of measuring stations are used for the assessment of ambient air quality. Techniques other than measurements can be used to assess ambient air quality and it is therefore necessary to define criteria for the use and required accuracy of such techniques.

(8) Detailed measurements of fine particulate matter at rural background locations should be made in order to understand better the impacts of this pollutant and to develop appropriate policies. Such measurements should be made in a manner consistent with those of the cooperative programme for monitoring and evaluation of the long range transmission of air pollutants in Europe (EMEP) set up under the 1979 Convention on Long-range Transboundary Air Pollution approved by Council Decision 81/462/EEC of 11 June 1981 [11].

(9) Air quality status should be maintained where it is already good, or improved. Where the objectives for ambient air quality laid down in this Directive are not met, Member States should take action in order to comply with the limit values and critical levels, and where possible, to attain the target values and long-term objectives.

(10) The risk posed by air pollution to vegetation and natural ecosystems is most important in places away from urban areas. The assessment of such risks and the compliance with critical levels for the protection of vegetation should therefore focus on places away from built-up areas.

(11) Fine particulate matter ($PM_{2,5}$) is responsible for significant negative impacts on human health. Further, there is as yet no identifiable threshold below which $PM_{2,5}$ would not pose a risk. As such, this pollutant should not be regulated in the same way as other air pollutants. The approach should aim at a general reduction of concentrations in the urban background to ensure that large sections of the population benefit from improved air quality. However, to ensure a minimum degree of health protection everywhere, that approach should be combined with a limit value, which is to be preceded in a first stage by a target value.

(12) The existing target values and long-term objectives of ensuring effective protection against harmful effects on human health and vegetation and ecosystems from exposure to ozone should remain unchanged. An alert threshold and an information threshold for ozone should be set for the protection of the general population and sensitive sections, respectively, from brief exposures to elevated ozone concentrations. Those thresholds should trigger the dissemination of information to the public on the risks of exposure and the implementation, if appropriate, of short-term measures to reduce ozone levels where the alert threshold is exceeded.

(13) Ozone is a transboundary pollutant formed in the atmosphere from the emission of primary pollutants addressed by Directive 2001/81/EC of the European Parliament and of the Council of 23 October 2001 on national emission ceilings for certain atmospheric pollutants [12]. Progress towards the air quality targets and long term objectives for ozone set in this Directive should be determined by the targets and emission ceilings provided for

in Directive 2001/81/EC and, if appropriate, by implementing air quality plans as provided for in this Directive.
(14) Fixed measurements should be mandatory in zones and agglomerations where the long-term objectives for ozone or the assessment thresholds for other pollutants are exceeded. Information from fixed measurements may be supplemented by modelling techniques and/or indicative measurements to enable point data to be interpreted in terms of geographical distribution of concentrations. The use of supplementary techniques of assessment should also allow for reduction of the required minimum number of fixed sampling points.
(15) Contributions from natural sources can be assessed but cannot be controlled. Therefore, where natural contributions to pollutants in ambient air can be determined with sufficient certainty, and where exceedances are due in whole or in part to these natural contributions, these may, under the conditions laid down in this Directive, be subtracted when assessing compliance with air quality limit values. Contributions to exceedances of particulate matter PM_{10} limit values attributable to winter-sanding or -salting of roads may also be subtracted when assessing compliance with air quality limit values provided that reasonable measures have been taken to lower concentrations.
(16) For zones and agglomerations where conditions are particularly difficult, it should be possible to postpone the deadline for compliance with the air quality limit values in cases where, notwithstanding the implementation of appropriate pollution abatement measures, acute compliance problems exist in specific zones and agglomerations. Any postponement for a given zone or agglomeration should be accompanied by a comprehensive plan to be assessed by the Commission to ensure compliance by the revised deadline. The availability of necessary Community measures reflecting the chosen ambition level in the Thematic Strategy on air pollution to reduce emissions at source will be important for an effective emission reduction by the timeframe established in this Directive for compliance with the limit values and should be taken into account when assessing requests to postpone deadlines for compliance.
(17) The necessary Community measures to reduce emissions at source, in particular measures to improve the effectiveness of Community legislation on industrial emissions, to limit the exhaust emissions of engines installed in heavy duty vehicles, to further reduce the Member States' permitted national emissions of key pollutants and the emissions associated with refuelling of petrol cars at service stations, and to address the sulphur content of fuels including marine fuels should be duly examined as a priority by all institutions involved.
(18) Air quality plans should be developed for zones and agglomerations within which concentrations of pollutants in ambient air exceed the relevant air quality target values or limit values, plus any temporary margins of tolerance, where applicable. Air pollutants are emitted from many different sources and activities. To ensure coherence between different policies, such air quality plans should where feasible be consistent, and integrated with plans and programmes prepared pursuant to Directive 2001/80/EC of the European Parliament and of the Council of 23 October 2001 on the limitation of emissions of certain pollutants into the air from large combustion plants [13], Directive 2001/81/EC, and Directive 2002/49/EC of the European Parliament and of the Council of 25 June 2002 relating to the assessment and management of environmental noise [14]. Full account will also be taken of the ambient air quality objectives provided for in this Directive, where permits are granted for industrial activities pursuant to Directive 2008/1/EC of the European Parliament and of the Council of 15 January 2008 concerning integrated pollution prevention and control [15].
(19) Action plans should be drawn up indicating the measures to be taken in the short term where there is a risk of an exceedance of one or more alert thresholds in order to reduce that risk and to limit its duration. When the risk applies to one or more limit values or target

values, Member States may, where appropriate, draw up such short-term action plans. In respect of ozone, such short-term action plans should take into account the provisions of Commission Decision 2004/279/EC of 19 March 2004 concerning guidance for implementation of Directive 2002/3/EC of the European Parliament and of the Council relating to ozone in ambient air [16].

(20) Member States should consult with one another if, following significant pollution originating in another Member State, the level of a pollutant exceeds, or is likely to exceed, the relevant air quality objectives plus the margin of tolerance where applicable or, as the case may be, the alert threshold. The transboundary nature of specific pollutants, such as ozone and particulate matter, may require coordination between neighbouring Member States in drawing up and implementing air quality plans and short-term action plans and in informing the public. Where appropriate, Member States should pursue cooperation with third countries, with particular emphasis on the early involvement of candidate countries.

(21) It is necessary for the Member States and the Commission to collect, exchange and disseminate air quality information in order to understand better the impacts of air pollution and develop appropriate policies. Up-to-date information on concentrations of all regulated pollutants in ambient air should also be readily available to the public.

(22) In order to facilitate the handling and comparison of air quality information, data should be made available to the Commission in a standardised form.

(23) It is necessary to adapt procedures for data provision, assessment and reporting of air quality to enable electronic means and the Internet to be used as the main tools to make information available, and so that such procedures are compatible with Directive 2007/2/EC of the European Parliament and the Council of 14 March 2007 establishing an infrastructure for spatial information in the European Community (INSPIRE) [17].

(24) It is appropriate to provide for the possibility of adapting the criteria and techniques used for the assessment of the ambient air quality to scientific and technical progress and adapting thereto the information to be provided.

(25) Since the objectives of this Directive cannot be sufficiently achieved by the Member States by reason of the transboundary nature of air pollutants and can therefore be better achieved at Community level, the Community may adopt measures, in accordance with the principle of subsidiarity as set out in Article 5 of the Treaty. In accordance with the principle of proportionality, as set out in that Article, this Directive does not go beyond what is necessary in order to achieve those objectives.

(26) Member States should lay down rules on penalties applicable to infringements of the provisions of this Directive and ensure that they are implemented. The penalties should be effective, proportionate and dissuasive.

(27) Certain provisions of the acts repealed by this Directive should remain in force in order to ensure the continuance of existing air quality limits for nitrogen dioxide until they are replaced from 1 January 2010, the continuance of air quality reporting provisions until new implementing measures are adopted, and the continuance of obligations relating to the preliminary assessments of air quality required under Directive 2004/107/EC.

(28) The obligation to transpose this Directive into national law should be confined to those provisions which represent a substantive change as compared with the earlier Directives.

(29) In accordance with point 34 of the Interinstitutional Agreement on better lawmaking [18], Member States are encouraged to draw up, for themselves and in the interest of the Community, their own tables illustrating, as far as possible, the correlation between the Directive and the transposition measures, and to make them public.

(30) This Directive respects the fundamental rights and observes the principles recognised in particular by the Charter of Fundamental Rights of the European Union. In particular, this Directive seeks to promote the integration into the policies of the Union of a high level of

environmental protection and the improvement of the quality of the environment in accordance with the principle of sustainable development as laid down in Article 37 of the Charter of Fundamental Rights of the European Union.
(31) The measures necessary for the implementation of this Directive should be adopted in accordance with Council Decision 1999/468/EC of 28 June 1999 laying down the procedures for the exercise of implementing powers conferred on the Commission [19].
(32) The Commission should be empowered to amend Annexes I to VI, Annexes VIII to X and Annex XV. Since those measures are of general scope and are designed to amend non-essential elements of this Directive, they must be adopted in accordance with the regulatory procedure with scrutiny provided for in Article 5a of Decision 1999/468/EC.
(33) The transposition clause requires Member States to ensure that the necessary urban background measurements are in place well in time to define the Average Exposure Indicator, in order to guarantee that the requirements related to the assessment of the National Exposure Reduction Target and to the calculation of the Average Exposure Indicator are met,

HAVE ADOPTED THIS DIRECTIVE:

CHAPTER I
GENERAL PROVISIONS

Article 1
Subject matter

This Directive lays down measures aimed at the following:

1. defining and establishing objectives for ambient air quality designed to avoid, prevent or reduce harmful effects on human health and the environment as a whole;
2. assessing the ambient air quality in Member States on the basis of common methods and criteria;
3. obtaining information on ambient air quality in order to help combat air pollution and nuisance and to monitor long-term trends and improvements resulting from national and Community measures;
4. ensuring that such information on ambient air quality is made available to the public;
5. maintaining air quality where it is good and improving it in other cases;
6. promoting increased cooperation between the Member States in reducing air pollution.

Article 2
Definitions

For the purposes of this Directive:

1. "ambient air" shall mean outdoor air in the troposphere, excluding workplaces as defined by Directive 89/654/EEC [20] where provisions concerning health and safety at work apply and to which members of the public do not have regular access;
2. "pollutant" shall mean any substance present in ambient air and likely to have harmful effects on human health and/or the environment as a whole;
3. "level" shall mean the concentration of a pollutant in ambient air or the deposition thereof on surfaces in a given time;

4. "assessment" shall mean any method used to measure, calculate, predict or estimate levels;
5. "limit value" shall mean a level fixed on the basis of scientific knowledge, with the aim of avoiding, preventing or reducing harmful effects on human health and/or the environment as a whole, to be attained within a given period and not to be exceeded once attained;
6. "critical level" shall mean a level fixed on the basis of scientific knowledge, above which direct adverse effects may occur on some receptors, such as trees, other plants or natural ecosystems but not on humans;
7. "margin of tolerance" shall mean the percentage of the limit value by which that value may be exceeded subject to the conditions laid down in this Directive;
8. "air quality plans" shall mean plans that set out measures in order to attain the limit values or target values;
9. "target value" shall mean a level fixed with the aim of avoiding, preventing or reducing harmful effects on human health and/or the environment as a whole, to be attained where possible over a given period;
10. "alert threshold" shall mean a level beyond which there is a risk to human health from brief exposure for the population as a whole and at which immediate steps are to be taken by the Member States;
11. "information threshold" shall mean a level beyond which there is a risk to human health from brief exposure for particularly sensitive sections of the population and for which immediate and appropriate information is necessary;
12. "upper assessment threshold" shall mean a level below which a combination of fixed measurements and modelling techniques and/or indicative measurements may be used to assess ambient air quality;
13. "lower assessment threshold" shall mean a level below which modelling or objective-estimation techniques alone may be used to assess ambient air quality;
14. "long-term objective" shall mean a level to be attained in the long term, save where not achievable through proportionate measures, with the aim of providing effective protection of human health and the environment;
15. "contributions from natural sources" shall mean emissions of pollutants not caused directly or indirectly by human activities, including natural events such as volcanic eruptions, seismic activities, geothermal activities, wild-land fires, high-wind events, sea sprays or the atmospheric re-suspension or transport of natural particles from dry regions;
16. "zone" shall mean part of the territory of a Member State, as delimited by that Member State for the purposes of air quality assessment and management;
17. "agglomeration" shall mean a zone that is a conurbation with a population in excess of 250000 inhabitants or, where the population is 250000 inhabitants or less, with a given population density per km^2 to be established by the Member States;
18. "PM_{10}" shall mean particulate matter which passes through a size-selective inlet as defined in the reference method for the sampling and measurement of PM10, EN 12341, with a 50 % efficiency cut-off at 10 μm aerodynamic diameter;
19. "$PM_{2,5}$" shall mean particulate matter which passes through a size-selective inlet as defined in the reference method for the sampling and measurement of PM2,5, EN 14907, with a 50 % efficiency cut-off at 2,5 μm aerodynamic diameter;
20. "average exposure indicator" shall mean an average level determined on the basis of measurements at urban background locations throughout the territory of a Member State and which reflects population exposure. It is used to calculate the national exposure reduction target and the exposure concentration obligation;

21. "exposure concentration obligation" shall mean a level fixed on the basis of the average exposure indicator with the aim of reducing harmful effects on human health, to be attained over a given period;
22. "national exposure reduction target" shall mean a percentage reduction of the average exposure of the population of a Member State set for the reference year with the aim of reducing harmful effects on human health, to be attained where possible over a given period;
23. "urban background locations" shall mean places in urban areas where levels are representative of the exposure of the general urban population;
24. "oxides of nitrogen" shall mean the sum of the volume mixing ratio (ppbv) of nitrogen monoxide (nitric oxide) and nitrogen dioxide expressed in units of mass concentration of nitrogen dioxide (µg/m^3);
25. "fixed measurements" shall mean measurements taken at fixed sites, either continuously or by random sampling, to determine the levels in accordance with the relevant data quality objectives;
26. "indicative measurements" shall mean measurements which meet data quality objectives that are less strict than those required for fixed measurements;
27. "volatile organic compounds" (VOC) shall mean organic compounds from anthropogenic and biogenic sources, other than methane, that are capable of producing photochemical oxidants by reactions with nitrogen oxides in the presence of sunlight;
28. "ozone precursor substances" means substances which contribute to the formation of ground-level ozone, some of which are listed in Annex X.

Article 3
Responsibilities

Member States shall designate at the appropriate levels the competent authorities and bodies responsible for the following:

(a) assessment of ambient air quality;

(b) approval of measurement systems (methods, equipment, networks and laboratories);

(c) ensuring the accuracy of measurements;

(d) analysis of assessment methods;

(e) coordination on their territory if Community-wide quality assurance programmes are being organised by the Commission;

(f) cooperation with the other Member States and the Commission.

Where relevant, the competent authorities and bodies shall comply with Section C of Annex I.

Article 4
Establishment of zones and agglomerations

Member States shall establish zones and agglomerations throughout their territory. Air quality assessment and air quality management shall be carried out in all zones and agglomerations.

CHAPTER II
ASSESSMENT OF AMBIENT AIR QUALITY

SECTION 1
Assessment of ambient air quality in relation to sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter, lead, benzene and carbon monoxide

Article 5
Assessment regime

1. The upper and lower assessment thresholds specified in Section A of Annex II shall apply to sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter (PM_{10} and $PM_{2,5}$), lead, benzene and carbon monoxide.
Each zone and agglomeration shall be classified in relation to those assessment thresholds.

2. The classification referred to in paragraph 1 shall be reviewed at least every five years in accordance with the procedure laid down in Section B of Annex II.
However, classifications shall be reviewed more frequently in the event of significant changes in activities relevant to the ambient concentrations of sulphur dioxide, nitrogen dioxide or, where relevant, oxides of nitrogen, particulate matter (PM_{10}, $PM_{2,5}$), lead, benzene or carbon monoxide.

Article 6
Assessment criteria

1. Member States shall assess ambient air quality with respect to the pollutants referred to in Article 5 in all their zones and agglomerations, in accordance with the criteria laid down in paragraphs 2, 3 and 4 of this Article and in accordance with the criteria laid down in Annex III.

2. In all zones and agglomerations where the level of pollutants referred to in paragraph 1 exceeds the upper assessment threshold established for those pollutants, fixed measurements shall be used to assess the ambient air quality. Those fixed measurements may be supplemented by modelling techniques and/or indicative measurements to provide adequate information on the spatial distribution of the ambient air quality.

3. In all zones and agglomerations where the level of pollutants referred to in paragraph 1 is below the upper assessment threshold established for those pollutants, a combination of fixed measurements and modelling techniques and/or indicative measurements may be used to assess the ambient air quality.

4. In all zones and agglomerations where the level of pollutants referred to in paragraph 1 is below the lower assessment threshold established for those pollutants, modelling techniques or objective-estimation techniques or both shall be sufficient for the assessment of the ambient air quality.

5. In addition to the assessments referred to in paragraphs 2, 3 and 4, measurements shall be made, at rural background locations away from significant sources of air pollution, for the purposes of providing, as a minimum, information on the total mass concentration and the chemical speciation concentrations of fine particulate matter ($PM_{2,5}$) on an annual average basis and shall be conducted using the following criteria:

(a) one sampling point shall be installed every 100000 km^2;

(b) each Member State shall set up at least one measuring station or may, by agreement with adjoining Member States, set up one or several common measuring stations, covering the relevant neighbouring zones, to achieve the necessary spatial resolution;

(c) where appropriate, monitoring shall be coordinated with the monitoring strategy and measurement programme of the Cooperative Programme for Monitoring and Evaluation of the Long-range Transmission of Air Pollutants in Europe (EMEP);

(d) Sections A and C of Annex I shall apply in relation to the data quality objectives for mass concentration measurements of particulate matter and Annex IV shall apply in its entirety.

Member States shall inform the Commission of the measurement methods used in the measurement of the chemical composition of fine particulate matter ($PM_{2,5}$).

Article 7
Sampling points

1. The location of sampling points for the measurement of sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter (PM_{10}, $PM_{2,5}$), lead, benzene and carbon monoxide in ambient air shall be determined using the criteria listed in Annex III.

2. In each zone or agglomeration where fixed measurements are the sole source of information for assessing air quality, the number of sampling points for each relevant pollutant shall not be less than the minimum number of sampling points specified in Section A of Annex V.

3. For zones and agglomerations within which information from fixed measurement sampling points is supplemented by information from modelling and/or indicative measurement, the total number of sampling points specified in Section A of Annex V may be reduced by up to 50 %, provided that the following conditions are met:

(a) the supplementary methods provide sufficient information for the assessment of air quality with regard to limit values or alert thresholds, as well as adequate information for the public;

(b) the number of sampling points to be installed and the spatial resolution of other techniques are sufficient for the concentration of the relevant pollutant to be established in accordance with the data quality objectives specified in Section A of Annex I and enable assessment results to meet the criteria specified in Section B of Annex I.

The results of modelling and/or indicative measurement shall be taken into account for the assessment of air quality with respect to the limit values.

4. The application in Member States of the criteria for selecting sampling points shall be monitored by the Commission so as to facilitate the harmonised application of those criteria throughout the European Union.

Article 8
Reference measurement methods

1. Member States shall apply the reference measurement methods and criteria specified in Section A and Section C of Annex VI.

2. Other measurement methods may be used subject to the conditions set out in Section B of Annex VI.

SECTION 2
Assessment of ambient air quality in relation to ozone

Article 9
Assessment criteria

1. Where, in a zone or agglomeration, concentrations of ozone have exceeded the long-term objectives specified in Section C of Annex VII during any of the previous five years of measurement, fixed measurements shall be taken.

2. Where fewer than five years' data are available, Member States may, for the purposes of determining whether the long-term objectives referred to in paragraph 1 have been exceeded during those five years, combine the results from measurement campaigns of short duration carried out when and where levels are likely to be at their highest, with the results obtained from emission inventories and modelling.

Article 10
Sampling points

1. The siting of sampling points for the measurement of ozone shall be determined using the criteria set out in Annex VIII.

2. The sampling points for fixed measurements of ozone in each zone or agglomeration within which measurement is the sole source of information for assessing air quality shall not be less than the minimum number of sampling points specified in Section A of Annex IX.

3. For zones and agglomerations within which information from sampling points for fixed measurements is supplemented by information from modelling and/or indicative measurements, the number of sampling points specified in Section A of Annex IX may be reduced provided that the following conditions are met:

(a) the supplementary methods provide sufficient information for the assessment of air quality with regard to target values, long-term objectives, information and alert thresholds;

(b) the number of sampling points to be installed and the spatial resolution of other techniques are sufficient for the concentration of ozone to be established in accordance with the data quality objectives specified in Section A of Annex I and enable assessment results to meet the criteria specified in Section B of Annex I;

(c) the number of sampling points in each zone or agglomeration amounts to at least one sampling point per two million inhabitants or one sampling point per 50000 km^2, whichever produces the greater number of sampling points, but must not be less than one sampling point in each zone or agglomeration;

(d) nitrogen dioxide is measured at all remaining sampling points except at rural background stations as referred to in Section A of Annex VIII.

The results of modelling and/or indicative measurement shall be taken into account for the assessment of air quality with respect to the target values.

4. Nitrogen dioxide shall be measured at a minimum of 50 % of the ozone sampling points required under Section A of Annex IX. That measurement shall be continuous except at rural background stations, as referred to in Section A of Annex VIII, where other measurement methods may be used.

5. In zones and agglomerations where, during each of the previous five years of measurement, concentrations are below the long-term objectives, the number of sampling points for fixed measurements shall be determined in accordance with Section B of Annex IX.

6. Each Member State shall ensure that at least one sampling point is installed and operated in its territory to supply data on concentrations of the ozone precursor substances listed in Annex X. Each Member State shall choose the number and siting of the stations at which ozone precursor substances are to be measured, taking into account the objectives and methods laid down in Annex X.

Article 11
Reference measurement methods

1. Member States shall apply the reference method for measurement of ozone, set out in point 8 of Section A of Annex VI. Other measuring methods may be used subject to the conditions set out in Section B of Annex VI.

2. Each Member State shall inform the Commission of the methods it uses to sample and measure VOC, as listed in Annex X.

CHAPTER III
AMBIENT AIR QUALITY MANAGEMENT

Article 12
Requirements where levels are lower than the limit values

In zones and agglomerations where the levels of sulphur dioxide, nitrogen dioxide, PM_{10}, $PM_{2,5}$, lead, benzene and carbon monoxide in ambient air are below the respective limit values specified in Annexes XI and XIV, Member States shall maintain the levels of those pollutants below the limit values and shall endeavour to preserve the best ambient air quality, compatible with sustainable development.

Article 13
Limit values and alert thresholds for the protection of human health

1. Member States shall ensure that, throughout their zones and agglomerations, levels of sulphur dioxide, PM_{10}, lead, and carbon monoxide in ambient air do not exceed the limit values laid down in Annex XI.
In respect of nitrogen dioxide and benzene, the limit values specified in Annex XI may not be exceeded from the dates specified therein.
Compliance with these requirements shall be assessed in accordance with Annex III.
The margins of tolerance laid down in Annex XI shall apply in accordance with Article 22(3) and Article 23(1).

2. The alert thresholds for concentrations of sulphur dioxide and nitrogen dioxide in ambient air shall be those laid down in Section A of Annex XII.

Article 14
Critical levels

1. Member States shall ensure compliance with the critical levels specified in Annex XIII as assessed in accordance with Section A of Annex III.

2. Where fixed measurements are the sole source of information for assessing air quality, the number of sampling points shall not be less than the minimum number specified in Section C of Annex V. Where that information is supplemented by indicative measurements or modelling, the minimum number of sampling points may be reduced by up to 50 % so long as the assessed concentrations of the relevant pollutant can be established in accordance with the data quality objectives specified in Section A of Annex I.

Article 15
National $PM_{2,5}$ exposure reduction target for the protection of human health

1. Member States shall take all necessary measures not entailing disproportionate costs to reduce exposure to $PM_{2,5}$ with a view to attaining the national exposure reduction target laid down in Section B of Annex XIV by the year specified therein.

2. Member States shall ensure that the average exposure indicator for the year 2015 established in accordance with Section A of Annex XIV does not exceed the exposure concentration obligation laid down in Section C of that Annex.

3. The average exposure indicator for $PM_{2,5}$ shall be assessed in accordance with Section A of Annex XIV.

4. Each Member State shall, in accordance with Annex III, ensure that the distribution and the number of sampling points on which the average exposure indicator for $PM_{2,5}$ is based reflect the general population exposure adequately. The number of sampling points shall be no less than that determined by application of Section B of Annex V.

Article 16
$PM_{2,5}$ target value and limit value for the protection of human health

1. Member States shall take all necessary measures not entailing disproportionate costs to ensure that concentrations of $PM_{2,5}$ in ambient air do not exceed the target value laid down in Section D of Annex XIV as from the date specified therein.

2. Member States shall ensure that concentrations of $PM_{2,5}$ in ambient air do not exceed the limit value laid down in Section E of Annex XIV throughout their zones and agglomerations as from the date specified therein. Compliance with this requirement shall be assessed in accordance with Annex III.

3. The margin of tolerance laid down in Section E of Annex XIV shall apply in accordance with Article 23(1).

Article 17
Requirements in zones and agglomerations where ozone concentrations exceed the target values and long-term objectives

1. Member States shall take all necessary measures not entailing disproportionate costs to ensure that the target values and long-term objectives are attained.

2. For zones and agglomerations in which a target value is exceeded, Member States shall ensure that the programme prepared pursuant to Article 6 of Directive 2001/81/EC and, if appropriate, an air quality plan is implemented in order to attain the target values, save where not achievable through measures not entailing disproportionate costs, as from the date specified in Section B of Annex VII to this Directive.

3. For zones and agglomerations in which the levels of ozone in ambient air are higher than the long-term objectives but below, or equal to, the target values, Member States shall prepare and implement cost-effective measures with the aim of achieving the long-term objectives. Those measures shall, at least, be consistent with all the air quality plans and the programme referred to in paragraph 2.

Article 18
Requirements in zones and agglomerations where ozone concentrations meet the long-term objectives

In zones and agglomerations in which ozone levels meet the long-term objectives, Member States shall, in so far as factors including the transboundary nature of ozone pollution and meteorological conditions permit, maintain those levels below the long-term objectives and shall preserve through proportionate measures the best ambient air quality compatible with sustainable development and a high level of environmental and human health protection.

Article 19
Measures required in the event of information or alert thresholds being exceeded

Where the information threshold specified in Annex XII or any of the alert thresholds laid down therein is exceeded, Member States shall take the necessary steps to inform the public by means of radio, television, newspapers or the Internet.
Member States shall also forward to the Commission, on a provisional basis, information concerning the levels recorded and the duration of the periods during which the alert threshold or information threshold was exceeded.

Article 20
Contributions from natural sources

1. Member States shall transmit to the Commission, for a given year, lists of zones and agglomerations where exceedances of limit values for a given pollutant are attributable to natural sources. Member States shall provide information on concentrations and sources and the evidence demonstrating that the exceedances are attributable to natural sources.

2. Where the Commission has been informed of an exceedance attributable to natural sources in accordance with paragraph 1, that exceedance shall not be considered as an exceedance for the purposes of this Directive.

3. The Commission shall by 11 June 2010 publish guidelines for demonstration and subtraction of exceedances attributable to natural sources.

Article 21
Exceedances attributable to winter-sanding or -salting of roads

1. Member States may designate zones or agglomerations within which limit values for PM_{10} are exceeded in ambient air due to the re-suspension of particulates following winter-sanding or -salting of roads.

2. Member States shall send the Commission lists of any such zones or agglomerations together with information on concentrations and sources of PM_{10} therein.

3. When informing the Commission in accordance with Article 27, Member States shall provide the necessary evidence to demonstrate that any exceedances are due to re-suspended particulates and that reasonable measures have been taken to lower the concentrations.

4. Without prejudice to Article 20, in the case of zones and agglomerations referred to in paragraph 1 of this Article, Member States need to establish the air quality plan provided for in Article 23 only in so far as exceedances are attributable to PM_{10} sources other than winter-sanding or -salting of roads.

5. The Commission shall by 11 June 2010 publish guidelines for determination of contributions from the re-suspension of particulates following winter-sanding or -salting of roads.

Article 22
Postponement of attainment deadlines and exemption from the obligation to apply certain limit values

1. Where, in a given zone or agglomeration, conformity with the limit values for nitrogen dioxide or benzene cannot be achieved by the deadlines specified in Annex XI, a Member State may postpone those deadlines by a maximum of five years for that particular zone or agglomeration, on condition that an air quality plan is established in accordance with Article 23 for the zone or agglomeration to which the postponement would apply; such air quality plan shall be supplemented by the information listed in Section B of Annex XV related to the pollutants concerned and shall demonstrate how conformity will be achieved with the limit values before the new deadline.

2. Where, in a given zone or agglomeration, conformity with the limit values for PM_{10} as specified in Annex XI cannot be achieved because of site-specific dispersion characteristics, adverse climatic conditions or transboundary contributions, a Member State shall be exempt from the obligation to apply those limit values until 11 June 2011 provided that the conditions laid down in paragraph 1 are fulfilled and that the Member State shows that all

appropriate measures have been taken at national, regional and local level to meet the deadlines.

3. Where a Member State applies paragraphs 1 or 2, it shall ensure that the limit value for each pollutant is not exceeded by more than the maximum margin of tolerance specified in Annex XI for each of the pollutants concerned.

4. Member States shall notify the Commission where, in their view, paragraphs 1 or 2 are applicable, and shall communicate the air quality plan referred to in paragraph 1 including all relevant information necessary for the Commission to assess whether or not the relevant conditions are satisfied. In its assessment, the Commission shall take into account estimated effects on ambient air quality in the Member States, at present and in the future, of measures that have been taken by the Member States as well as estimated effects on ambient air quality of current Community measures and planned Community measures to be proposed by the Commission.
Where the Commission has raised no objections within nine months of receipt of that notification, the relevant conditions for the application of paragraphs 1 or 2 shall be deemed to be satisfied.
If objections are raised, the Commission may require Member States to adjust or provide new air quality plans.

CHAPTER IV
PLANS

Article 23
Air quality plans

1. Where, in given zones or agglomerations, the levels of pollutants in ambient air exceed any limit value or target value, plus any relevant margin of tolerance in each case, Member States shall ensure that air quality plans are established for those zones and agglomerations in order to achieve the related limit value or target value specified in Annexes XI and XIV.

In the event of exceedances of those limit values for which the attainment deadline is already expired, the air quality plans shall set out appropriate measures, so that the exceedance period can be kept as short as possible. The air quality plans may additionally include specific measures aiming at the protection of sensitive population groups, including children.
Those air quality plans shall incorporate at least the information listed in Section A of Annex XV and may include measures pursuant to Article 24. Those plans shall be communicated to the Commission without delay, but no later than two years after the end of the year the first exceedance was observed.
Where air quality plans must be prepared or implemented in respect of several pollutants, Member States shall, where appropriate, prepare and implement integrated air quality plans covering all pollutants concerned.

2. Member States shall, to the extent feasible, ensure consistency with other plans required under Directive 2001/80/EC, Directive 2001/81/EC or Directive 2002/49/EC in order to achieve the relevant environmental objectives.

Article 24
Short-term action plans

1. Where, in a given zone or agglomeration, there is a risk that the levels of pollutants will exceed one or more of the alert thresholds specified in Annex XII, Member States shall draw up action plans indicating the measures to be taken in the short term in order to reduce the risk or duration of such an exceedance. Where this risk applies to one or more limit values or target values specified in Annexes VII, XI and XIV, Member States may, where appropriate, draw up such short-term action plans.
However, where there is a risk that the alert threshold for ozone specified in Section B of Annex XII will be exceeded, Member States shall only draw up such short-term action plans when in their opinion there is a significant potential, taking into account national geographical, meteorological and economic conditions, to reduce the risk, duration or severity of such an exceedance. When drawing up such a short-term action plan Member States shall take account of Decision 2004/279/EC.

2. The short-term action plans referred to in paragraph 1 may, depending on the individual case, provide for effective measures to control and, where necessary, suspend activities which contribute to the risk of the respective limit values or target values or alert threshold being exceeded. Those action plans may include measures in relation to motor-vehicle traffic, construction works, ships at berth, and the use of industrial plants or products and domestic heating. Specific actions aiming at the protection of sensitive population groups, including children, may also be considered in the framework of those plans.

3. When Member States have drawn up a short-term action plan, they shall make available to the public and to appropriate organisations such as environmental organisations, consumer organisations, organisations representing the interests of sensitive population groups, other relevant health-care bodies and the relevant industrial federations both the results of their investigations on the feasibility and the content of specific short-term action plans as well as information on the implementation of these plans.

4. For the first time before 11 June 2010 and at regular intervals thereafter, the Commission shall publish examples of best practices for the drawing-up of short-term action plans, including examples of best practices for the protection of sensitive population groups, including children.

Article 25
Trans-boundary air pollution

1. Where any alert threshold, limit value or target value plus any relevant margin of tolerance or long-term objective is exceeded due to significant trans-boundary transport of air pollutants or their precursors, the Member States concerned shall cooperate and, where appropriate, draw up joint activities, such as the preparation of joint or coordinated air quality plans pursuant to Article 23 in order to remove such exceedances through the application of appropriate but proportionate measures.

2. The Commission shall be invited to be present and to assist in any cooperation referred to in paragraph 1. Where appropriate, the Commission shall, taking into account the reports established pursuant to Article 9 of Directive 2001/81/EC, consider whether further action

should be taken at Community level in order to reduce precursor emissions responsible for trans-boundary pollution.

3. Member States shall, if appropriate pursuant to Article 24, prepare and implement joint short-term action plans covering neighbouring zones in other Member States. Member States shall ensure that neighbouring zones in other Member States which have developed short-term action plans receive all appropriate information.

4. Where the information threshold or alert thresholds are exceeded in zones or agglomerations close to national borders, information shall be provided as soon as possible to the competent authorities in the neighbouring Member States concerned. That information shall also be made available to the public.

5. In drawing up plans as provided for in paragraphs 1 and 3 and in informing the public as referred to in paragraph 4, Member States shall, where appropriate, endeavour to pursue cooperation with third countries, and in particular with candidate countries.

CHAPTER V
INFORMATION AND REPORTING

Article 26
Public information

1. Member States shall ensure that the public as well as appropriate organisations such as environmental organisations, consumer organisations, organisations representing the interests of sensitive populations, other relevant health-care bodies and the relevant industrial federations are informed, adequately and in good time, of the following:

(a) ambient air quality in accordance with Annex XVI;

(b) any postponement decisions pursuant to Article 22(1);

(c) any exemptions pursuant to Article 22(2);

(d) air quality plans as provided for in Article 22(1) and Article 23 and programmes referred to in Article 17(2).

The information shall be made available free of charge by means of any easily accessible media including the Internet or any other appropriate means of telecommunication, and shall take into account the provisions laid down in Directive 2007/2/EC.

2. Member States shall make available to the public annual reports for all pollutants covered by this Directive.
Those reports shall summarise the levels exceeding limit values, target values, long-term objectives, information thresholds and alert thresholds, for the relevant averaging periods. That information shall be combined with a summary assessment of the effects of those exceedances. The reports may include, where appropriate, further information and assessments on forest protection as well as information on other pollutants for which monitoring provisions are specified in this Directive, such as, inter alia, selected non-regulated ozone precursor substances as listed in Section B of Annex X.

3. Member States shall inform the public of the competent authority or body designated in relation to the tasks referred to in Article 3.

Article 27
Transmission of information and reporting

1. Member States shall ensure that information on ambient air quality is made available to the Commission within the required timescale as determined by the implementing measures referred to in Article 28(2).

2. In any event, for the specific purpose of assessing compliance with the limit values and critical levels and the attainment of target values, such information shall be made available to the Commission no later than nine months after the end of each year and shall include:

(a) the changes made in that year to the list and delimitation of zones and agglomerations established under Article 4;
(b) the list of zones and agglomerations in which the levels of one or more pollutants are higher than the limit values plus the margin of tolerance where applicable or higher than target values or critical levels; and for these zones and agglomerations:
(i) levels assessed and, if relevant, the dates and periods when such levels were observed;
(ii) if appropriate, an assessment on contributions from natural sources and from re-suspension of particulates following winter-sanding or -salting of roads to the levels assessed, as declared to the Commission under Articles 20 and 21.

3. Paragraphs 1 and 2 shall apply to information collected as from the beginning of the second calendar year after the entry into force of the implementing measures referred to in Article 28(2).

Article 28
Implementing measures

1. Measures designed to amend the non-essential elements of this Directive, namely Annexes I to VI, Annexes VIII to X and Annex XV, shall be adopted in accordance with the regulatory procedure with scrutiny referred to in Article 29(3).
However, the amendments may not have the effect of directly or indirectly modifying either of the following:
(a) the limit values, exposure reduction targets, critical levels, target values, information or alert thresholds or long-term objectives specified in Annex VII and Annexes XI to XIV;
(b) the dates for compliance with any of the parameters referred to in point (a).

2. The Commission shall, in accordance with the regulatory procedure referred to in Article 29(2), determine the additional information to be made available by Member States pursuant to Article 27 as well as the timescales in which such information is to be communicated.
The Commission shall also identify ways of streamlining the way data are reported and the reciprocal exchange of information and data from networks and individual stations measuring ambient air pollution within the Member States, in accordance with the regulatory procedure referred to in Article 29(2).

3. The Commission shall draw up guidelines for the agreements on setting up common measuring stations as referred to in Article 6(5).

4. The Commission shall publish guidance on the demonstration of equivalence referred to in Section B of Annex VI.

CHAPTER VI
COMMITTEE, TRANSITIONAL AND FINAL PROVISIONS

Article 29
Committee

1. The Commission shall be assisted by a committee, "the Ambient Air Quality Committee".

2. Where reference is made to this paragraph, Articles 5 and 7 of Decision 1999/468/EC shall apply, having regard to the provisions of Article 8 thereof.
The period laid down in Article 5(6) of Decision 1999/468/EC shall be set at three months.

3. Where reference is made to this paragraph, Article 5a(1) to (4) and Article 7 of Decision 1999/468/EC shall apply, having regard to the provisions of Article 8 thereof.

Article 30
Penalties

Member States shall lay down the rules on penalties applicable to infringements of the national provisions adopted pursuant to this Directive and shall take all measures necessary to ensure that they are implemented. The penalties provided for must be effective, proportionate and dissuasive.

Article 31
Repeal and transitional provisions

1. Directives 96/62/EC, 1999/30/EC, 2000/69/EC and 2002/3/EC shall be repealed as from 11 June 2010, without prejudice to the obligations on the Member States relating to time-limits for transposition or application of those Directives.
However, from 11 June 2008, the following shall apply:

(a) in Directive 96/62/EC, paragraph 1 of Article 12 shall be replaced by the following:

"1. The detailed arrangements for forwarding the information to be provided under Article 11 shall be adopted in accordance with the procedure referred to in paragraph 3.";

(b) in Directive 1999/30/EC, Article 7(7), footnote 1 in point I of Annex VIII and point VI of Annex IX shall be deleted;

(c) in Directive 2000/69/EC, Article 5(7) and point III in Annex VII shall be deleted;

(d) in Directive 2002/3/EC, Article 9(5) and point II of Annex VIII shall be deleted.

2. Notwithstanding the first subparagraph of paragraph 1, the following Articles shall remain in force:

(a) Article 5 of Directive 96/62/EC until 31 December 2010;

(b) Article 11(1) of Directive 96/62/EC and Article 10(1), (2) and (3) of Directive 2002/3/EC until the end of the second calendar year following the entry into force of the implementing measures referred to in Article 28(2) of this Directive;

(c) Article 9(3) and (4) of Directive 1999/30/EC until 31 December 2009.

3. References made to the repealed Directives shall be construed as being made to this Directive and should be read in accordance with the correlation table in Annex XVII.

4. Decision 97/101/EC shall be repealed with effect from the end of the second calendar year following the entry into force of the implementing measures referred to in Article 28(2) of this Directive.
However, the third, fourth and fifth indents of Article 7 of Decision 97/101/EC shall be deleted with effect from 11 June 2008.

Article 32
Review

1. In 2013 the Commission shall review the provisions related to $PM_{2,5}$ and, as appropriate, other pollutants, and shall present a proposal to the European Parliament and the Council.
As regards $PM_{2,5}$, the review shall be undertaken with a view to establishing a legally binding national exposure reduction obligation in order to replace the national exposure reduction target and to review the exposure concentration obligation laid down in Article 15, taking into account, inter alia, the following elements:

- latest scientific information from WHO and other relevant organisations,
- air quality situations and reduction potentials in the Member States,
- the revision of Directive 2001/81/EC,
- progress made in implementing Community reduction measures for air pollutants,

2. The Commission shall take into account the feasibility of adopting a more ambitious limit value for $PM_{2,5}$, shall review the indicative limit value of the second stage for $PM_{2,5}$ and consider confirming or altering that value.

3. As part of the review, the Commission shall also prepare a report on the experience and on the necessity of monitoring of PM_{10} and $PM_{2,5}$, taking into account technical progress in automatic measuring techniques. If appropriate, new reference methods for the measurement of PM_{10} and $PM_{2,5}$ shall be proposed.

Article 33
Transposition

1. Member States shall bring into force the laws, regulations and administrative provisions necessary to comply with this Directive before 11 June 2010. They shall forthwith communicate to the Commission the text of those measures.

When Member States adopt these measures, they shall contain a reference to this Directive or shall be accompanied by such reference on the occasion of their official publication. The methods of making such reference shall be laid down by Member States.

2. However, Member States shall ensure that a sufficient number of urban background measurement stations of $PM_{2,5}$ necessary for the calculation of the Average Exposure Indicator, in accordance with Section B of Annex V, is established at the latest by 1 January 2009, in order to comply with the timeframe and the conditions indicated in Section A of Annex XIV.

3. Member States shall communicate to the Commission the text of the main provisions of national law which they adopt in the field covered by this Directive.

Article 34
Entry into force

This Directive shall enter into force on the day of its publication in the Official Journal of the European Union.

Article 35
Addressees

This Directive is addressed to the Member States.

Done at Strasbourg, 21 May 2008.

For the European Parliament
The President
H.-G. Pöttering

For the Council
The President
J. Lenarčič

[1] OJ C 195, 18.8.2006, p. 84.
[2] OJ C 206, 29.8.2006, p. 1.
[3] Opinion of the European Parliament of 26 September 2006 (OJ C 306 E, 15.12.2006, p. 103), Council Common Position of 25 June 2007 (OJ C 236 E, 6.11.2007, p. 1) and Position of the European Parliament of 11 December 2007. Council Decision of 14 April 2008.
[4] OJ L 242, 10.9.2002, p. 1.
[5] OJ L 296, 21.11.1996, p. 55. Directive as amended by Regulation (EC) No 1882/2003 of the European Parliament and of the Council (OJ L 284, 31.10.2003, p. 1).
[6] OJ L 163, 29.6.1999, p. 41. Directive as amended by Commission Decision 2001/744/EC (OJ L 278, 23.10.2001, p. 35).
[7] OJ L 313, 13.12.2000, p. 12.
[8] OJ L 67, 9.3.2002, p. 14.
[9] OJ L 35, 5.2.1997, p. 14. Decision as amended by Commission Decision 2001/752/EC (OJ L 282, 26.10.2001, p. 69).
[10] OJ L 23, 26.1.2005, p. 3.
[11] OJ L 171, 27.6.1981, p. 11.
[12] OJ L 309, 27.11.2001, p. 22. Directive as last amended by Council Directive 2006/105/EC (OJ L 363, 20.12.2006, p. 368).
[13] OJ L 309, 27.11.2001, p. 1. Directive as last amended by Directive 2006/105/EC.
[14] OJ L 189, 18.7.2002, p. 12.
[15] OJ L 24, 29.1.2008, p. 8.
[16] OJ L 87, 25.3.2004, p. 50.
[17] OJ L 108, 25.4.2007, p. 1.
[18] OJ C 321, 31.12.2003, p. 1.
[19] OJ L 184, 17.7.1999, p. 23. Decision as amended by Decision 2006/512/EC (OJ L 200, 22.7.2006, p. 11).
[20] Council Directive 89/654/EEC of 30 November 1989 concerning the minimum safety and health requirements for the workplace (OJ L 393, 30.12.1989, p. 1). Directive as amended by Directive 2007/30/EC of the European Parliament and of the Council (OJ L 165, 27.6.2007, p. 21).

ANNEX I
DATA QUALITY OBJECTIVES

A. Data quality objectives for ambient air quality assessment

	Sulphur dioxide, nitrogen dioxide and oxides of nitrogen and carbon monoxide	Benzene	Particulate matter (PM_{10}/$PM_{2,5}$) and lead	Ozone and related NO and NO_2
Fixed measurements[1]				
Uncertainty	15 %	25 %	25 %	15 %
Minimum data capture	90 %	90 %	90 %	90 % during summer 75 % during winter
Minimum time coverage:				
—urban background and traffic	—	35 % [2]	—	—
—industrial sites	—	90 %	—	—
Indicative measurements				
Uncertainty	25 %	30 %	50 %	30 %
Minimum data capture	90 %	90 %	90 %	90 %
Minimum time coverage	14 % [4]	14 %[3]	14 %[4]	> 10 % during summer
Modelling uncertainty:				
Hourly	50 %	—	—	50 %
Eight-hour averages	50 %	—	—	50 %
Daily averages	50 %	—	not yet defined	—
Annual averages	30 %	50 %	50 %	—
Objective estimation Uncertainty	75 %	100 %	100 %	75 %

The uncertainty (expressed at a 95 % confidence level) of the assessment methods will be evaluated in accordance with the principles of the CEN Guide to the Expression of Uncertainty in Measurement (ENV 13005-1999), the methodology of ISO 5725:1994 and the guidance provided in the CEN report "Air Quality — Approach to Uncertainty Estimation for Ambient Air Reference Measurement Methods" (CR 14377:2002E). The percentages for uncertainty in the above table are given for individual measurements averaged over the period considered by the limit value (or target value in the case of ozone), for a 95 % confidence interval. The uncertainty for the fixed measurements shall be interpreted as being applicable in the region of the appropriate limit value (or target value in the case of ozone).

The uncertainty for modelling is defined as the maximum deviation of the measured and calculated concentration levels for 90 % of individual monitoring points, over the period considered, by the limit value (or target value in the case of ozone), without taking into account the timing of the events. The uncertainty for modelling shall be interpreted as being applicable in the region of the appropriate limit value (or target value in the case of ozone). The fixed measurements that have to be selected for comparison with modelling results shall be representative of the scale covered by the model.

The uncertainty for objective estimation is defined as the maximum deviation of the measured and calculated concentration levels, over the period considered, by the limit value (or target value in the case of ozone), without taking into account the timing of the events.

The requirements for minimum data capture and time coverage do not include losses of data due to the regular calibration or the normal maintenance of the instrumentation.

B. Results of air quality assessment

The following information shall be compiled for zones or agglomerations within which sources other than measurement are employed to supplement information from measurement or as the sole means of air quality assessment:

- a description of assessment activities carried out,
- the specific methods used, with references to descriptions of the method,
- the sources of data and information,
- a description of results, including uncertainties and, in particular, the extent of any area or, if relevant, the length of road within the zone or agglomeration over which concentrations exceed any limit value, target value or long-term objective plus margin of tolerance, if applicable, and of any area within which concentrations exceed the upper assessment threshold or the lower assessment threshold,
- the population potentially exposed to levels in excess of any limit value for protection of human health.

C. Quality assurance for ambient air quality assessment: data validation

1. To ensure accuracy of measurements and compliance with the data quality objectives laid down in Section A, the appropriate competent authorities and bodies designated pursuant to Article 3 shall ensure the following:

- that all measurements undertaken in relation to the assessment of ambient air quality pursuant to Articles 6 and 9 are traceable in accordance with the requirements set out in Section 5.6.2.2 of the ISO/IEC 17025:2005,

- that institutions operating networks and individual stations have an established quality assurance and quality control system which provides for regular maintenance to assure the accuracy of measuring devices,
- that a quality assurance/quality control process is established for the process of data collection and reporting and that institutions appointed for this task actively participate in the related Community-wide quality assurance programmes,
- that the national laboratories, when appointed by the appropriate competent authority or body designated pursuant to Article 3, that are taking part in Community-wide intercomparisons covering pollutants regulated in this Directive, are accredited according to EN/ISO 17025 by 2010 for the reference methods referred to in Annex VI. These laboratories shall be involved in the coordination on Member States territory of the Community wide quality assurance programmes to be organised by the Commission and shall also coordinate, on the national level, the appropriate realisation of reference methods and the demonstration of equivalence of non-reference methods.

2. All reported data under Article 27 shall be deemed to be valid except data flagged as provisional.

[1] Member States may apply random measurements instead of continuous measurements for benzene, lead and particulate matter if they can demonstrate to the Commission that the uncertainty, including the uncertainty due to random sampling, meets the quality objective of 25 % and the time coverage is still larger than the minimum time coverage for indicative measurements. Random sampling must be evenly distributed over the year in order to avoid skewing of results. The uncertainty due to random sampling may be determined by the procedure laid down in ISO 11222 (2002) "Air Quality — Determination of the Uncertainty of the Time Average of Air Quality Measurements". If random measurements are used to assess the requirements of the PM_{10} limit value, the 90,4 percentile (to be lower than or equal to 50 µg/m^3) should be evaluated instead of the number of exceedances, which is highly influenced by data coverage.
[2] Distributed over the year to be representative of various conditions for climate and traffic.
[3] One day's measurement a week at random, evenly distributed over the year, or eight weeks evenly distributed over the year.
[4] One measurement a week at random, evenly distributed over the year, or eight weeks evenly distributed over the year.

ANNEX II
Determination of requirements for assessment of concentrations of sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter (PM_{10} and $PM_{2,5}$), lead, benzene and carbon monoxide in ambient air within a zone or agglomeration

A. Upper and lower assessment thresholds

The following upper and lower assessment thresholds will apply:

1. Sulphur dioxide

	Health protection	Vegetation protection
Upper assessment	60 % of 24-hour limit value (75 µg/m^3, not to	60 % of winter

threshold	be exceeded more than 3 times in any calendar year)	critical level (12 µg/m^3)
Lower assessment threshold	40 % of 24-hour limit value (50 µg/m^3, not to be exceeded more than three times in any calendar year)	40 % of winter critical level (8 µg/m^3)

2. Nitrogen dioxide and oxides of nitrogen

	Hourly limit value for the protection of human health (NO_2)	Annual limit value for the protection of human health (NO_2)	Annual critical level for the protection of vegetation and natural ecosystems (NO_x)
Upper assessment threshold	70 % of limit value (140 µg/m^3, not to be exceeded more than 18 times in any calendar year)	80 % of limit value (32 µg/m^3)	80 % of critical level (24 µg/m^3)
Lower assessment threshold	50 % of limit value (100 µg/m^3, not to be exceeded more than 18 times in any calendar year)	65 % of limit value (26 µg/m^3)	65 % of critical level (19,5 µg/m^3)

3. Particulate matter (PM_{10}/$PM_{2,5}$)

	24-hour average PM_{10}	Annual average PM_{10}	Annual average $PM_{2,5}$ [1]
Upper assessment threshold	70 % of limit value (35 µg/m^3, not to be exceeded more than 35 times in any calendar year)	70 % of limit value (28 µg/m^3)	70 % of limit value (17 µg/m^3)
Lower assessment threshold	50 % of limit value (25 µg/m^3, not to be exceeded more than 35 times in any calendar year)	50 % of limit value (20 µg/m^3)	50 % of limit value (12 µg/m^3)

4. Lead

	Annual average
Upper assessment threshold	70 % of limit value (0,35 µg/m^3)
Lower assessment threshold	50 % of limit value (0,25 µg/m^3)

5. Benzene

	Annual average
Upper assessment threshold	70 % of limit value (3,5 µg/m^3)
Lower assessment threshold	40 % of limit value (2 µg/m^3)

6. Carbon monoxide

	Eight-hour average
Upper assessment threshold	70 % of limit value (7 mg/m^3)
Lower assessment threshold	50 % of limit value (5 mg/m^3)

B. Determination of exceedances of upper and lower assessment thresholds

Exceedances of upper and lower assessment thresholds shall be determined on the basis of concentrations during the previous five years where sufficient data are available. An assessment threshold shall be deemed to have been exceeded if it has been exceeded during at least three separate years out of those previous five years.

Where fewer than five years' data are available, Member States may combine measurement campaigns of short duration during the period of the year and at locations likely to be typical of the highest pollution levels with results obtained from information from emission inventories and modelling to determine exceedances of the upper and lower assessment thresholds.

[1] The upper assessment threshold and the lower assessment threshold for PM2,5 do not apply to the measurements to assess compliance with the PM2,5 exposure reduction target for the protection of human health.

ANNEX III

Assessment of ambient air quality and location of sampling points for the measurement of sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter (PM_{10} and $PM_{2,5}$), lead, benzene and carbon monoxide in ambient air

A. General

Ambient air quality shall be assessed in all zones and agglomerations in accordance with the following criteria:

1. Ambient air quality shall be assessed at all locations except those listed in paragraph 2, in accordance with the criteria established by Sections B and C for the location of sampling points for fixed measurement. The principles established by Sections B and C shall also apply in so far as they are relevant in identifying the specific locations in which concentration of the relevant pollutants are established where ambient air quality is assessed by indicative measurement or modelling.

2. Compliance with the limit values directed at the protection of human health shall not be assessed at the following locations:

(a) any locations situated within areas where members of the public do not have access and there is no fixed habitation;

(b) in accordance with Article 2(1), on factory premises or at industrial installations to which all relevant provisions concerning health and safety at work apply;

(c) on the carriageway of roads; and on the central reservations of roads except where there is normally pedestrian access to the central reservation.

B. Macroscale siting of sampling points

1. Protection of human health

(a) Sampling points directed at the protection of human health shall be sited in such a way as to provide data on the following:

- the areas within zones and agglomerations where the highest concentrations occur to which the population is likely to be directly or indirectly exposed for a period which is significant in relation to the averaging period of the limit value(s),

- levels in other areas within the zones and agglomerations which are representative of the exposure of the general population,

(b) Sampling points shall in general be sited in such a way as to avoid measuring very small micro-environments in their immediate vicinity, which means that a sampling point must be sited in such a way that the air sampled is representative of air quality for a street segment no less than 100 m length at traffic-orientated sites and at least 250 m × 250 m at industrial sites, where feasible;

(c) Urban background locations shall be located so that their pollution level is influenced by the integrated contribution from all sources upwind of the station. The pollution level should not be dominated by a single source unless such a situation is typical for a larger urban area. Those sampling points shall, as a general rule, be representative for several square kilometres;

(d) Where the objective is to assess rural background levels, the sampling point shall not be influenced by agglomerations or industrial sites in its vicinity, i.e. sites closer than five kilometres;

(e) Where contributions from industrial sources are to be assessed, at least one sampling point shall be installed downwind of the source in the nearest residential area. Where the background concentration is not known, an additional sampling point shall be situated within the main wind direction;

(f) Sampling points shall, where possible, also be representative of similar locations not in their immediate vicinity;

(g) Account shall be taken of the need to locate sampling points on islands where that is necessary for the protection of human health.

2. Protection of vegetation and natural ecosystems

Sampling points targeted at the protection of vegetation and natural ecosystems shall be sited more than 20 km away from agglomerations or more than 5 km away from other built-up areas, industrial installations or motorways or major roads with traffic counts of more than 50000 vehicles per day, which means that a sampling point must be sited in such a way that the air sampled is representative of air quality in a surrounding area of at least 1000 km². A Member State may provide for a sampling point to be sited at a lesser distance or to be representative of air quality in a less extended area, taking account of geographical conditions or of the opportunities to protect particularly vulnerable areas.

Account shall be taken of the need to assess air quality on islands.

C. Microscale siting of sampling points

In so far as is practicable, the following shall apply:

- the flow around the inlet sampling probe shall be unrestricted (free in an arc of at least 270°) without any obstructions affecting the airflow in the vicinity of the sampler (normally some metres away from buildings, balconies, trees and other obstacles and at least 0,5 m from the nearest building in the case of sampling points representing air quality at the building line),

- in general, the inlet sampling point shall be between 1,5 m (the breathing zone) and 4 m above the ground. Higher positions (up to 8 m) may be necessary in some circumstances. Higher siting may also be appropriate if the station is representative of a large area,

- the inlet probe shall not be positioned in the immediate vicinity of sources in order to avoid the direct intake of emissions unmixed with ambient air,
- the sampler's exhaust outlet shall be positioned so that recirculation of exhaust air to the sampler inlet is avoided,
- for all pollutants, traffic-orientated sampling probes shall be at least 25 m from the edge of major junctions and no more than 10 m from the kerbside.,

The following factors may also be taken into account:

- interfering sources,
- security,
- access,
- availability of electrical power and telephone communications,
- visibility of the site in relation to its surroundings,
- safety of the public and operators,
- the desirability of co-locating sampling points for different pollutants,
- planning requirements.,

D. Documentation and review of site selection

The site-selection procedures shall be fully documented at the classification stage by such means as compass-point photographs of the surrounding area and a detailed map. Sites shall be reviewed at regular intervals with repeated documentation to ensure that selection criteria remain valid over time.

ANNEX IV
MEASUREMENTS AT RURAL BACKGROUND LOCATIONS IRRESPECTIVE OF CONCENTRATION

A. Objectives

The main objectives of such measurements are to ensure that adequate information is made available on levels in the background. This information is essential to judge the enhanced levels in more polluted areas (such as urban background, industry related locations, traffic related locations), assess the possible contribution from long-range transport of air pollutants, support source apportionment analysis and for the understanding of specific pollutants such as particulate matter. It is also essential for the increased use of modelling also in urban areas.

B. Substances

Measurement of $PM_{2,5}$ must include at least the total mass concentration and concentrations of appropriate compounds to characterise its chemical composition. At least the list of chemical species given below shall be included.

SO_4^{2-}	Na^+	NH_4^+	Ca^{2+}	elemental carbon (EC)
NO_3^-	K^+	Cl^-	Mg^{2+}	organic carbon (OC)

C. Siting

Measurements should be taken in particular in rural background areas in accordance with parts A, B and C of Annex III.

ANNEX V
Criteria for determining minimum numbers of sampling points for fixed measurement of concentrations of sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter (PM_{10}, $PM_{2,5}$), lead, benzene and carbon monoxide in ambient air

A. Minimum number of sampling points for fixed measurement to assess compliance with limit values for the protection of human health and alert thresholds in zones and agglomerations where fixed measurement is the sole source of information

1. Diffuse sources

Population of agglomeration or zone (thousands)	If maximum concentrations exceed the upper assessment threshold [1]		If maximum concentrations are between the upper and lower assessment thresholds	
	Pollutants except PM	PM [2] (sum of PM_{10} and $PM_{2,5}$)	Pollutants except PM	PM [2] (sum of PM_{10} and $PM_{2,5}$)
0-249	1	2	1	1
250-499	2	3	1	2
500-749	2	3	1	2
750-999	3	4	1	2
1000-1499	4	6	2	3
1500-1999	5	7	2	3
2000-2749	6	8	3	4
2750-3749	7	10	3	4
3750-4749	8	11	3	6
4750-5999	9	13	4	6
≥ 6000	10	15	4	7

2. Point sources

For the assessment of pollution in the vicinity of point sources, the number of sampling points for fixed measurement shall be calculated taking into account emission densities, the likely distribution patterns of ambient-air pollution and the potential exposure of the population.

B. Minimum number of sampling points for fixed measurement to assess compliance with the $PM_{2,5}$ exposure reduction target for the protection of human health

One sampling point per million inhabitants summed over agglomerations and additional urban areas in excess of 100000 inhabitants shall be operated for this purpose. Those sampling points may coincide with sampling points under Section A.

C. Minimum number of sampling points for fixed measurements to assess compliance with critical levels for the protection of vegetation in zones other than agglomerations

If maximum concentrations exceed the upper assessment threshold	If maximum concentrations are between upper and lower assessment threshold
1 station every 20000 km²	1 station every 40000 km²

In island zones the number of sampling points for fixed measurement should be calculated taking into account the likely distribution patterns of ambient-air pollution and the potential exposure of vegetation.

[1] For nitrogen dioxide, particulate matter, benzene and carbon monoxide: to include at least one urban background monitoring station and one traffic-orientated station provided this does not increase the number of sampling points. For these pollutants, the total number of urban-background stations and the total number of traffic oriented stations in a Member State required under Section A(1) shall not differ by more than a factor of 2. Sampling points with exceedances of the limit value for PM_{10} within the last three years shall be maintained, unless a relocation is necessary owing to special circumstances, in particular spatial development.
[2] Where $PM_{2,5}$ and PM_{10} are measured in accordance with Article 8 at the same monitoring station, these shall count as two separate sampling points. The total number of $PM_{2,5}$ and PM_{10} sampling points in a Member State required under Section A(1) shall not differ by more than a factor of 2, and the number of $PM_{2,5}$ sampling points in the urban background of agglomerations and urban areas shall meet the requirements under Section B of Annex V.

ANNEX VI
Reference methods for assessment of concentrations of sulphur dioxide, nitrogen dioxide and oxides of nitrogen, particulate matter (PM_{10} and $PM_{2,5}$), lead, benzene, carbon monoxide, and ozone

A. Reference measurement methods

1. Reference method for the measurement of sulphur dioxide
The reference method for the measurement of sulphur dioxide is that described in EN 14212:2005 "Ambient air quality — Standard method for the measurement of the concentration of sulphur dioxide by ultraviolet fluorescence".

2. Reference method for the measurement of nitrogen dioxide and oxides of nitrogen
The reference method for the measurement of nitrogen dioxide and oxides of nitrogen is that described in EN 14211:2005 "Ambient air quality — Standard method for the measurement of the concentration of nitrogen dioxide and nitrogen monoxide by chemiluminescence".

3. Reference method for the sampling and measurement of lead
The reference method for the sampling of lead is that described in Section A(4) of this Annex. The reference method for the measurement of lead is that described in EN 14902:2005 "Standard method for measurement of Pb/Cd/As/Ni in the PM_{10} fraction of suspended particulate matter".

4. Reference method for the sampling and measurement of PM_{10}
The reference method for the sampling and measurement of PM_{10} is that described in EN 12341:1999 "Air Quality — Determination of the PM_{10} fraction of suspended particulate matter — Reference method and field test procedure to demonstrate reference equivalence of measurement methods".

5. Reference method for the sampling and measurement of $PM_{2,5}$
The reference method for the sampling and measurement of $PM_{2,5}$ is that described in EN 14907:2005 "Standard gravimetric measurement method for the determination of the $PM_{2,5}$ mass fraction of suspended particulate matter".

6. Reference method for the sampling and measurement of benzene
The reference method for the measurement of benzene is that described in EN 14662:2005, parts 1, 2 and 3 "Ambient air quality — Standard method for measurement of benzene concentrations".

7. Reference method for the measurement of carbon monoxide
The reference method for the measurement of carbon monoxide is that described in EN 14626:2005 "Ambient air quality — Standard method for the measurement of the concentration of carbon monoxide by non-dispersive infrared spectroscopy".

8. Reference method for measurement of ozone
The reference method for the measurement of ozone is that described in EN 14625:2005 "Ambient air quality — Standard method for the measurement of the concentration of ozone by ultraviolet photometry".

B. Demonstration of equivalence

1. A Member State may use any other method which it can demonstrate gives results equivalent to any of the methods referred to in Section A or, in the case of particulate matter, any other method which the Member State concerned can demonstrate displays a consistent relationship to the reference method. In that event the results achieved by that method must be corrected to produce results equivalent to those that would have been achieved by using the reference method.

2. The Commission may require the Member States to prepare and submit a report on the demonstration of equivalence in accordance with paragraph 1.

3. When assessing the acceptability of the report mentioned in paragraph 2, the Commission will make reference to its guidance on the demonstration of equivalence (to be published). Where Member States have been using interim factors to approximate equivalence, the latter shall be confirmed and/or amended with reference to the Commission's guidance.

4. Member States should ensure that whenever appropriate, the correction is also applied retroactively to past measurement data in order to achieve better data comparability.

C. Standardisation

For gaseous pollutants the volume must be standardised at a temperature of 293 K and an atmospheric pressure of 101,3 kPa. For particulate matter and substances to be analysed in particulate matter (e.g. lead) the sampling volume refers to ambient conditions in terms of temperature and atmospheric pressure at the date of measurements.

D. Introduction of new equipment

All new equipment purchased for implementation of this Directive must comply with the reference method or equivalent by 11 June 2010.
All equipment used in fixed measurements must comply with the reference method or equivalent by 11 June 2013.

E. Mutual recognition of data

In carrying out the type approval to demonstrate that equipment meets the performance requirements of the reference methods listed in Section A, competent authorities and bodies designated pursuant to Article 3 shall accept test reports issued in other Member States by laboratories accredited to EN ISO 17025 for carrying out such testing.

ANNEX VII
OZONE TARGET VALUES AND LONG-TERM OBJECTIVES

A. Definitions and criteria

1. Definitions
AOT40 (expressed in (μg/m^3) · hours) means the sum of the difference between hourly concentrations greater than 80 μg/m^3 (= 40 parts per billion) and 80 μg/m^3 over a given period using only the one-hour values measured between 8.00 and 20.00 Central European Time (CET) each day.

2. Criteria
The following criteria shall be used for checking validity when aggregating data and calculating statistical parameters:

Parameter	Required proportion of valid data
One hour values	75 % (i.e. 45 minutes)
Eight hours values	75 % of values (i.e. six hours)
Maximum daily 8 hours mean from hourly running 8 hours	75 % of the hourly running eight hours averages (i.e. 18 eight-hourly averages per day)
AOT40	90 % of the one hour values over the time period defined for calculating the AOT40 value [1]
Annual mean	75 % of the one hour values over summer (April to September) and 75 % over winter (January to March, October to December) seasons separately
Number of exceedances and maximum values per month	90 % of the daily maximum eight hours mean values (27 available daily values per month) 90 % of the one hour

	values between 8.00 and 20.00 CET
Number of exceedances and maximum values per year	five out of six months over the summer season (April to September)

B. Target values

Objective	Averaging period	Target value	Date by which target value should be met [2]
Protection of human health	Maximum daily eight-hour mean [3]	120 µg/m^3 not to be exceeded on more than 25 days per calendar year averaged over three years [4]	1.1.2010
Protection of vegetation	May to July	AOT40 (calculated from 1 h values) 18000 µg/m^3 · h averaged over five years [4]	1.1.2010

C. Long-term objectives

Objective	Averaging period	Longterm objective	Date by which the longterm objective should be met
Protection of human health	Maximum daily eight-hour mean within a calendar year	120 µg/m^3	not defined
Protection of vegetation	May to July	AOT40 (calculated from 1 h values) 6000 µg/m^3 · h	not defined

[1] In cases where all possible measured data are not available, the following factor shall be used to calculate AOT40 values:(*)being the number of hours within the time period of AOT40 definition, (i.e. 08:00 to 20:00 CET from 1 May to 31 July each year, for vegetation protection and from 1 April to 30 September each year for forest protection).
[2] Compliance with target values will be assessed as of this date. That is, 2010 will be the first year the data for which is used in calculating compliance over the following three or five years, as appropriate.
[3] The maximum daily eight-hour mean concentration shall be selected by examining eight-hour running averages, calculated from hourly data and updated each hour. Each eight - hour average so calculated shall be assigned to the day on which it ends. i.e. the first calculation period for any one day will be the period from 17:00 on the previous day to 01:00 on that day; the last calculation period for any one day will be the period from 16:00 to 24:00 on the day.
[4] If the three or five year averages cannot be determined on the basis of a full and consecutive set of annual data, the minimum annual data required for checking compliance with the target values will be as follows:

- for the target value for the protection of human health: valid data for one year,
- for the target value for the protection of vegetation: valid data for three years.

ANNEX VIII
Criteria for classifying and locating sampling points for assessments of ozone concentrations

The following apply to fixed measurements:

A. Macroscale siting

Type of station	Objectives of measurement	Representativeness [1]	Macroscale siting criteria
Urban	Protection of human health: to assess the exposure of the urban population to ozone, i.e. where population density and ozone concentration are relatively high and representative of the exposure of the general population	A few km^2	Away from the influence of local emissions such as traffic, petrol stations, etc.; vented locations where well mixed levels can be measured; locations such as residential and commercial areas of cities, parks (away from the trees), big streets or squares with very little or no traffic, open areas characteristic of educational, sports or recreation facilities
Suburban	Protection of human health and vegetation: to assess the exposure of the population and vegetation located in the outskirts of the agglomeration, where the highest ozone levels, to which the population and vegetation are likely to be directly or indirectly exposed occur	Some tens of km^2	At a certain distance from the area of maximum emissions, downwind following the main wind direction/directions during conditions favourable to ozone formation; where population, sensitive crops or natural ecosystems located in the outer fringe of an agglomeration are exposed to high ozone levels; where appropriate, some suburban stations

			also upwind of the area of maximum emissions, in order to determine the regional background levels of ozone
Rural	Protection of human health and vegetation: to assess the exposure of population, crops and natural ecosystems to sub-regional scale ozone concentrations	Sub-regional levels (some hundreds of km^2)	Stations can be located in small settlements and/or areas with natural ecosystems, forests or crops; representative for ozone away from the influence of immediate local emissions such as industrial installations and roads; at open area sites, but not on summits of higher mountains
Rural background	Protection of vegetation and human health: to assess the exposure of crops and natural ecosystems to regional-scale ozone concentrations as well as exposure of the population	Regional/national/continental levels (1000 to 10000 km^2)	Station located in areas with lower population density, e.g. with natural ecosystems, forests, at a distance of at least 20 km from urban and industrial areas and away from local emissions; avoid locations which are subject to locally enhanced formation of ground-near inversion conditions, also summits of higher mountains; coastal sites with pronounced diurnal wind cycles of local character are not recommended.

For rural and rural background stations the location shall, where appropriate, be coordinated with the monitoring requirements of Commission Regulation (EC) No 1737/2006 of 7 November 2006 laying down detailed rules for the implementation of Regulation (EC) No 2152/2003 of the European Parliament and of the Council concerning monitoring of forests and environmental interactions in the Community [2].

B. Microscale siting

In so far as is practicable the procedure on microscale siting in Section C of Annex III shall be followed, ensuring also that the inlet probe is positioned well away from such sources as furnaces and incineration flues and more than 10 m from the nearest road, with distance increasing as a function of traffic intensity.

C. Documentation and review of site selection

The procedures in Section D of Annex III shall be followed, applying proper screening and interpretation of the monitoring data in the context of the meteorological and photochemical processes affecting the ozone concentrations measured at the respective sites.

[1] Sampling points should, where possible, be representative of similar locations not in their immediate vicinity.
[2] OJ L 334, 30.11.2006, p. 1.

ANNEX IX
Criteria for determining the minimum number of sampling points for fixed measurement of concentrations of ozone

A. Minimum number of sampling points for fixed continuous measurements to assess compliance with target values, long-term objectives and information and alert thresholds where such measurements are the sole source of information

Population (× 1000)	Agglomerations (urban and suburban) [1]	Other zones (suburban and rural) [1]	Rural background
< 250		1	1 station/50000 km² as an average density over all zones per country [2]
< 500	1	2	
< 1000	2	2	
< 1500	3	3	
< 2000	3	4	
< 2750	4	5	
< 3750	5	6	
> 3750	One additional station per 2 million inhabitants	One additional station per 2 million inhabitants	

B. Minimum number of sampling points for fixed measurements for zones and agglomerations attaining the long-term objectives

The number of sampling points for ozone shall, in combination with other means of supplementary assessment such as air quality modelling and collocated nitrogen dioxide measurements, be sufficient to examine the trend of ozone pollution and check compliance with the long-term objectives. The number of stations located in agglomerations and other zones may be reduced to one-third of the number specified in Section A. Where information from fixed measurement stations is the sole source of information, at least one monitoring station shall be kept. If, in zones where there is supplementary assessment, the result of this is that a zone has no remaining station, coordination with the number of stations in neighbouring zones shall ensure adequate assessment of ozone concentrations against long-term objectives. The number of rural background stations shall be one per 100000 km².

[1] At least 1 station in suburban areas, where the highest exposure of the population is likely to occur. In agglomerations at least 50 % of the stations shall be located in suburban areas.
[2] 1 station per 25000 km² for complex terrain is recommended.

ANNEX X
MEASUREMENTS OF OZONE PRECURSOR SUBSTANCES

A. Objectives

The main objectives of such measurements are to analyse any trend in ozone precursors, to check the efficiency of emission reduction strategies, to check the consistency of emission inventories and to help attribute emission sources to observed pollution concentrations.
An additional aim is to support the understanding of ozone formation and precursor dispersion processes, as well as the application of photochemical models.

B. Substances

Measurement of ozone precursor substances shall include at least nitrogen oxides (NO and NO_2), and appropriate volatile organic compounds (VOC). A list of volatile organic compounds recommended for measurement is given below:

	1-Butene	Isoprene	Ethyl benzene
Ethane	Trans-2-Butene	n-Hexane	m + p-Xylene
Ethylene	cis-2-Butene	i-Hexane	o-Xylene
Acetylene	1,3-Butadiene	n-Heptane	1,2,4-Trimethylebenzene
Propane	n-Pentane	n-Octane	1,2,3-Trimethylebenzene
Propene	i-Pentane	i-Octane	1,3,5-Trimethylebenzene
n-Butane	1-Pentene	Benzene	Formaldehyde
i-Butane	2-Pentene	Toluene	Total non-methane hydrocarbons

C. Siting

Measurements shall be taken in particular in urban or suburban areas at any monitoring site set up in accordance with the requirements of this Directive and considered appropriate with regard to the monitoring objectives referred to in Section A.

ANNEX XI
LIMIT VALUES FOR THE PROTECTION OF HUMAN HEALTH

A. Criteria

Without prejudice to Annex I, the following criteria shall be used for checking validity when aggregating data and calculating statistical parameters:

Parameter	Required proportion of valid data
One hour values	75 % (i.e. 45 minutes)
Eight hours values	75 % of values (i.e. 6 hours)
Maximum daily 8-hour mean	75 % of the hourly running eight hour averages (i.e. 18 eight hour averages per day)
24-hour values	75 % of the hourly averages (i.e. at least 18 hour values)
Annual mean	90 % [1] of the one hour values or (if not available) 24-hour values over the year

B. Limit values

Averaging Period	Limit value	Margin of tolerance	Date by which limit value is to be met
Sulphur dioxide			
One hour	350 µg/m^3, not to be exceeded more than 24 times a calendar year	150 µg/m^3 (43 %)	— [2]
One day	125 µg/m^3, not to be exceeded more than 3 times a calendar year	None	— [2]
Nitrogen dioxide			
One hour	200 µg/m^3, not to be exceeded more than 18 times a calendar year	50 % on 19 July 1999, decreasing on 1 January 2001 and every 12 months thereafter by equal annual percentages to reach 0 % by 1 January 2010	1 January 2010
Calendar year	40 µg/m^3	50 % on 19 July 1999, decreasing on 1 January 2001 and every 12 months thereafter by equal annual percentages to reach 0 % by 1 January 2010	1 January 2010
Benzene			
Calendar year	5 µg/m^3	5 µg/m^3 (100 %) on 13 December 2000, decreasing on 1 January 2006 and every 12 months thereafter by 1 µg/m3 to reach 0 % by 1 January 2010	1 January 2010
Carbon monoxide			
maximum daily eight hour mean [3]	10 mg/m^3	60 %	— [2]
Lead			
Calendar year	0,5 µg/m^3 [4]	100 %	— [4]
PM_{10}			
One day	50 µg/m^3, not to be exceeded more than 35 times a calendar year	50 %	— [2]
Calendar year	40 µg/m^3	20 %	— [2]

[1] The requirements for the calculation of annual mean do not include losses of data due to the regular calibration or the normal maintenance of the instrumentation.
[2] Already in force since 1 January 2005
[3] The maximum daily eight hour mean concentration will be selected by examining eight hour running averages, calculated from hourly data and updated each hour. Each eight hour average so calculated will be assigned to the day on which it ends i.e. the first calculation period for any one day will be the period from 17:00 on the previous day to 01:00 on that day; the last calculation period for any one day will be the period from 16:00 to 24:00 on that day.
[4] Already in force since 1 January 2005. Limit value to be met only by 1 January 2010 in the immediate vicinity of the specific industrial sources situated on sites contaminated by decades of industrial activities. In such cases, the limit value until 1 January 2010 will be 1,0 µg/m^3. The area in which higher limit values apply must not extend further than 1000 m from such specific sources.

ANNEX XII
INFORMATION AND ALERT THRESHOLDS

A. Alert thresholds for pollutants other than ozone

To be measured over three consecutive hours at locations representative of air quality over at least 100 km² or an entire zone or agglomeration, whichever is the smaller.

Pollutant	Alert threshold
Sulphur dioxide	500 µg/m^3
Nitrogen dioxide	400 µg/m^3

B. Information and alert thresholds for ozone

Purpose	Averaging period	Threshold
Information	1 hour	180 µg/m^3
Alert	1 hour [1]	240 µg/m^3

[1] For the implementation of Article 24, the exceedance of the threshold is to be measured or predicted for three consecutive hours.

ANNEX XIII
CRITICAL LEVELS FOR THE PROTECTION OF VEGETATION

Averaging period	Critical level	Margin of tolerance
Sulphur dioxide		
Calendar year and winter (1 October to 31 March)	20 µg/m^3	None
Oxides of nitrogen		
Calendar year	30 µg/m^3 NO_x	None

ANNEX XIV
NATIONAL EXPOSURE REDUCTION TARGET, TARGET VALUE AND LIMIT VALUE FOR $PM_{2,5}$

A. Average exposure indicator

The Average Exposure Indicator expressed in $\mu g/m^3$ (AEI) shall be based upon measurements in urban background locations in zones and agglomerations throughout the territory of a Member State. It should be assessed as a three-calendar year running annual mean concentration averaged over all sampling points established pursuant to Section B of Annex V. The AEI for the reference year 2010 shall be the mean concentration of the years 2008, 2009 and 2010.

However, where data are not available for 2008, Member States may use the mean concentration of the years 2009 and 2010 or the mean concentration of the years 2009, 2010 and 2011. Member States making use of these possibilities shall communicate their decisions to the Commission by 11 September 2008.

The AEI for the year 2020 shall be the three-year running mean concentration averaged over all those sampling points for the years 2018, 2019 and 2020. The AEI is used for the examination whether the national exposure reduction target is met.

The AEI for the year 2015 shall be the three-year running mean concentration averaged over all those sampling points for the years 2013, 2014 and 2015. The AEI is used for the examination whether the exposure concentration obligation is met.

B. National exposure reduction target

Exposure reduction target relative to the AEI in 2010		Year by which the exposure reduction target should be met
Initial concentration in $\mu g/m^3$	Reduction target in percent	2020
< 8,5 = 8,5	0 %	
> 8,5 — < 13	10 %	
= 13 — < 18	15 %	
= 18 — < 22	20 %	
≥ 22	All appropriate measures to achieve 18 $\mu g/m^3$	

Where the AEI in the reference year is 8,5 $\mu g/m^3$ or less the exposure reduction target shall be zero. The reduction target shall be zero also in cases where the AEI reaches the level of 8,5 $\mu g/m^3$ at any point of time during the period from 2010 to 2020 and is maintained at or below that level.

C. Exposure concentration obligation

Exposure concentration obligation	Year by which the obligation value is to be met
20 $\mu g/m^3$	2015

D. Target value

Averaging period	Target value	Date by which target value should be met
Calendar year	25 $\mu g/m^3$	1 January 2010

E. Limit value

Averaging period	Limit value	Margin of tolerance	Date by which limit value is to be met
STAGE 1			
Calendar year	25 µg/m^3	20 % on 11 June 2008, decreasing on the next 1 January and every 12 months thereafter by equal annual percentages to reach 0 % by 1 January 2015	1 January 2015
STAGE 2 [1]			
Calendar year	20 µg/m^3		1 January 2020

[1] Stage 2 — indicative limit value to be reviewed by the Commission in 2013 in the light of further information on health and environmental effects, technical feasibility and experience of the target value in Member States.

ANNEX XV
Information to be included in the local, regional or national air quality plans for improvement in ambient air quality

A. Information to be provided under article 23 (air quality plans)

1. Localisation of excess pollution
   (a) region;
   (b) city (map);
   (c) measuring station (map, geographical coordinates).

2. General information
   (a) type of zone (city, industrial or rural area);
   (b) estimate of the polluted area (km²) and of the population exposed to the pollution;
   (c) useful climatic data;
   (d) relevant data on topography;
   (e) sufficient information on the type of targets requiring protection in the zone.

3. Responsible authorities
Names and addresses of persons responsible for the development and implementation of improvement plans.

4. Nature and assessment of pollution
   (a) concentrations observed over previous years (before the implementation of the improvement measures);
   (b) concentrations measured since the beginning of the project;
   (c) techniques used for the assessment.

5. Origin of pollution

(a) list of the main emission sources responsible for pollution (map);
(b) total quantity of emissions from these sources (tonnes/year);
(c) information on pollution imported from other regions.

6. Analysis of the situation
(a) details of those factors responsible for the exceedance (e.g. transport, including cross-border transport, formation of secondary pollutants in the atmosphere);
(b) details of possible measures for the improvement of air quality.

7. Details of those measures or projects for improvement which existed prior to 11 June 2008, i.e:
(a) local, regional, national, international measures;
(b) observed effects of these measures.

8. Details of those measures or projects adopted with a view to reducing pollution following the entry into force of this Directive:
(a) listing and description of all the measures set out in the project;
(b) timetable for implementation;
(c) estimate of the improvement of air quality planned and of the expected time required to attain these objectives.

9. Details of the measures or projects planned or being researched for the long term.

10. List of the publications, documents, work, etc., used to supplement information required under this Annex.

B. Information to be provided under article 22(1)

1. All information as laid down in Section A.

2. Information concerning the status of implementation of the following Directives:

1. Council Directive 70/220/EEC of 20 March 1970 on the approximation of the laws of the Member States on measures to be taken against air pollution by emissions from motor vehicles [1];
2. Directive 94/63/EC of the European Parliament and of the Council of 20 December 1994 on the control of volatile organic compound (VOC) emissions resulting from the storage of petrol and its distribution from terminals to service stations [2];
3. Directive 2008/1/EC of the European Parliament and of the Council of 15 January 2008 concerning integrated pollution prevention and control [3];
4. Directive 97/68/EC of the European Parliament and of the Council of 16 December 1997 on the approximation of the laws of the Member States relating to measures against the emission of gaseous and particulate pollutants from internal combustion engines to be installed in non-road mobile machinery [4];
5. Directive 98/70/EC of the European Parliament and of the Council of 13 October 1998 relating to the quality of petrol and diesel fuels [5];
6. Council Directive 1999/13/EC of 11 March 1999 on the limitation of emissions of volatile organic compounds due to the use of organic solvents in certain activities and installations [6];

7. Council Directive 1999/32/EC of 26 April 1999 relating to a reduction in the sulphur content of certain liquid fuels [7];

8. Directive 2000/76/EC of the European Parliament and of the Council of 4 December 2000 on the incineration of waste [8];

9. Directive 2001/80/EC of the European Parliament and of the Council of 23 October 2001 on the limitation of emissions of certain pollutants into the air from large combustion plants;

10. Directive 2001/81/EC of the European Parliament and of the Council of 23 October 2001 on national emission ceilings for certain atmospheric pollutants;

11. Directive 2004/42/EC of the European Parliament and of the Council of 21 April 2004 on the limitation of emissions of volatile organic compounds due to the use of organic solvents in certain paints and varnishes and vehicle refinishing products [9];

12. Directive 2005/33/EC of the European Parliament and of the Council of 6 July 2005 amending Directive 1999/32/EC as regards the sulphur content of marine fuels [10];

13. Directive 2005/55/EC of the European Parliament and of the Council of 28 September 2005 on the approximation of the laws of the Member States relating to the measures to be taken against the emission of gaseous and particulate pollutants from compression-ignition engines for use in vehicles, and the emission of gaseous pollutants from positive-ignition engines fuelled with natural gas or liquefied petroleum gas for use in vehicles [11];

14. Directive 2006/32/EC of the European Parliament and of the Council of 5 April 2006 on energy end-use efficiency and energy services [12].

3. Information on all air pollution abatement measures that have been considered at appropriate local, regional or national level for implementation in connection with the attainment of air quality objectives, including:

(a) reduction of emissions from stationary sources by ensuring that polluting small and medium sized stationary combustion sources (including for biomass) are fitted with emission control equipment or replaced;

(b) reduction of emissions from vehicles through retrofitting with emission control equipment. The use of economic incentives to accelerate take-up should be considered;

(c) procurement by public authorities, in line with the handbook on environmental public procurement, of road vehicles, fuels and combustion equipment to reduce emissions, including the purchase of:

- new vehicles, including low emission vehicles,
- cleaner vehicle transport services,
- low emission stationary combustion sources,
- low emission fuels for stationary and mobile sources,

(d) measures to limit transport emissions through traffic planning and management (including congestion pricing, differentiated parking fees or other economic incentives; establishing low emission zones);

(e) measures to encourage a shift of transport towards less polluting modes;

(f) ensuring that low emission fuels are used in small, medium and large scale stationary sources and in mobile sources;

(g) measures to reduce air pollution through the permit system under Directive 2008/1/EC, the national plans under Directive 2001/80/EC, and through the use of economic instruments such as taxes, charges or emission trading.

(h) where appropriate, measures to protect the health of children or other sensitive groups.

[1] OJ L 76, 6.4.1970, p. 1. Directive as last amended by Directive 2006/96/EC (OJ L 363, 20.12.2006, p. 81).
[2] OJ L 365, 31.12.1994, p. 24. Directive as amended by Regulation (EC) No 1882/2003 (OJ L 284, 31.10.2003, p. 1).
[3] OJ L 24, 29.1.2008, p. 8.
[4] OJ L 59, 27.2.1998, p. 1. Directive as last amended by Directive 2006/105/EC.
[5] OJ L 350, 28.12.1998, p. 58. Directive as amended by Regulation (EC) No 1882/2003.
[6] OJ L 85, 29.3.1999, p. 1. Directive as last amended by Directive 2004/42/EC of the European Parliament and of the Council (OJ L 143, 30.4.2004, p. 87).
[7] OJ L 121, 11.5.1999, p. 13. Directive as last amended by Directive 2005/33/EC of the European Parliament and of the Council (OJ L 191, 22.7.2005, p. 59).
[8] OJ L 332, 28.12.2000, p. 91.
[9] OJ L 143, 30.4.2004, p. 87.
[10] OJ L 191, 22.7.2005, p. 59.
[11] OJ L 275, 20.10.2005, p. 1. Directive as last amended by Regulation (EC) No 715/2007 (OJ L 171, 29.6.2007, p. 1).
[12] OJ L 114, 27.4.2006, p. 64.

ANNEX XVI
PUBLIC INFORMATION

1. Member States shall ensure that up-to-date information on ambient concentrations of the pollutants covered by this Directive is routinely made available to the public.

2. Ambient concentrations provided shall be presented as average values according to the appropriate averaging period as laid down in Annex VII and Annexes XI to XIV. The information shall at least indicate any levels exceeding air quality objectives including limit values, target values, alert thresholds, information thresholds or long term objectives of the regulated pollutant. It shall also provide a short assessment in relation to the air quality objectives and appropriate information regarding effects on health, or, where appropriate, vegetation.

3. Information on ambient concentrations of sulphur dioxide, nitrogen dioxide, particulate matter (at least PM_{10}), ozone and carbon monoxide shall be updated on at least a daily basis, and, wherever practicable, information shall be updated on an hourly basis. Information on ambient concentrations of lead and benzene, presented as an average value for the last 12 months, shall be updated on a three-monthly basis, and on a monthly basis, wherever practicable.

4. Member States shall ensure that timely information about actual or predicted exceedances of alert thresholds, and any information threshold is provided to the public. Details supplied shall include at least the following information:

(a) information on observed exceedance(s):
- location or area of the exceedance,
- type of threshold exceeded (information or alert),
- start time and duration of the exceedance,
- highest one hour concentration and in addition highest eight hour mean concentration in the case of ozone;

(b) forecast for the following afternoon/day(s):

- geographical area of expected exceedances of information and/or alert threshold,
- expected changes in pollution (improvement, stabilisation or deterioration), together with the reasons for those changes;

(c) information on the type of population concerned, possible health effects and recommended behaviour:

- information on population groups at risk,
- description of likely symptoms,
- recommended precautions to be taken by the population concerned,
- where to find further information;

(d) information on preventive action to reduce pollution and/or exposure to it: indication of main source sectors; recommendations for action to reduce emissions;

(e) in the case of predicted exceedances, Member State shall take steps to ensure that such details are supplied to the extent practicable.

ANNEX XVII
CORRELATION TABLE

This Directive	Directive 96/62/EC	Directive 1999/30/EC	Directive 2000/69/EC	Directive 2002/3/EC
Article 1	Article 1	Article 1	Article 1	Article 1
Article 2(1) to (5)	Article 2(1) to (5)	—	—	—
Article 2(6) and (7)	—	—	—	—
Article 2(8)	Article 2(8)	Article 2(7)	—	—
Article 2(9)	Article 2(6)	—	—	Article 2(9)
Article 2(10)	Article 2(7)	Article 2(6)	—	Article 2(11)
Article 2(11)	—	—	—	Article 2(12)
Article 2(12) and (13)	—	Article 2(13) and (14)	Article 2(a) and (b)	—
Article 2(14)	—	—	—	Article 2(10)
Article 2(15) and (16)	Article 2(9) and (10)	Article 2(8) and (9)	—	Article 2(7) and (8)
Article 2(17) and (18)	—	Article 2(11) and (12)	—	—
Article 2(19), (20), (21), (22) and (23)	—	—	—	—
Article 2(24)	—	Article 2(10)	—	—
Article 2(25) and (26)	Article 6(5)	—	—	—
Article 2(27)	—	—	—	Article 2(13)
Article 2(28)	—	—	—	Article 2(3)
Article 3, with the exception of paragraph (1)(f)	Article 3	—	—	—
Article 3(1)(f)	—	—	—	—
Article 4	Article 2(9) and (10), Article 6(1)	—	—	—
Article 5	—	Article 7(1)	Article 5(1)	—
Article 6(1) to (4)	Article 6(1) to (4)	—	—	—
Article 6(5)	—	—	—	—
Article 7	—	Article 7(2) and (3) with amendments	Article 5(2) and (3)	—

			with amendments	
Article 8	—	Article 7(5)	Article 5(5)	—
Article 9	—	—	—	Article 9(1) first and second subparagraphs
Article 10	—	—	—	Article 9(1) to (3) with amendments
Article 11(1)	—	—	—	Article 9(4)
Article 11(2)	—	—	—	—
Article 12	Article 9	—	—	—
Article 13(1)	—	Articles 3(1), 4(1), 5(1) and 6	Articles 3(1) and 4	—
Article 13(2)	—	Articles 3(2) and 4(2)	—	—
Article 13(3)	—	Article 5(5)	—	—
Article 14	—	Articles 3(1) and 4(1) with amendments	—	—
Article 15	—	—	—	—
Article 16	—	—	—	—
Article 17(1)	—	—	—	Articles 3(1) and 4(1)
Article 17(2)	—	—	—	Article 3(2) and (3)
Article 17(3)	—	—	—	Article 4(2)
Article 18	—	—	—	Article 5
Article 19	Article 10 with amendments	Article 8(3)	—	Article 6 with amendments
Article 20	—	Articles 3(4) and 5(4) with amendments	—	—
Article 21	—	—	—	—
Article 22	—	—	—	—
Article 23	Article 8(1) to (4) with amendments	—	—	—
Article 24	Article 7(3) with amendments	—	—	Article 7 with amendments
Article 25	Article 8(5) with amendments	—	—	Article 8 with amendments

Article 26	—	Article 8 with amendments	Article 7 with amendments	Article 6 with amendments
Article 27	Article 11 with amendments	Article 5(2) second subparagraph	—	Article 10 with amendments
Article 28(1)	Article 12(1) with amendments	—	—	—
Article 28(2)	Article 11 with amendments	—	—	—
Article 28(3)	—	—	—	—
Article 28(4)	—	Annex IX with amendments	—	—
Article 29	Article 12(2)	—	—	—
Article 30	—	Article 11	Article 9	Article 14
Article 31	—	—	—	—
Article 32	—	—	—	—
Article 33	Article 13	Article 12	Article 10	Article 15
Article 34	Article 14	Article 13	Article 11	Article 17
Article 35	Article 15	Article 14	Article 12	Article 18
Annex I	—	Annex VIII with amendments	Annex VI	Annex VII
Annex II	—	Annex V with amendments	Annex III	—
Annex III	—	Annex VI	Annex IV	—
Annex IV	—	—	—	—
Annex V	—	Annex VII with amendments	Annex V	—
Annex VI	—	Annex IX with amendments	Annex VII	Annex VIII
Annex VII	—	—	—	Annex I, Annex III section II
Annex VIII	—	—	—	Annex IV
Annex IX	—	—	—	Annex V
Annex X	—	—	—	Annex VI
Annex XI	—	Annex I, section I, Annex II, section I and Annex III (with amendments); Annex IV (unchanged)	Annex I, Annex II	—

Annex XII	—	Annex I, section II, Annex II, section II,	—	Annex II, section I
Annex XIII	—	Annex I, section I, Annex II, section I	—	—
Annex XIV	—	—	—	—
Annex XV Section A	Annex IV	—	—	—
Annex XV Section B	—	—	—	—
Annex XVI	—	Article 8	Article 7	Article 6 with amendments

STATEMENT BY THE COMMISSION

The Commission takes note of the text adopted by the Council and the European Parliament for the Directive on ambient air quality and cleaner air for Europe. In particular, the Commission notes the importance attributed by the European Parliament and the Member States in Article 22(4) and recital 16 to Community measures for the abatement of air pollutant emissions at source.

The Commission recognises the need to reduce the emissions of harmful air pollutants if significant progress is to be delivered towards the objectives established in the Sixth Environmental Action Programme. The Commission's communication on a thematic strategy on air pollution sets out a significant number of possible Community measures. Significant progress on these and other measures has been made since the adoption of the strategy:

- the Council and Parliament have already adopted new legislation limiting the exhaust emissions of light duty vehicles,
- the Commission has adopted a proposal for new legislation to improve the effectiveness of Community industrial emissions legislation including intensive agricultural installations and measures to tackle smaller scale industrial combustion sources,
- the Commission has adopted a proposal for new legislation limiting the exhaust emissions of engines installed in heavy duty vehicles,
- in 2008 the Commission foresees new legislative proposals that would:
- further reduce the Member States' permitted national emissions of key pollutants,
- reduce emissions associated with refuelling of petrol cars at service stations,
- address the sulphur content of fuels including marine fuels,
- preparatory work is also underway to investigate the feasibility of:
- improving the eco-design and reducing the emissions of domestic boilers and water heaters,
- reducing the solvent content of paints, varnishes and vehicle refinishing products,
- reducing the exhaust emissions of non-road mobile machinery and thereby maximise the benefit of lower sulphur non-road fuels already proposed by the Commission,
- The Commission also continues to push for substantial emissions reductions from ships at the International Maritime Organisation and it is committed to bringing forward proposals for Community measures should the IMO fail to deliver sufficiently ambitious proposals as foreseen in 2008.

The Commission is, however, committed to the aims of its Better Regulation initiative and the need for proposals to be underpinned by a comprehensive assessment of the impacts and benefits. In this regard and in accordance with the Treaty establishing the European Community, the Commission will continue to evaluate the need to bring forward new legislative proposals but reserves its right to decide if and when it would be appropriate to present any such proposal.

STATEMENT BY THE NETHERLANDS

The Netherlands has always supported the development of ambitious and effective European policy on air quality and will continue to do so in the future. It is, therefore, happy with the compromise agreed by the Council and the European Parliament and compliments the Parliament, the Commission and the Presidency on the results achieved. The new

Directive on ambient air quality marks significant progress for both the environment and public health.

As the Netherlands pointed out when the Common Position was drawn up, the air quality in our country is strongly influenced by transboundary developments and will therefore benefit enormously from an effective European approach. The Netherlands' main concern has been that the Directive should contain a balanced package of European and national measures, as well as realistic time limits to achieve the air quality targets. Only then will Member States be able to achieve the ambitious targets that have been set.

The Netherlands is pleased with the Commission's statement that it will present Community measures in good time. Timely, EU-wide compliance with the air quality standards will depend on sound European policy tackling pollution at the source. The Netherlands would especially point to the lack of data and prevailing uncertainties about emissions and concentrations of fine particulates ($PM_{2,5}$). It will of course make every effort to meet the objectives of the Directive by the target date. On the basis of the knowledge currently at our command, this will largely be feasible. The Dutch government is developing a National Air Quality Cooperation Programme to tackle locations where emission ceilings are persistently exceeded, so that, there too, air quality standards may be met by the target date.

The Netherlands is pleased that the Council and the European Parliament concluded their second reading in time for the Directive to take effect as of early 2008. This is essential for our own national programme, as well as actions in the countries around us. The Netherlands will work hard to ensure that the national cooperation programme and all local and regional measures are sufficient.

II. Appendix 2: pollutant emission trends by country

II.1. $PM_{2.5}$

II.1.1. Data availability

Emission data are available online on the European Environmental agency website. These data are based on National Emissions reported to the Convention on Long-range Transboundary Air Pollution (LRTAP Convention). Emissions from several air pollutants are estimated annually in European countries. Raw emission data were downloaded from EEA website. Availability of $PM_{2.5}$ data is summarized below:

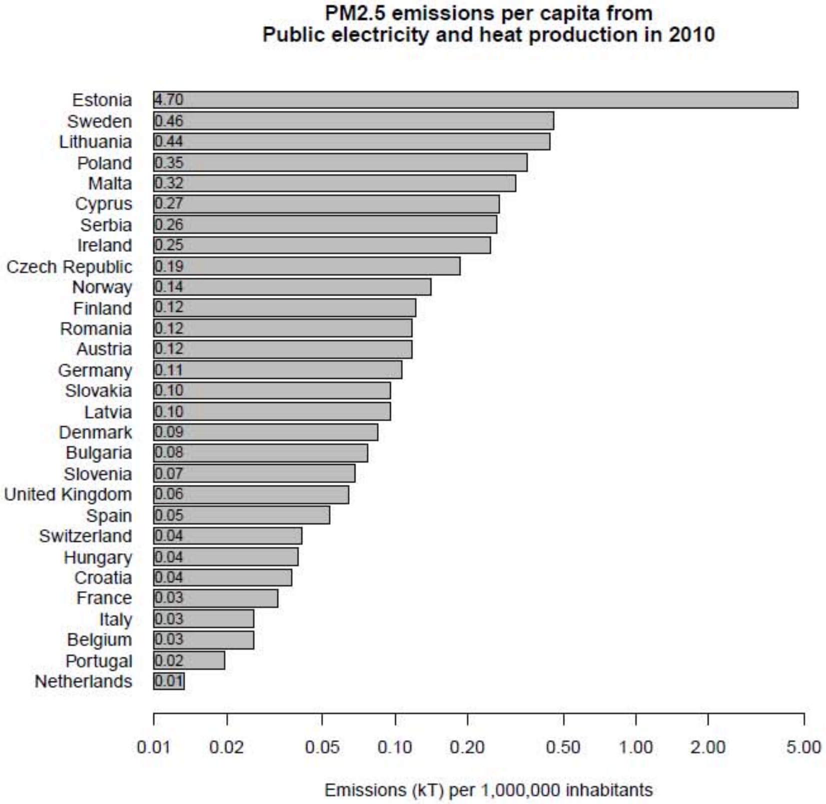
Country	From	To	Missing	x = no data										
				EP	EI	NRT	RT	CIH	IP	S	A	W	N	O
Albania	1990	2009	-			x				x		x		x
Austria	1990	2010	1991-1994; 1996-1999										x	
Belgium	2000	2010	2001-2004							x			x	
Bulgaria	1990	2010	-							x	x		x	x
Croatia	1990	2010	-							x		x	x	
Cyprus	2000	2010	-							x			x	
Czech Republic	2010	2010	-							x			x	
Denmark	2000	2010	-										x	x
Estonia	1990	2010	-										x	x
Finland	1980	2010	-										x	x
France	1990	2010	-											x
Germany	1995	2010	-											x
Hungary	2010	2010	-										x	x

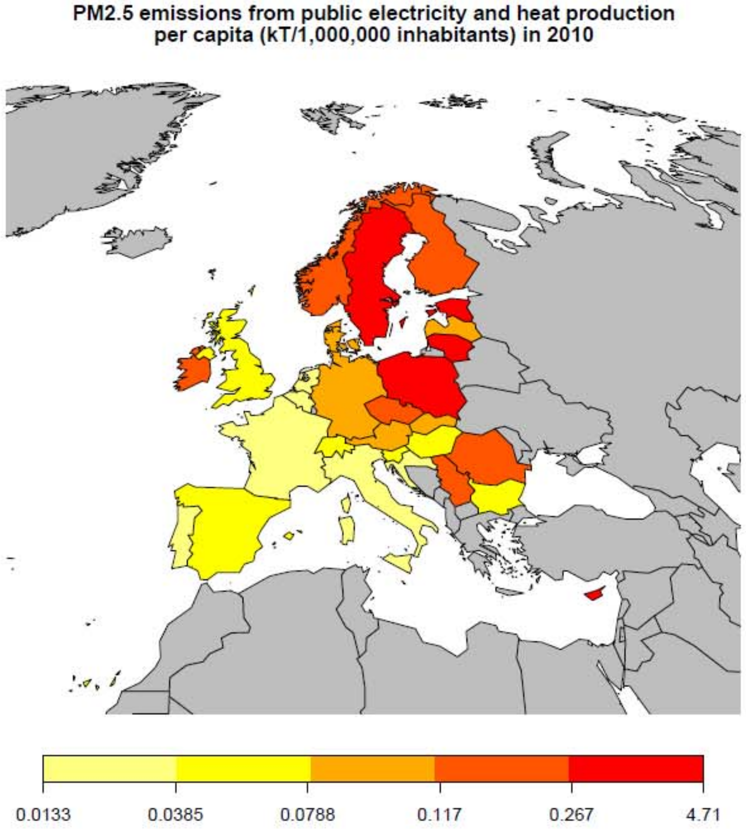
Country	From	To	Missing	EP	EI	NRT	RT	CIH	IP	S	A	W	N	O
				x = no data										
Ireland	1990	2010	-							x			x	
Italy	1990	2010	-							x				x
Iceland	2010	2010	-	x	x	x	x	x	x	x	x	x		x
Latvia	2000	2010	-							x			x	x
Lithuania	2008	2010	-						x	x	x	x	x	x
Malta	2000	2010	-						x	x			x	x
Montenegro	1990	2009	-							x		x		x
Netherlands	1990	2010	-										x	
Norway	1980	2010	1981-1986; 1988										x	
Poland	2005	2010	2006-2008							x				
Portugal	1990	2010	-							x			x	x
Romania	2005	2010	-				x			x			x	x
Serbia	2000	2010	-									x		x
Slovakia	2000	2010	-							x	x	x	x	x
Slovenia	2000	2010	-										x	x
Spain	2000	2010	-							x			x	x
Sweden	1990	2010	-										x	x
Switzerland	1990	2010	-											
United Kingdom	1980	2010	-											

Abbreviation list:

EP: energy production and distribution
EI: energy use in industry
NRT: non-road transport
RT: road transport
CIH: commercial, institutional and household energy use
IP: industrial processes
S: solvent and product use
A: agriculture
W: waste
N: natural emissions
O: other emissions

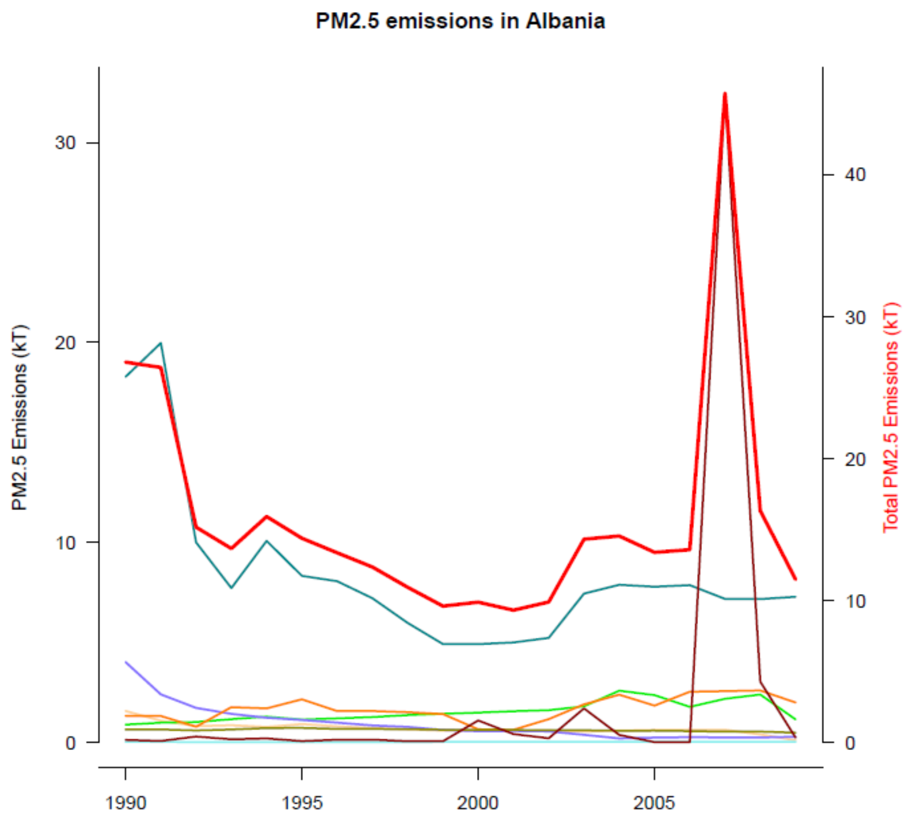
II.1.2. $PM_{2.5}$ emissions per capita from Public electricity and heat production sector in 2010

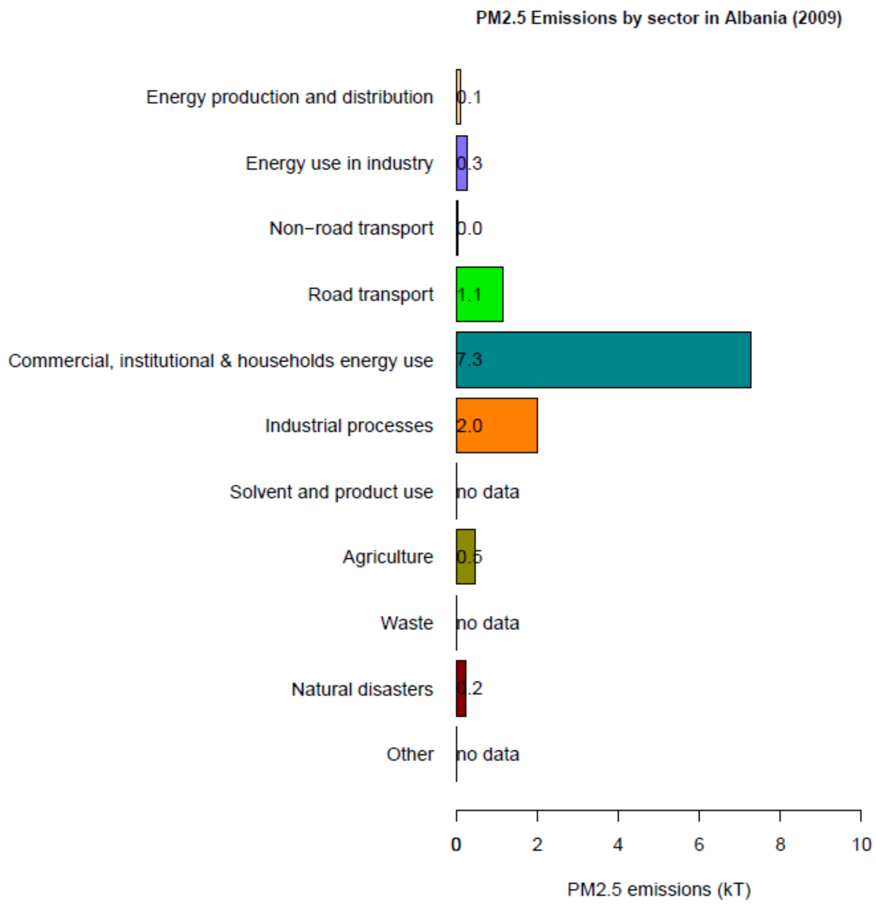




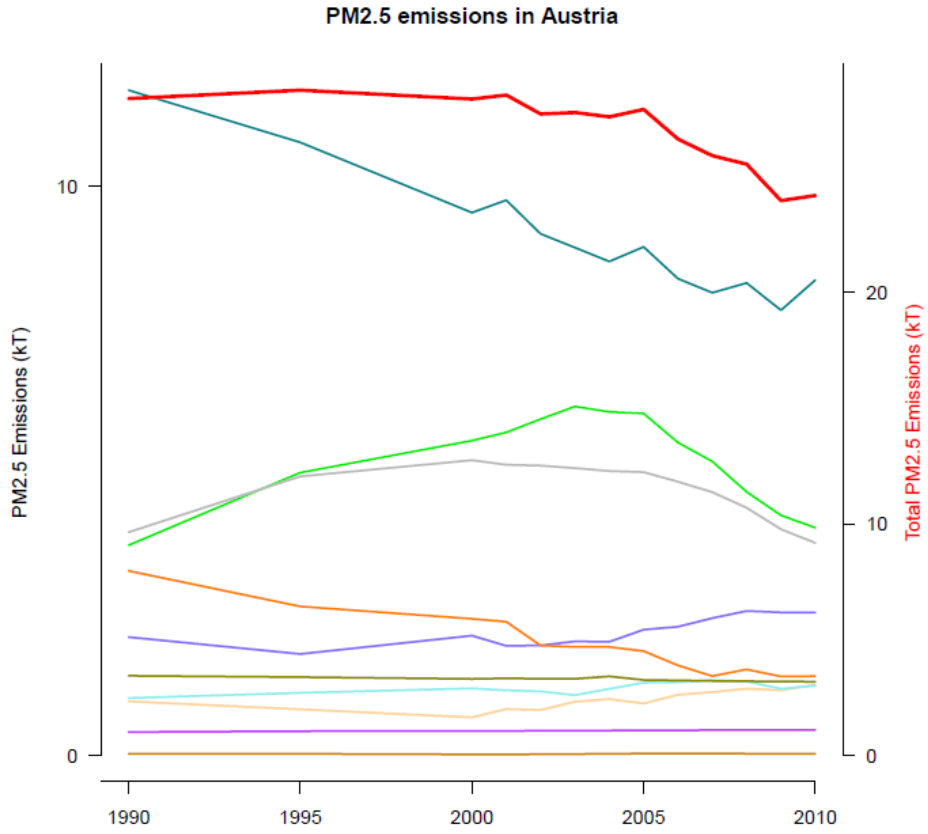
II.1.3. $PM_{2.5}$ emission trends by sector (left) and distribution of emissions by sector for the last available year (right)

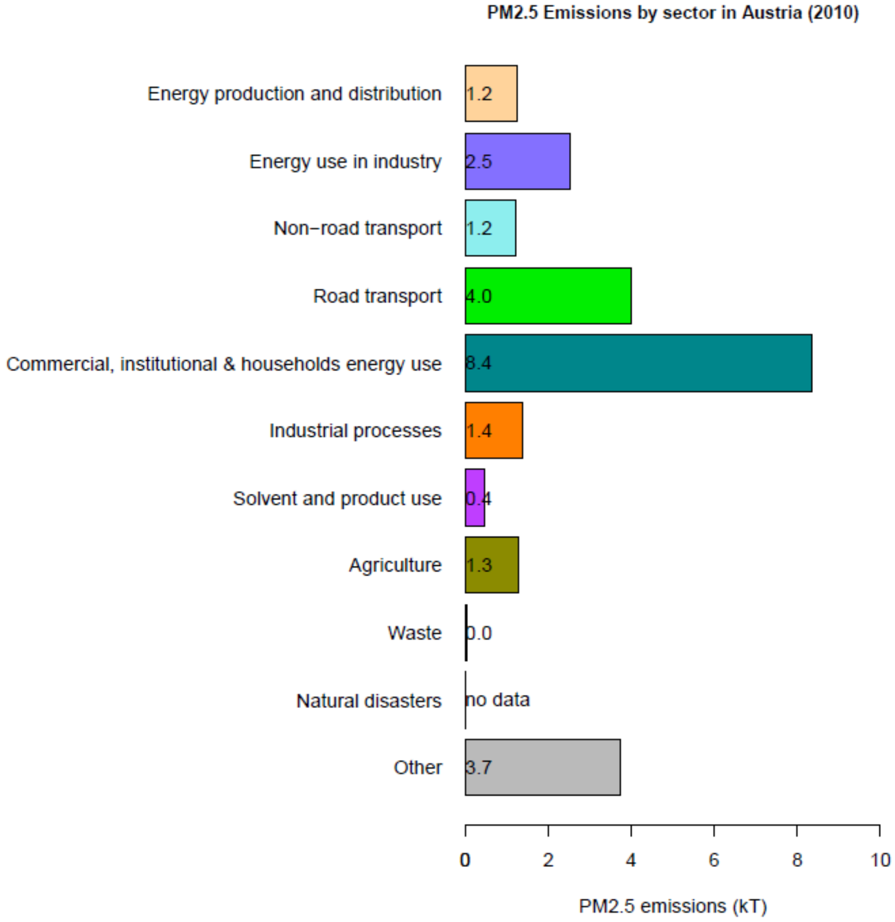
Albania ($PM_{2.5}$)



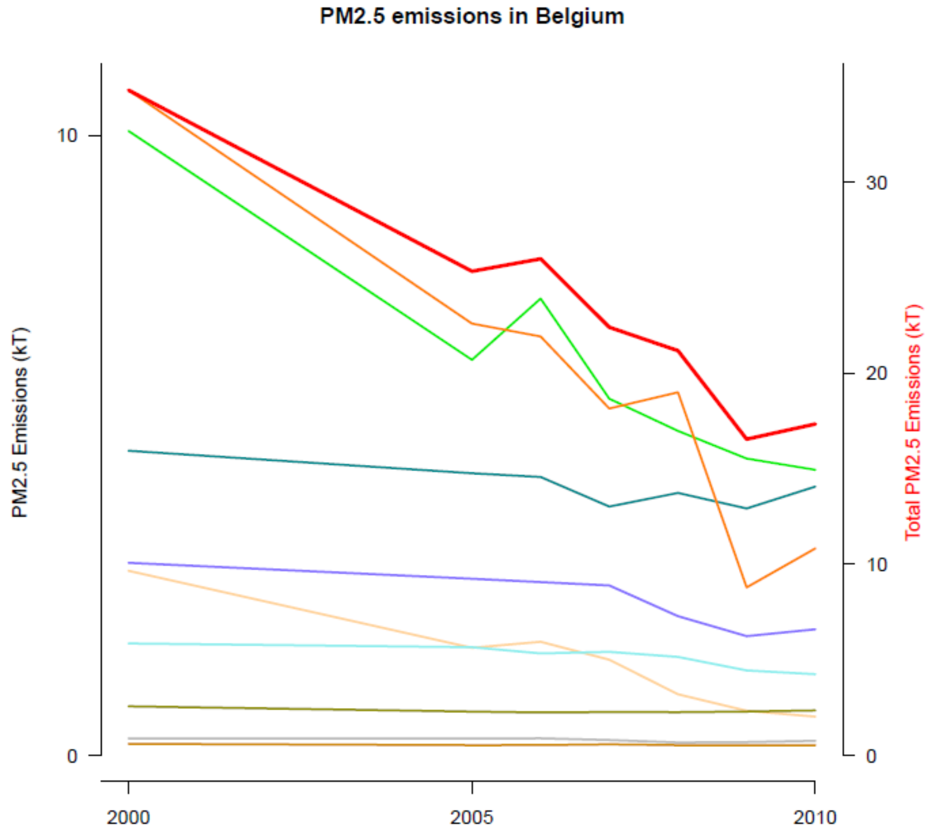


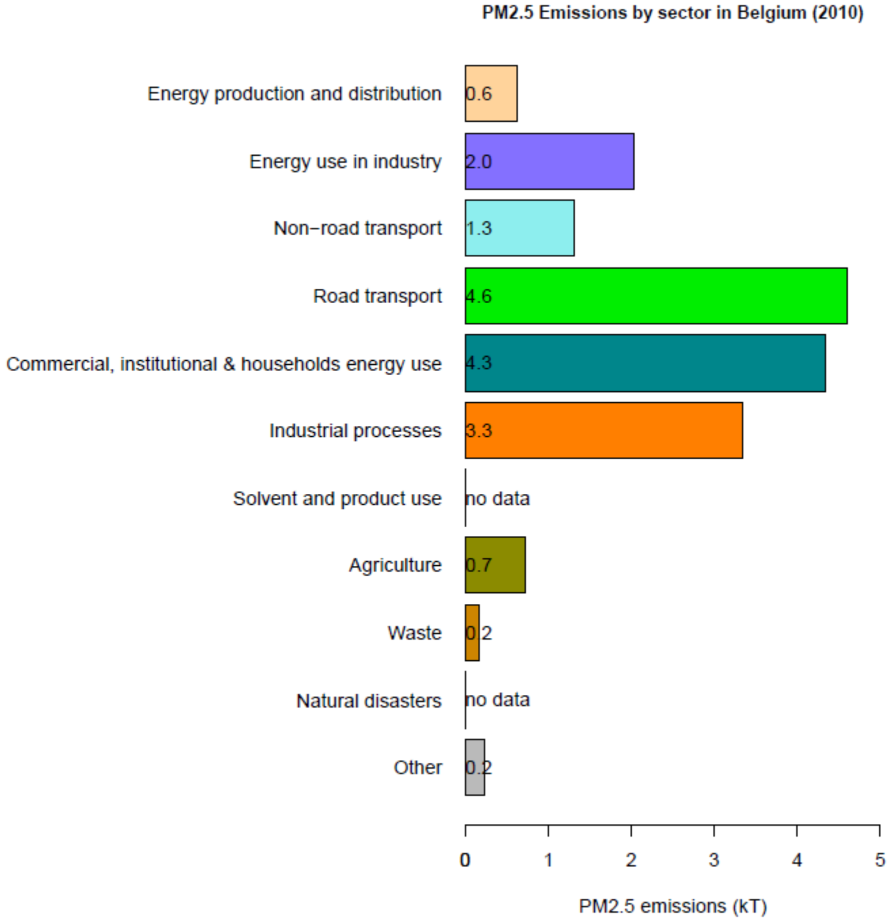
Austria ($PM_{2.5}$)



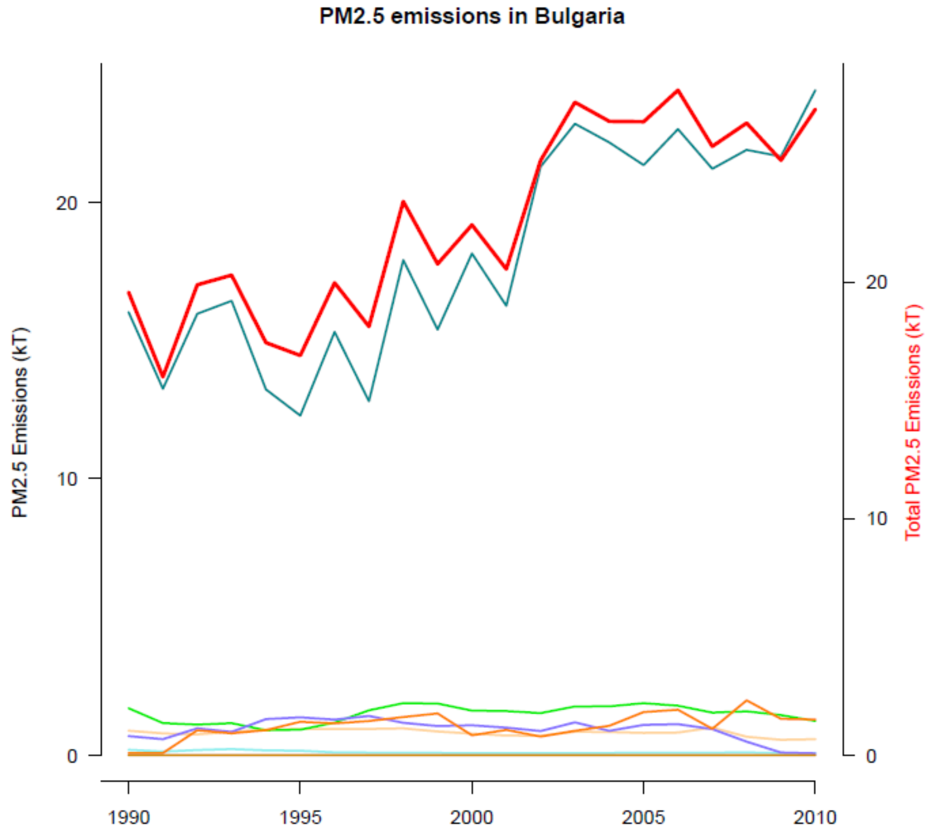


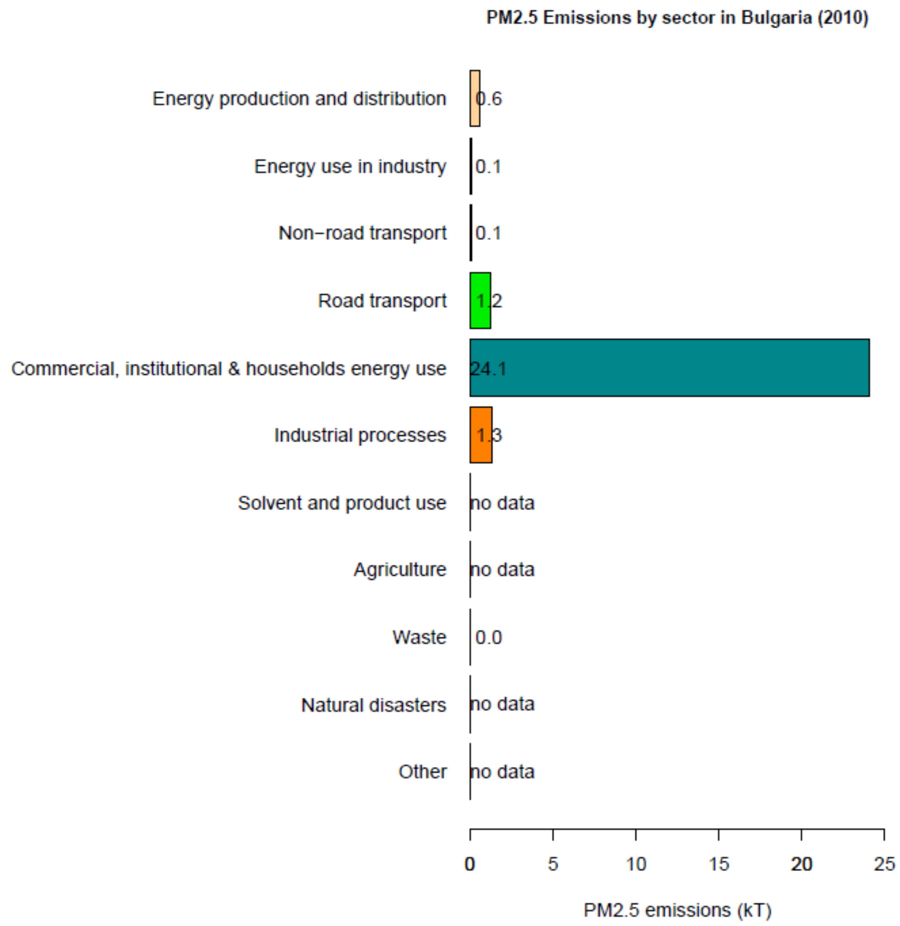
Belgium ($PM_{2.5}$)



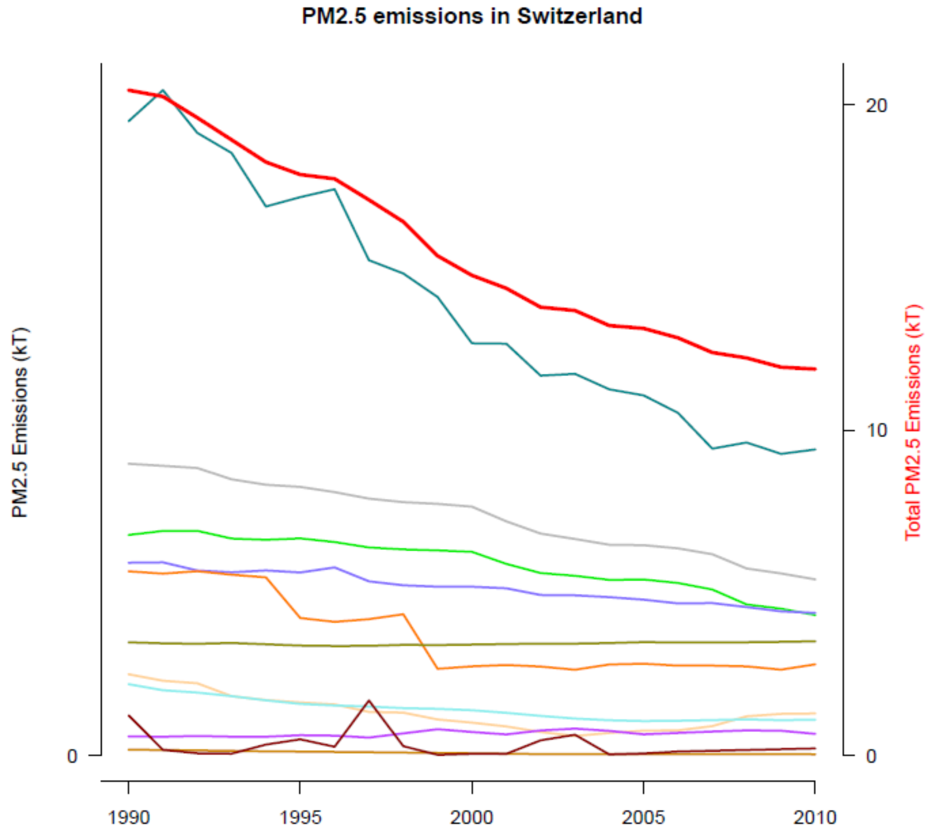


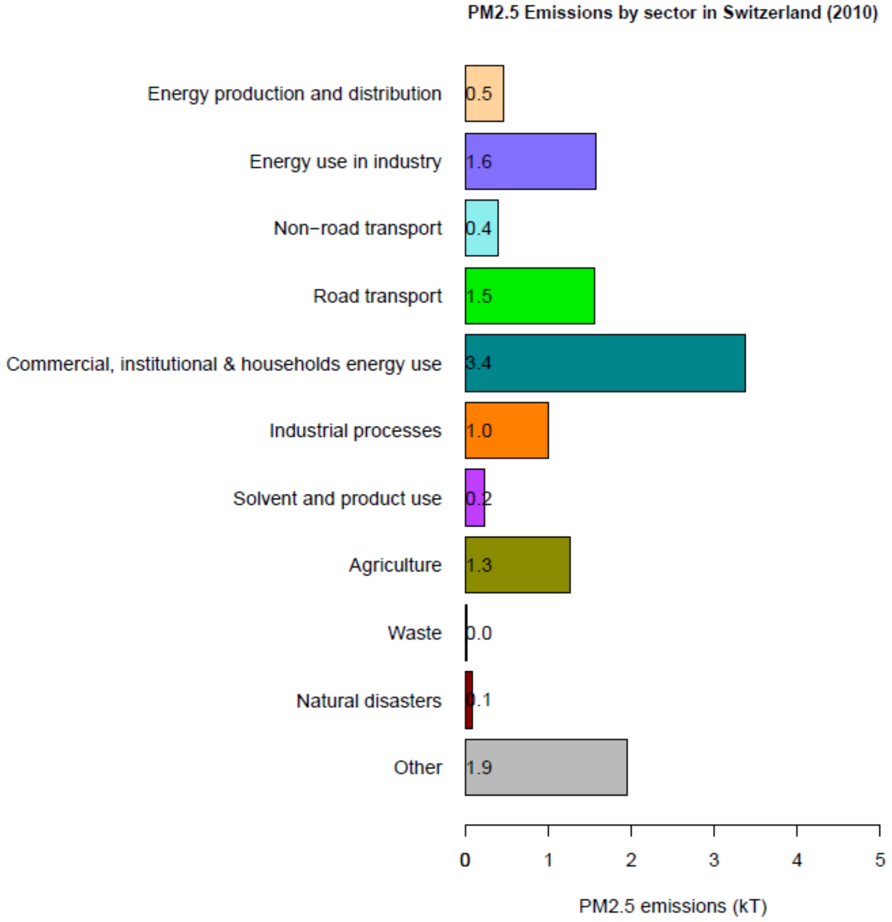
Bulgaria ($PM_{2.5}$)



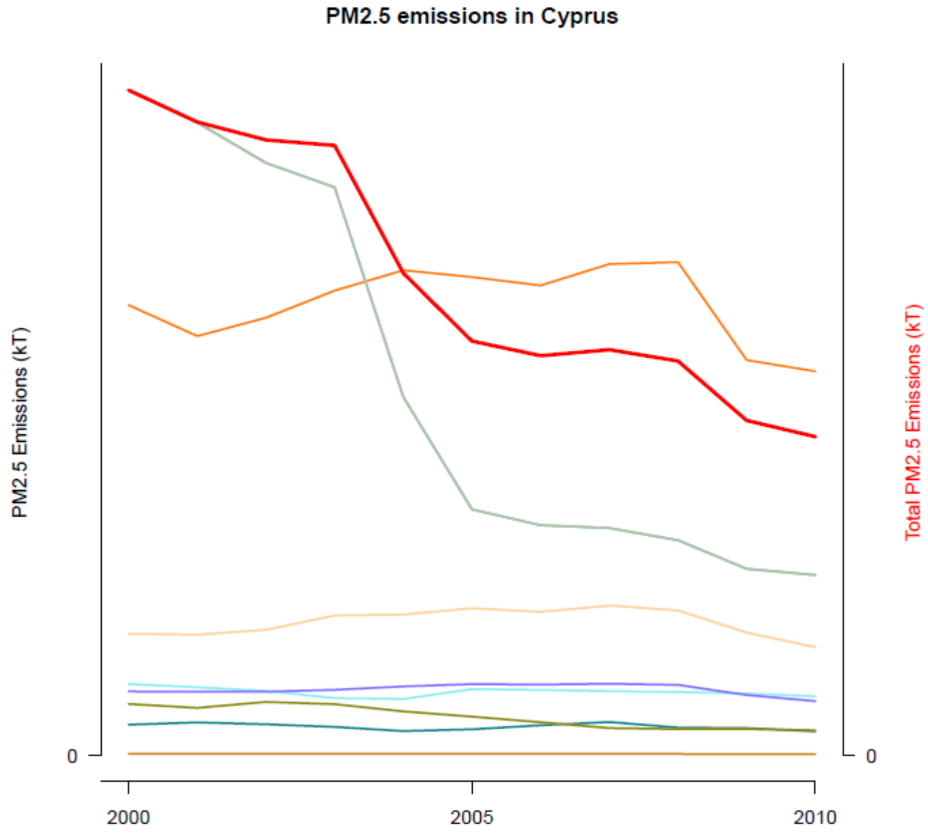


Switzerland ($PM_{2.5}$)

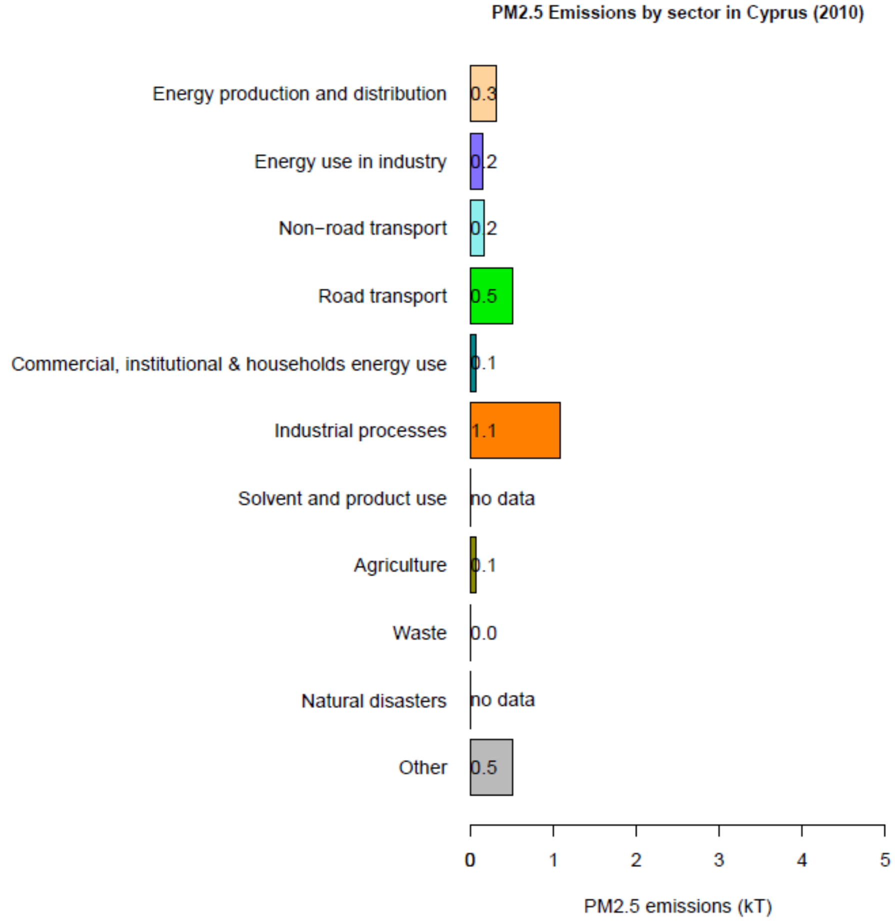




Cyprus ($PM_{2.5}$)

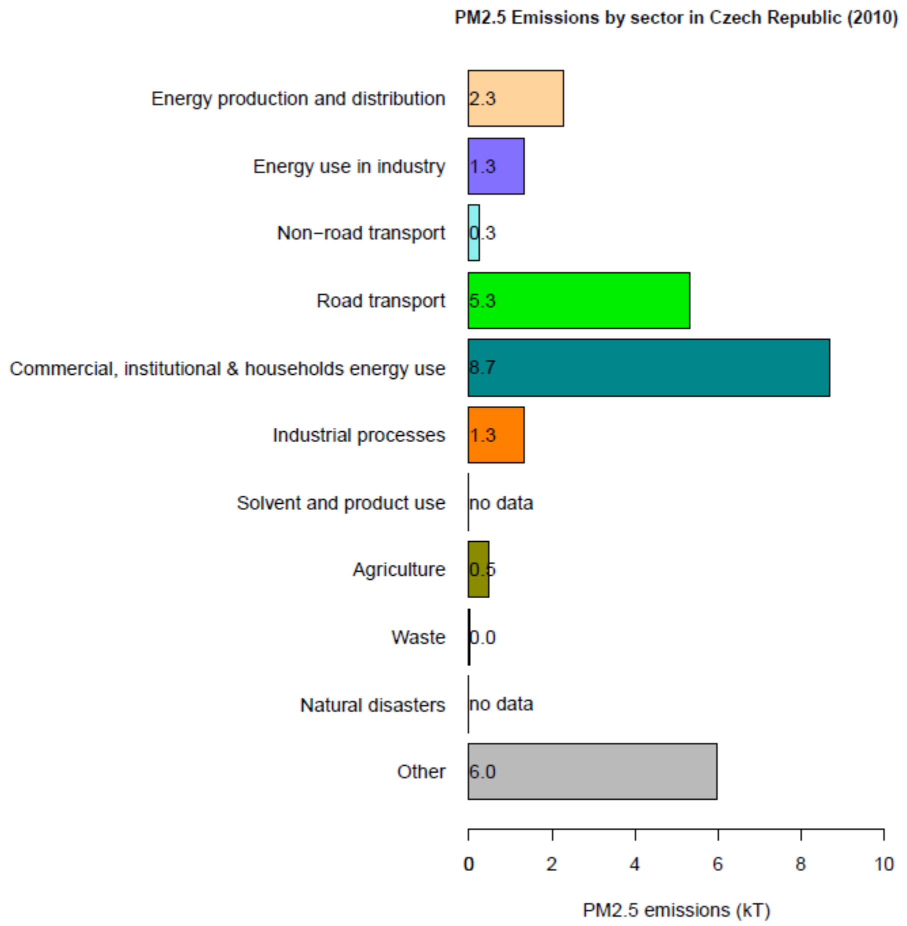


PM2.5 emissions in Cyprus
PM2.5 Emissions (kT)
Total PM2.5 Emissions (kT)
0
0
2000
2005
2010

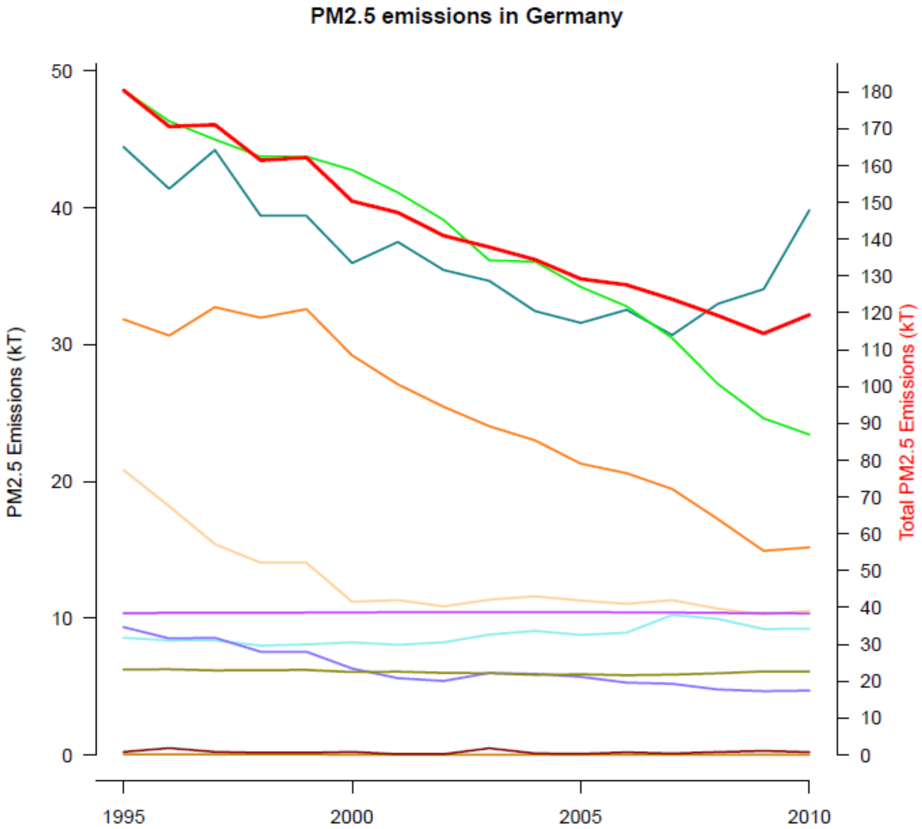


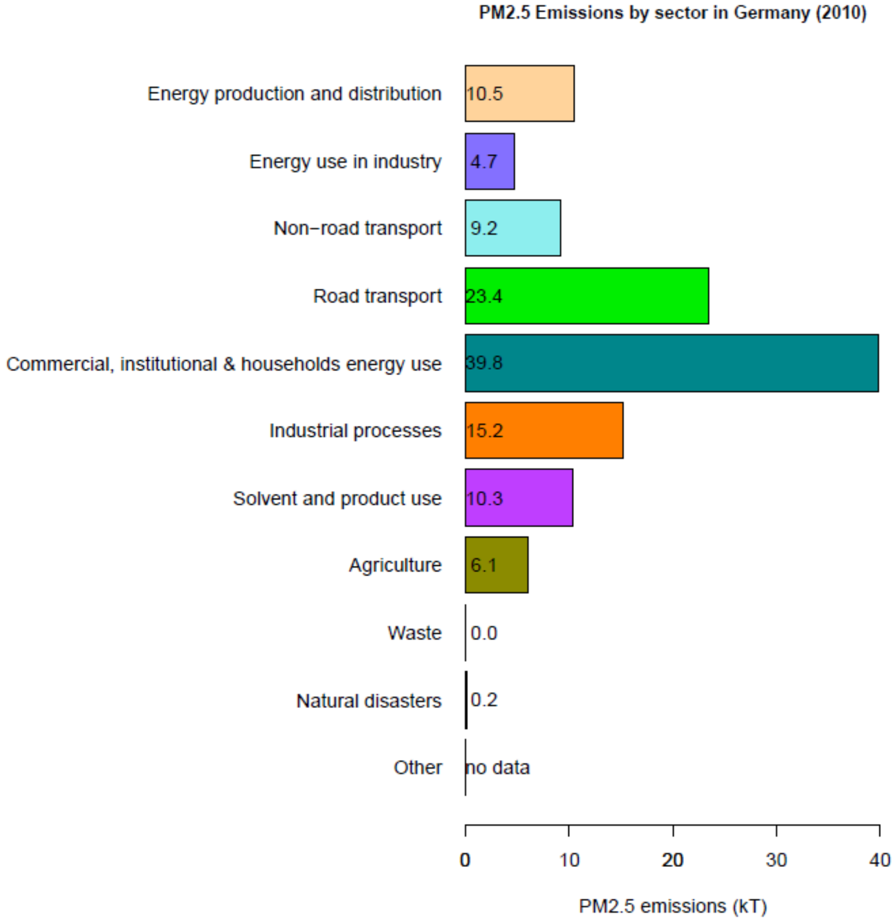
PM2.5 Emissions by sector in Cyprus (2010)
Energy production and distribution
0.3
Energy use in industry
0.2
Non−road transport
0.2
Road transport
0.5
Commercial, institutional & households energy use
0.1
Industrial processes
1.1
Solvent and product use
no data
Agriculture
0.1
Waste
0.0
Natural disasters
no data
Other
0.5
0
1
2
3
4
5
PM2.5 emissions (kT)

Czech Republic ($PM_{2.5}$)

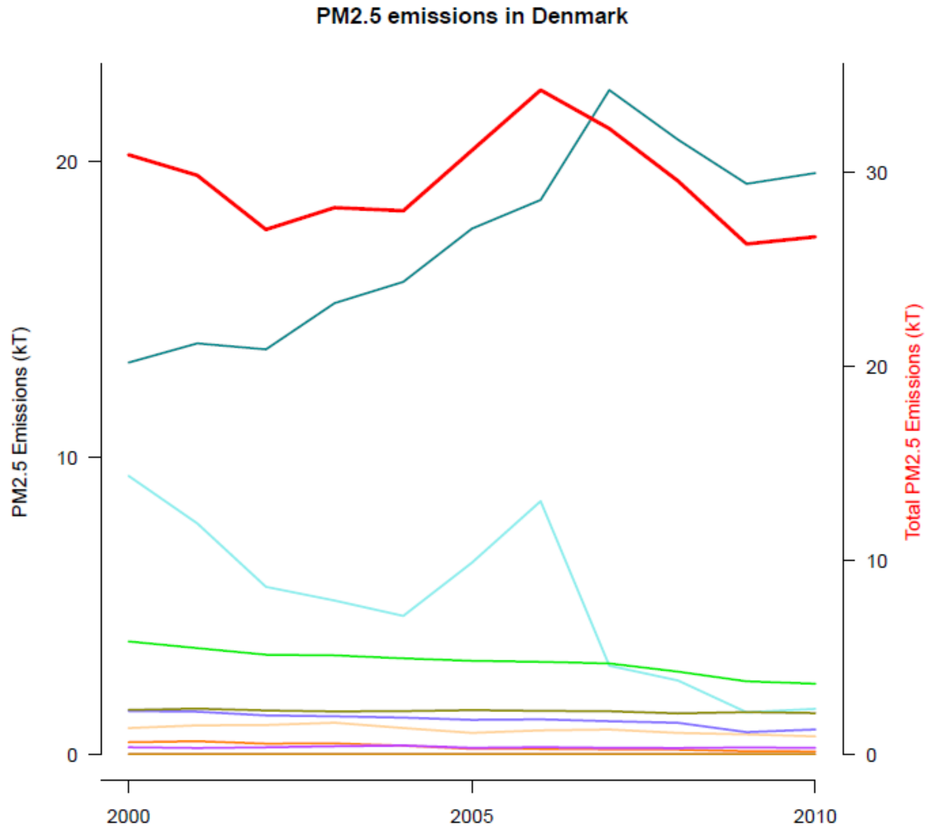


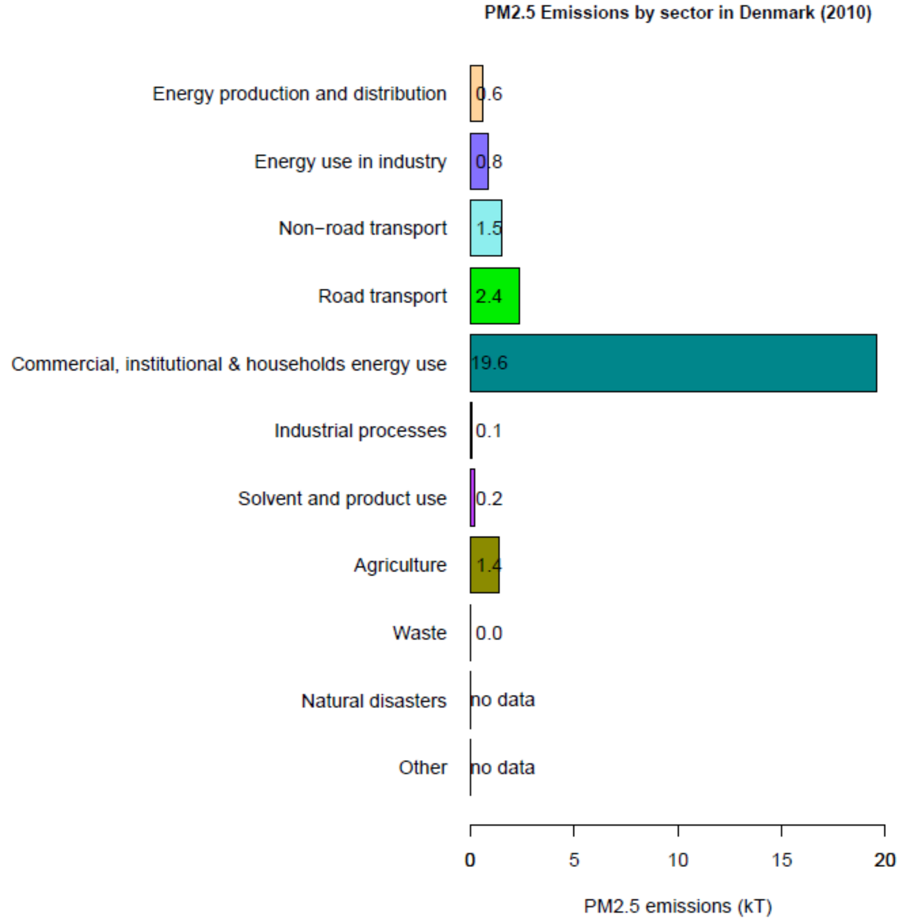
Germany ($PM_{2.5}$)



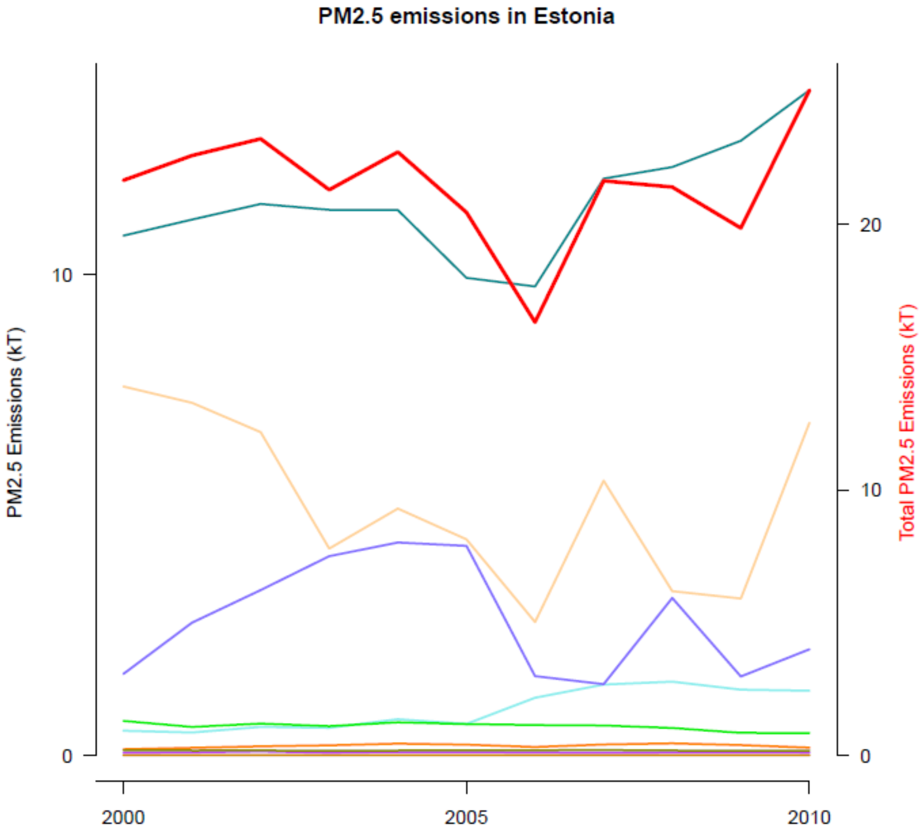


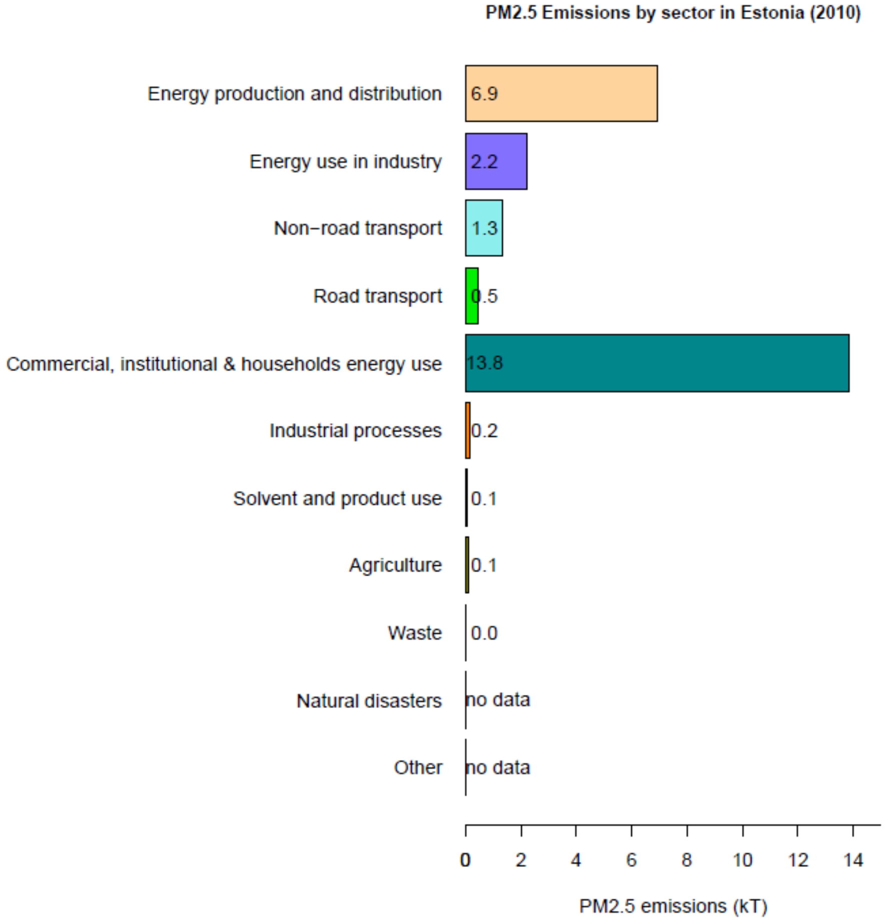
Denmark (PM$_{2.5}$)



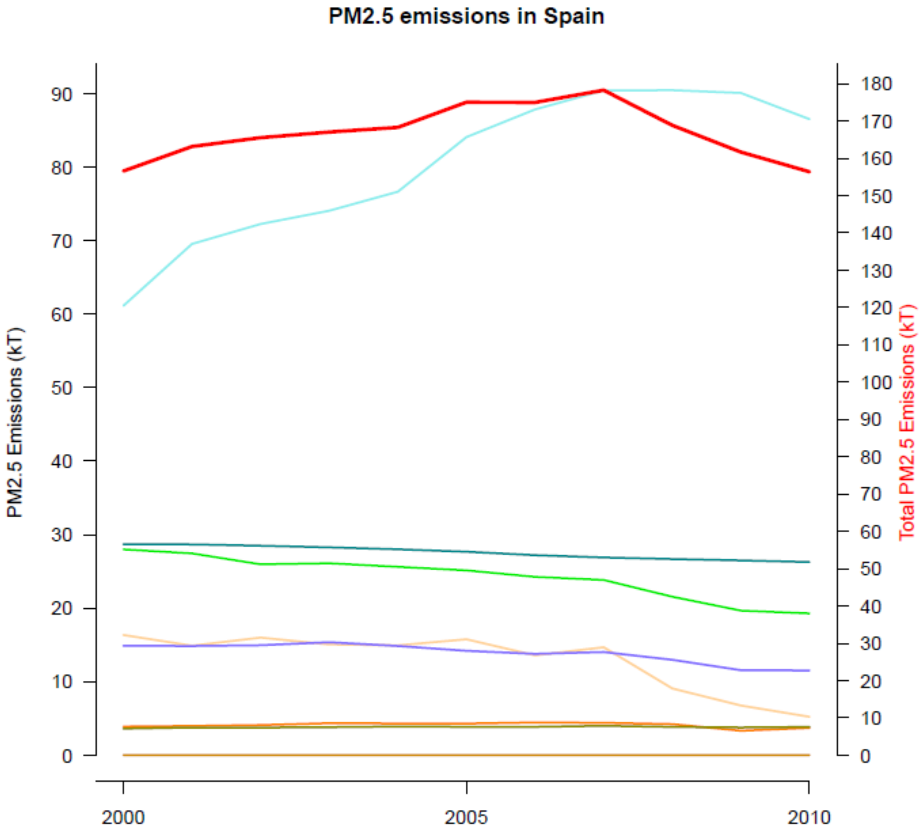


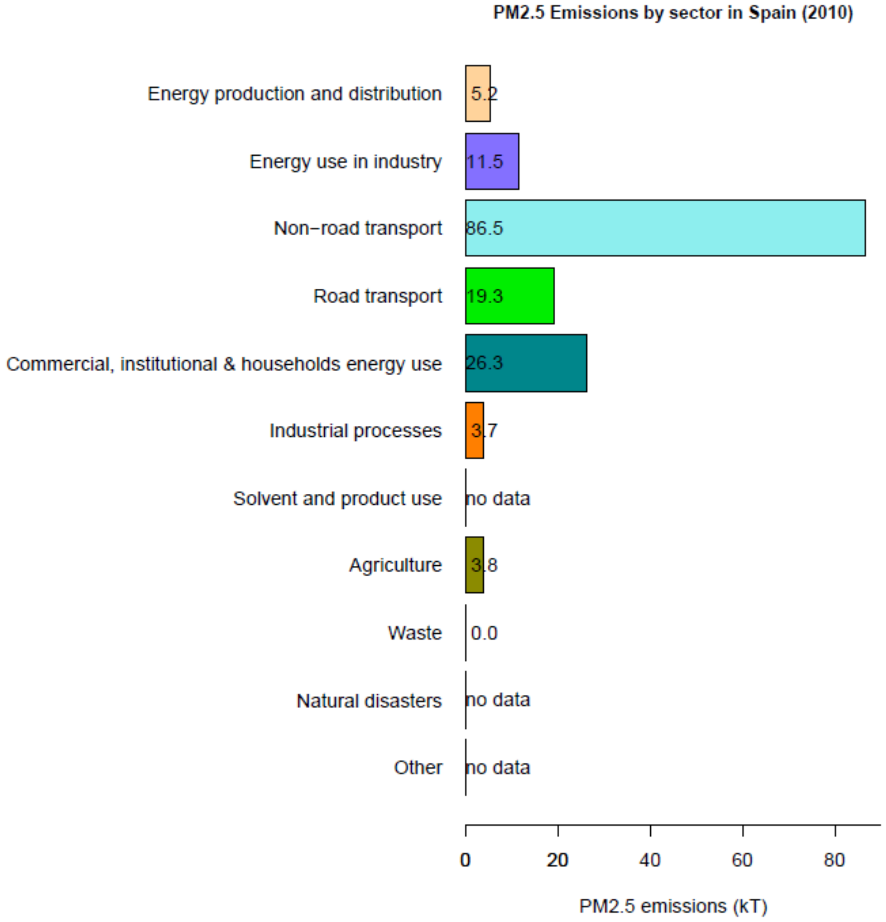
Estonia ($PM_{2.5}$)



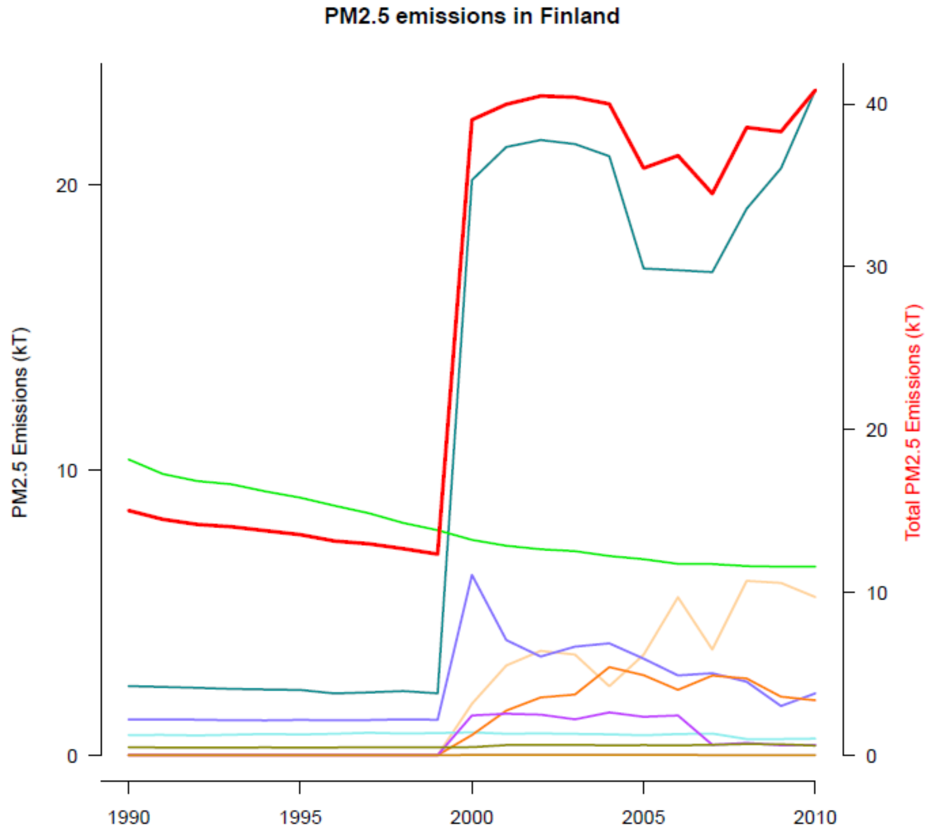


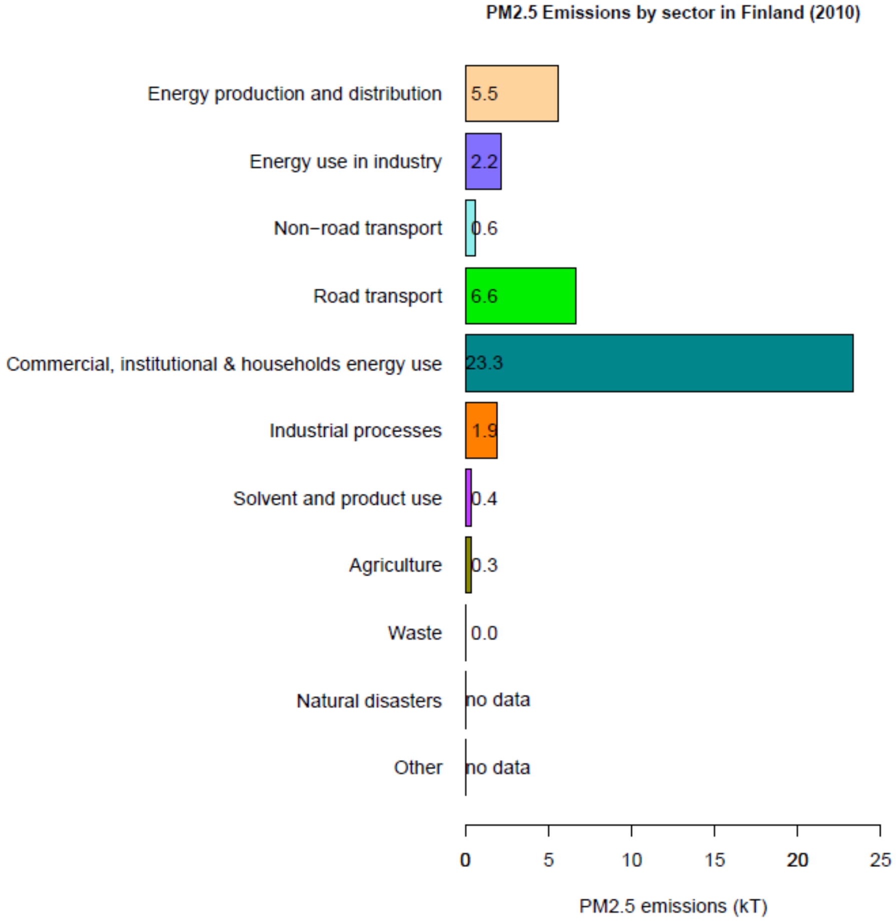
Spain ($PM_{2.5}$)


PM2.5 emissions in Spain
PM2.5 Emissions (kT)
Total PM2.5 Emissions (kT)
0
10
20
30
40
50
60
70
80
90
0
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
2000
2005
2010


PM2.5 Emissions by sector in Spain (2010)
Energy production and distribution
5.2
Energy use in industry
11.5
Non−road transport
86.5
Road transport
19.3
Commercial, institutional & households energy use
26.3
Industrial processes
3.7
Solvent and product use
no data
Agriculture
3.8
Waste
0.0
Natural disasters
no data
Other
no data
0
20
40
60
80
PM2.5 emissions (kT)

Finland ($PM_{2.5}$)





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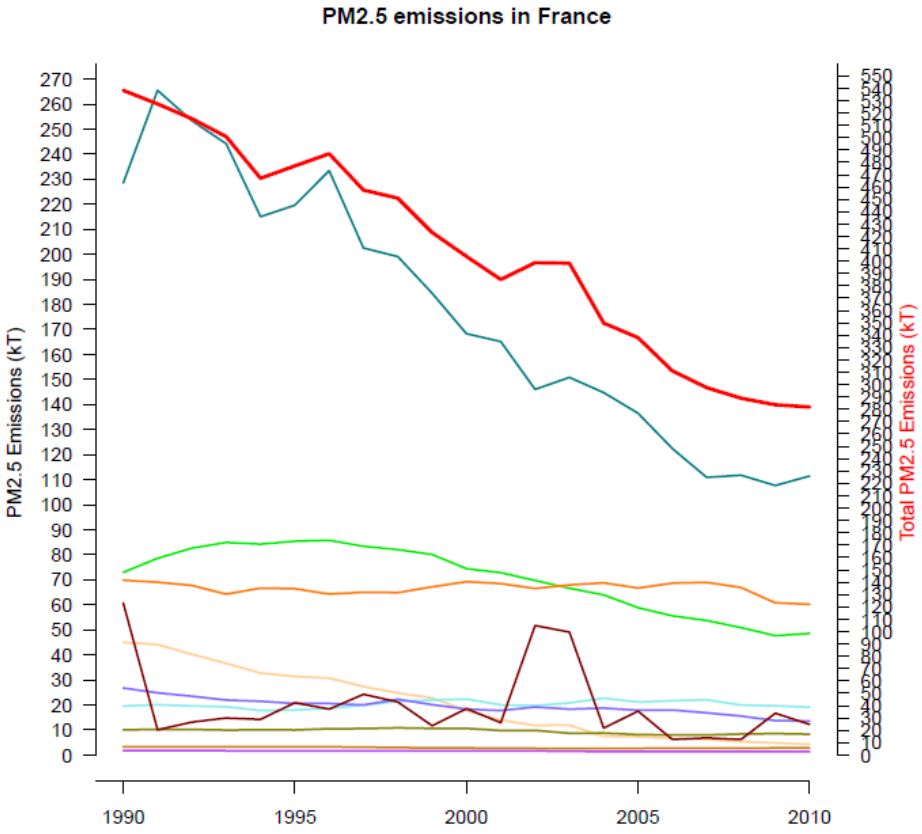
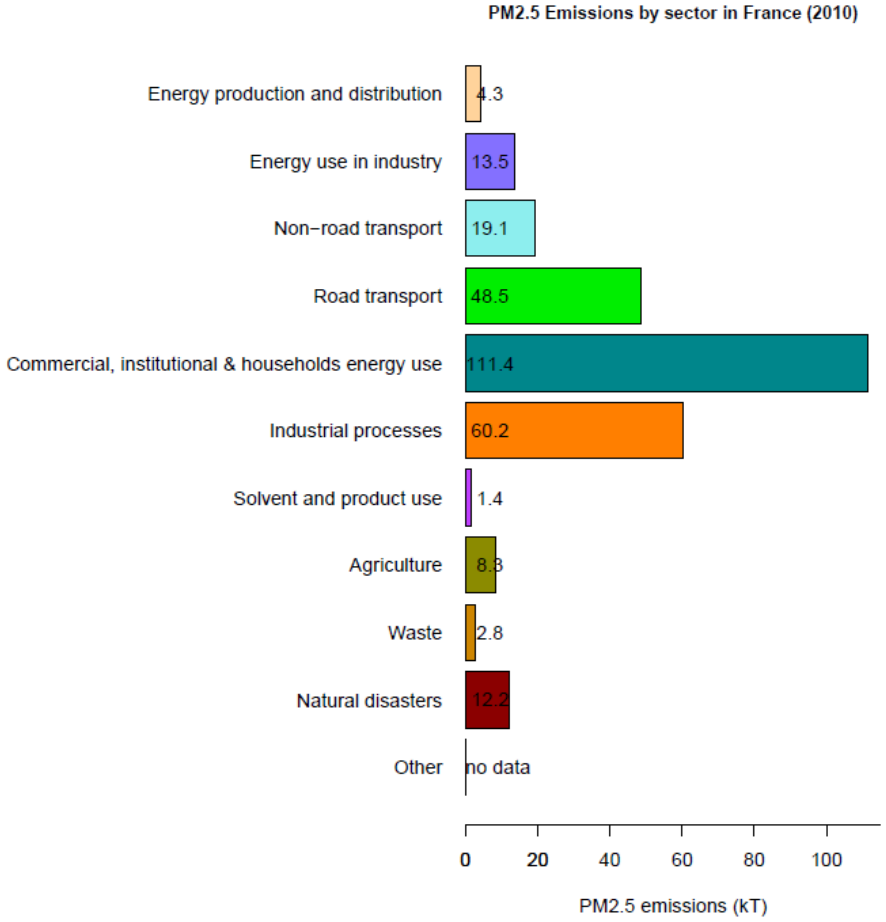
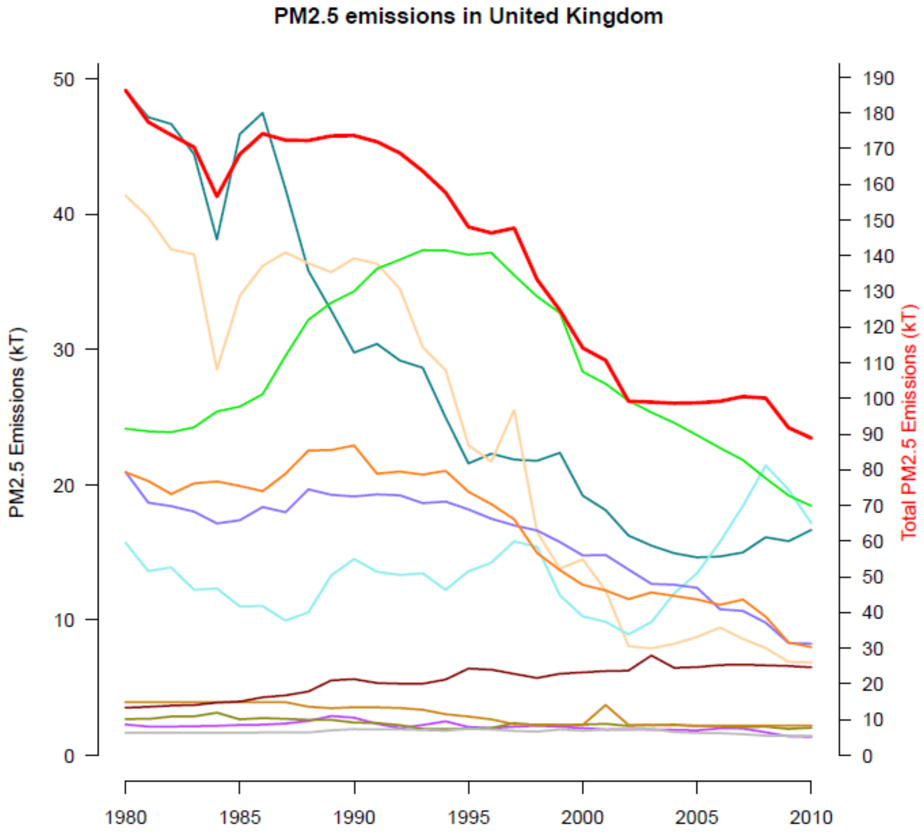
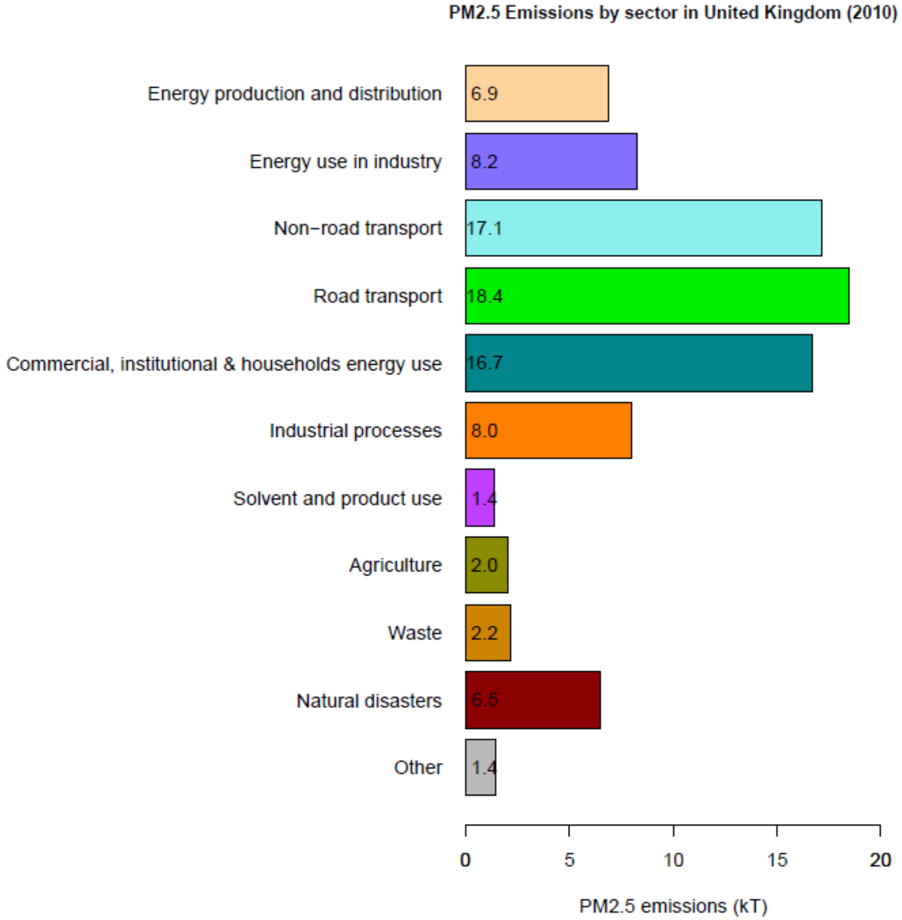
France (PM$_{2.5}$)



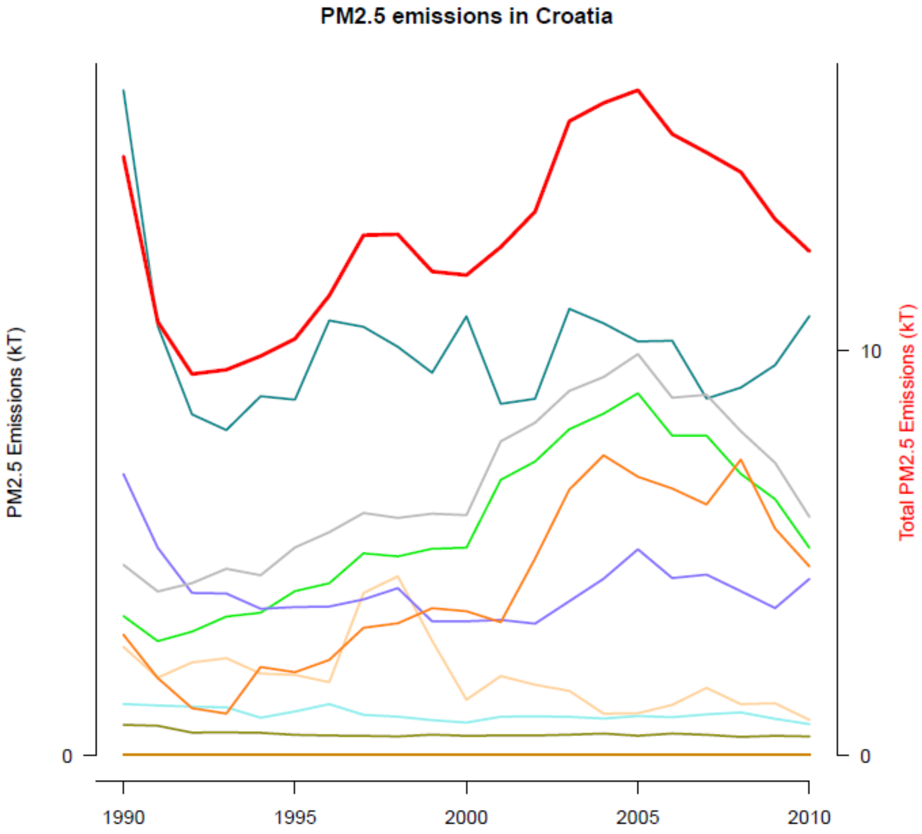


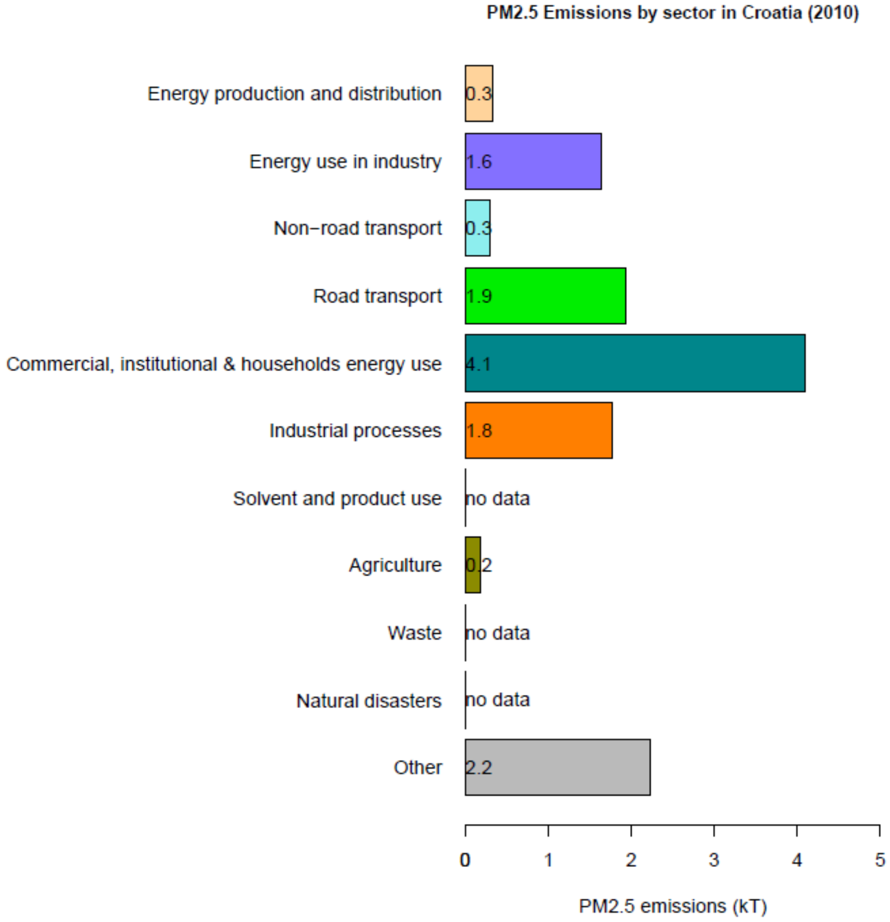
The United Kingdom ($PM_{2.5}$)



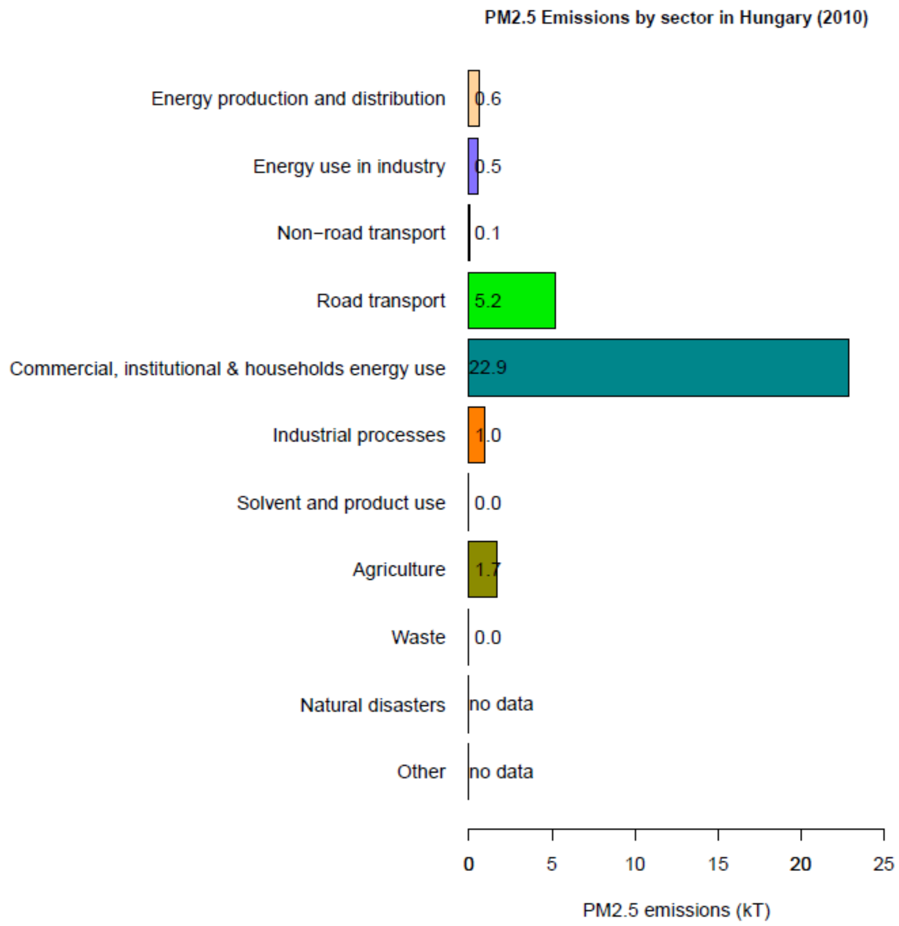


Croatia ($PM_{2.5}$)

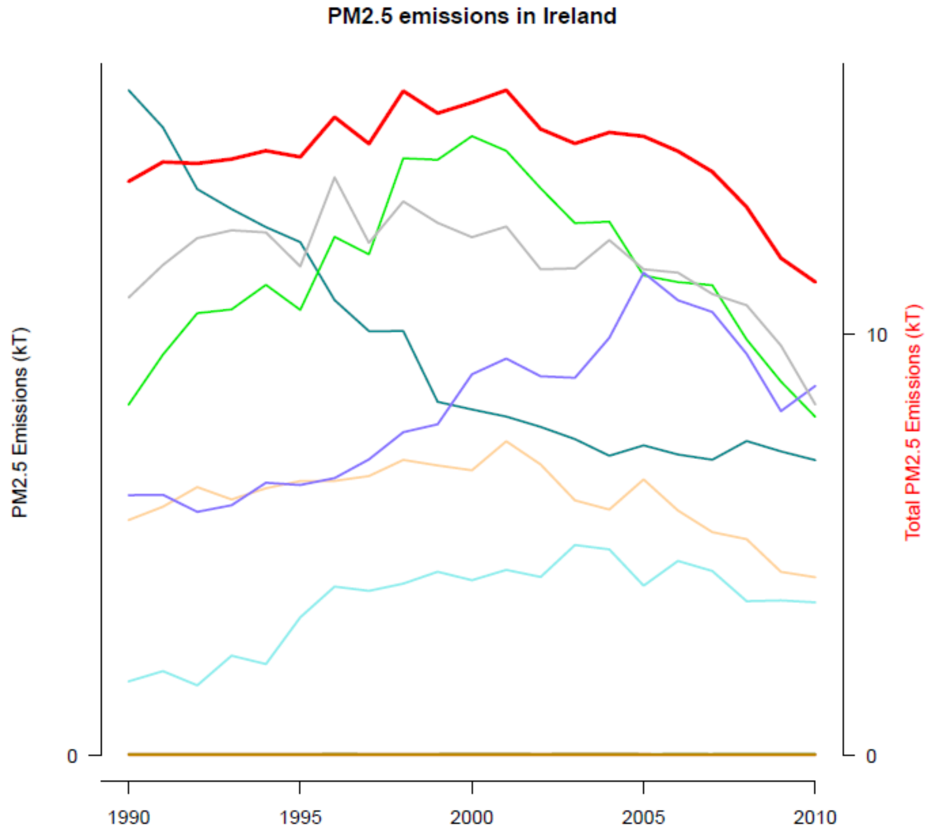


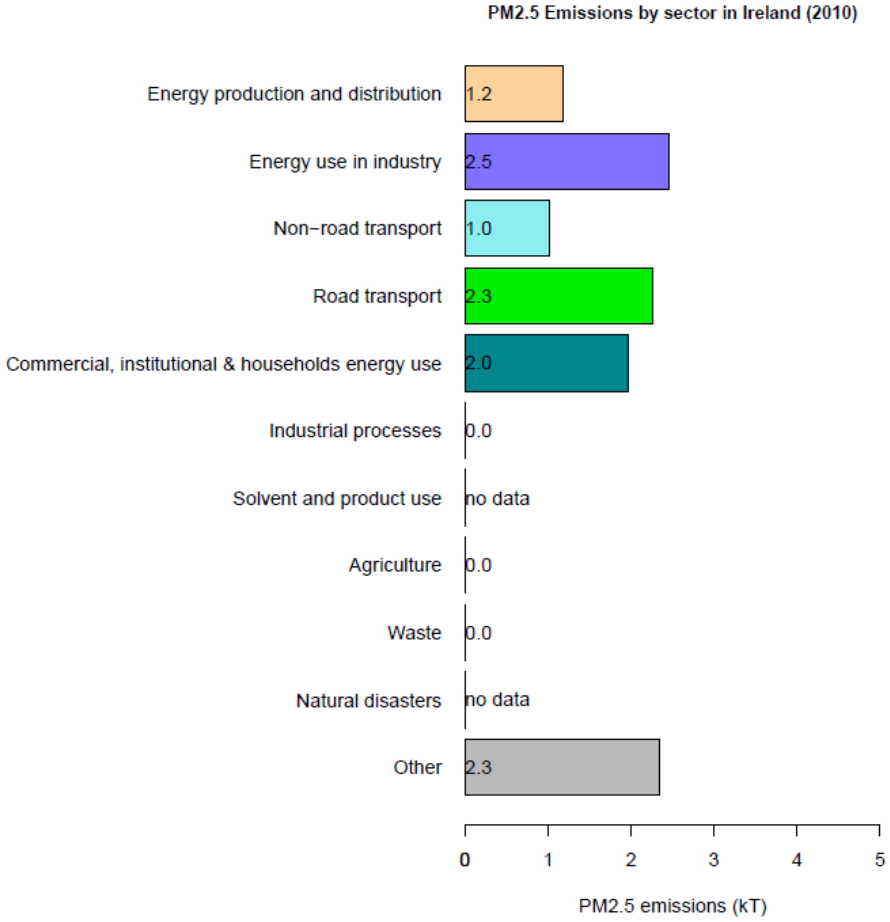


Hungary ($PM_{2.5}$)

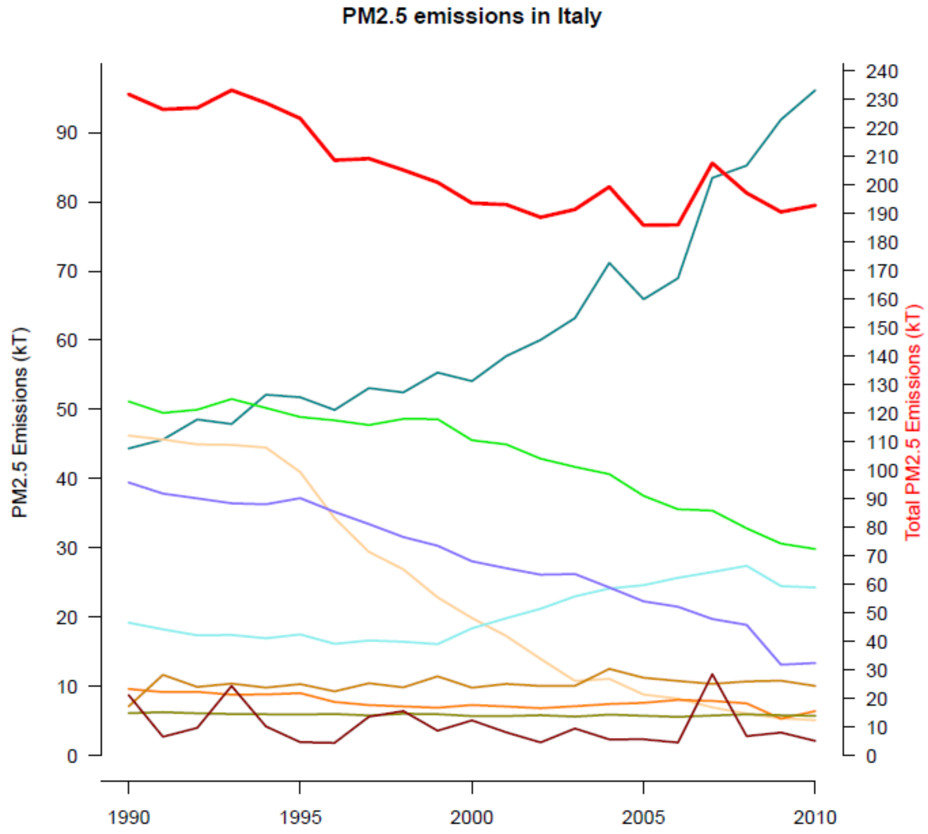


Ireland ($PM_{2.5}$)

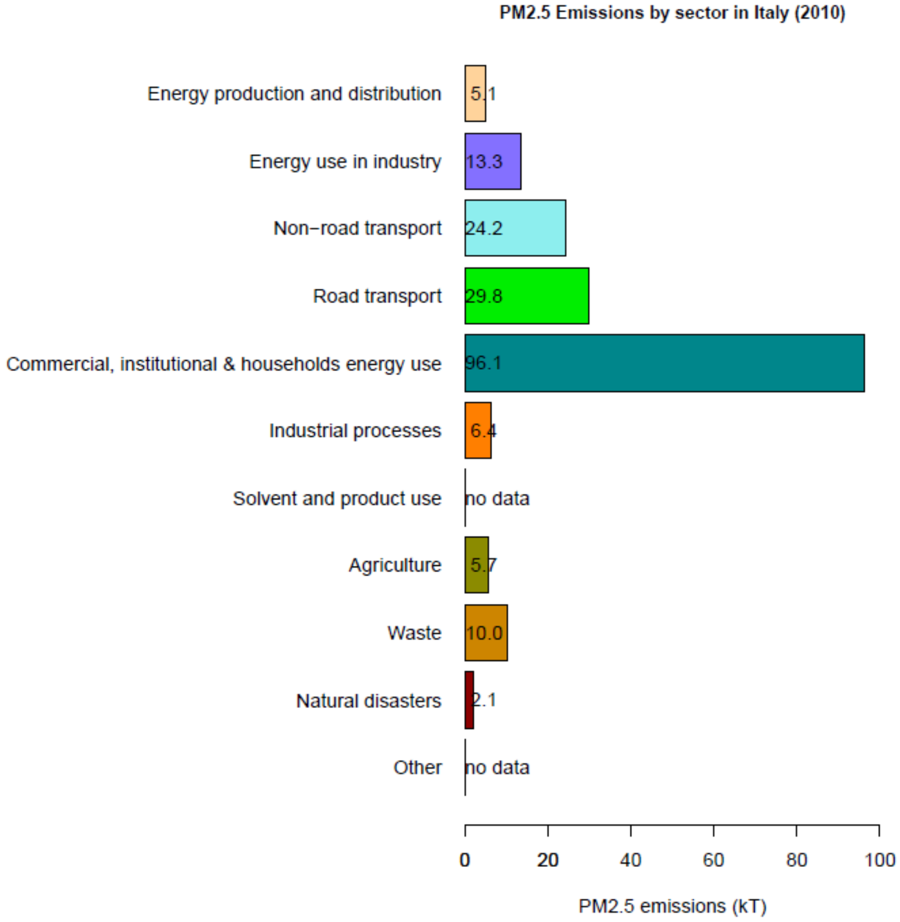




Italy ($PM_{2.5}$)

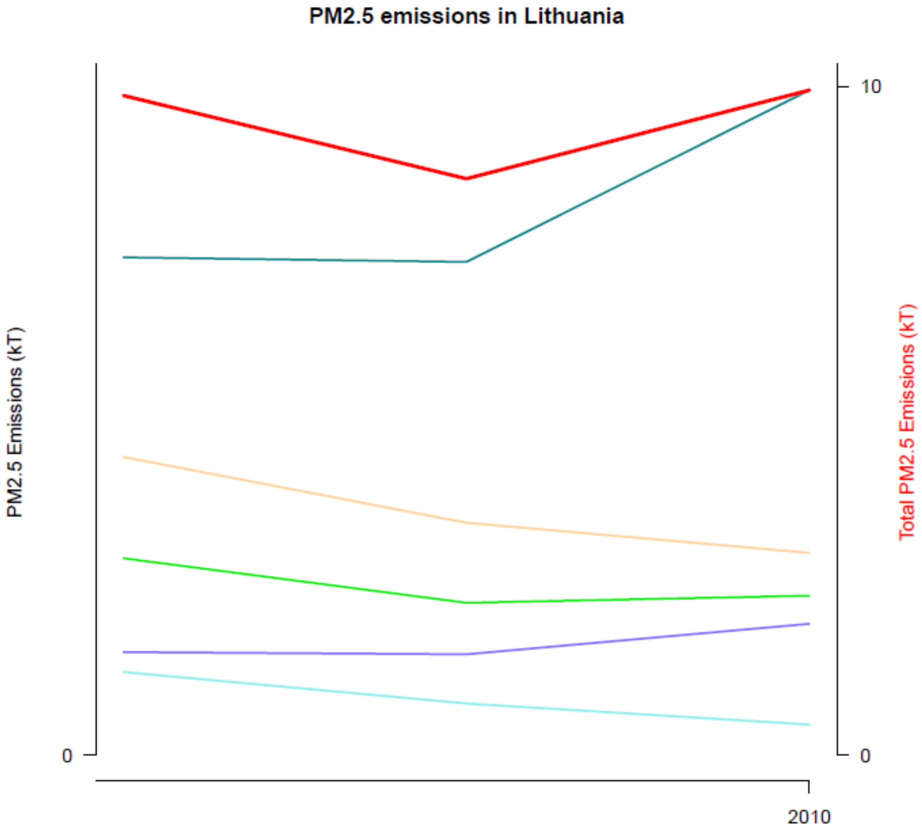


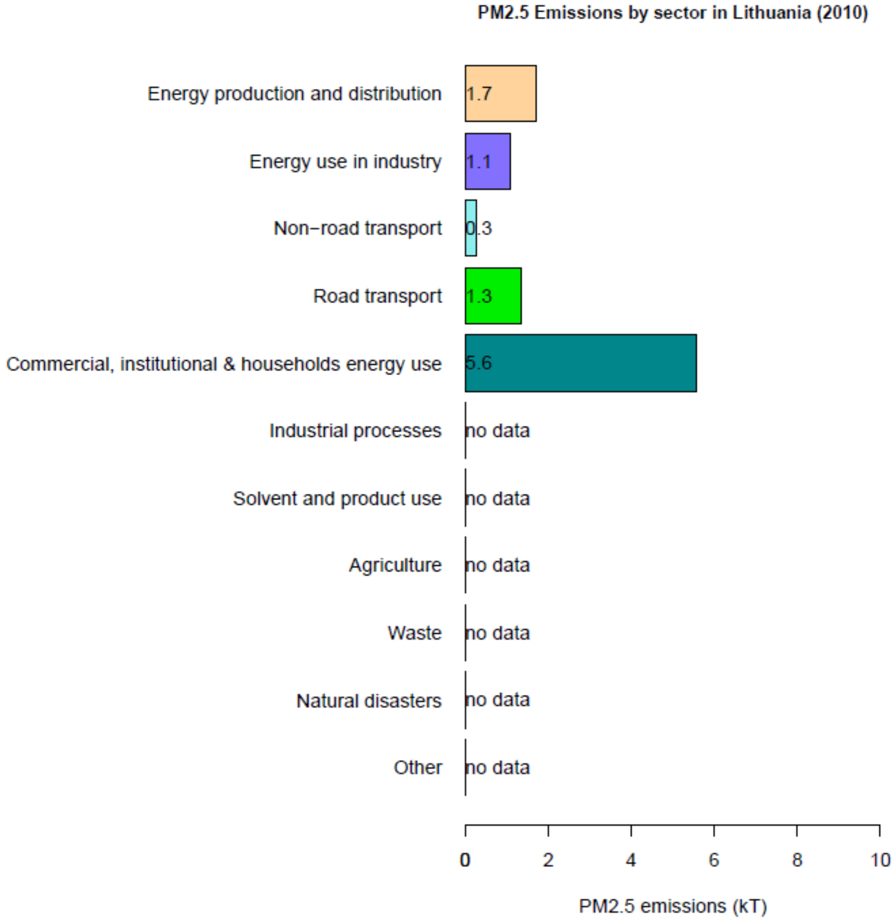
PM2.5 emissions in Italy
PM2.5 Emissions (kT)
Total PM2.5 Emissions (kT)
0
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
190
200
210
220
230
240
1990
1995
2000
2005
2010



PM2.5 Emissions by sector in Italy (2010)
Energy production and distribution
5.1
Energy use in industry
13.3
Non−road transport
24.2
Road transport
29.8
Commercial, institutional & households energy use
96.1
Industrial processes
6.4
Solvent and product use
no data
Agriculture
5.7
Waste
10.0
Natural disasters
2.1
Other
no data
0
20
40
60
80
100
PM2.5 emissions (kT)

Lithuania ($PM_{2.5}$)





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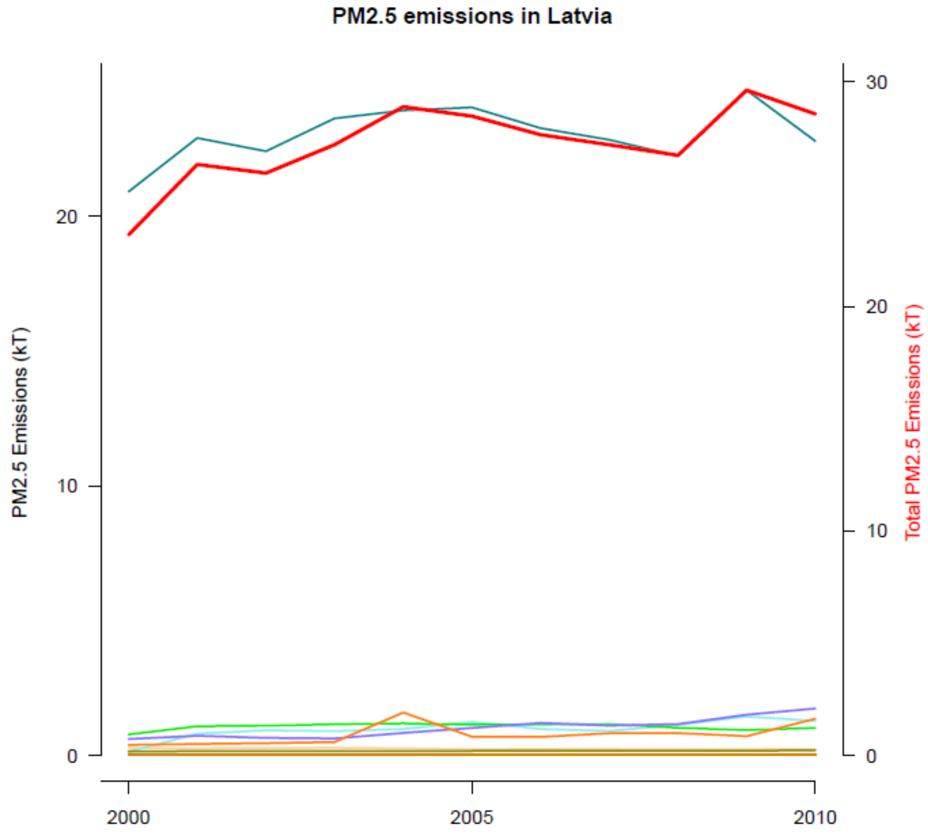
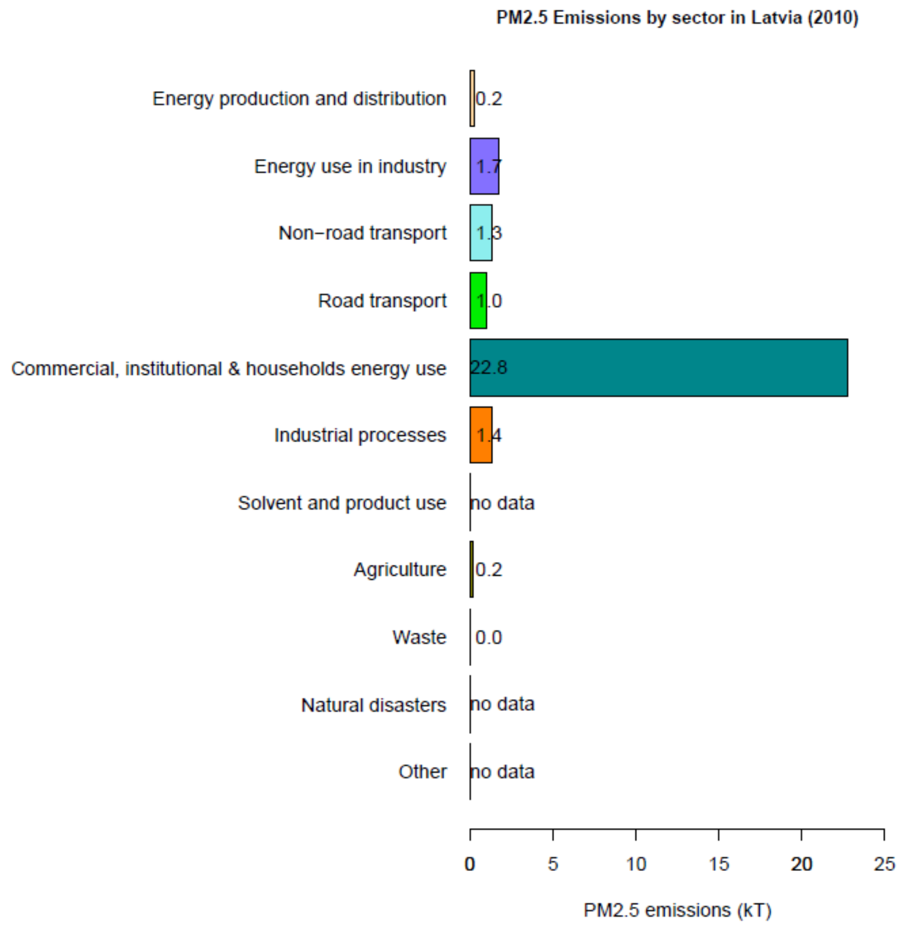
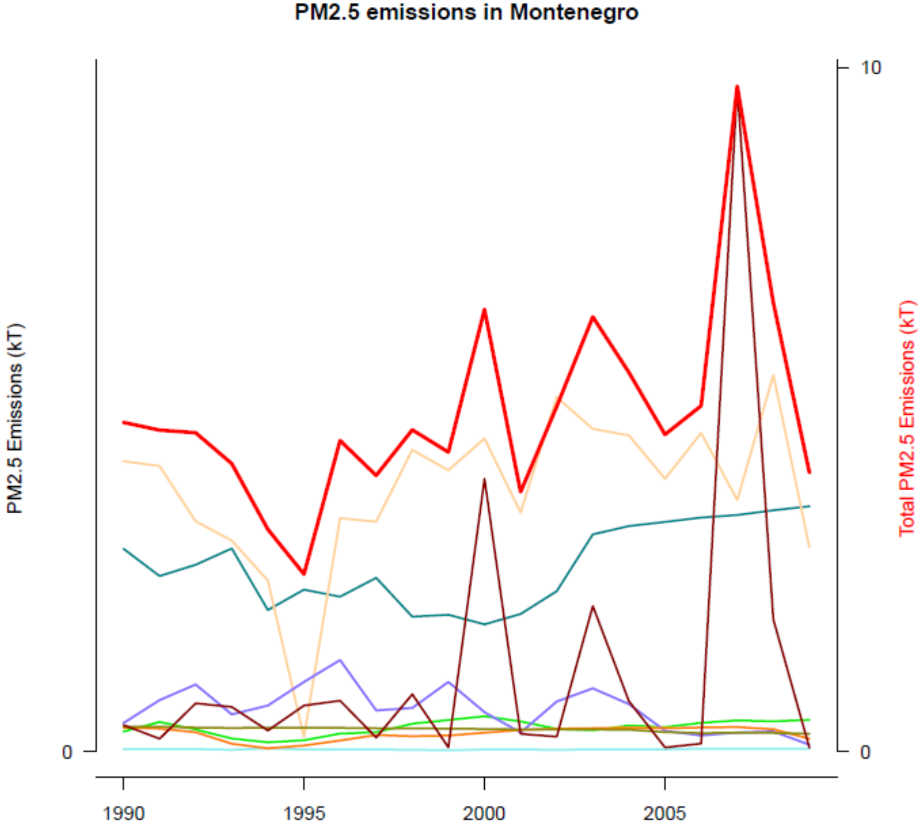
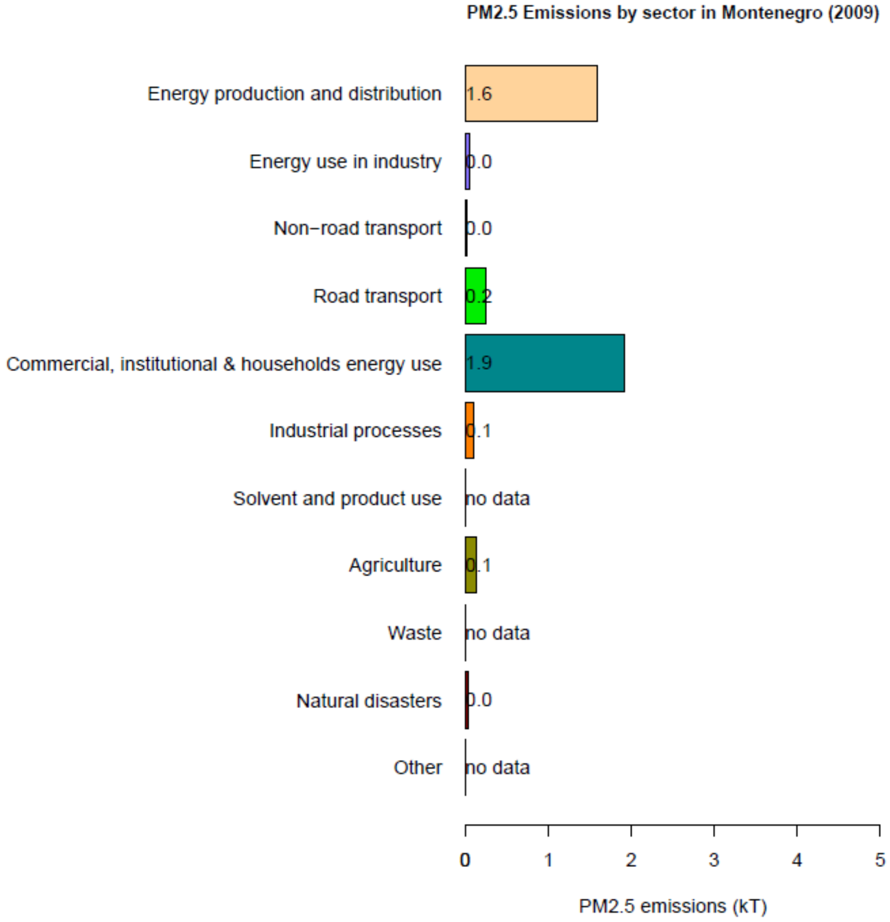
Latvia ($PM_{2.5}$)



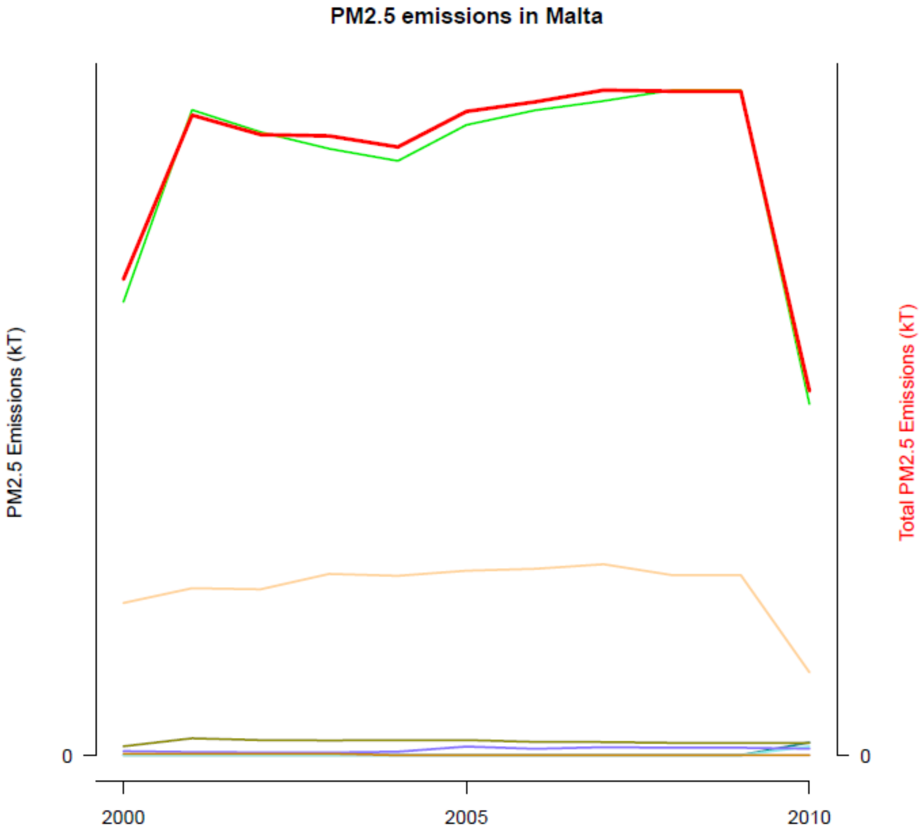


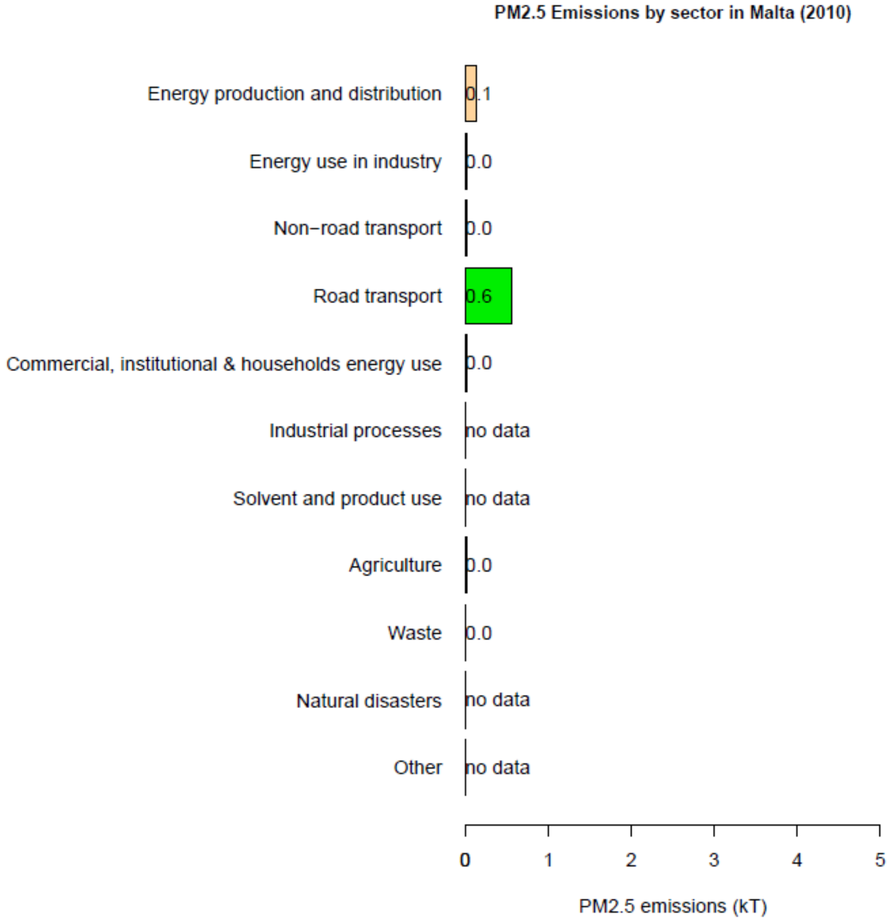
Montenegro ($PM_{2.5}$)



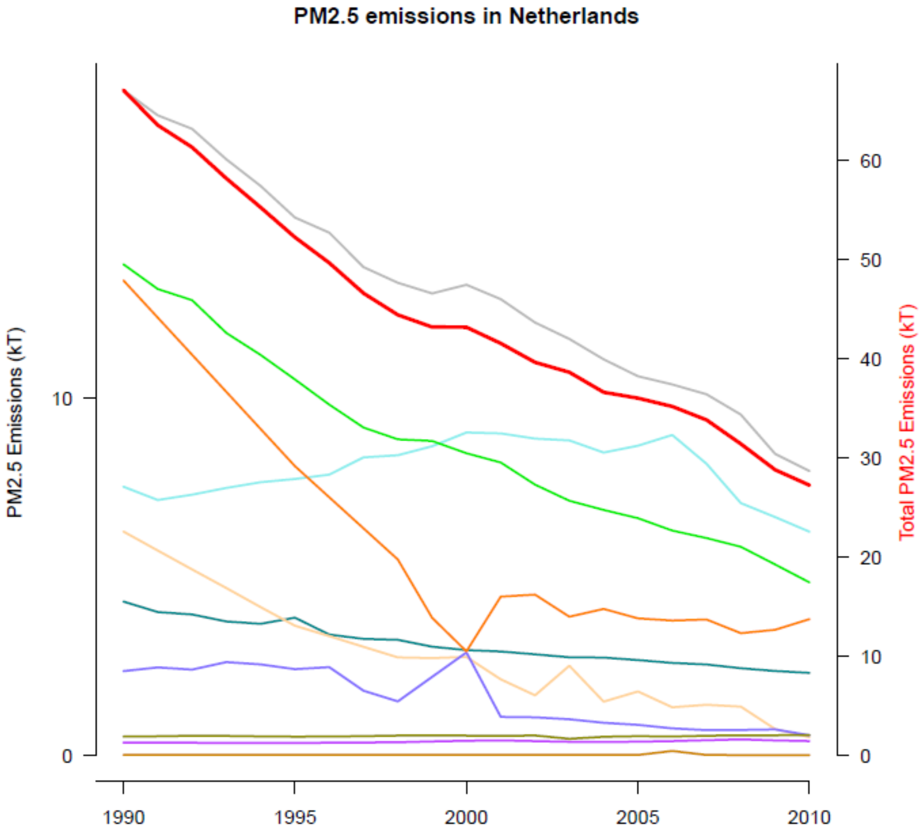


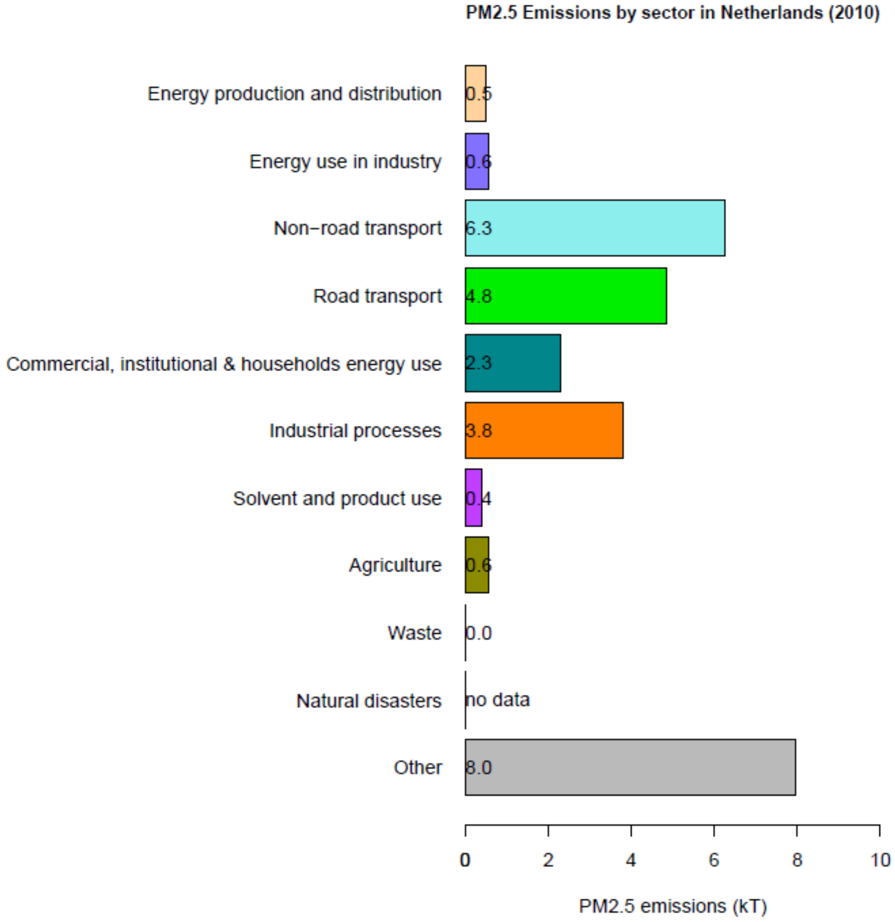
Malta ($PM_{2.5}$)


PM2.5 emissions in Malta
PM2.5 Emissions (kT)
Total PM2.5 Emissions (kT)
0
0
2000
2005
2010

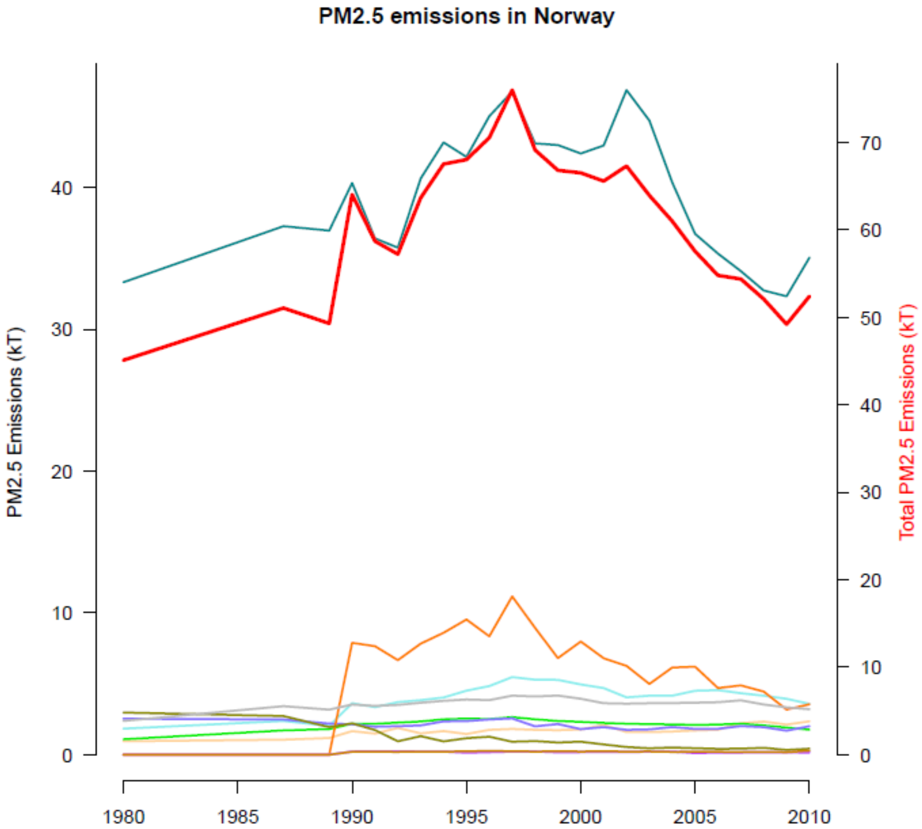

PM2.5 Emissions by sector in Malta (2010)
Energy production and distribution
0.1
Energy use in industry
0.0
Non−road transport
0.0
Road transport
0.6
Commercial, institutional & households energy use
0.0
Industrial processes
no data
Solvent and product use
no data
Agriculture
0.0
Waste
0.0
Natural disasters
no data
Other
no data
0
1
2
3
4
5
PM2.5 emissions (kT)

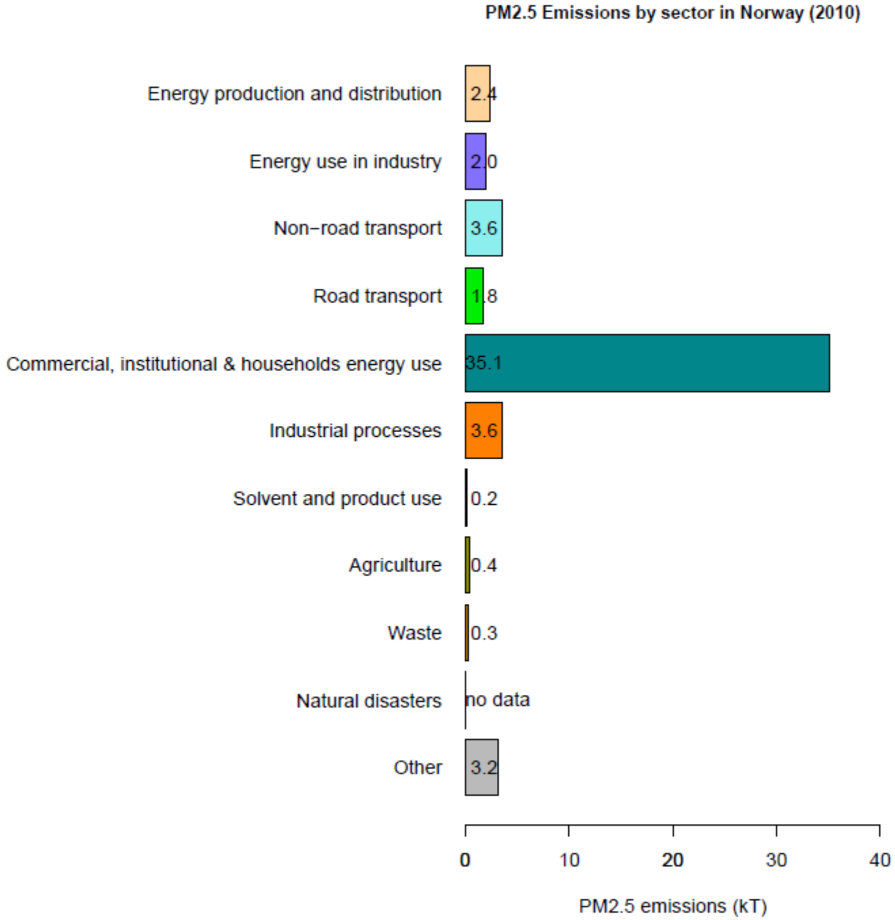
The Netherlands ($PM_{2.5}$)





Norway ($PM_{2.5}$)


PM2.5 emissions in Norway
PM2.5 Emissions (kT)
Total PM2.5 Emissions (kT)
0
10
20
30
40
0
10
20
30
40
50
60
70
1980
1985
1990
1995
2000
2005
2010


PM2.5 Emissions by sector in Norway (2010)
Energy production and distribution
2.4
Energy use in industry
2.0
Non−road transport
3.6
Road transport
1.8
Commercial, institutional & households energy use
35.1
Industrial processes
3.6
Solvent and product use
0.2
Agriculture
0.4
Waste
0.3
Natural disasters
no data
Other
3.2
0
10
20
30
40
PM2.5 emissions (kT)

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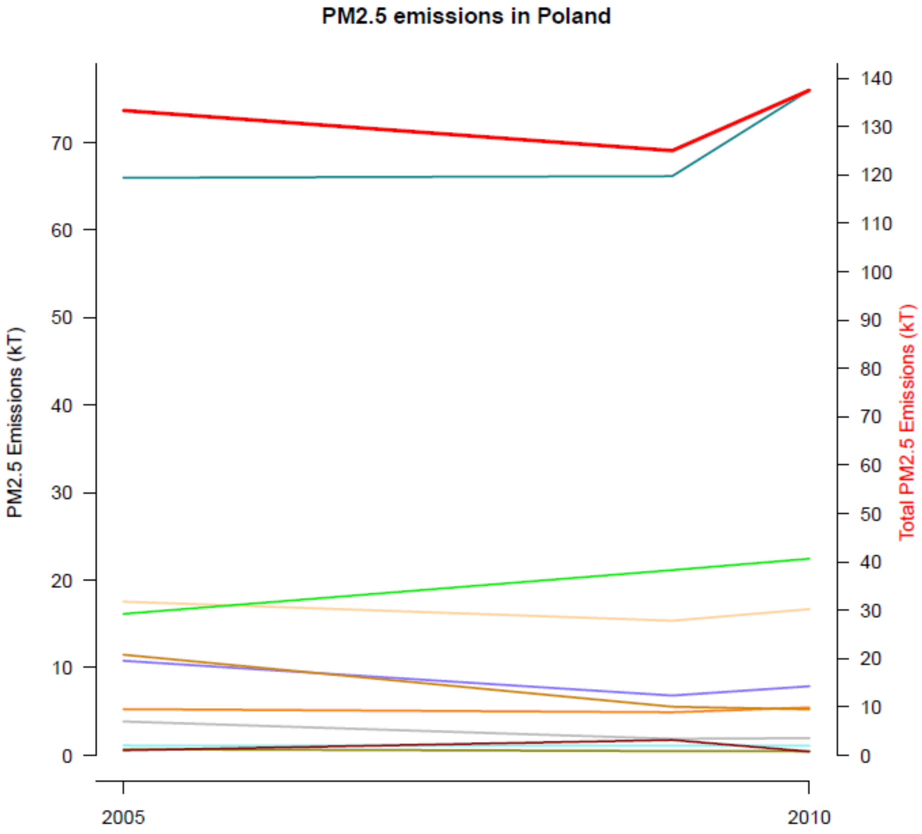
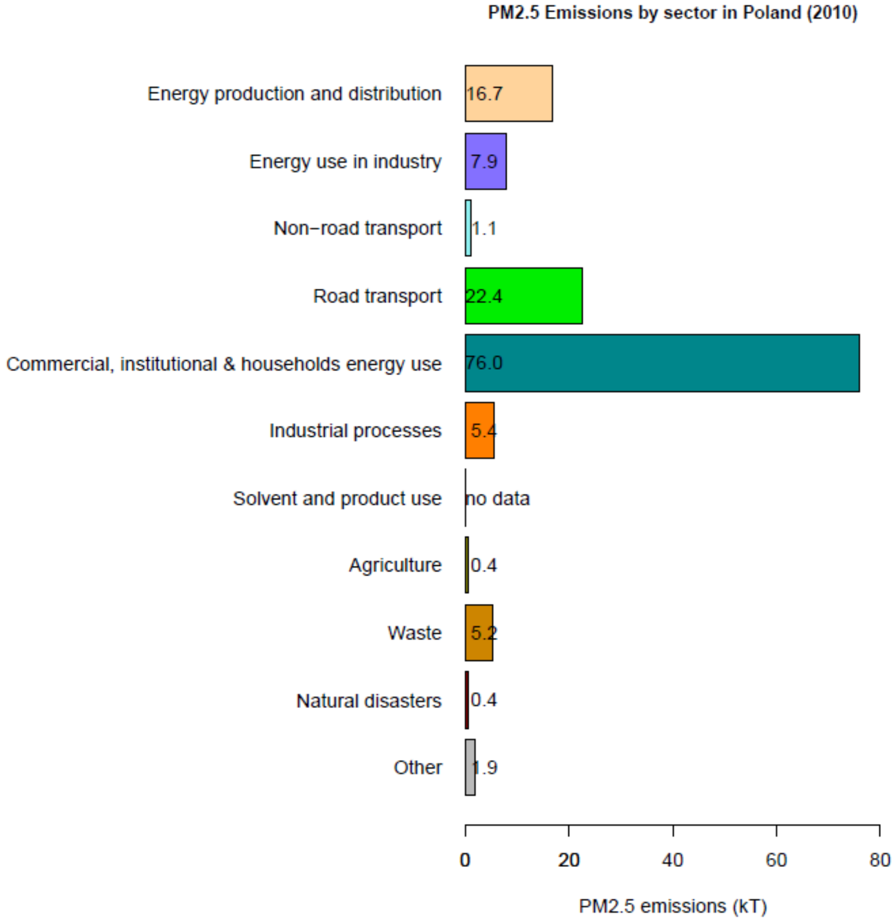
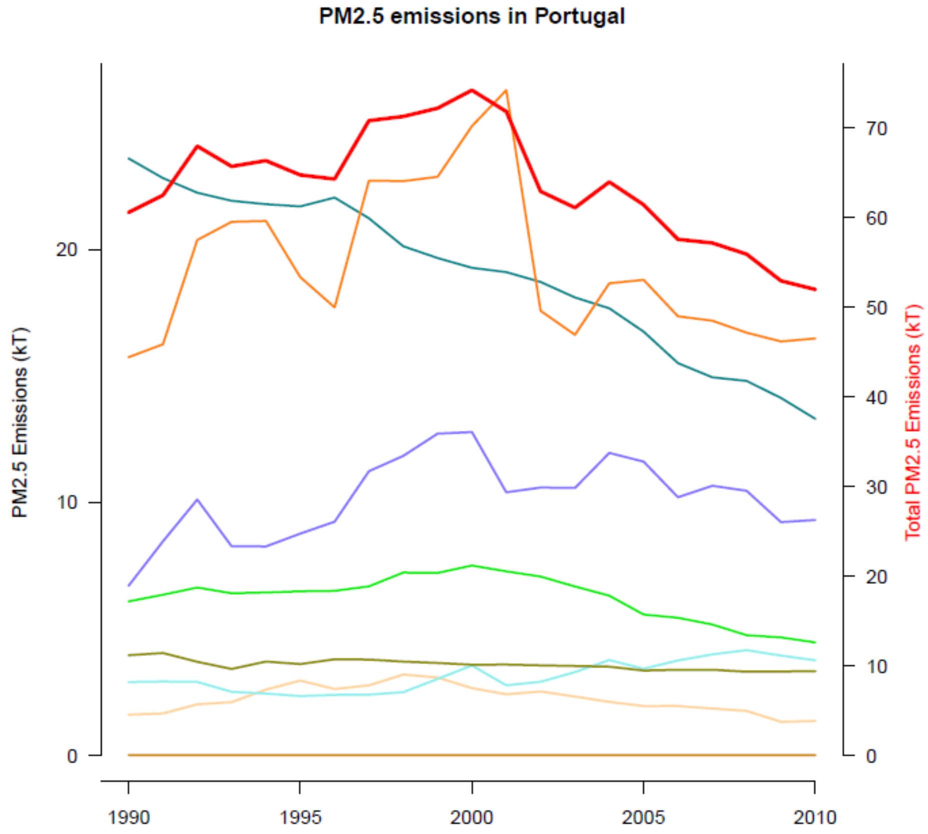
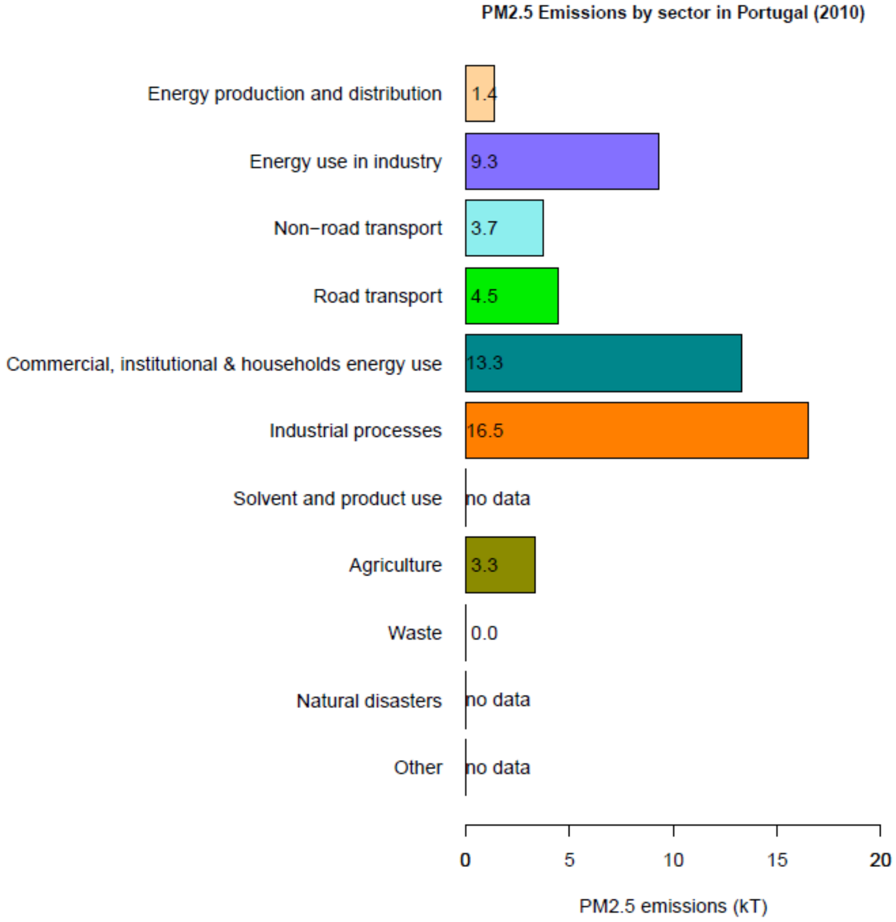
Poland ($PM_{2.5}$)



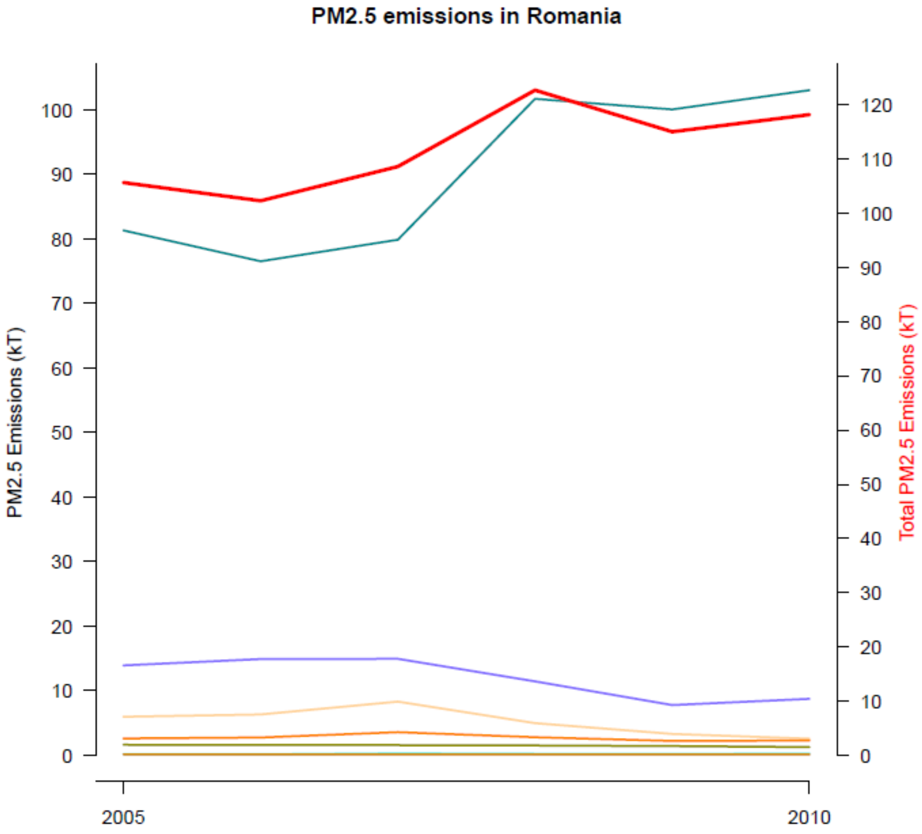


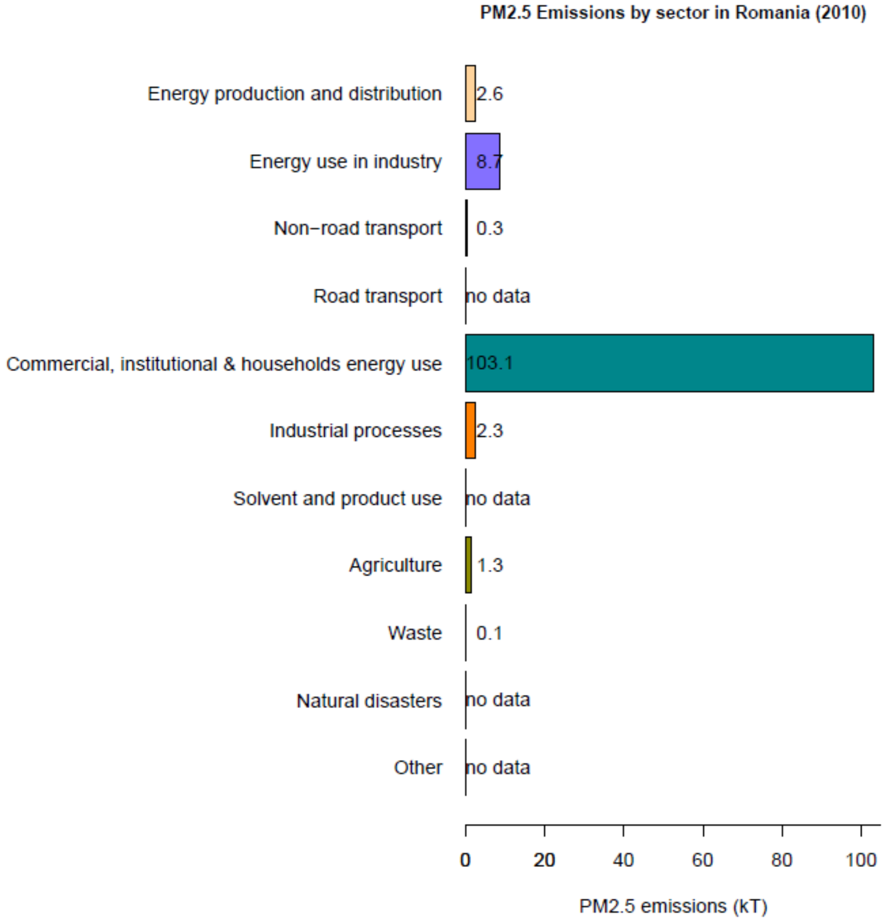
Portugal ($PM_{2.5}$)



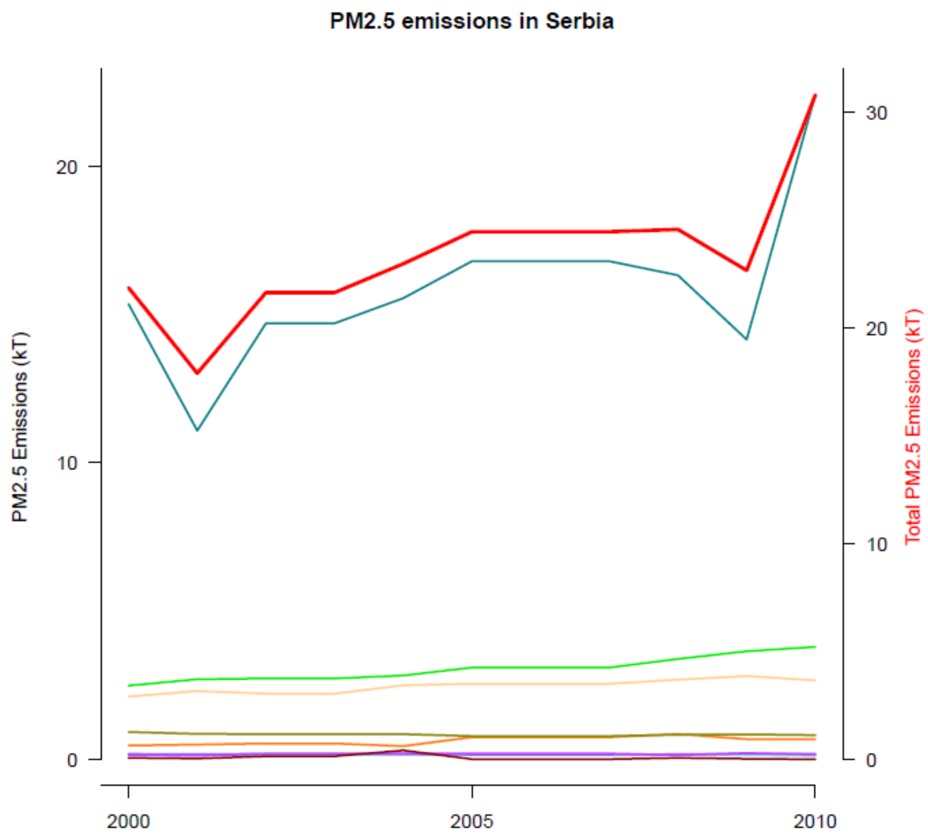


Romania ($PM_{2.5}$)

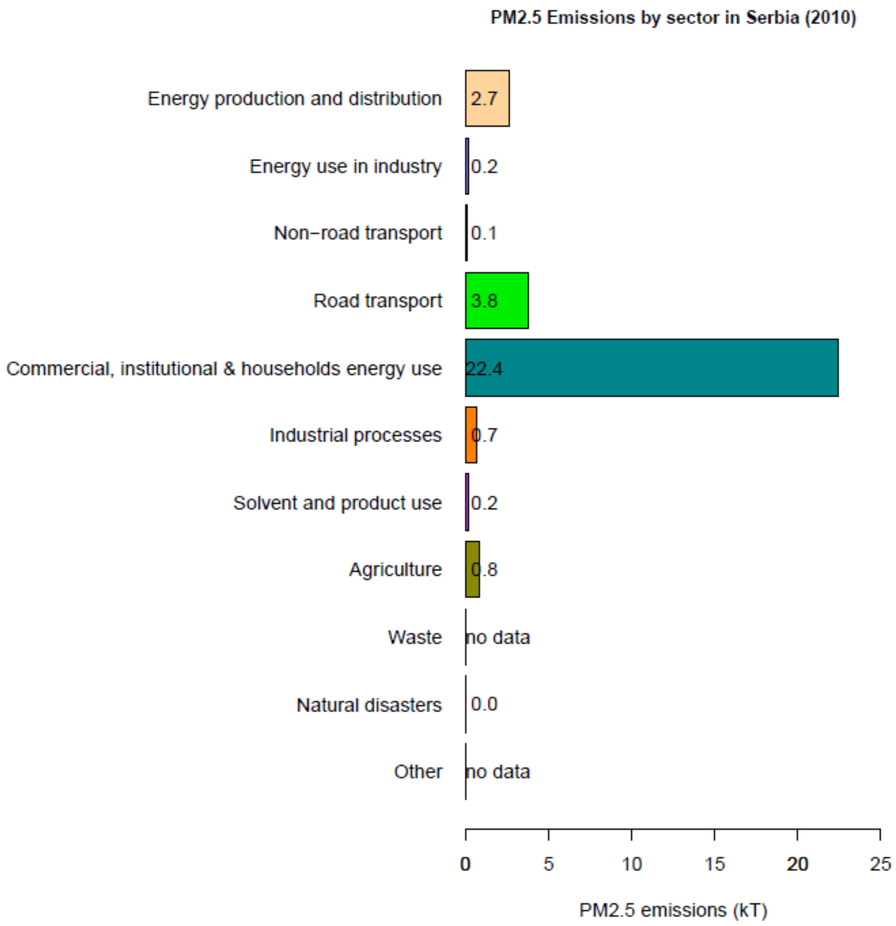




Serbia ($PM_{2.5}$)

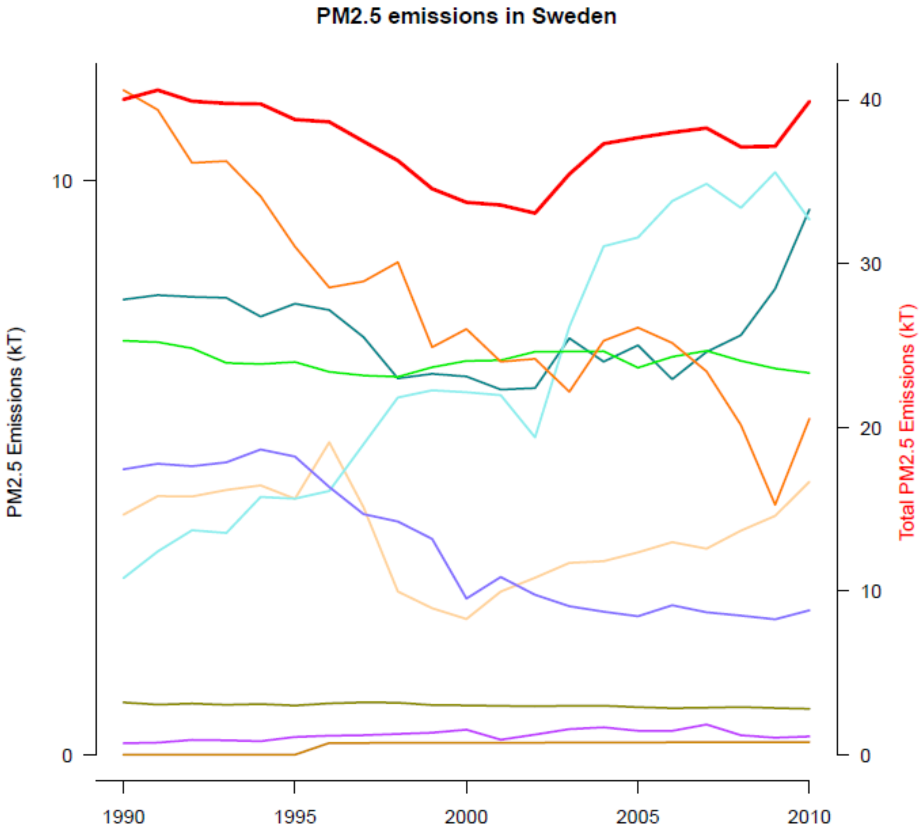


PM2.5 emissions in Serbia
PM2.5 Emissions (kT)
Total PM2.5 Emissions (kT)
0
10
20
30
2000
2005
2010

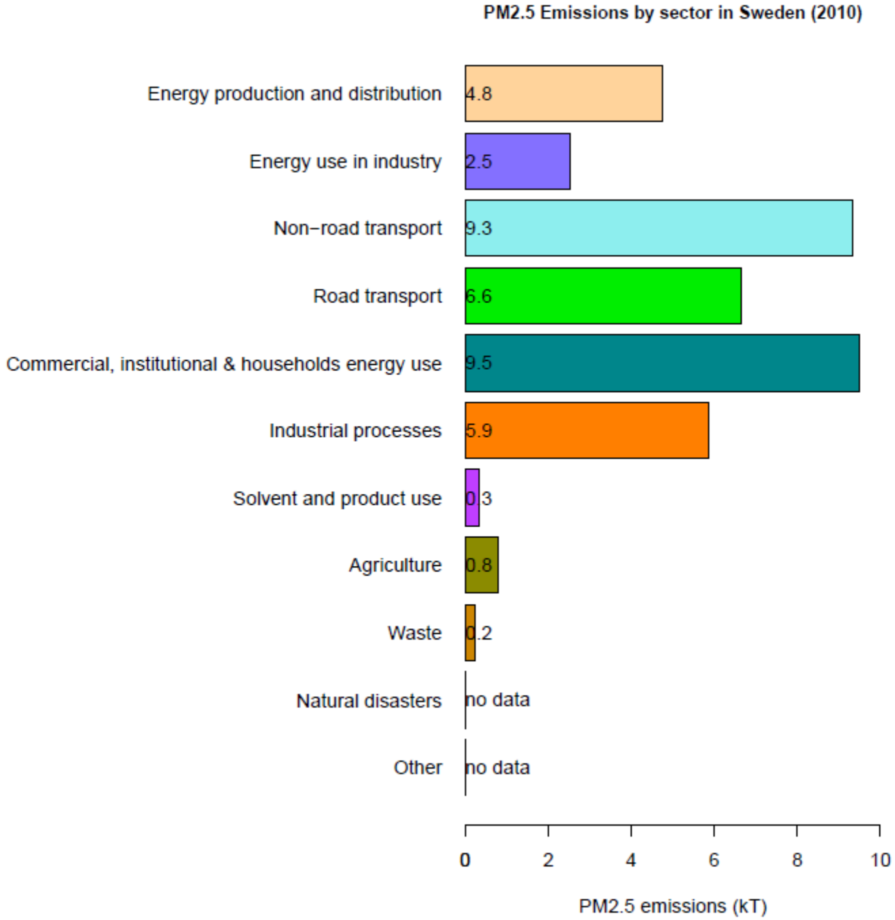


PM2.5 Emissions by sector in Serbia (2010)
Energy production and distribution
2.7
Energy use in industry
0.2
Non−road transport
0.1
Road transport
3.8
Commercial, institutional & households energy use
22.4
Industrial processes
0.7
Solvent and product use
0.2
Agriculture
0.8
Waste
no data
Natural disasters
0.0
Other
no data
0
5
10
15
20
25
PM2.5 emissions (kT)

Sweden ($PM_{2.5}$)

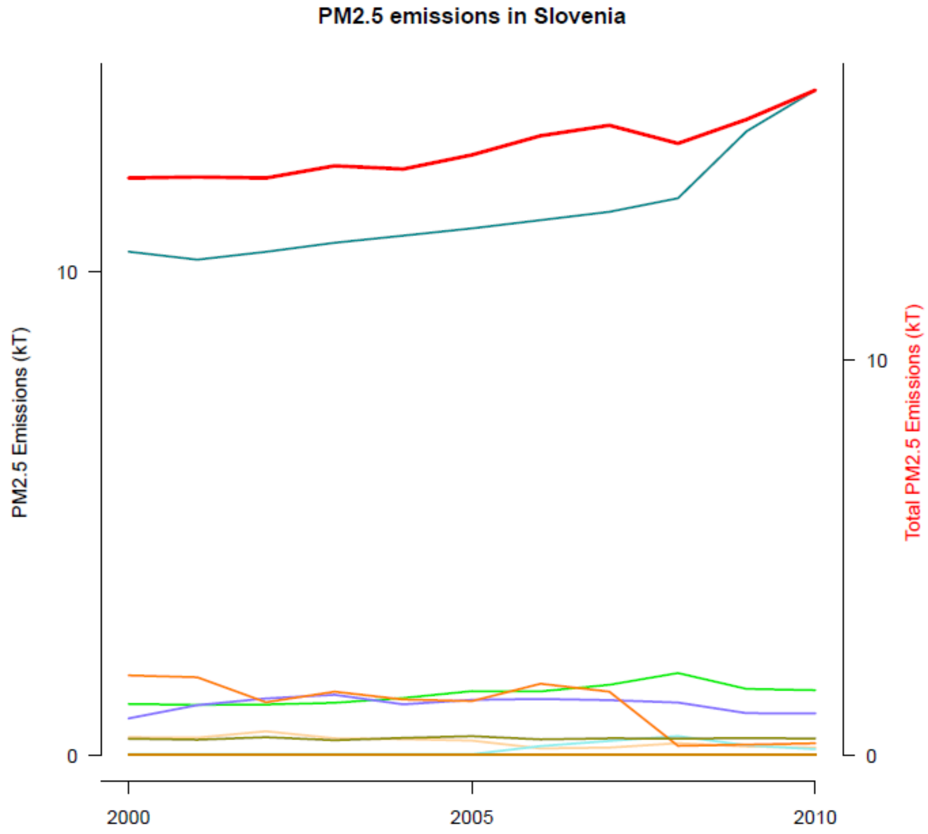


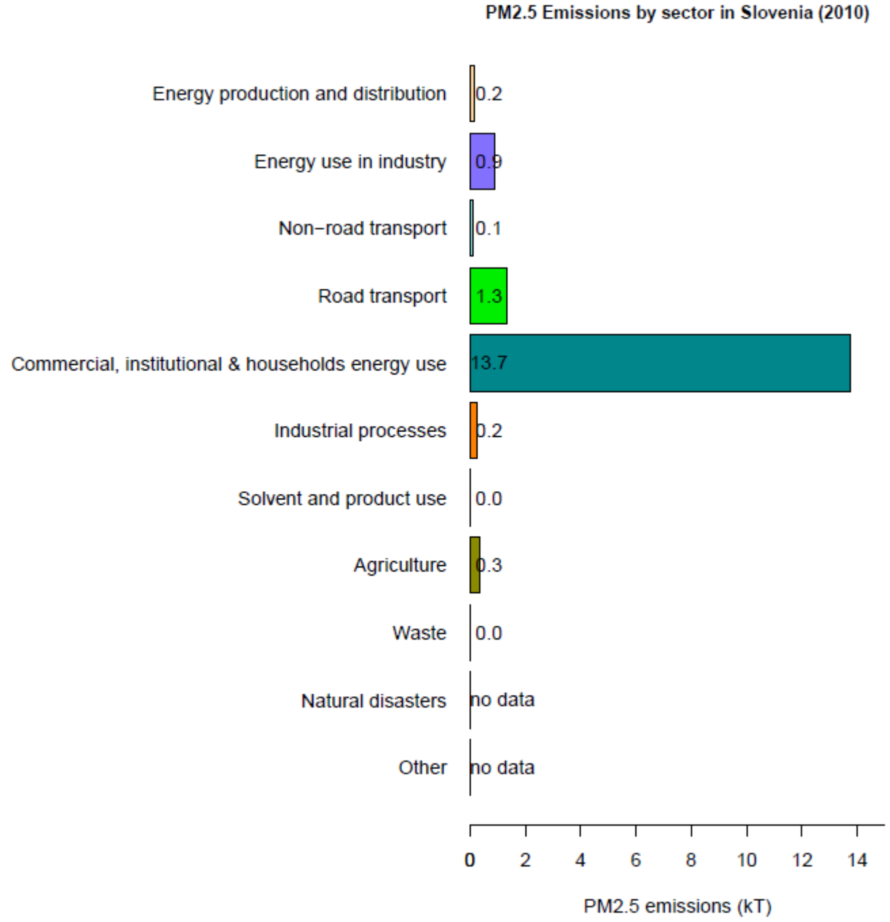
PM2.5 emissions in Sweden
PM2.5 Emissions (kT)
Total PM2.5 Emissions (kT)
10
0
40
30
20
10
0
1990
1995
2000
2005
2010



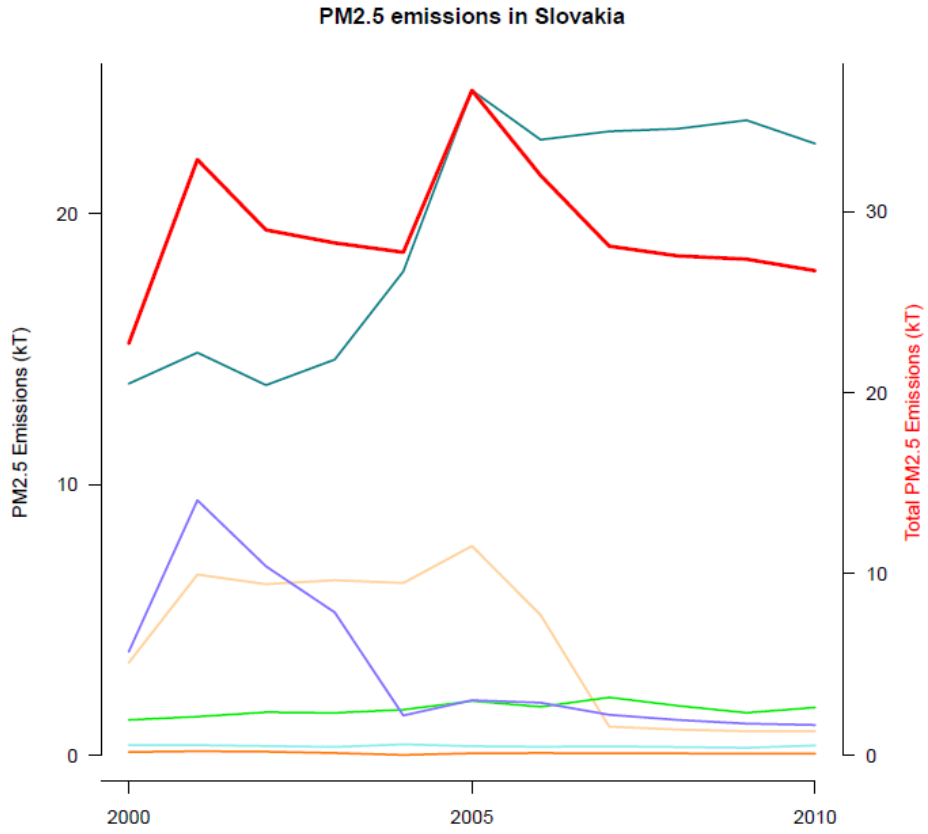
PM2.5 Emissions by sector in Sweden (2010)
Energy production and distribution
4.8
Energy use in industry
2.5
Non−road transport
9.3
Road transport
6.6
Commercial, institutional & households energy use
9.5
Industrial processes
5.9
Solvent and product use
0.3
Agriculture
0.8
Waste
0.2
Natural disasters
no data
Other
no data
0
2
4
6
8
10
PM2.5 emissions (kT)

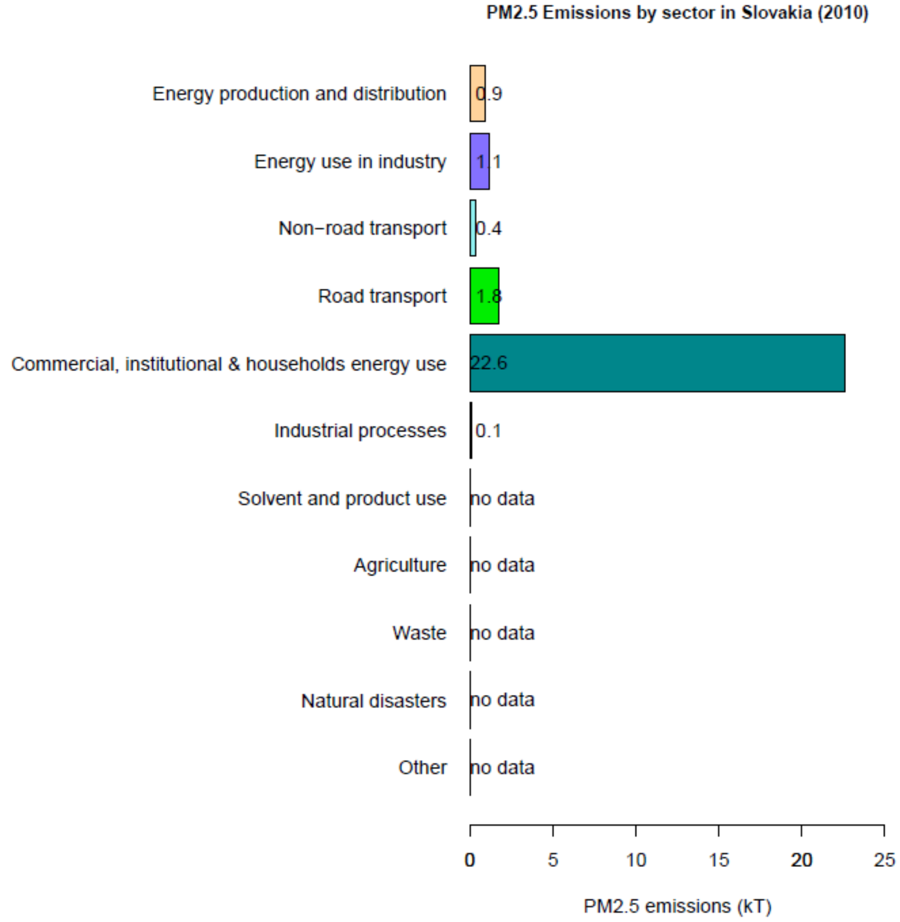
Slovenia ($PM_{2.5}$)



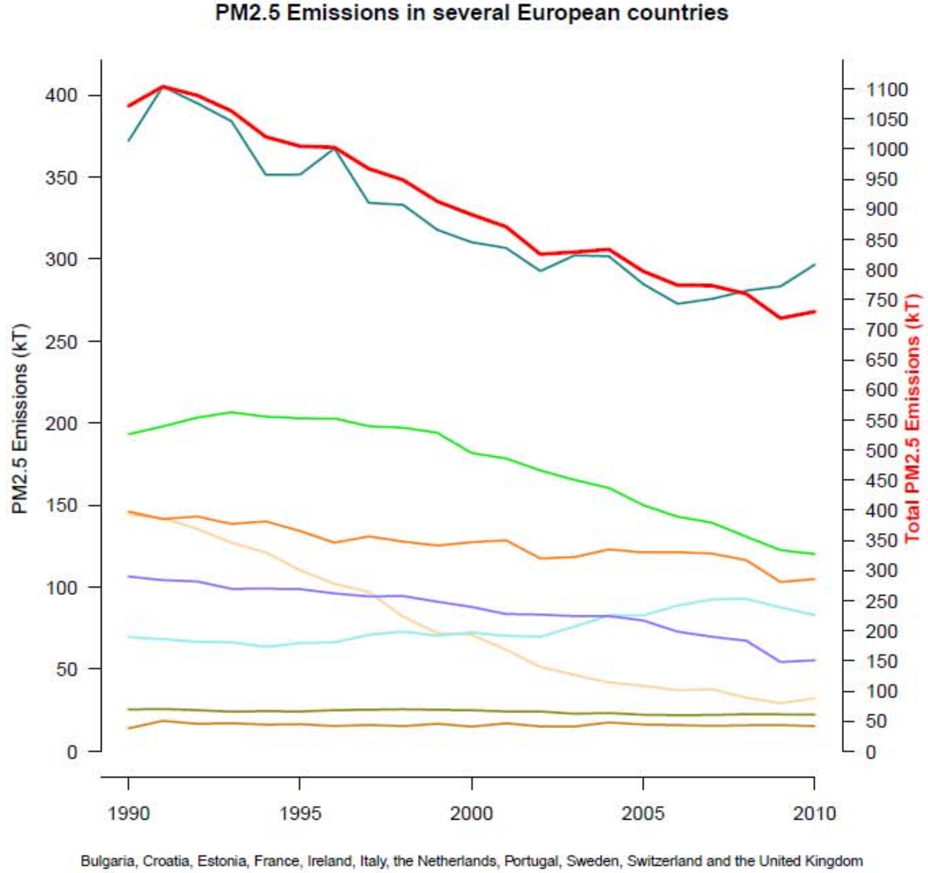


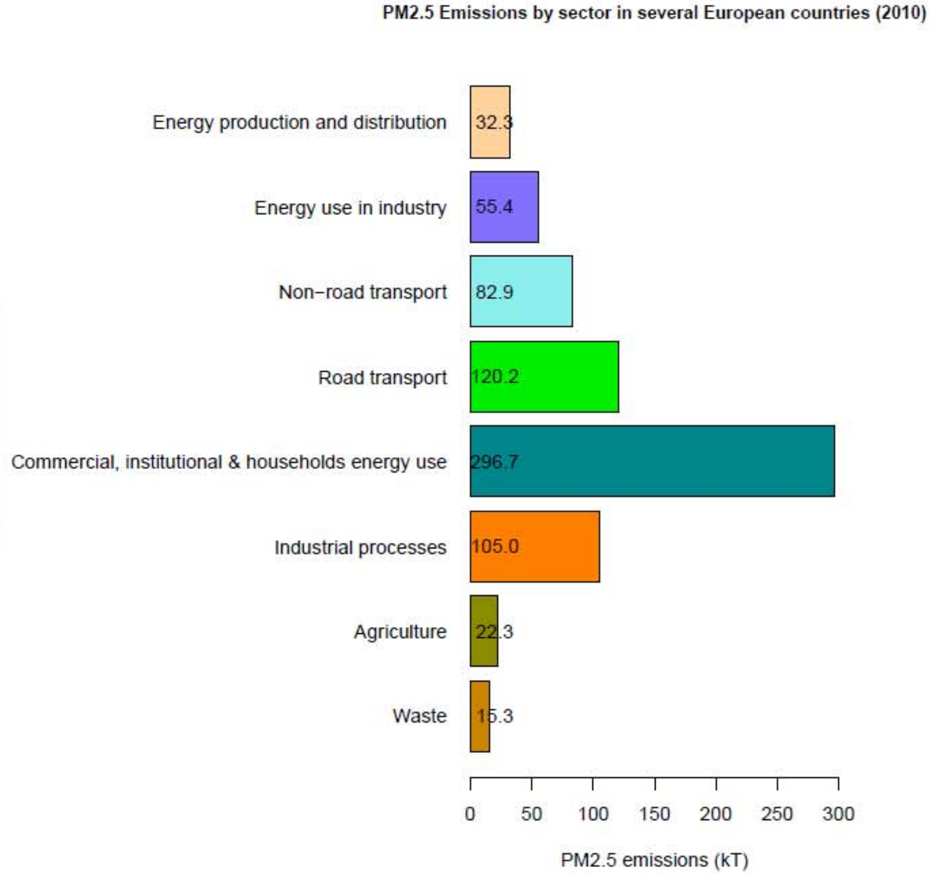
Slovakia ($PM_{2.5}$)





Bulgaria, Croatia, Estonia, France, Ireland, Italy, the Netherlands, Portugal, Sweden and the United Kingdom ($PM_{2.5}$)





II.2. PM_{10}

II.2.1. Data availability

Availability of PM_{10} data is described below:

Country	From	To	Missing years	x = no data										
				EP	EI	NRT	RT	CIH	IP	S	A	W	N	O
Albania	1990	2009	-							x		x		x
Austria	1990	2010	1991-1994; 1996-1999										x	
Belgium	2000	2010	2001-2004							x			x	
Bulgaria	1990	2010	-							x	x		x	x
Croatia	1990	2010	-							x		x	x	
Cyprus	2000	2010	-							x			x	
Czech Republic	2010	2010	-							x			x	
Denmark	2000	2010	-										x	x
Estonia	1990	2010	-										x	x
Finland	1980	2010	-										x	x
France	1990	2010	-											x
Germany	1995	2010	-											x
Hungary	2010	2010	-										x	x
Iceland	2010	2010	-	x	x	x	x	x	x	x	x	x		x
Ireland	1990	2010	-							x			x	
Italy	1990	2010	-							x				x
Latvia	2000	2010	-							x			x	x
Lithuania	2008	2010	-						x	x	x	x	x	x
Macedonia, Former Yugoslav Rep	2010	2010	-	x	x	x	x	x	x	x	x	x		x
Malta	2000	2010	-						x	x			x	x
Montenegro	1990	2009	-							x		x		x

Country	From	To	Missing years	x = no data										
				EP	EI	NRT	RT	CIH	IP	S	A	W	N	O
Netherlands	1990	2010	-										x	
Norway	1980	2010	1981-1986; 1988										x	
Poland	2009	2010	-							x				
Portugal	1990	2010	-							x			x	x
Romania	2005	2010	-							x			x	x
Serbia	2000	2010	-									x		x
Slovakia	2000	2010	-							x	x	x	x	x
Slovenia	2000	2010	-										x	x
Spain	2000	2010	-							x			x	x
Sweden	1990	2010	-										x	x
Switzerland	1990	2010	-											
United Kingdom	1980	2010	-											

Abbreviation list:

EP: energy production and distribution
EI: energy use in industry
NRT: non-road transport
RT: road transport
CIH: commercial, Institutional and Household energy use
IP: industrial processes
S: solvent and product use
A: agriculture
W: waste
N: natural emissions
O: other emissions

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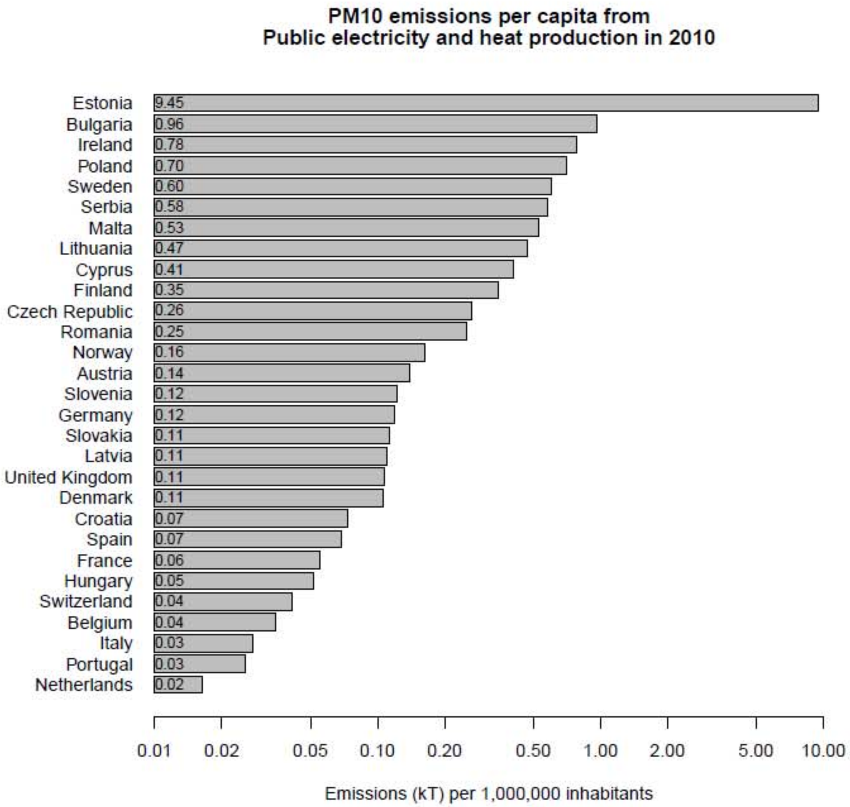
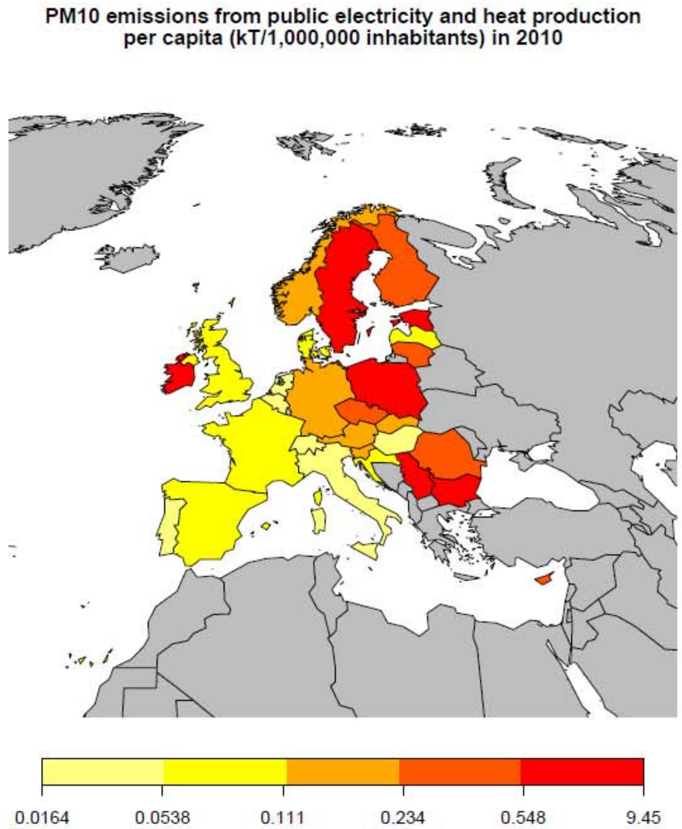
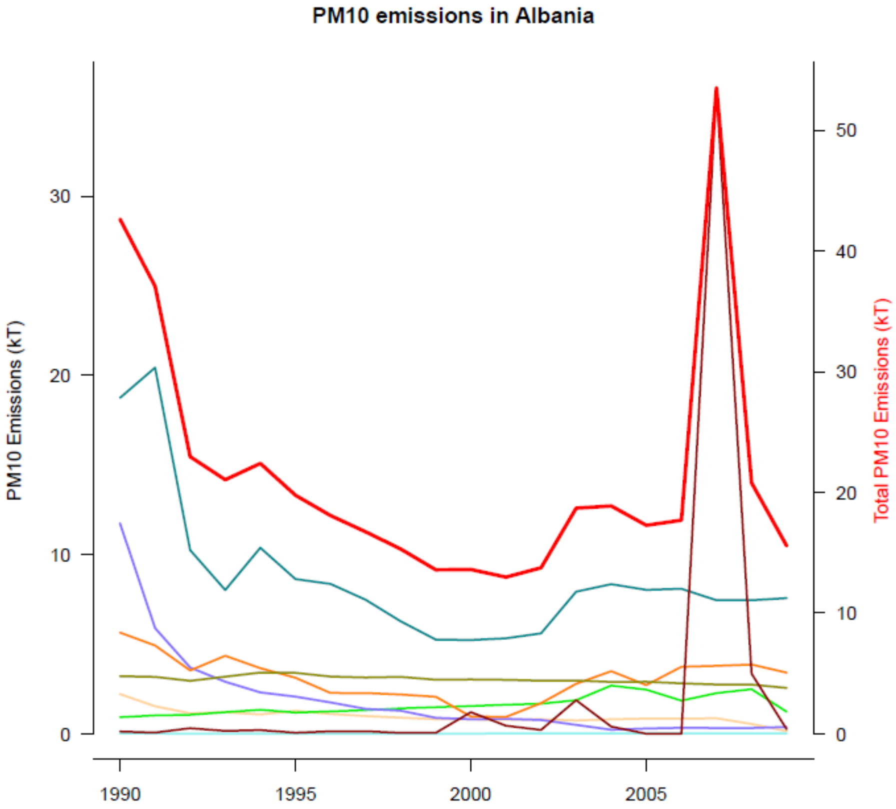
II.2.2. PM_{10} emissions per capita from Public electricity and heat production sector in 2010

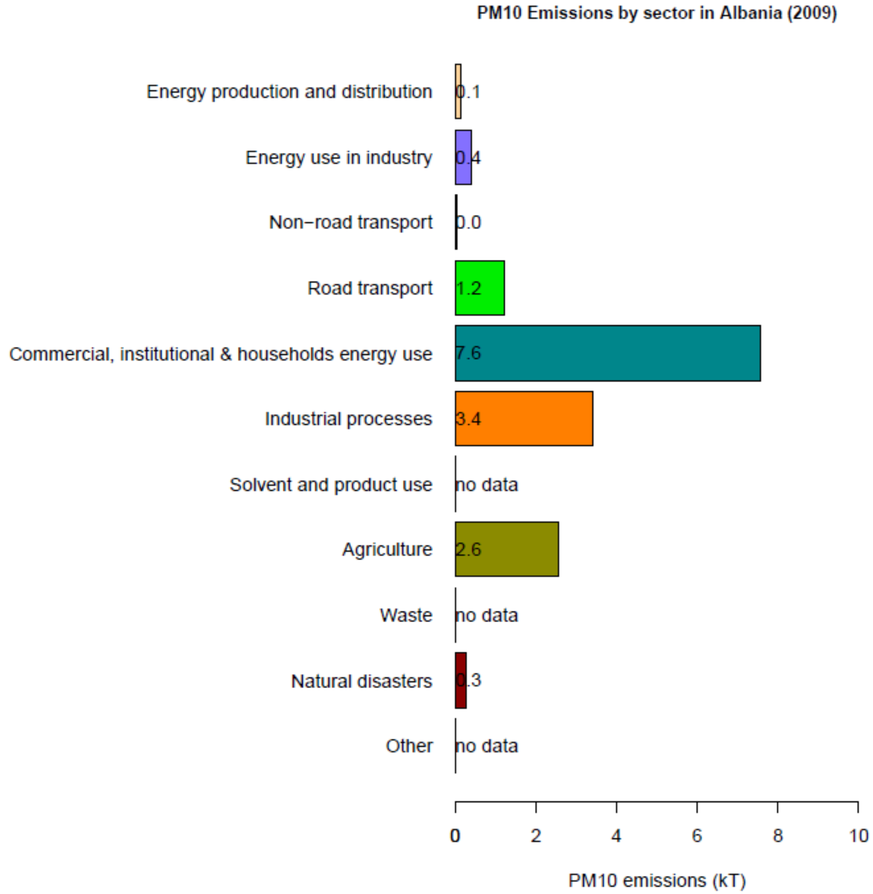




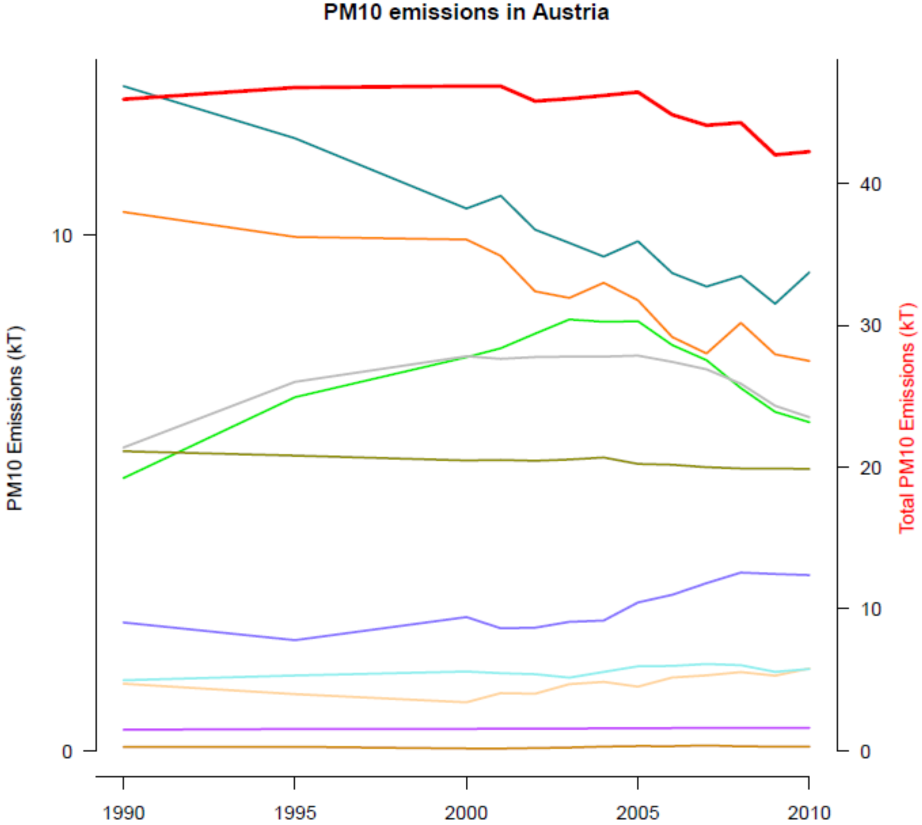
II.2.3. PM_{10} Trends in emissions by sector (left) and distribution of emissions by sector for the last available year (right)

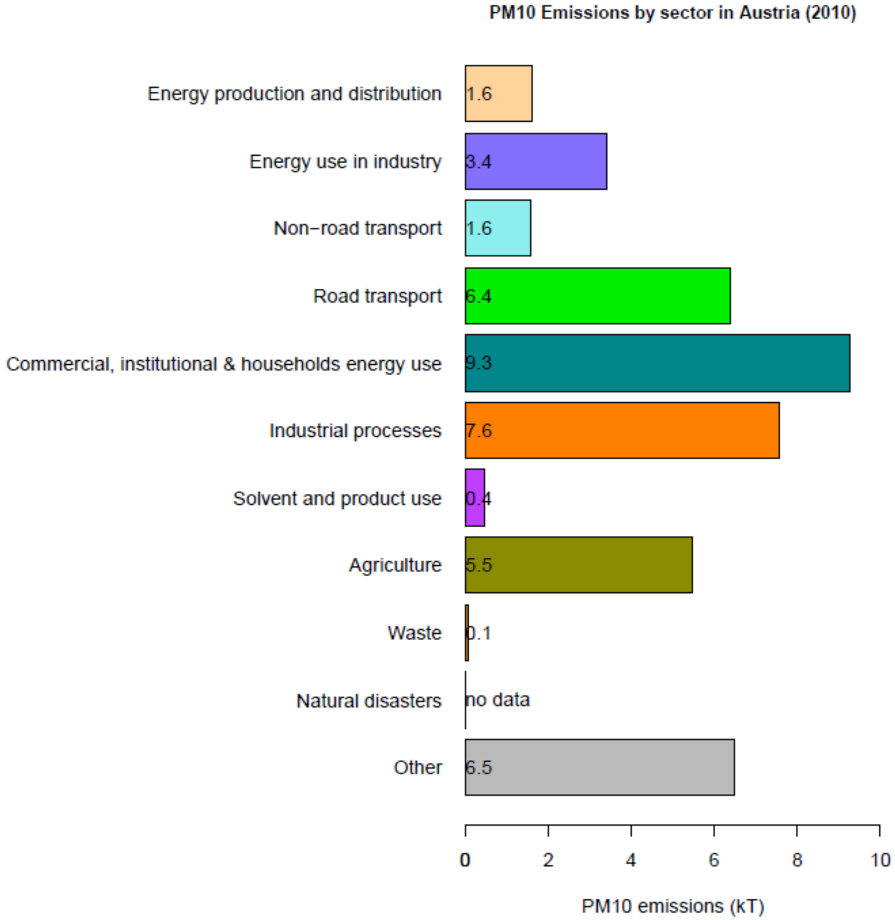
Albania (PM_{10})



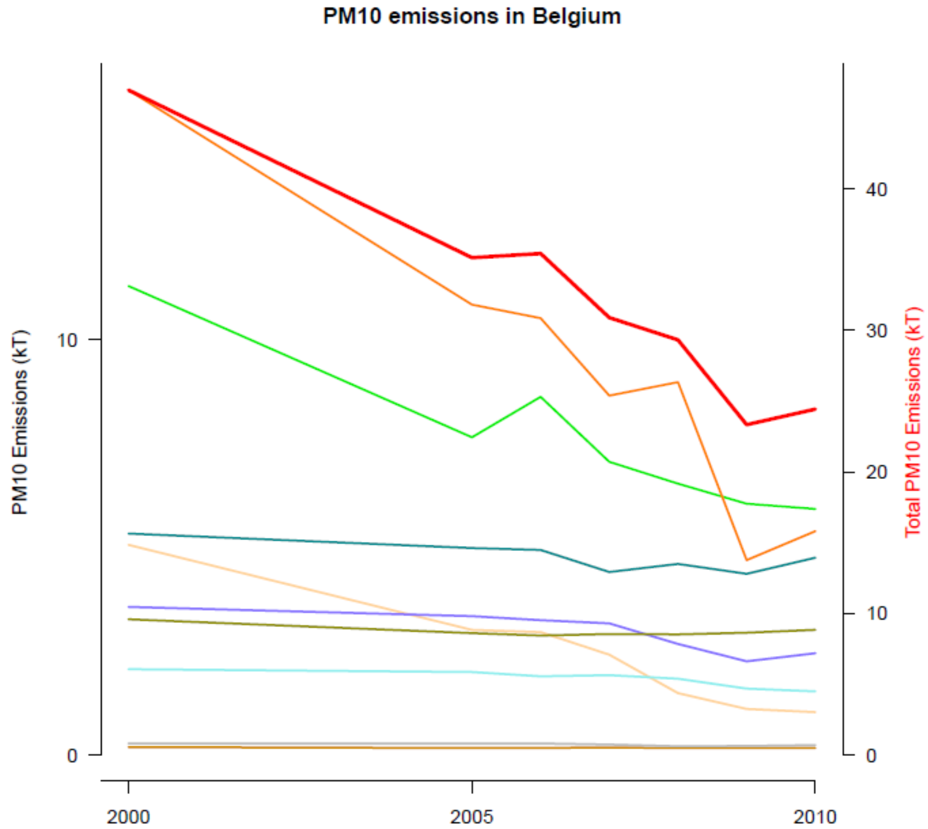


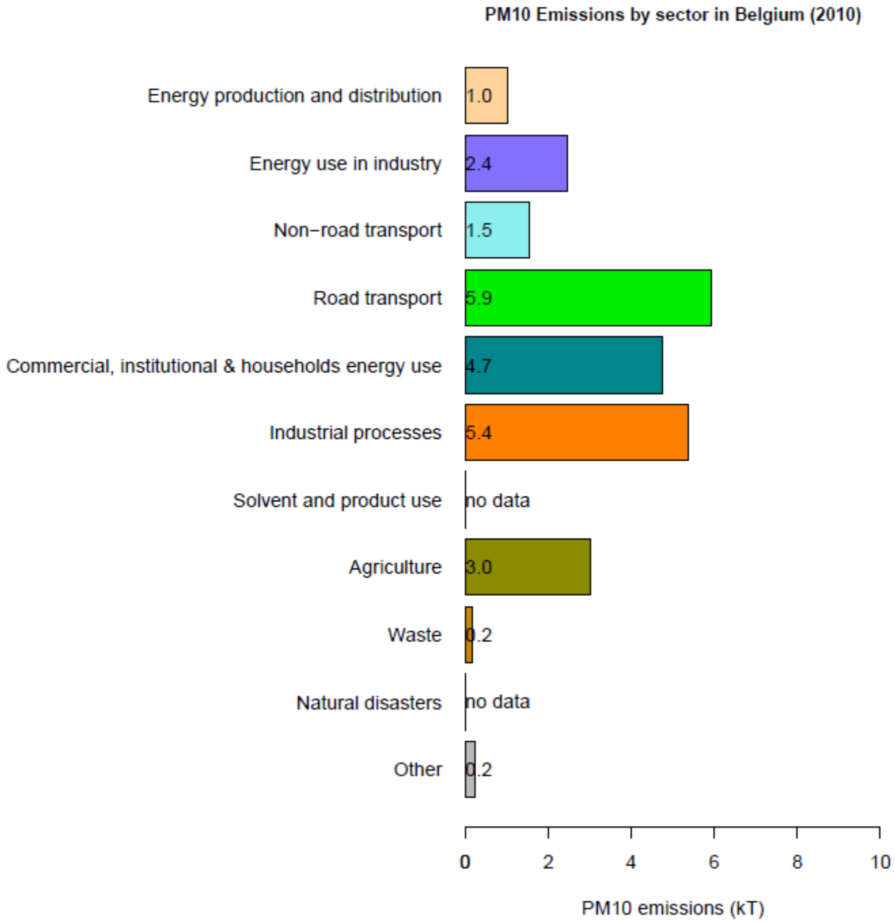
Austria (PM_{10})



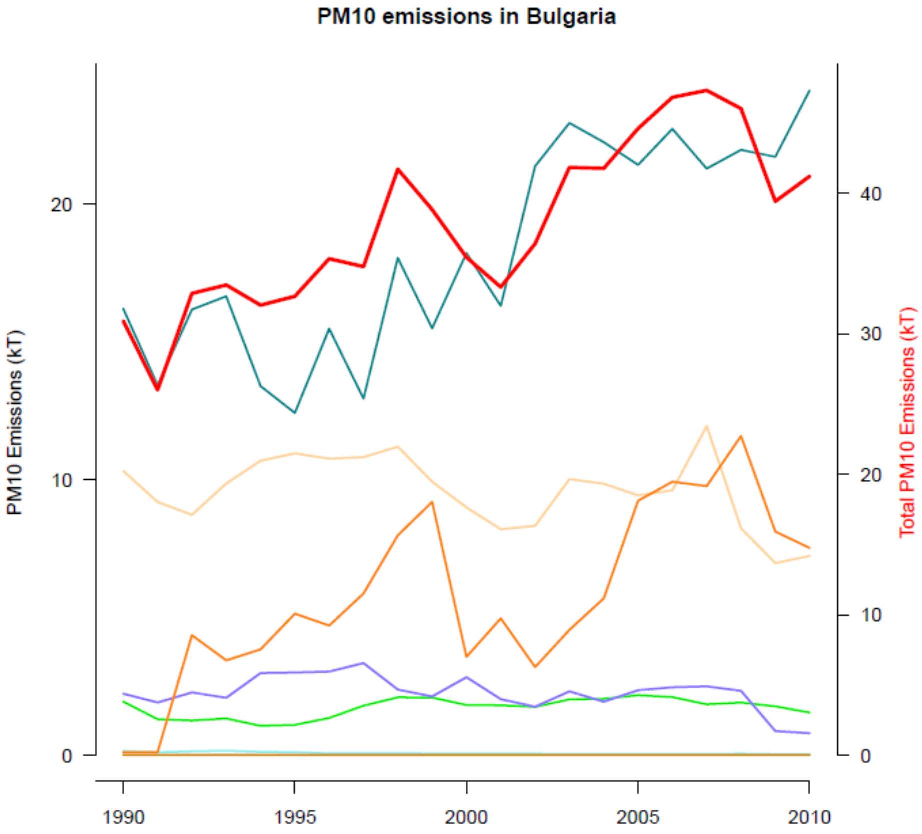


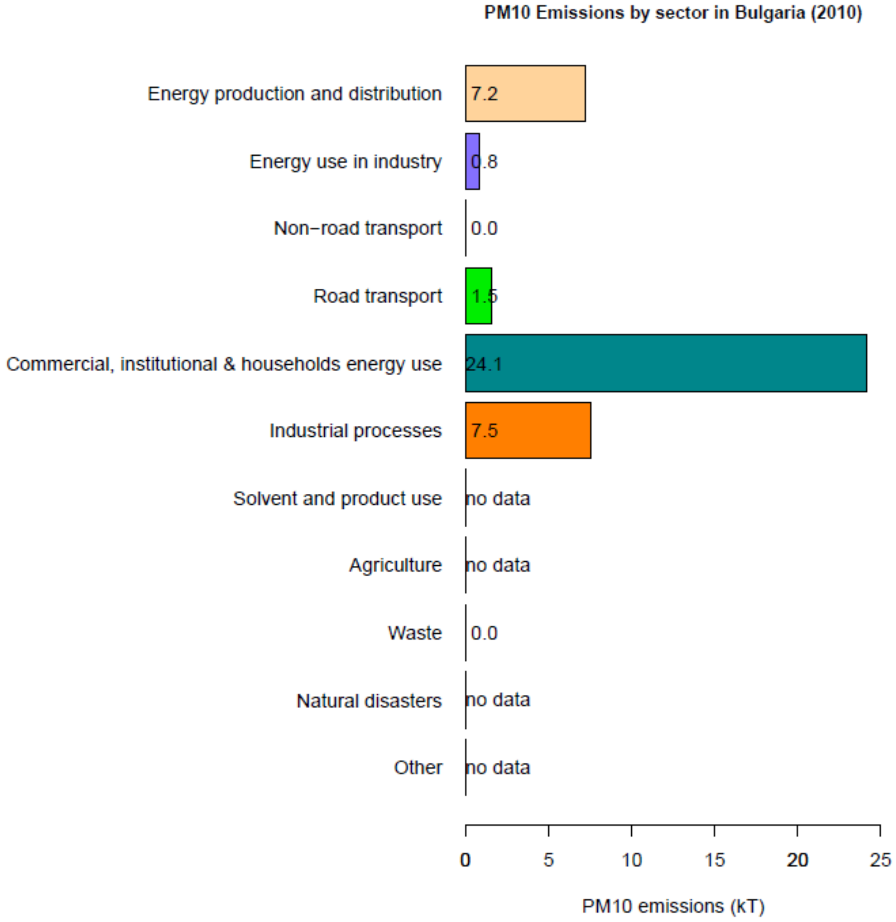
Belgium (PM_{10})



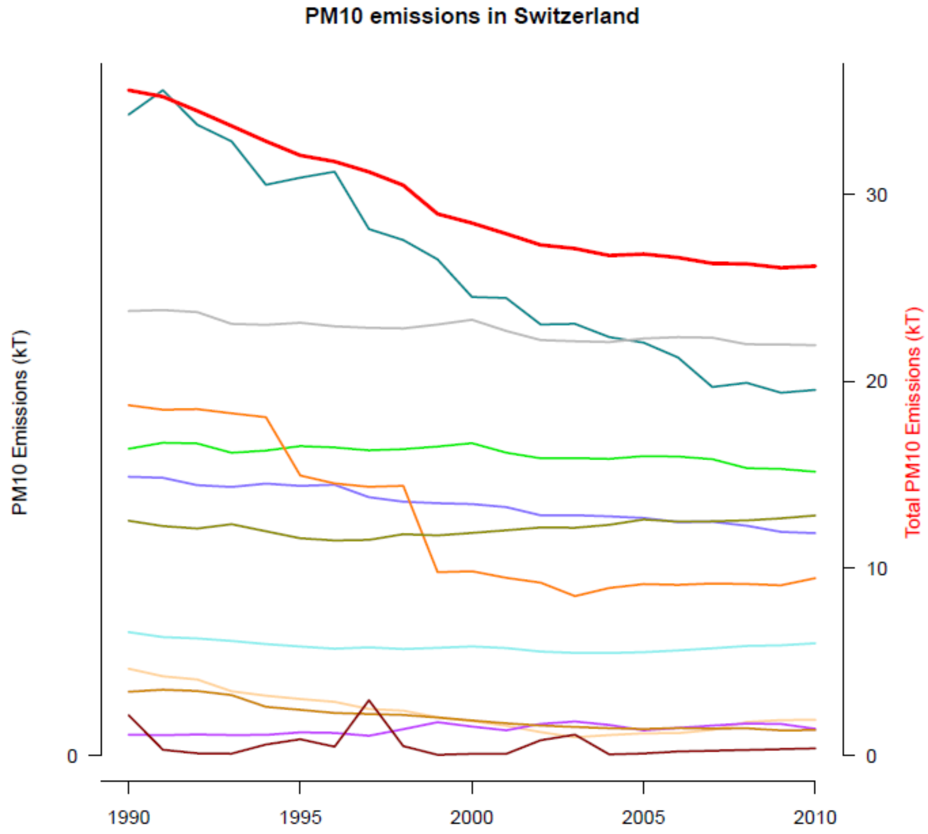


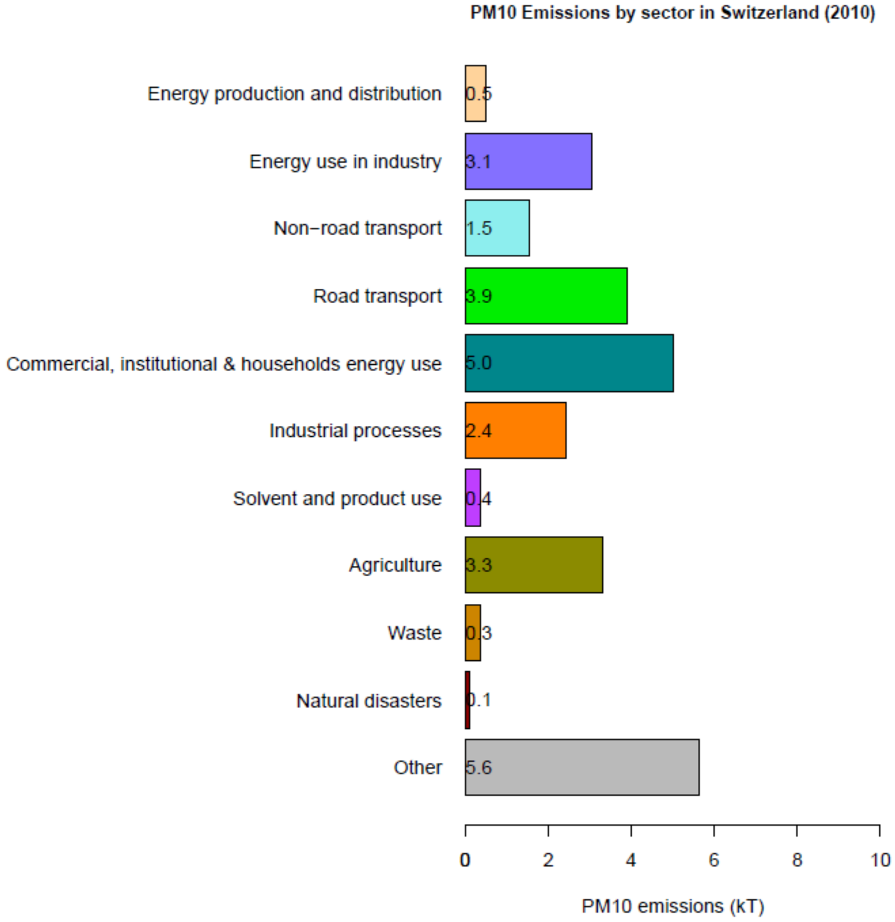
Bulgaria (PM_{10})



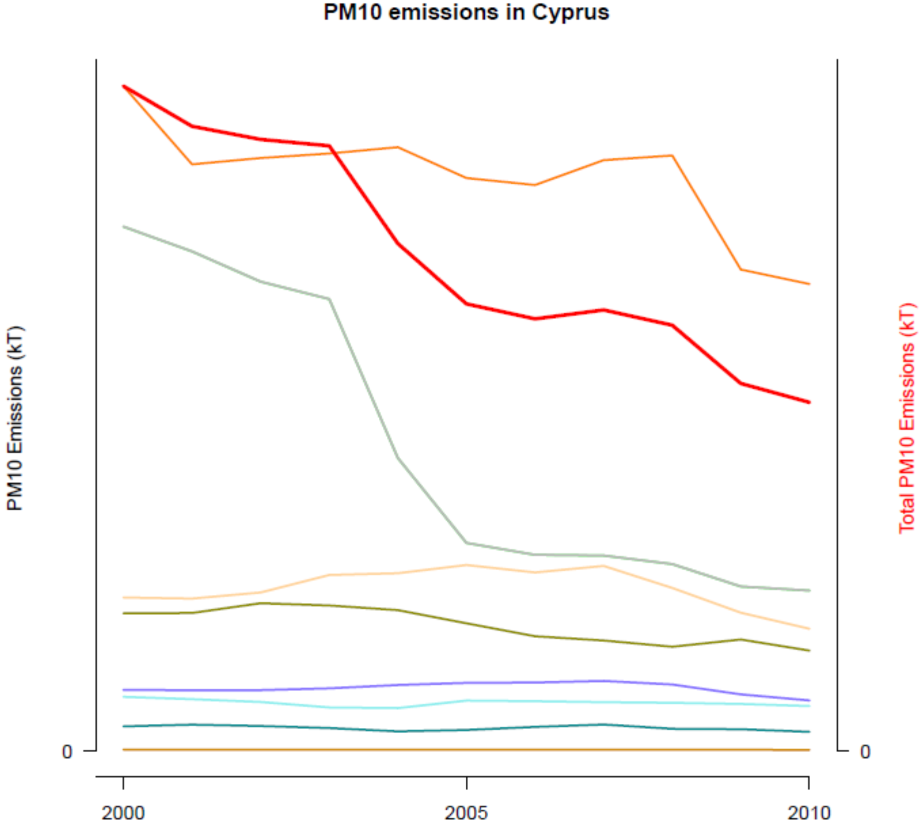


Switzerland (PM_{10})

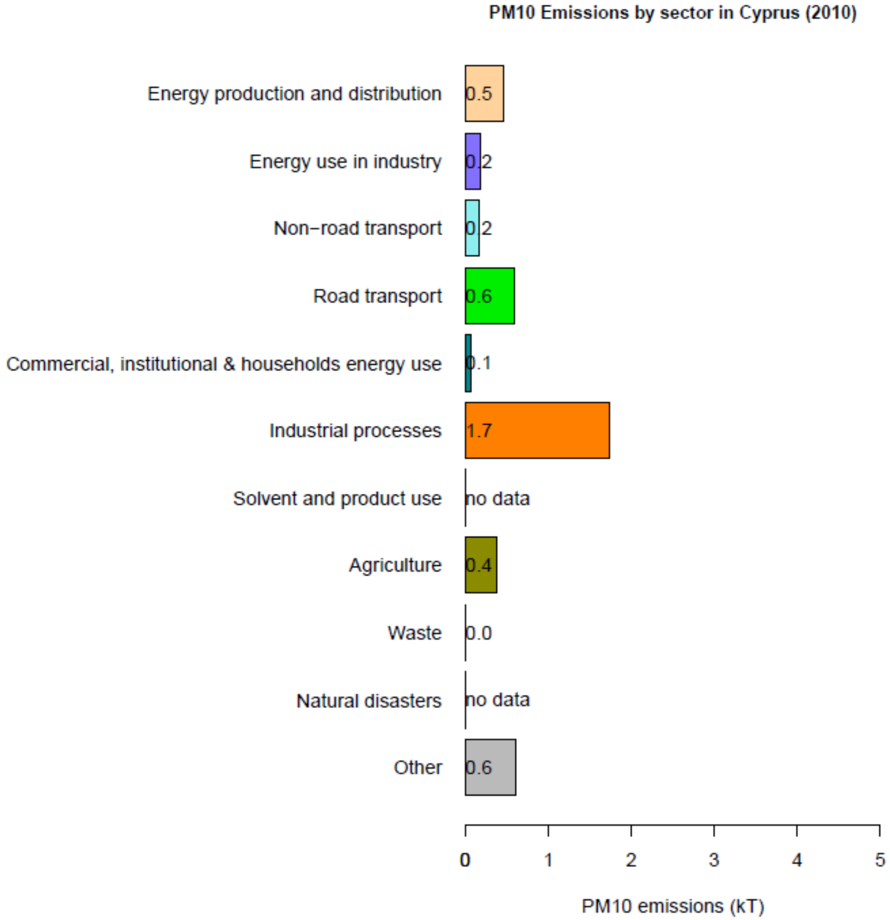




Cyprus (PM_{10})

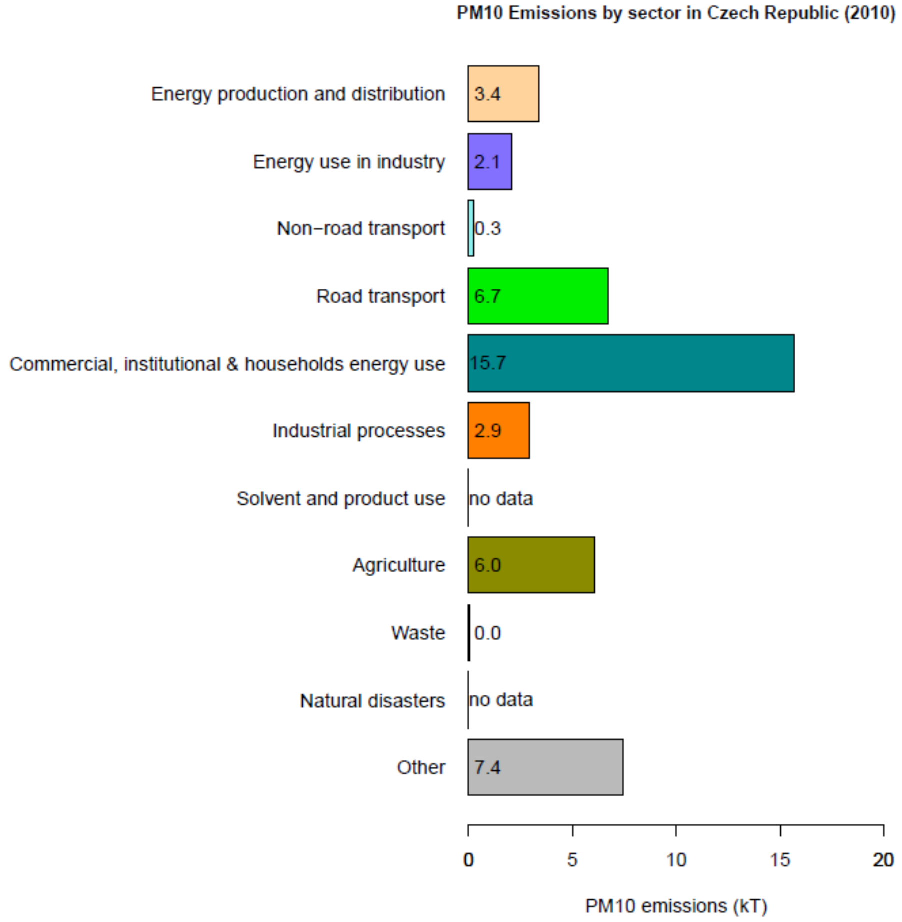


PM10 emissions in Cyprus
PM10 Emissions (kT)
Total PM10 Emissions (kT)
0
0
2000
2005
2010

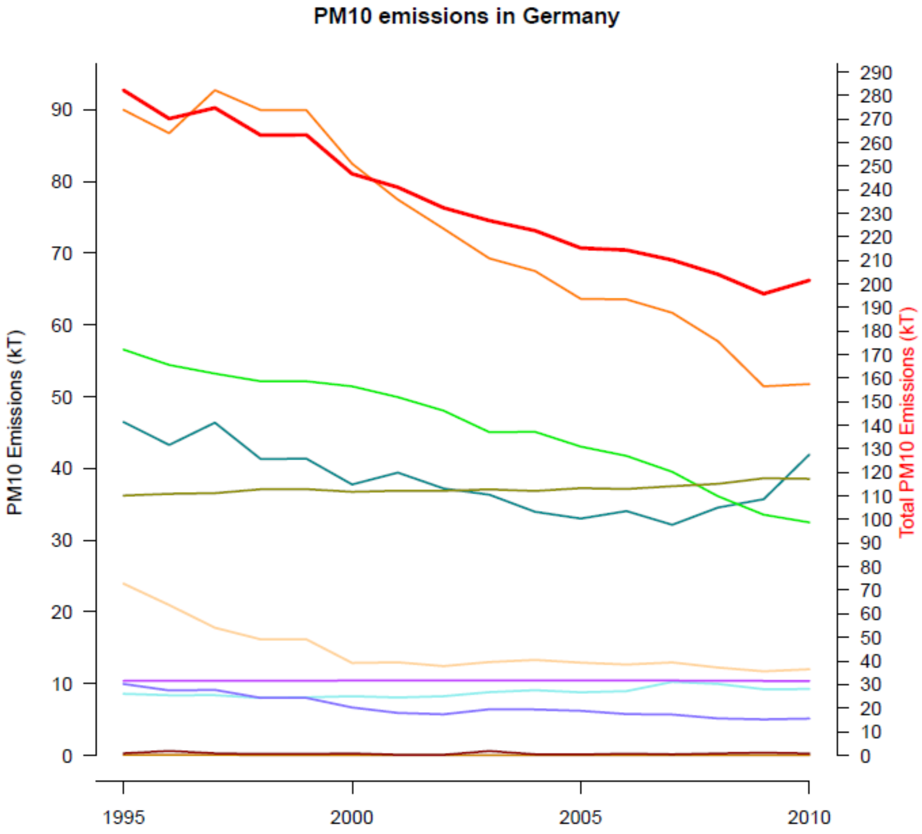


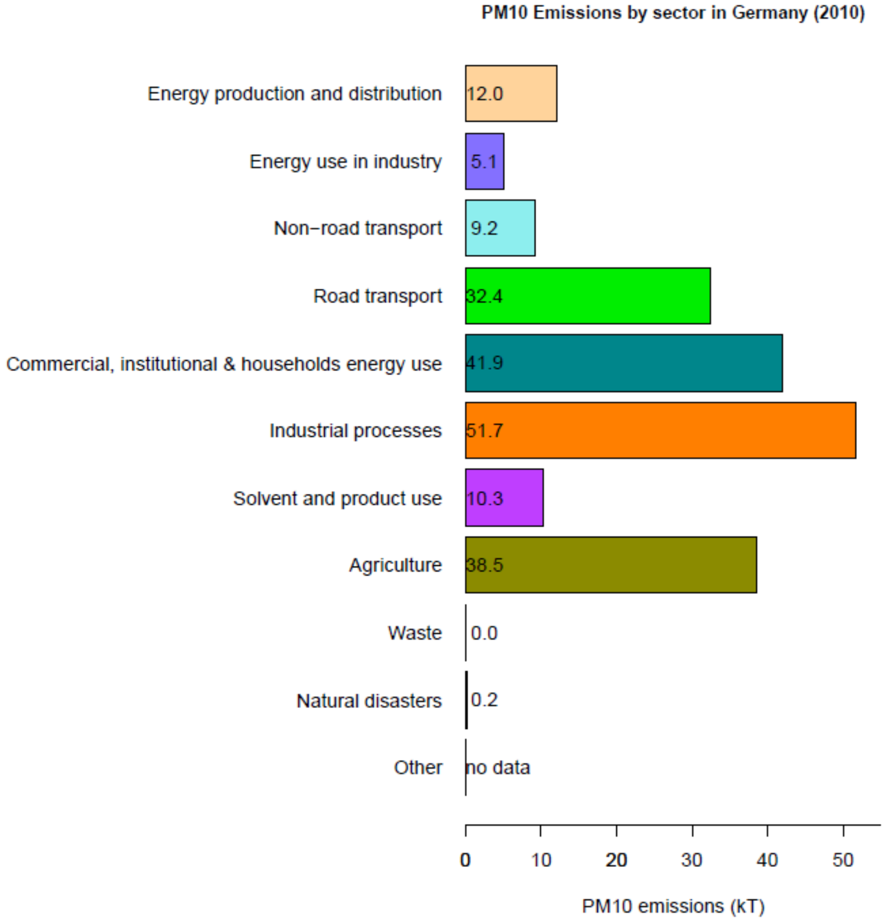
PM10 Emissions by sector in Cyprus (2010)
Energy production and distribution
0.5
Energy use in industry
0.2
Non−road transport
0.2
Road transport
0.6
Commercial, institutional & households energy use
0.1
Industrial processes
1.7
Solvent and product use
no data
Agriculture
0.4
Waste
0.0
Natural disasters
no data
Other
0.6
0
1
2
3
4
5
PM10 emissions (kT)

Czech Republic (PM_{10})

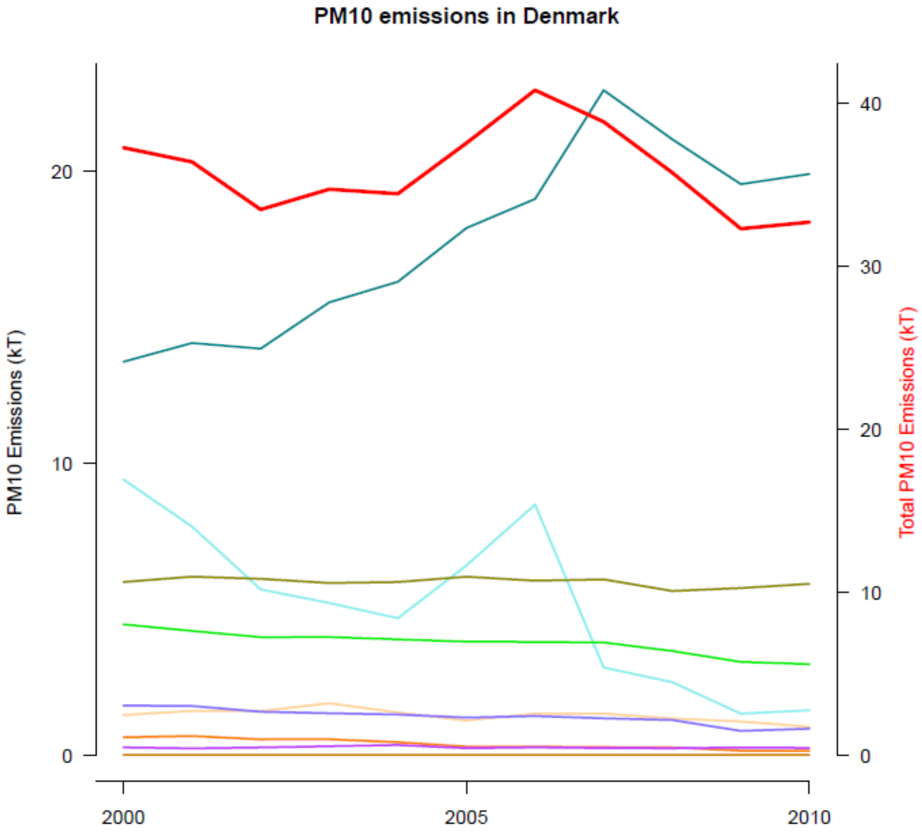


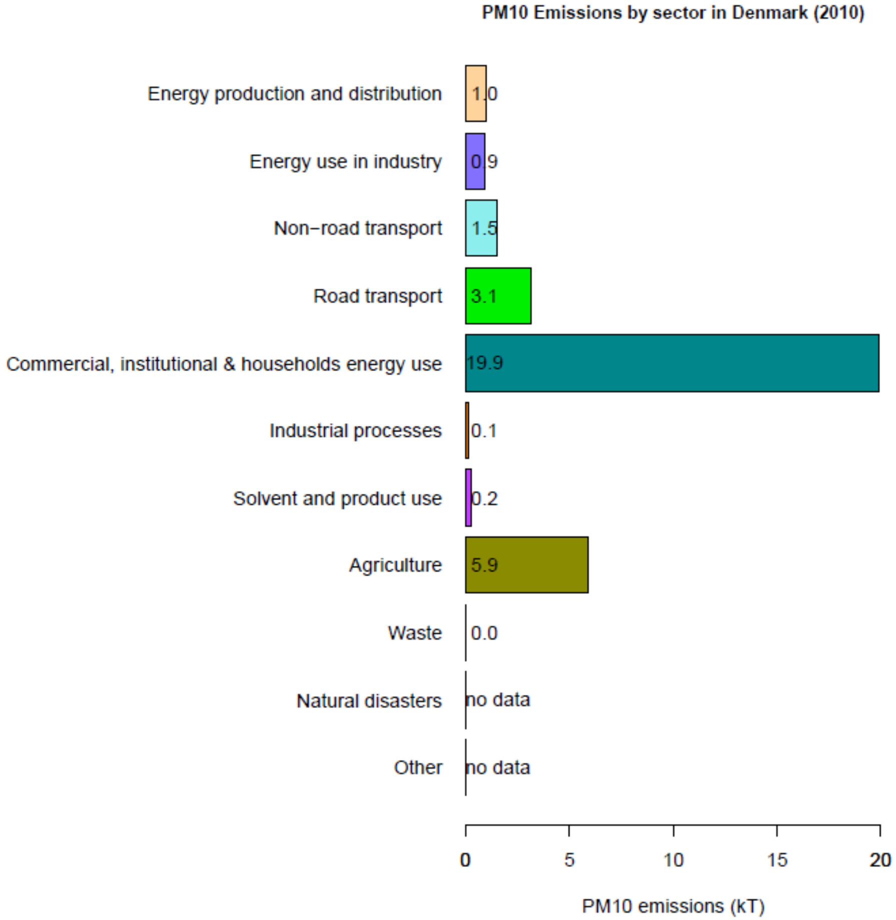
Germany (PM_{10})



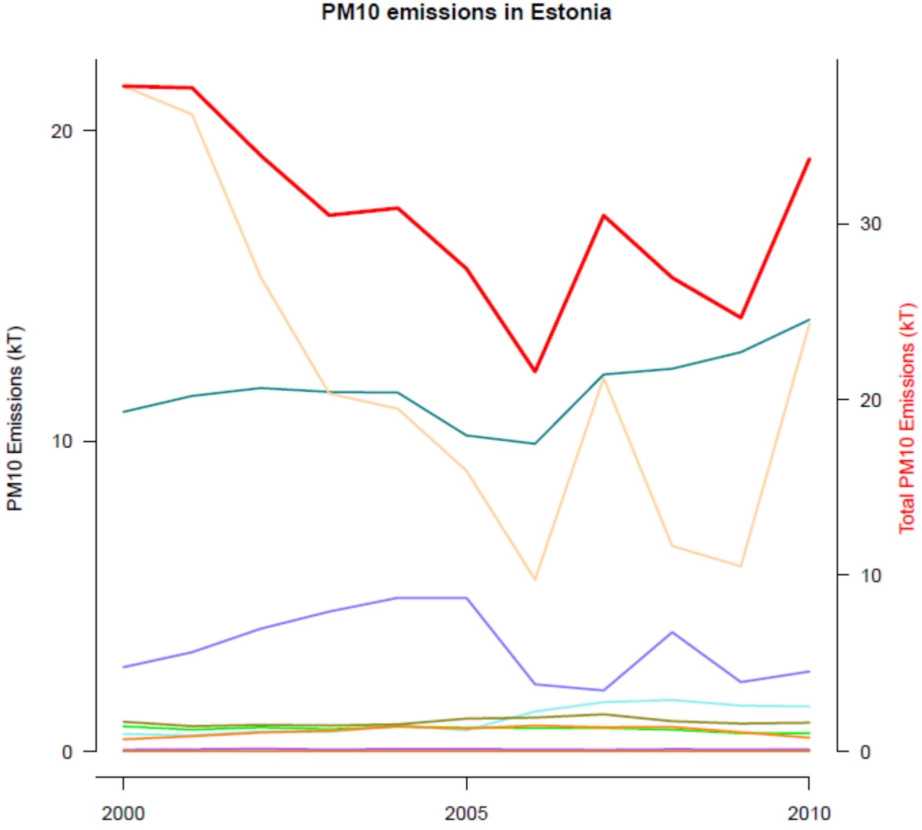


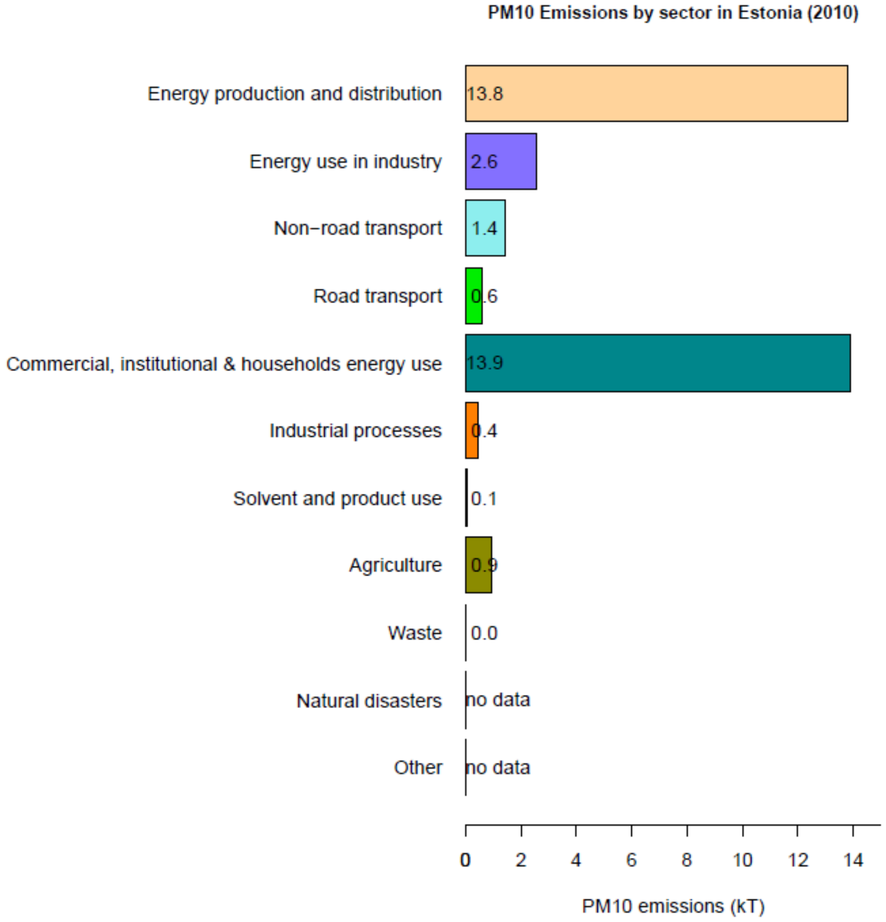
Denmark (PM_{10})





Estonia (PM_{10})





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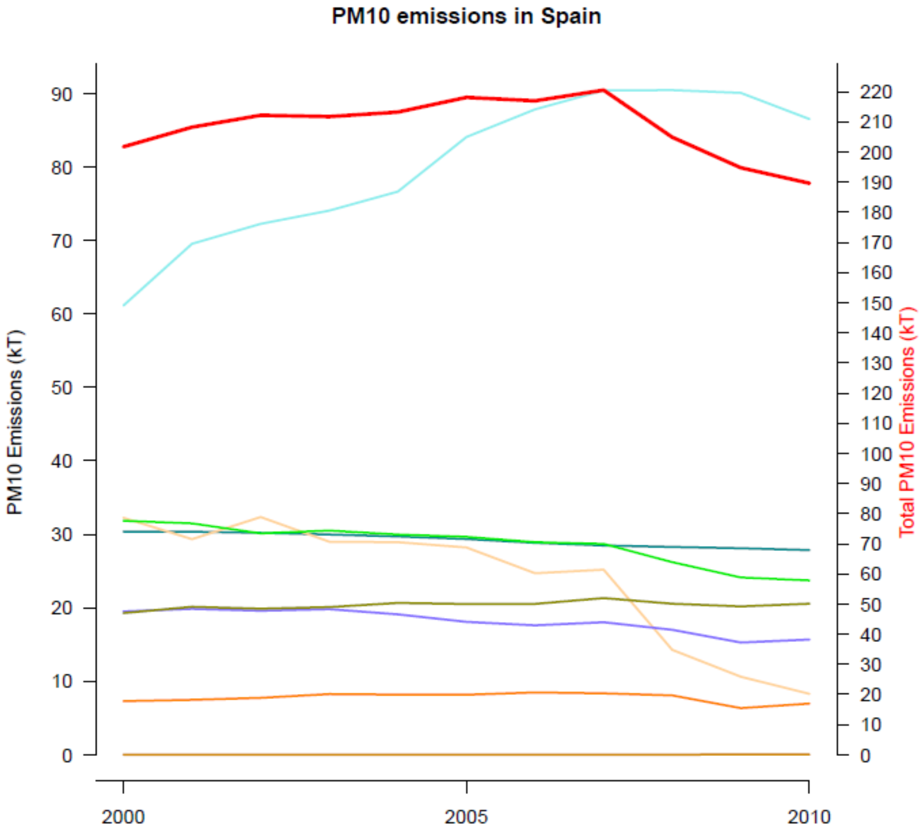
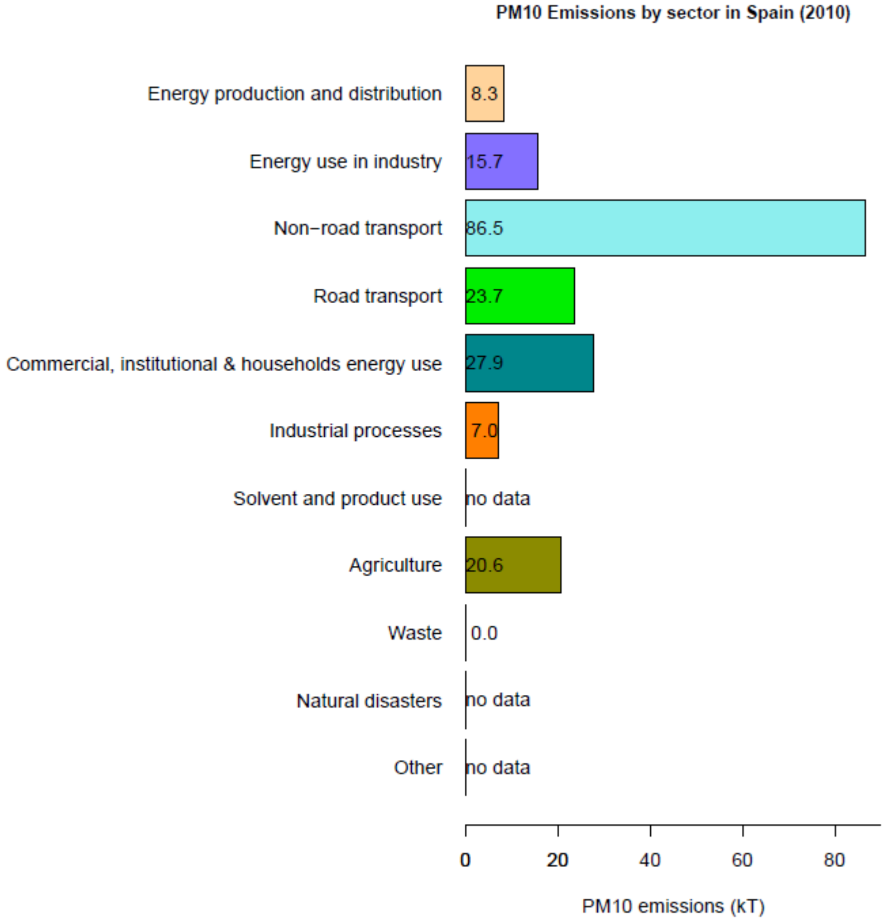
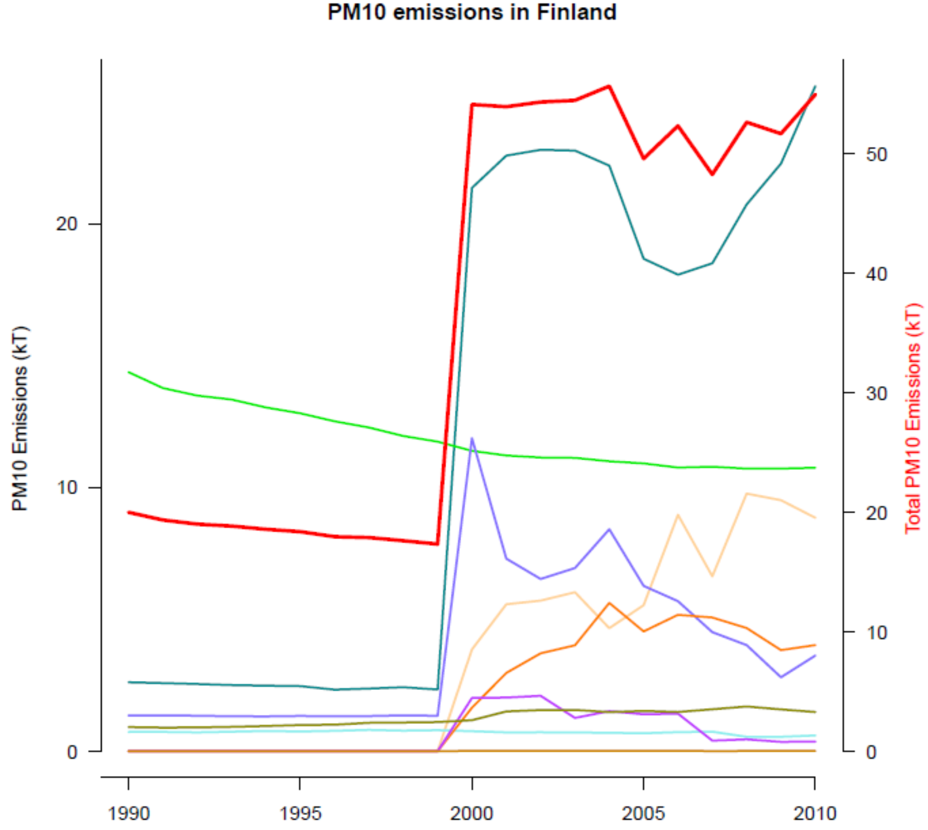
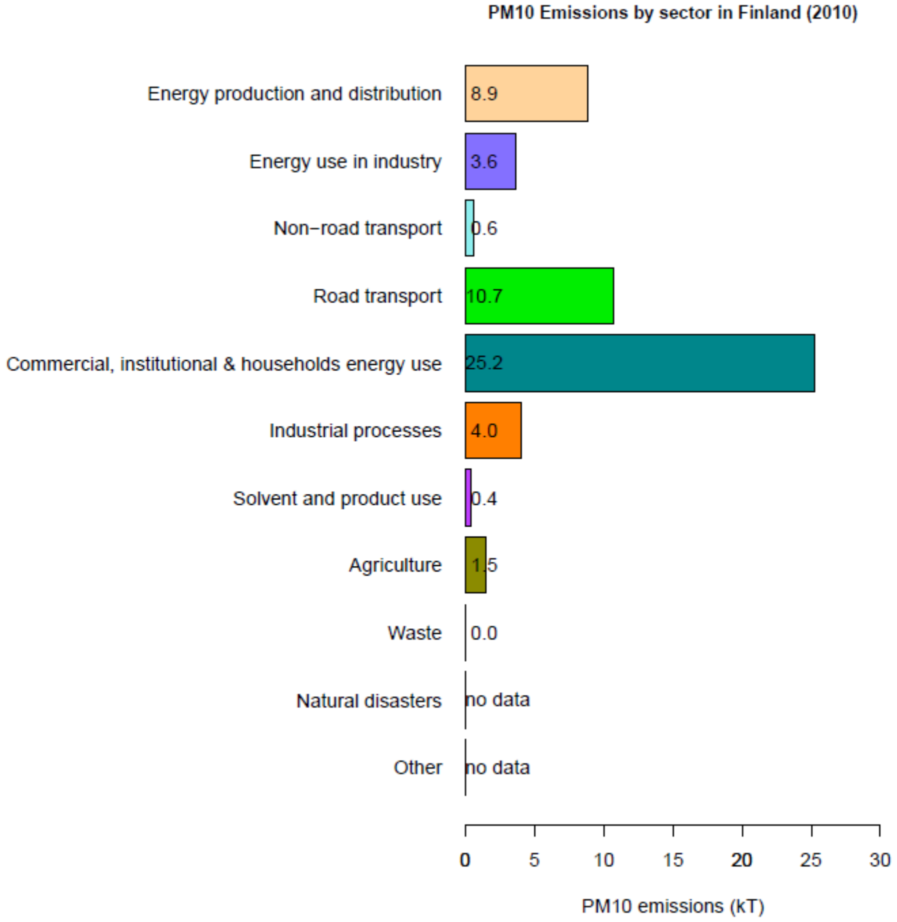
Spain (PM_{10})





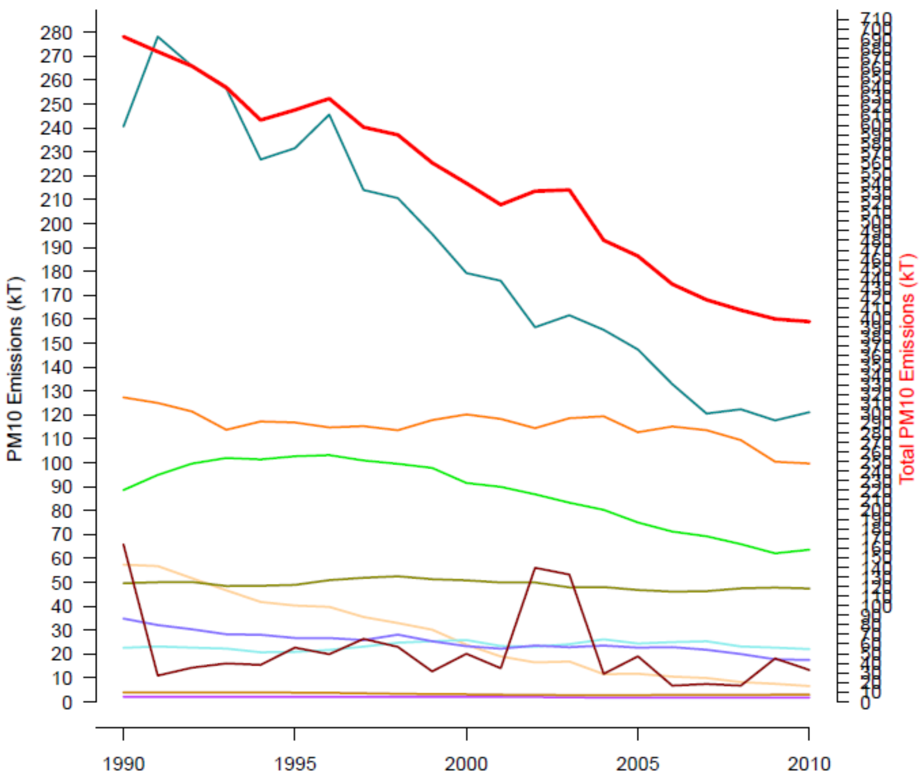
Finland (PM_{10})



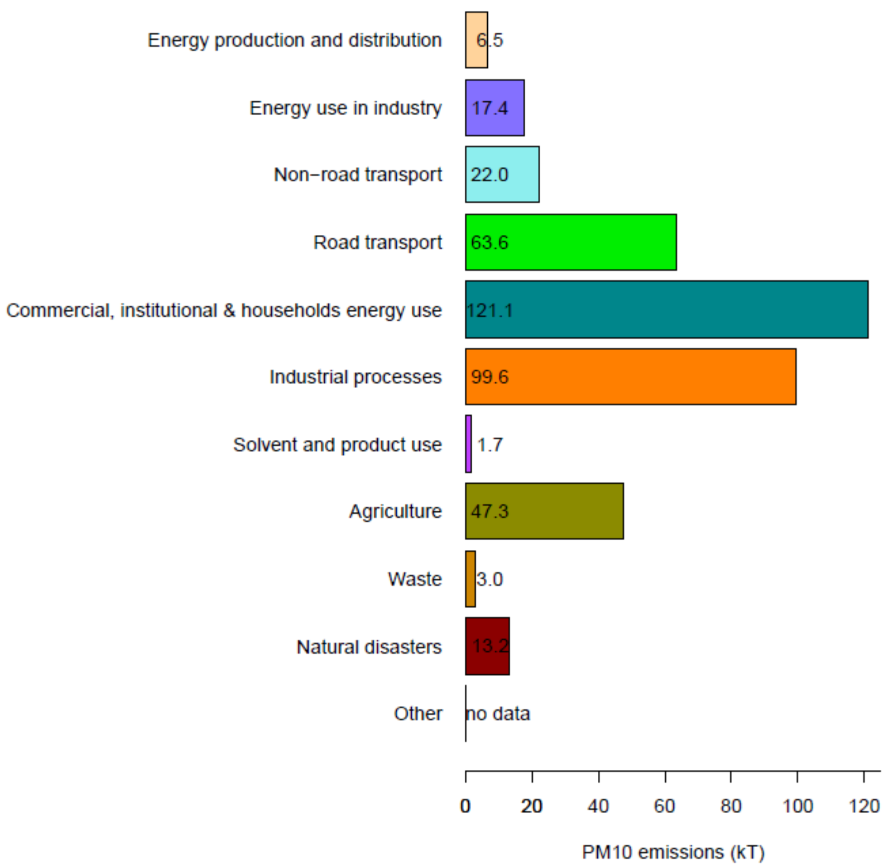


France (PM_{10})

PM10 emissions in France



PM10 Emissions by sector in France (2010)



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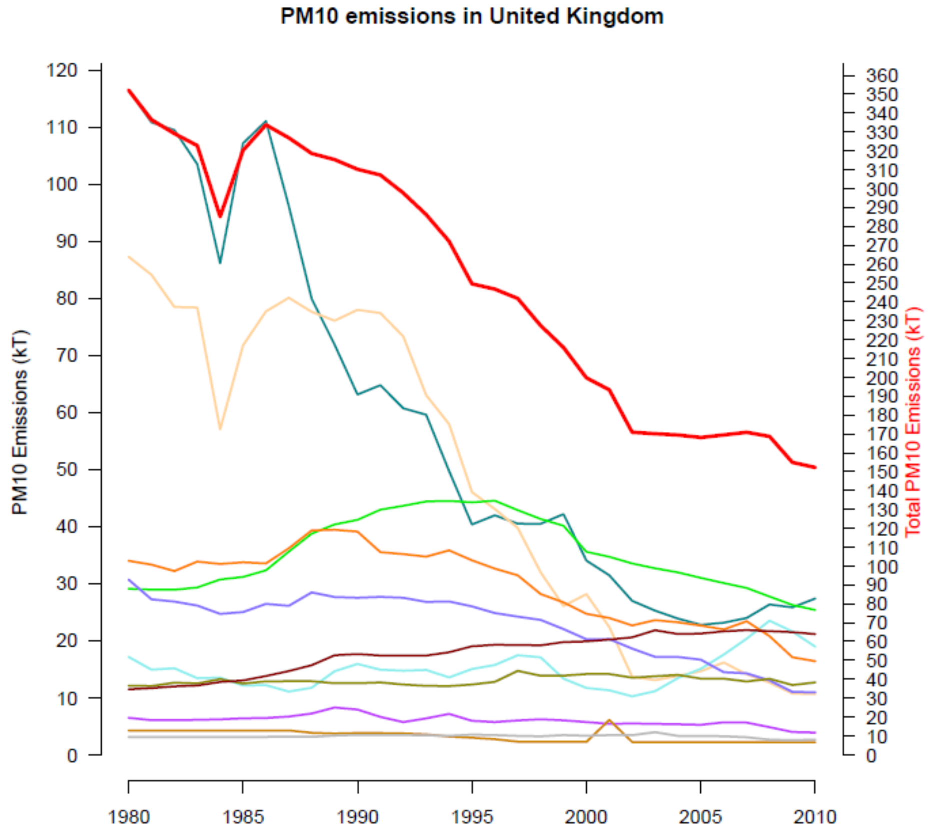
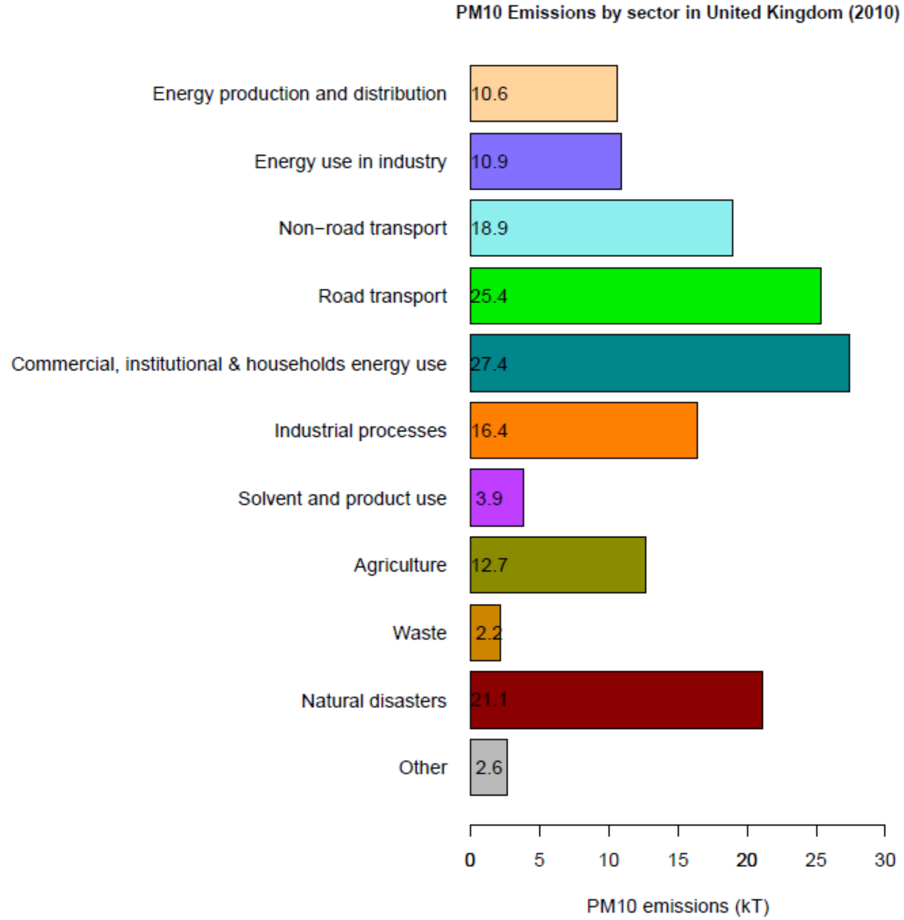
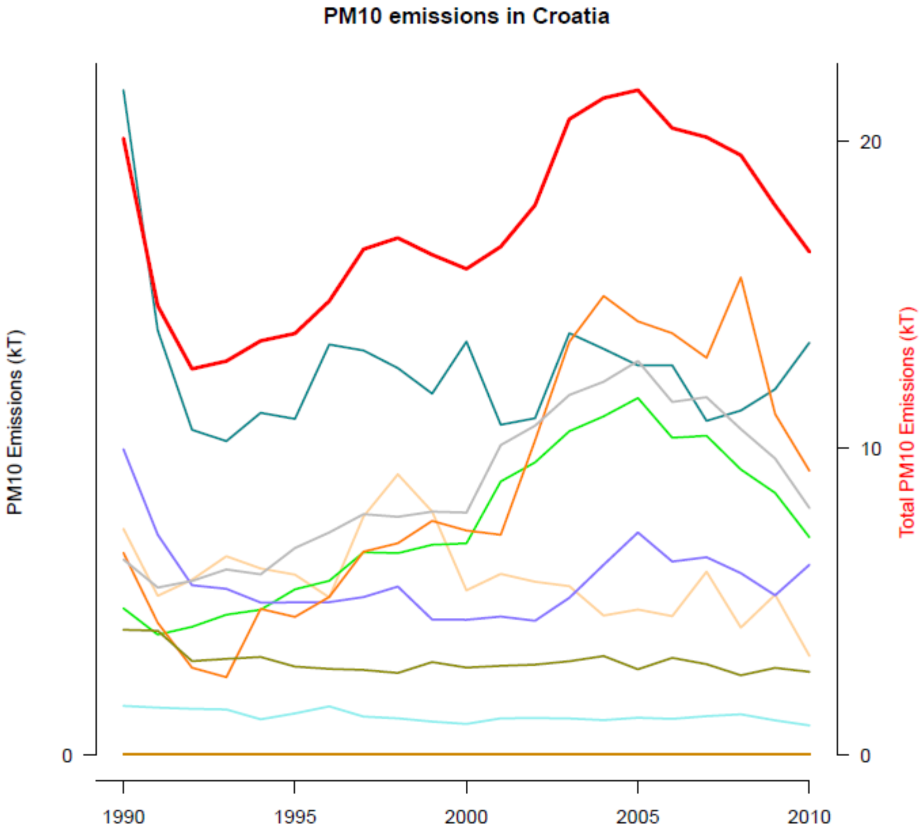
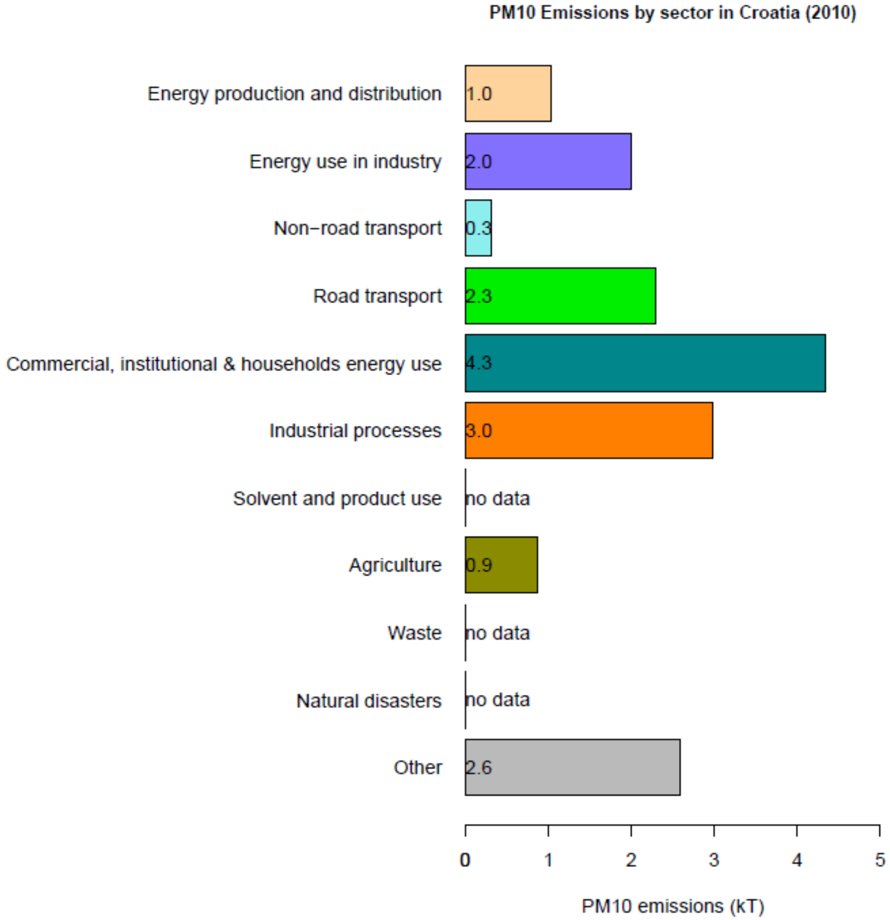
The United Kingdom (PM_{10})



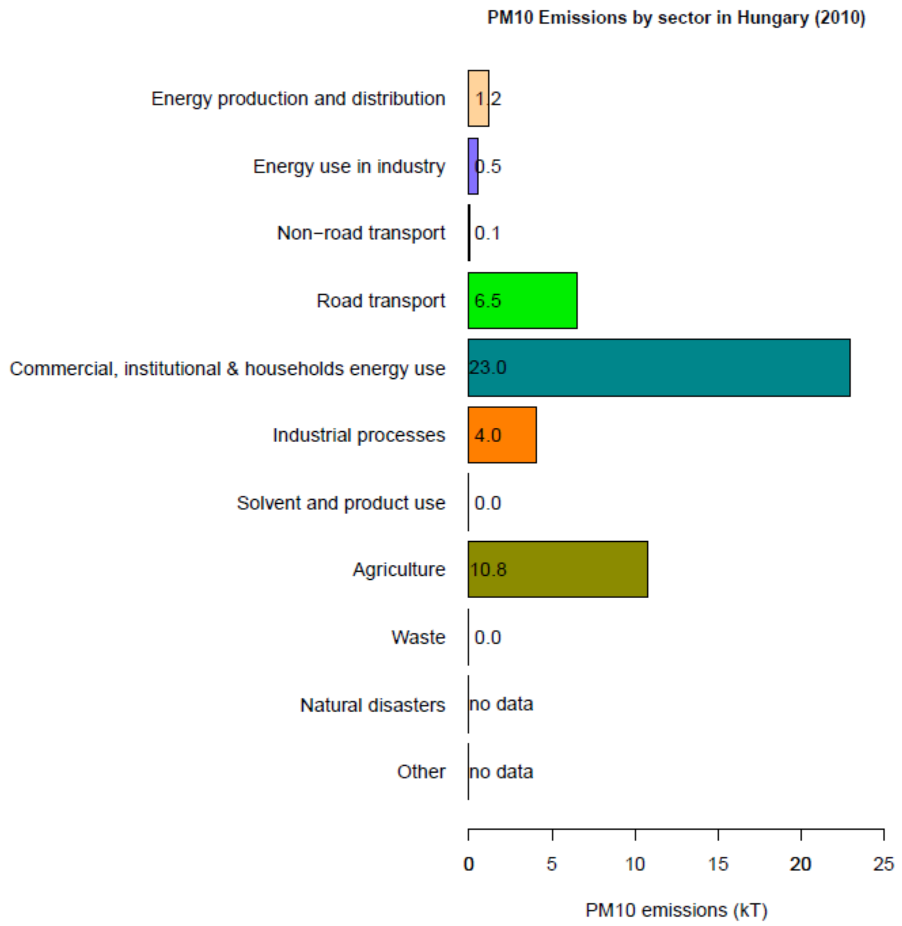


Croatia (PM_{10})

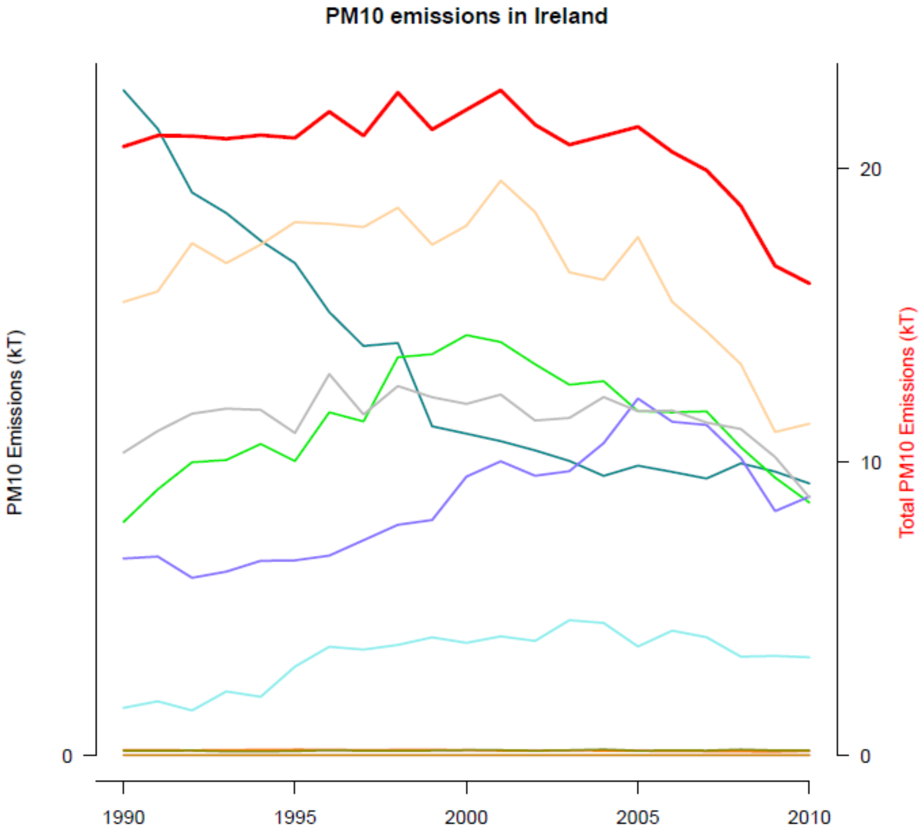


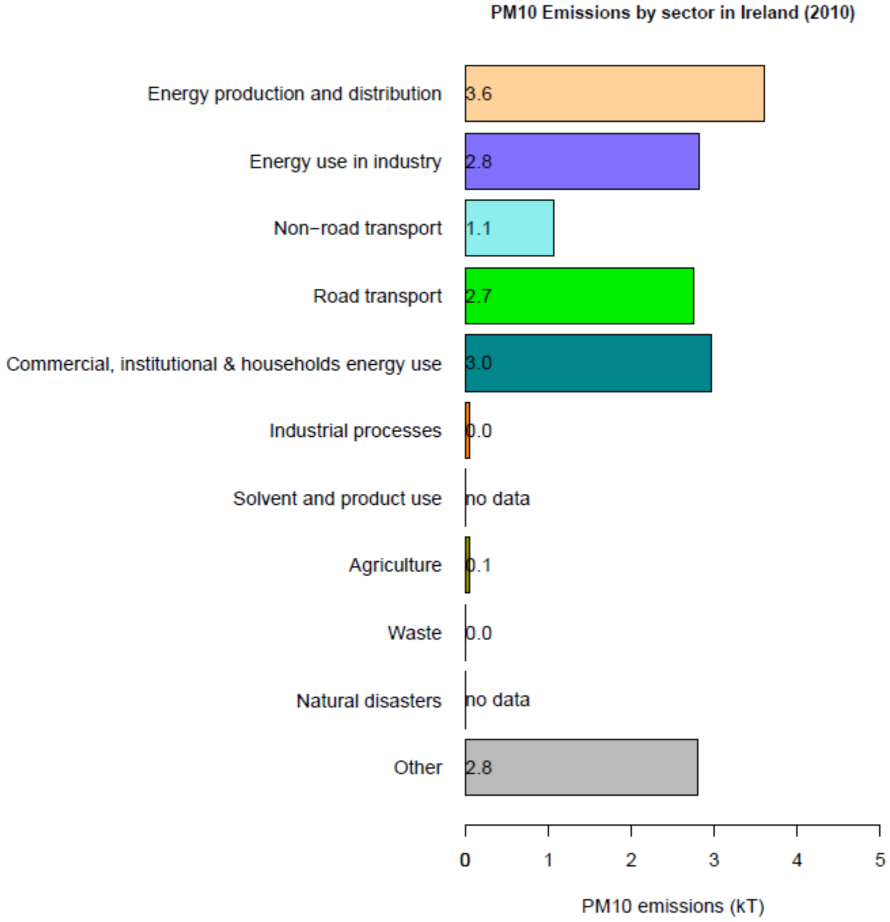


Hungary (PM_{10})

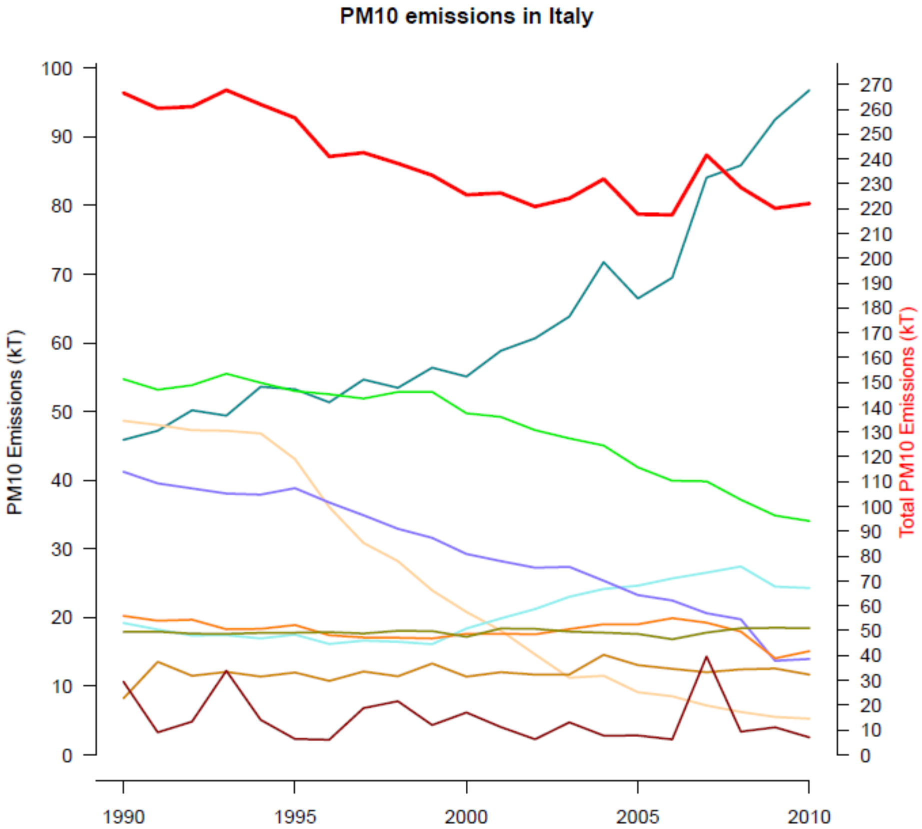


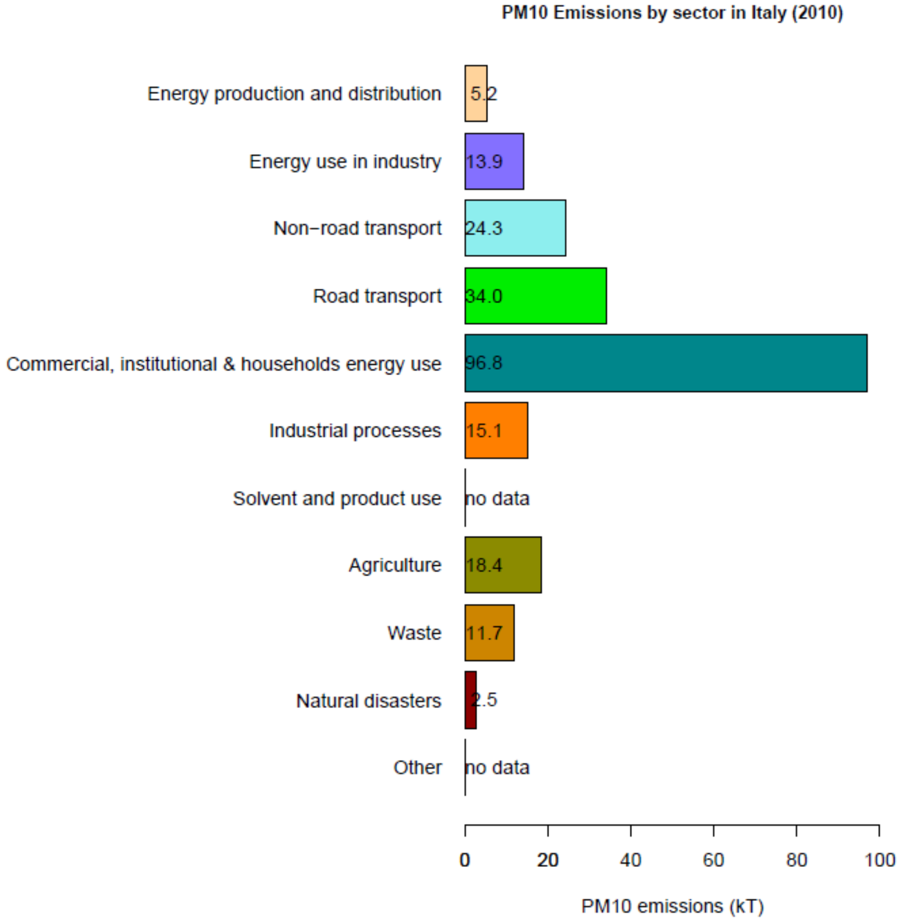
Ireland (PM_{10})



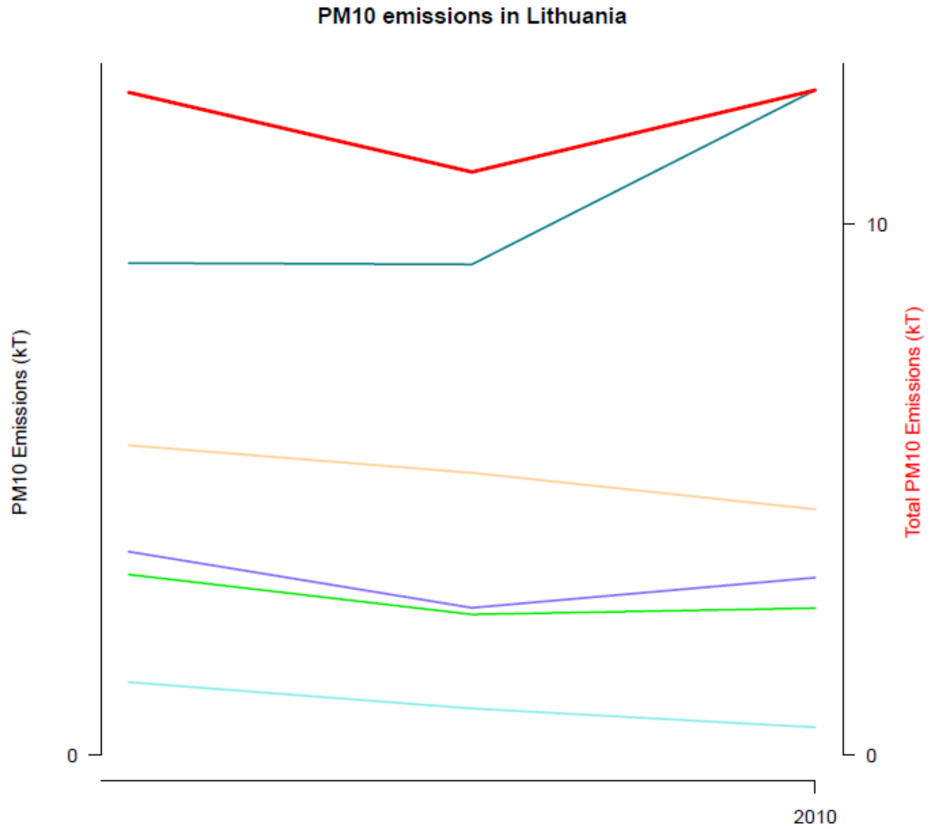


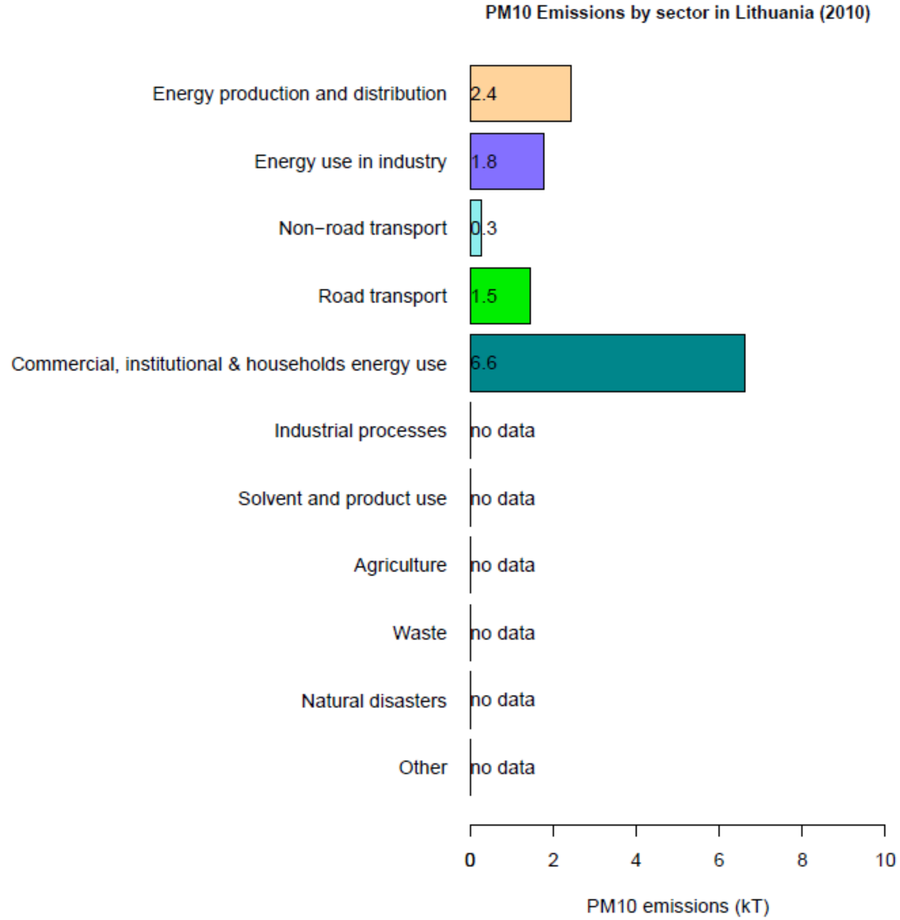
Italy (PM_{10})


PM10 emissions in Italy
PM10 Emissions (kT)
Total PM10 Emissions (kT)
100
90
80
70
60
50
40
30
20
10
0
270
260
250
240
230
220
210
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
1990
1995
2000
2005
2010

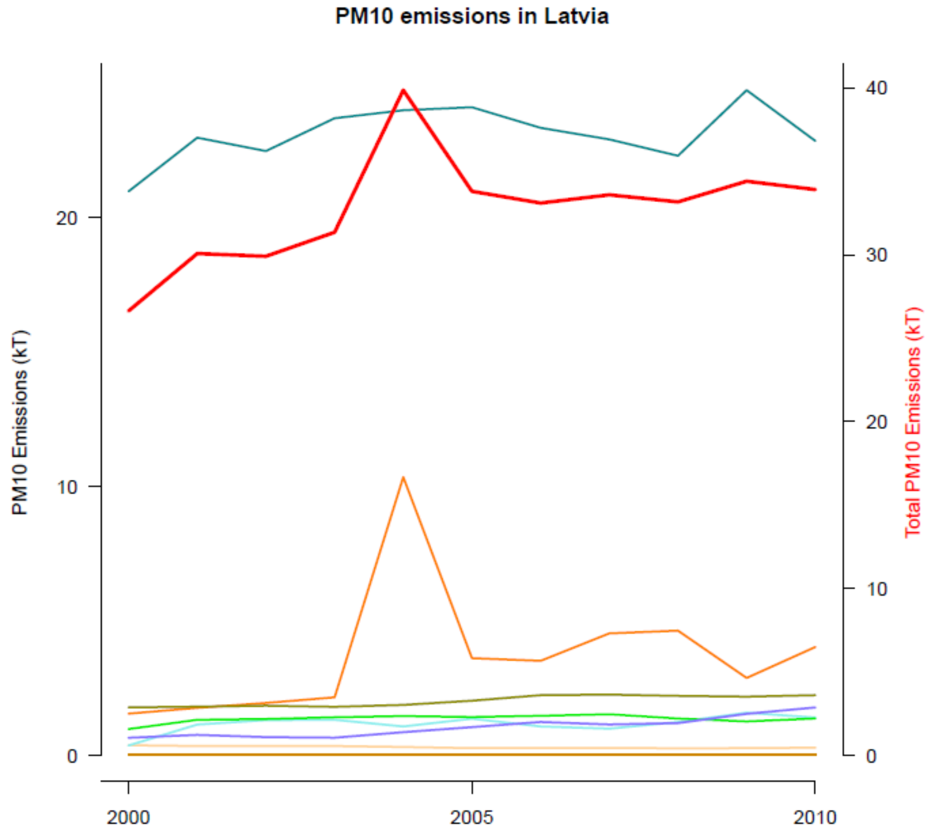

PM10 Emissions by sector in Italy (2010)
Energy production and distribution
5.2
Energy use in industry
13.9
Non−road transport
24.3
Road transport
34.0
Commercial, institutional & households energy use
96.8
Industrial processes
15.1
Solvent and product use
no data
Agriculture
18.4
Waste
11.7
Natural disasters
2.5
Other
no data
0
20
40
60
80
100
PM10 emissions (kT)

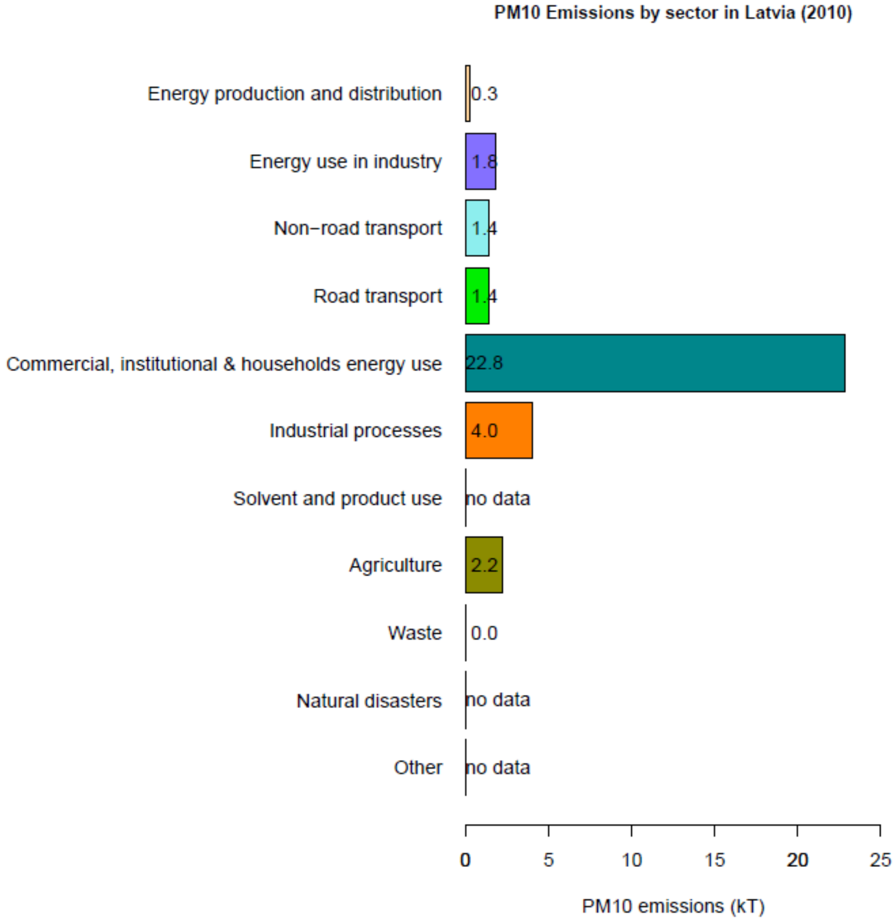
Lithuania (PM_{10})



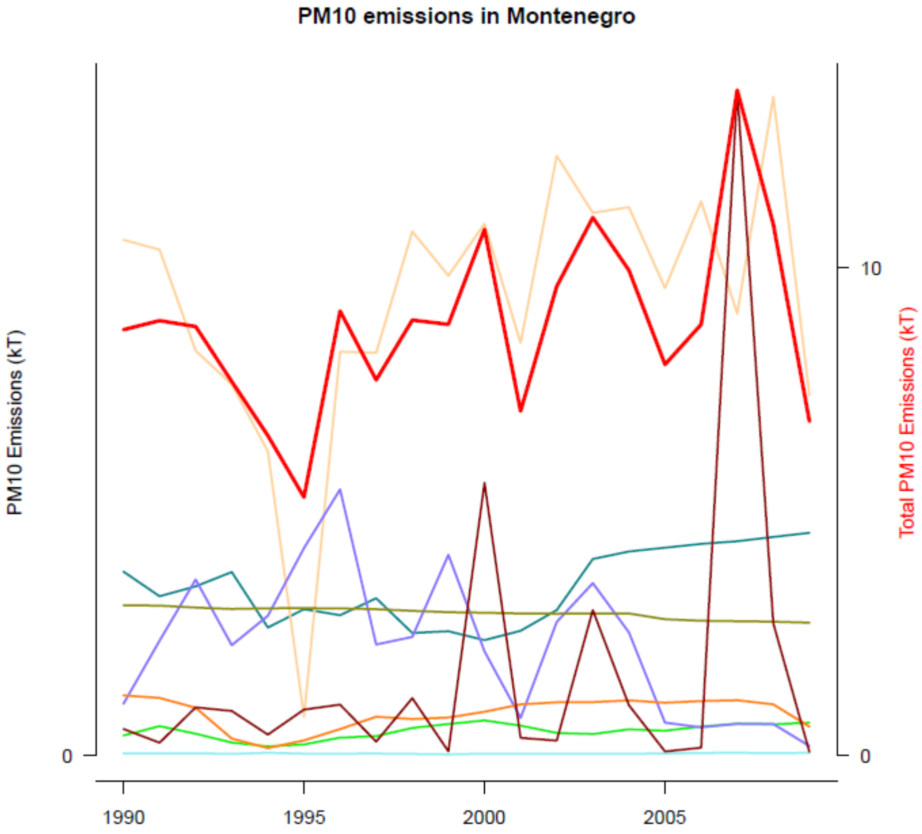


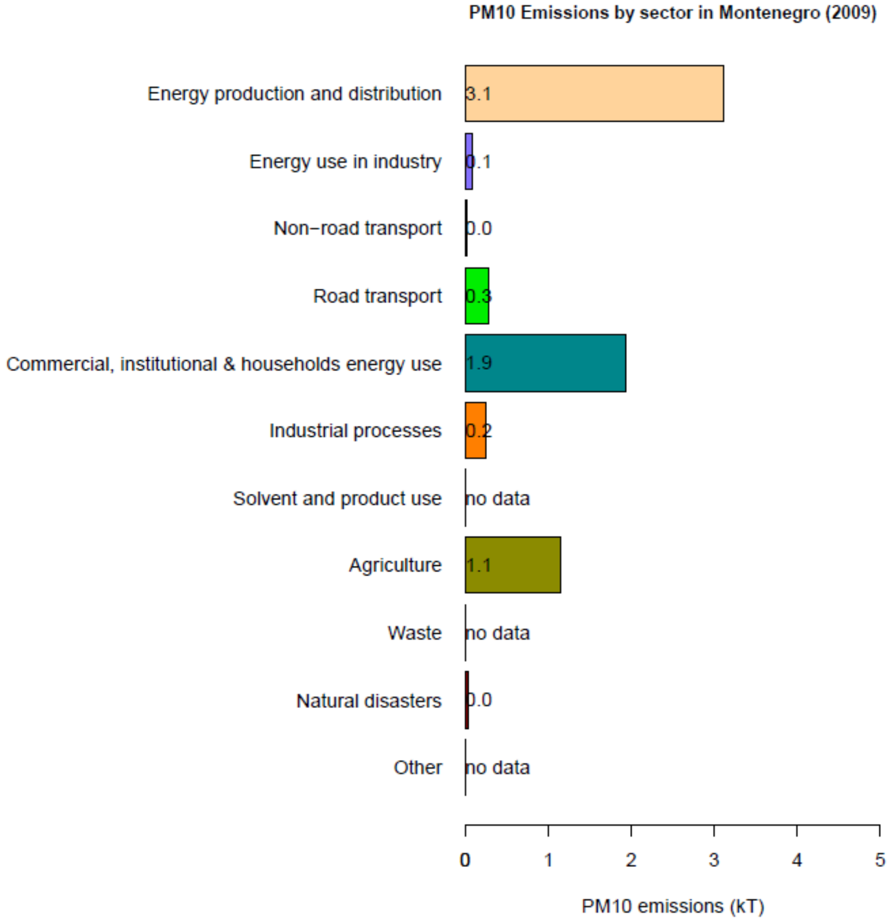
Latvia (PM_{10})


PM10 emissions in Latvia
PM10 Emissions (kT)
Total PM10 Emissions (kT)
0
10
20
0
10
20
30
40
2000
2005
2010

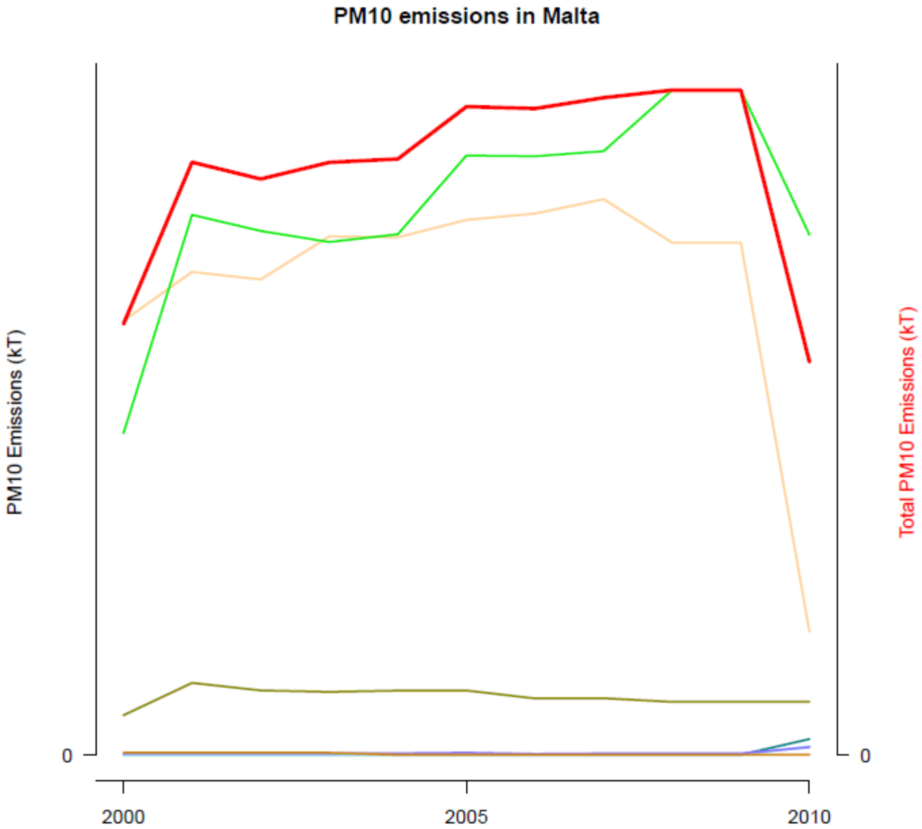

PM10 Emissions by sector in Latvia (2010)
Energy production and distribution
0.3
Energy use in industry
1.8
Non−road transport
1.4
Road transport
1.4
Commercial, institutional & households energy use
22.8
Industrial processes
4.0
Solvent and product use
no data
Agriculture
2.2
Waste
0.0
Natural disasters
no data
Other
no data
0
5
10
15
20
25
PM10 emissions (kT)

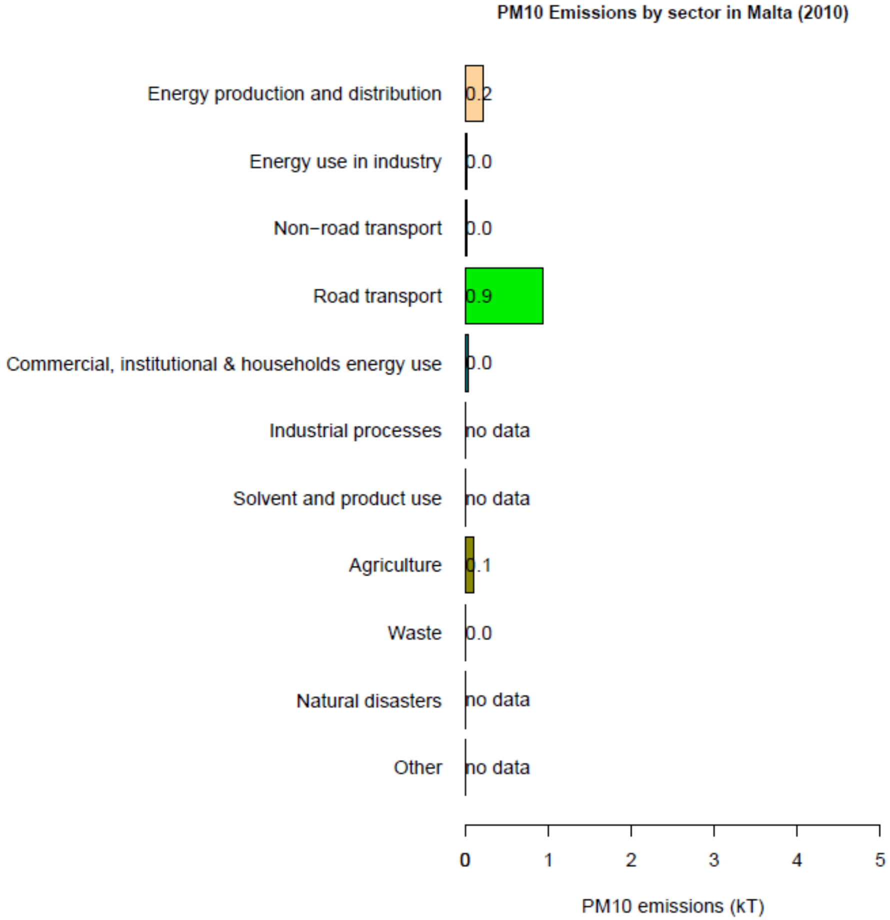
Montenegro (PM_{10})





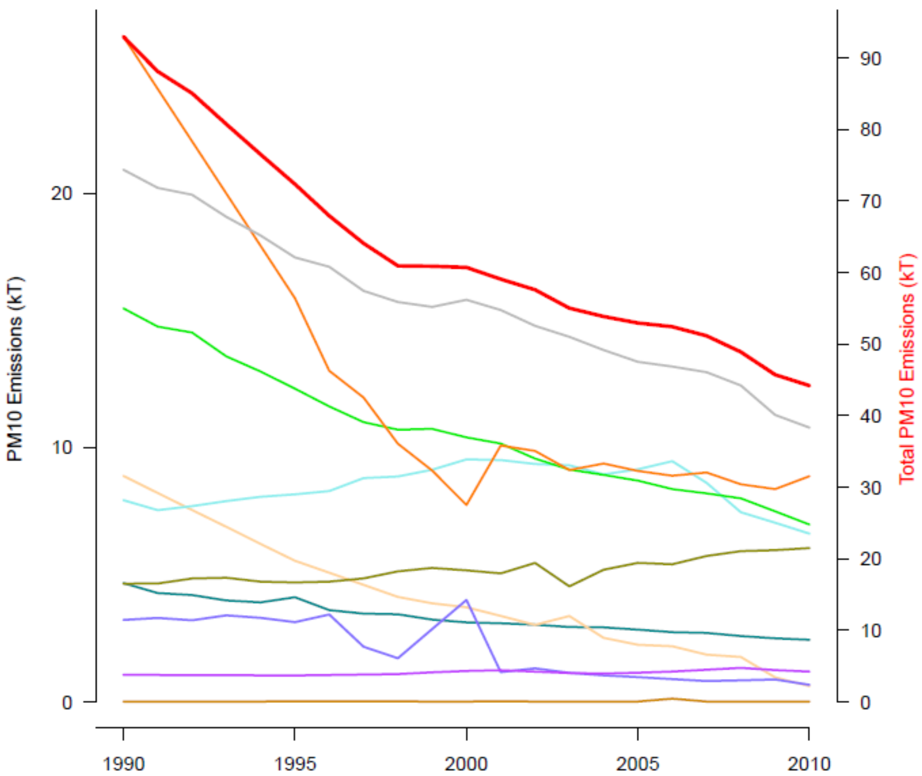
Malta (PM_{10})


PM10 emissions in Malta
PM10 Emissions (kT)
Total PM10 Emissions (kT)
0
0
2000
2005
2010

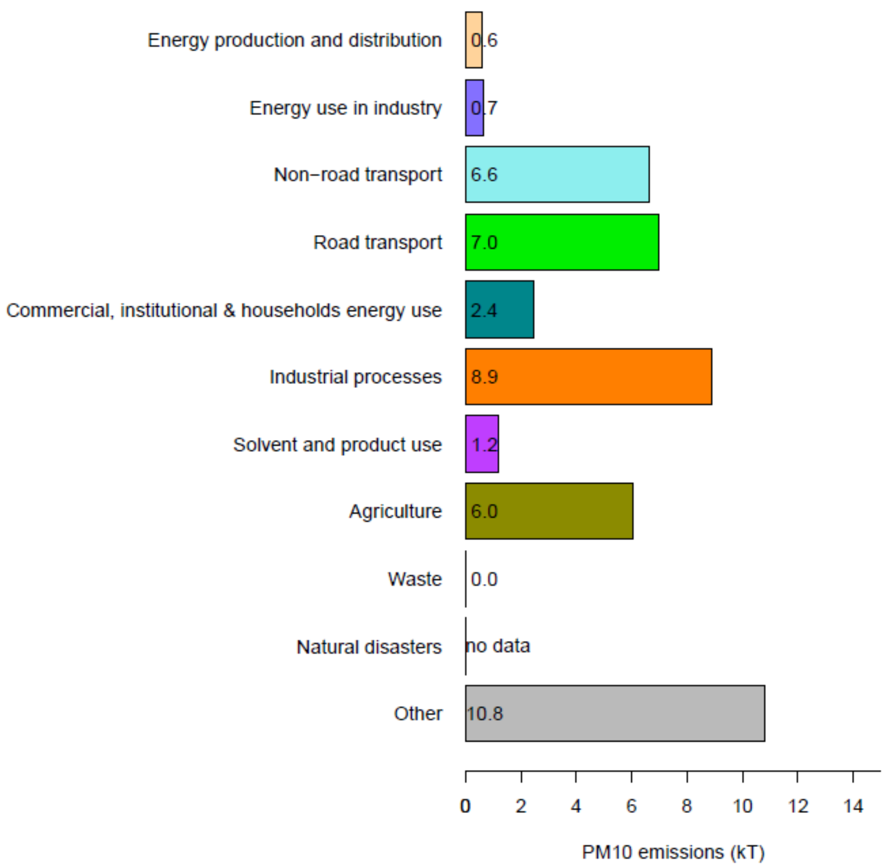

PM10 Emissions by sector in Malta (2010)
Energy production and distribution
0.2
Energy use in industry
0.0
Non−road transport
0.0
Road transport
0.9
Commercial, institutional & households energy use
0.0
Industrial processes
no data
Solvent and product use
no data
Agriculture
0.1
Waste
0.0
Natural disasters
no data
Other
no data
0
1
2
3
4
5
PM10 emissions (kT)

The Netherlands (PM_{10})

PM10 emissions in Netherlands



PM10 Emissions by sector in Netherlands (2010)



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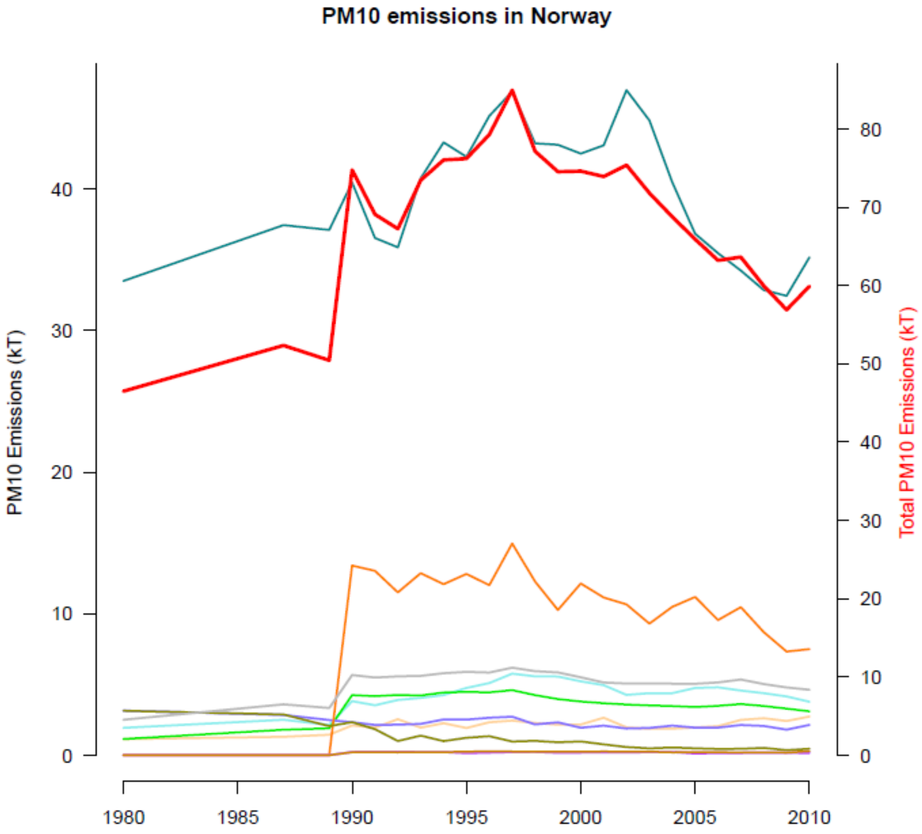
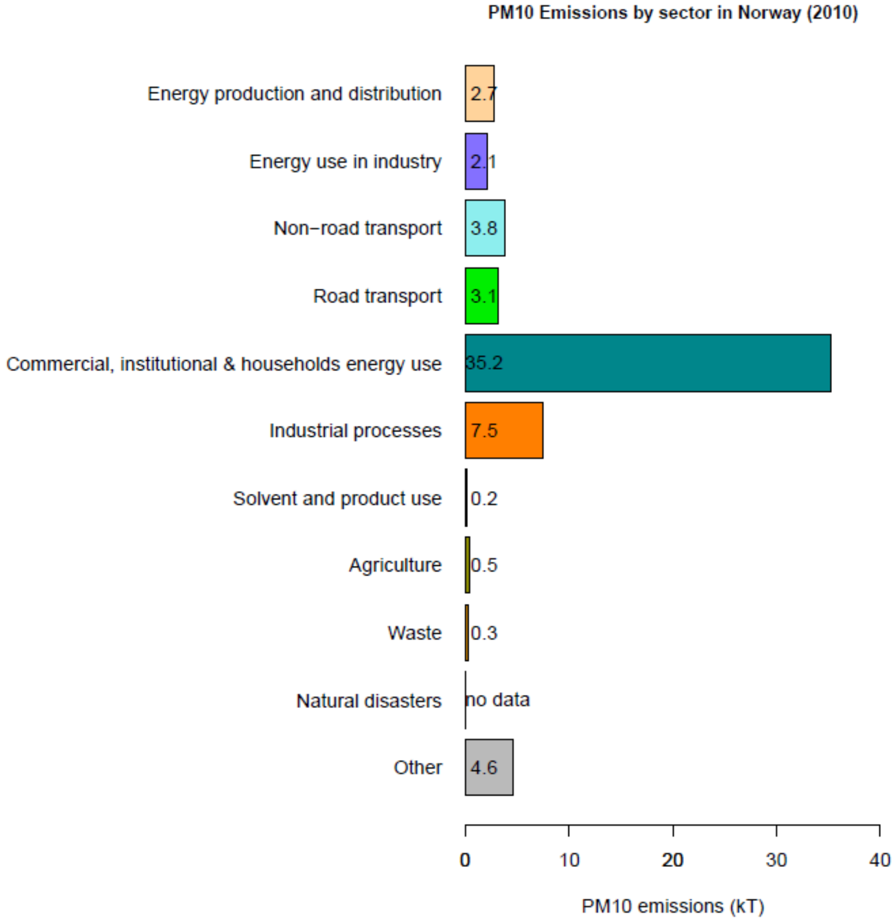
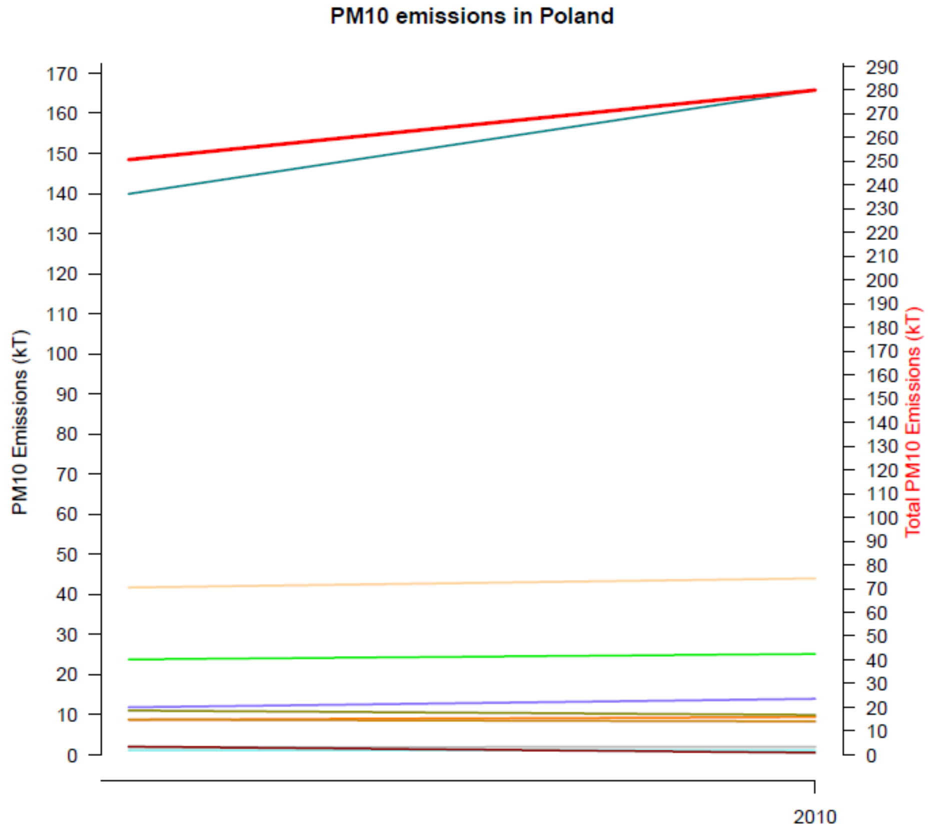
Norway (PM_{10})

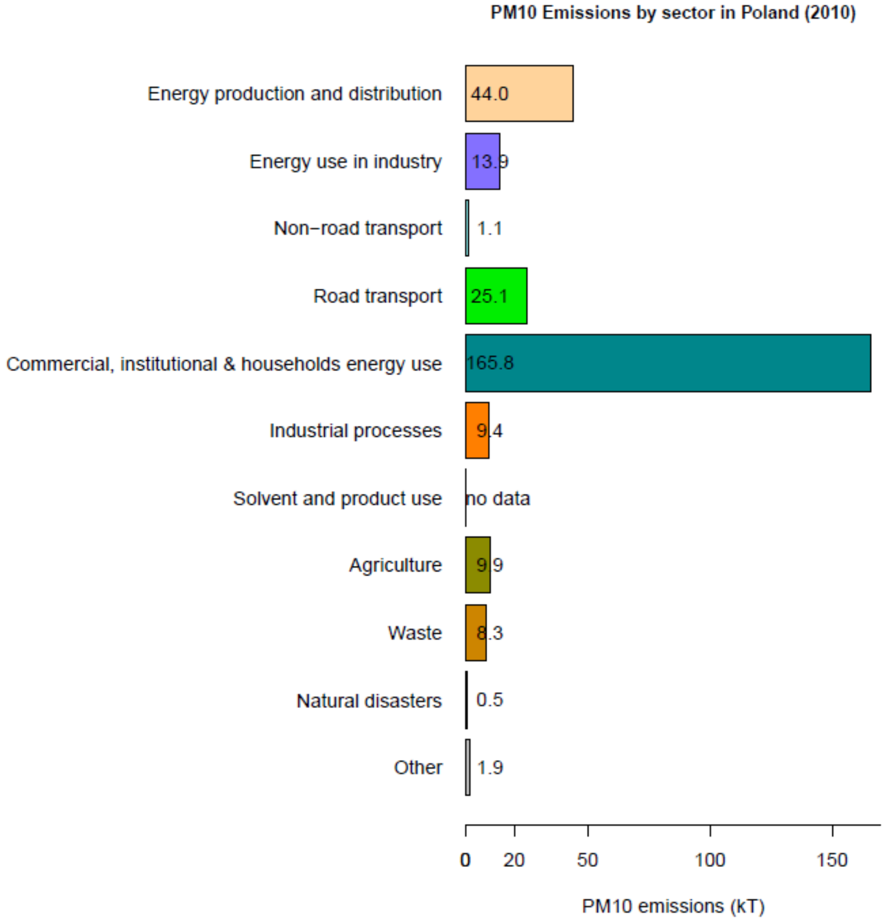




Poland (PM_{10})



PM10 emissions in Poland
PM10 Emissions (kT)
Total PM10 Emissions (kT)
170
160
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
290
280
270
260
250
240
230
220
210
200
190
180
170
160
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
2010



PM10 Emissions by sector in Poland (2010)
Energy production and distribution
44.0
Energy use in industry
13.9
Non−road transport
1.1
Road transport
25.1
Commercial, institutional & households energy use
165.8
Industrial processes
9.4
Solvent and product use
no data
Agriculture
9.9
Waste
8.3
Natural disasters
0.5
Other
1.9
0
20
50
100
150
PM10 emissions (kT)

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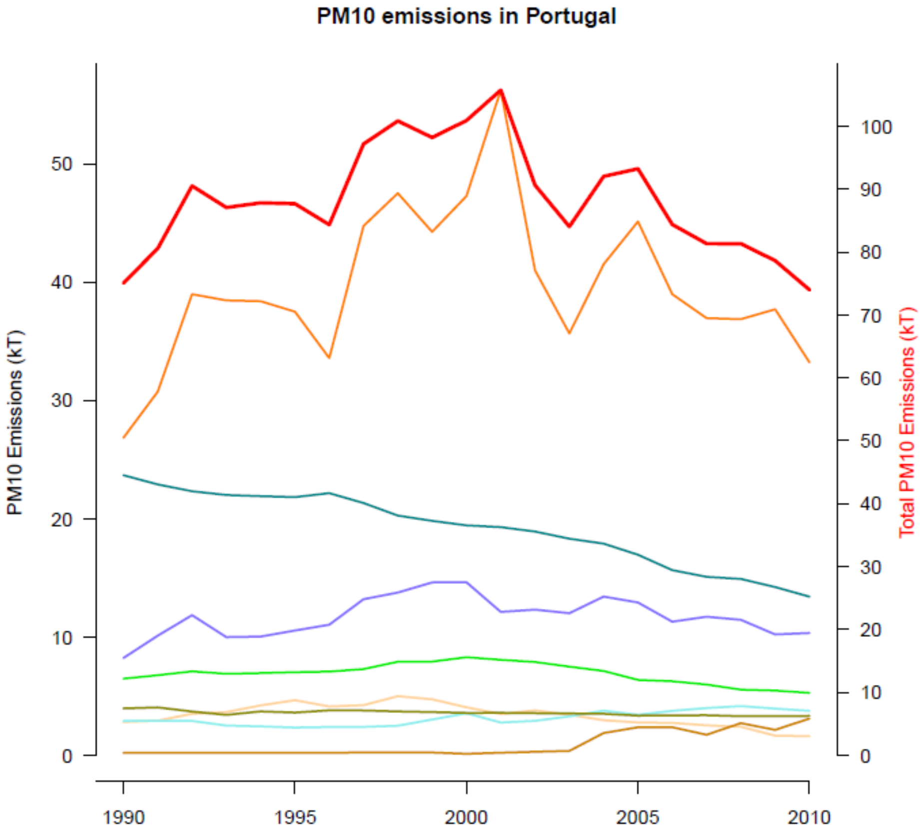
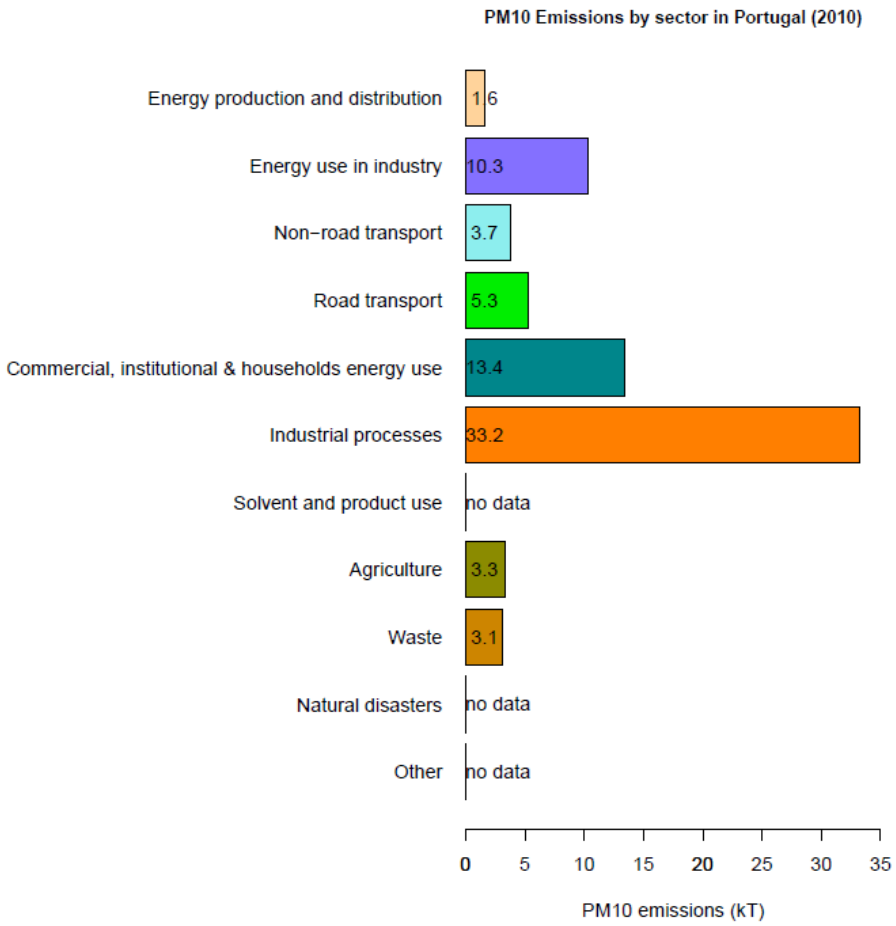
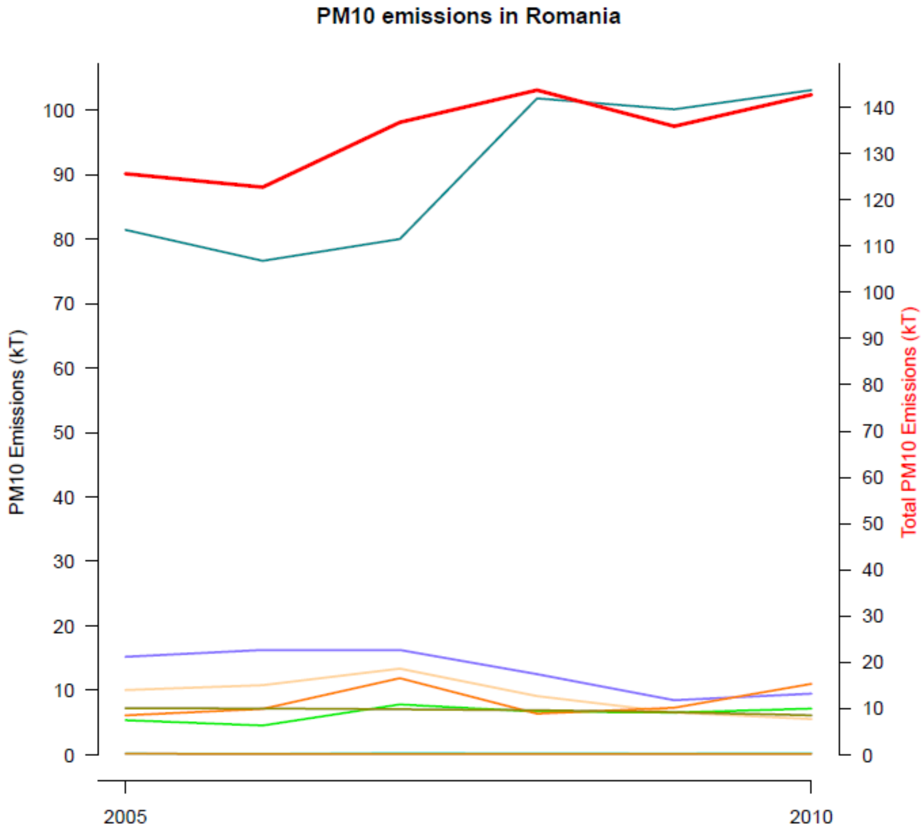
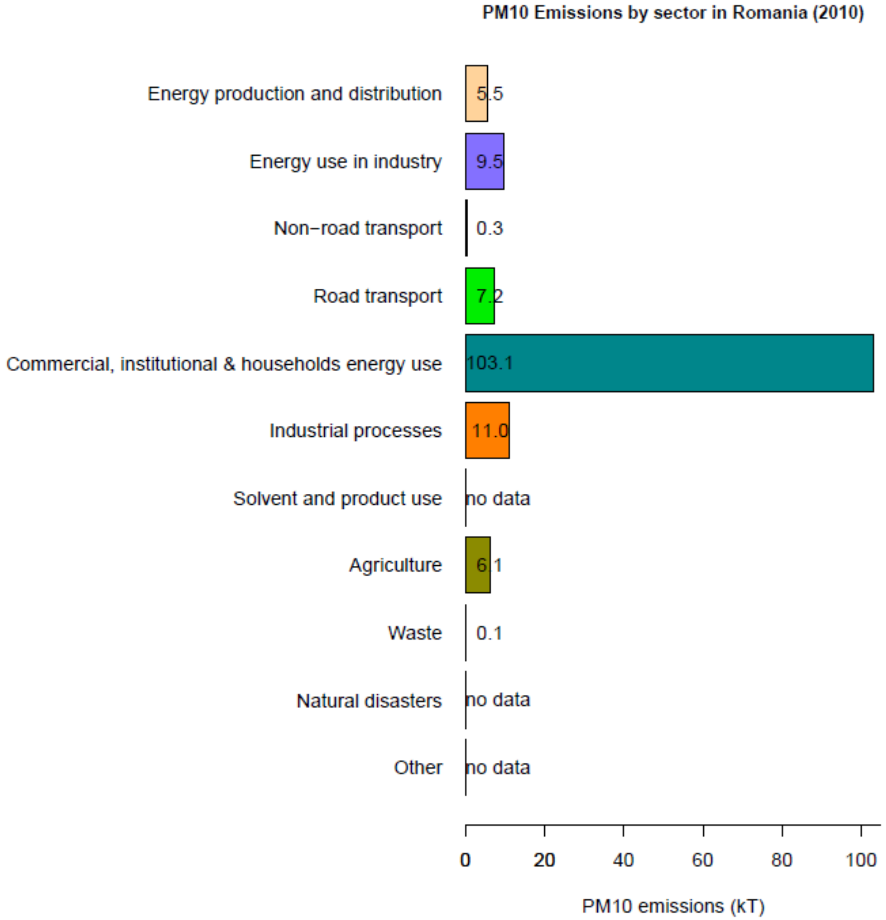
Portugal (PM_{10})





Romania (PM$_{10}$)





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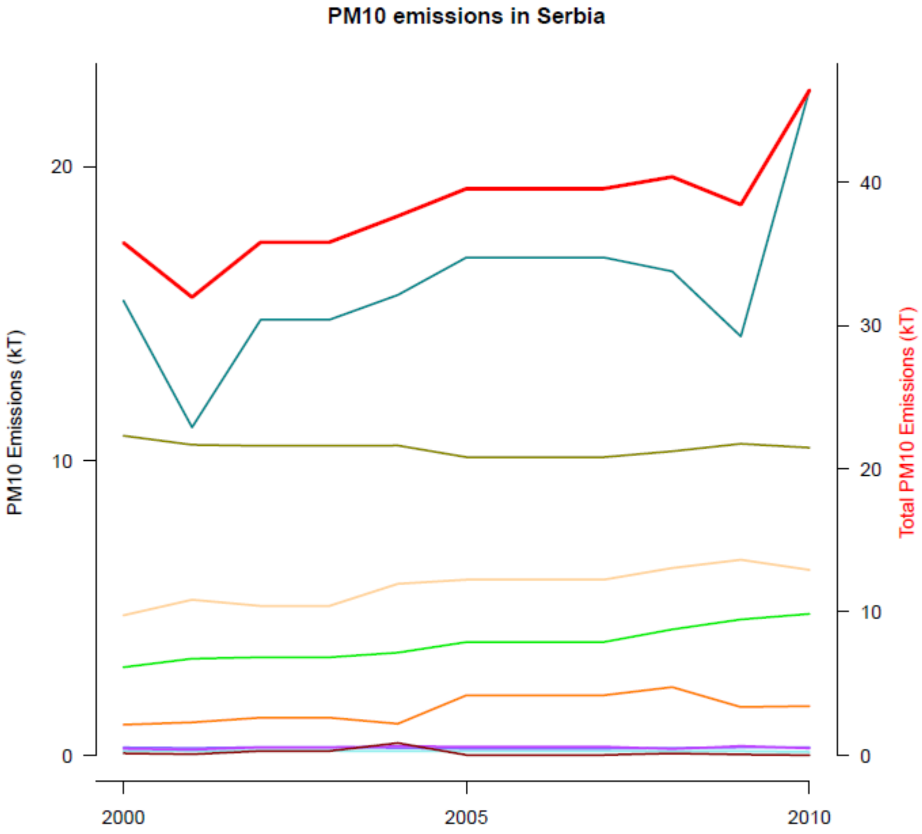
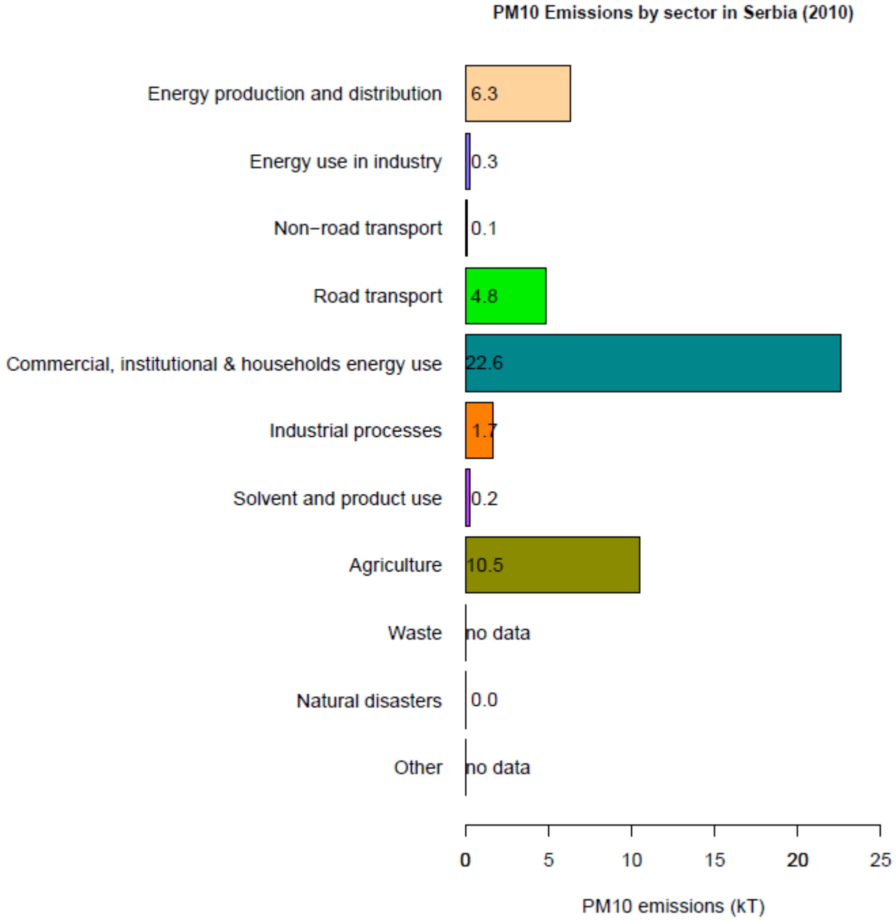
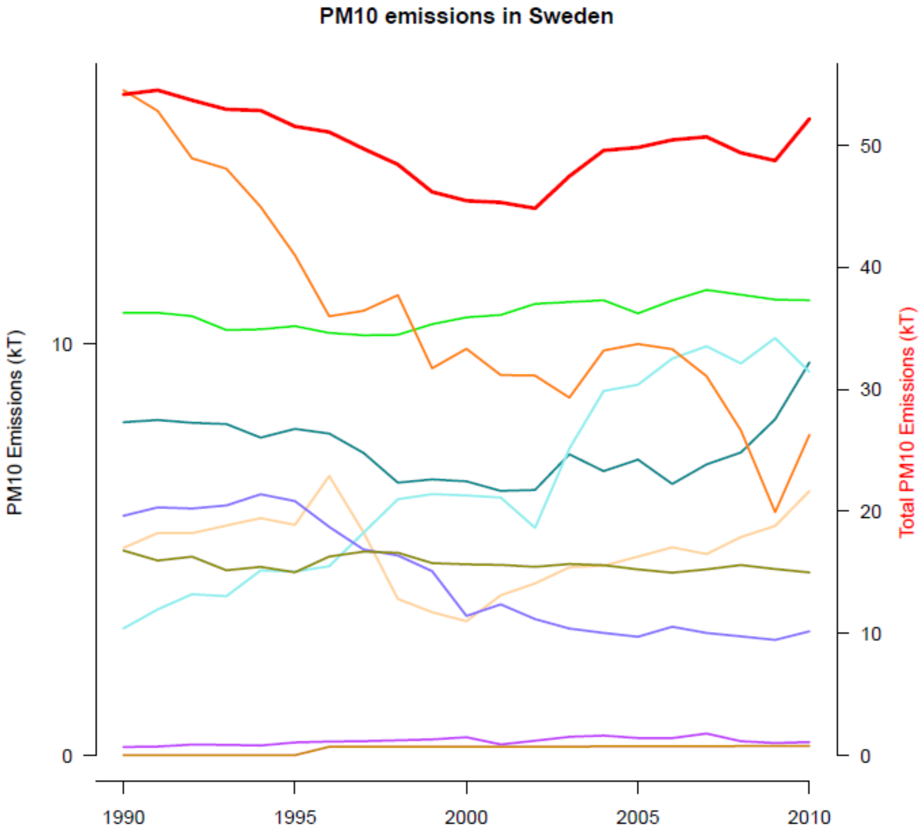
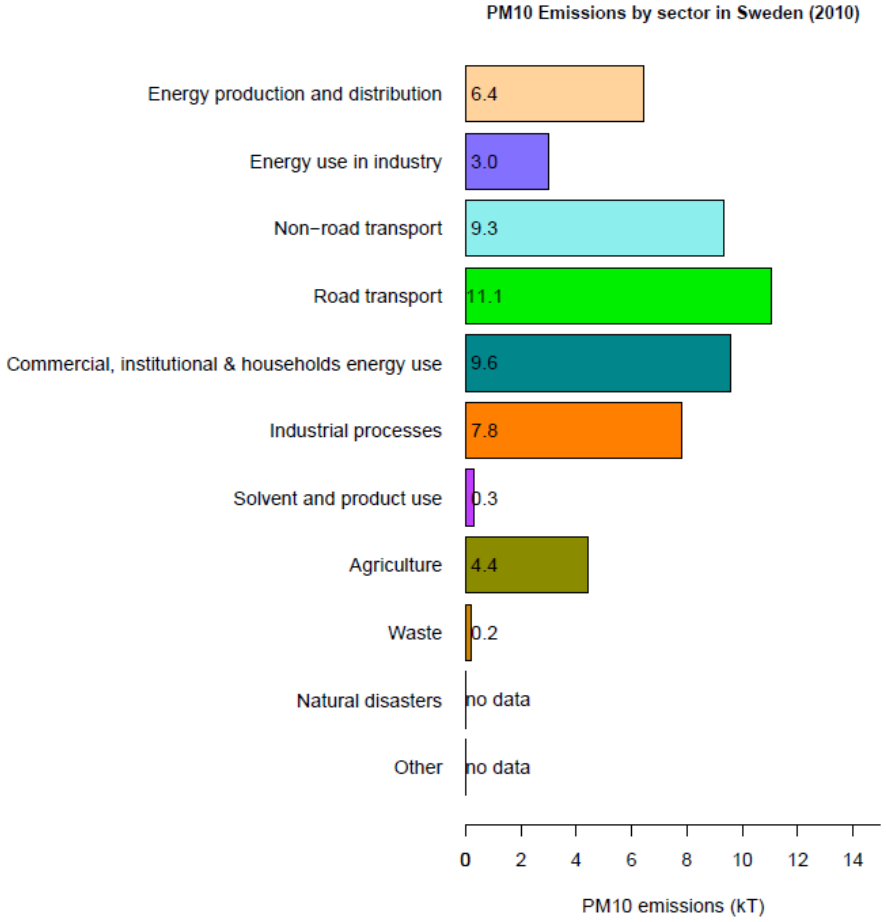
Serbia (PM_{10})



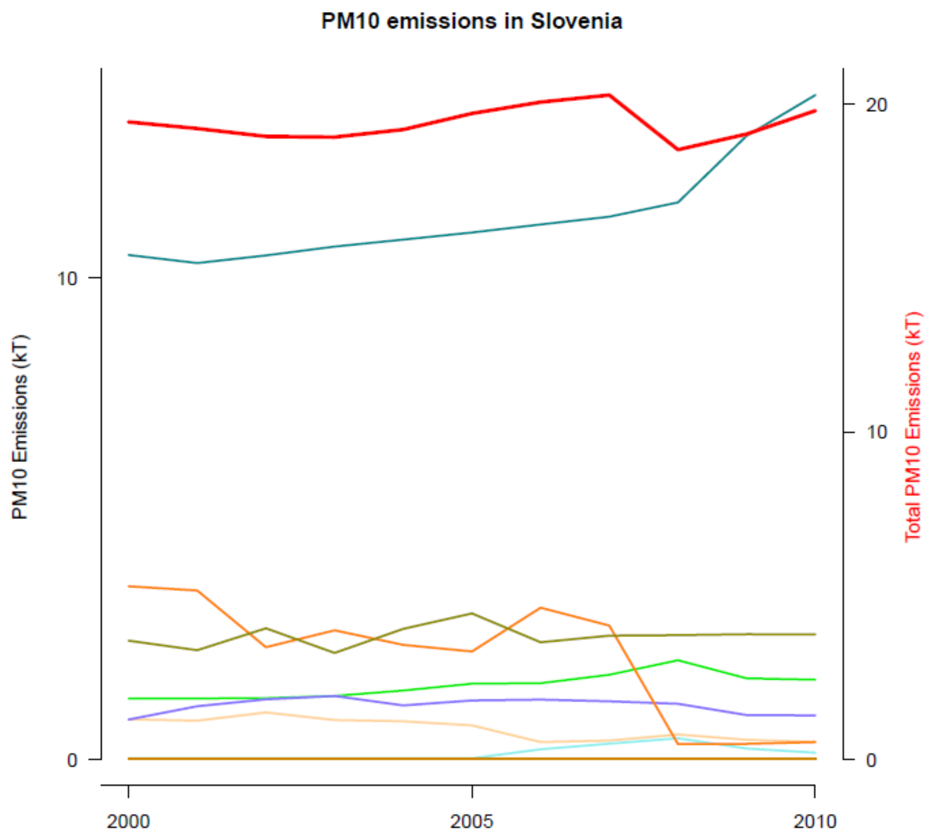


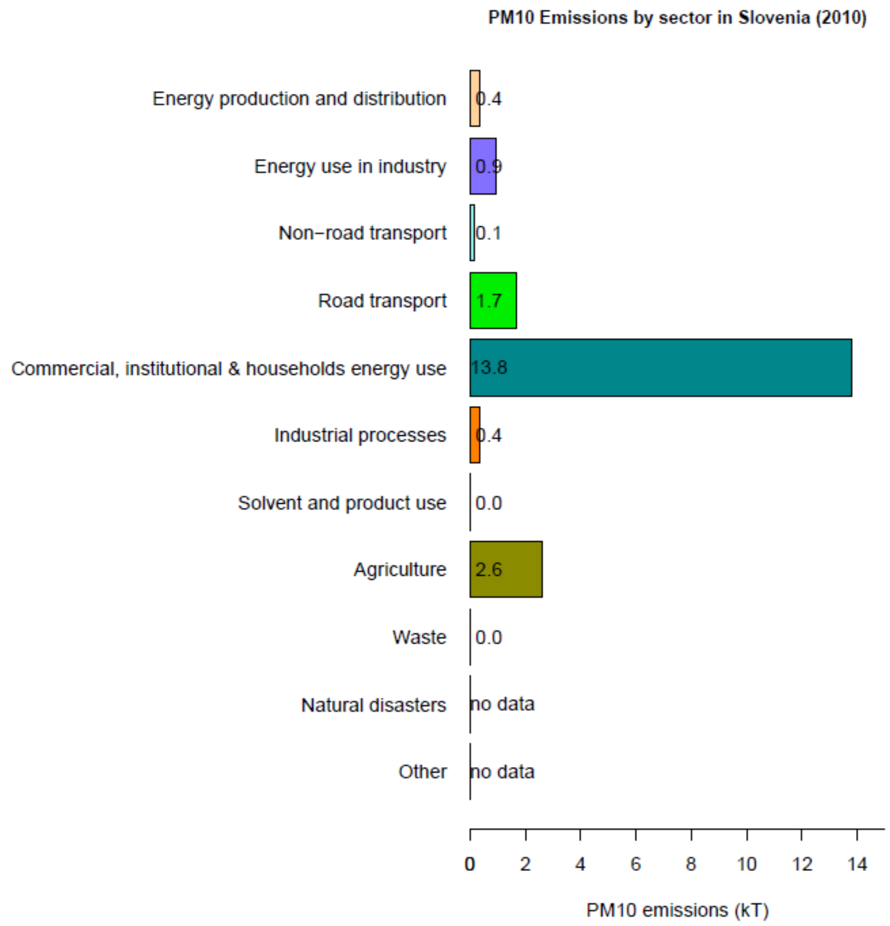
Sweden (PM_{10})


PM10 emissions in Sweden
PM10 Emissions (kT)
Total PM10 Emissions (kT)
10
0
50
40
30
20
10
0
1990
1995
2000
2005
2010

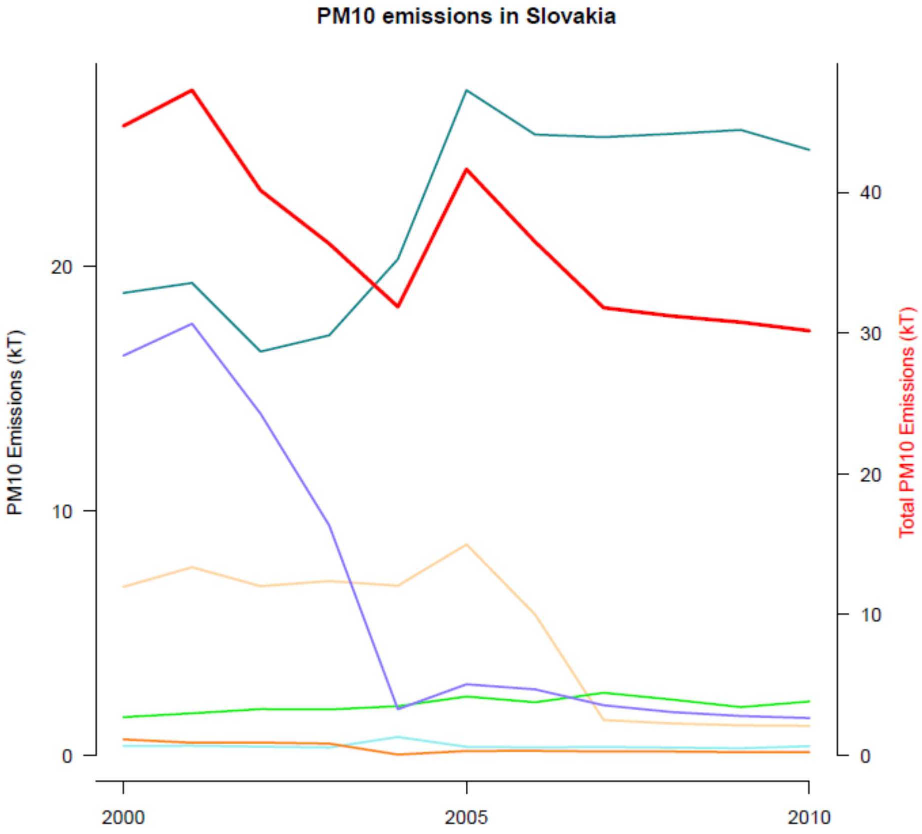

PM10 Emissions by sector in Sweden (2010)
Energy production and distribution
6.4
Energy use in industry
3.0
Non−road transport
9.3
Road transport
11.1
Commercial, institutional & households energy use
9.6
Industrial processes
7.8
Solvent and product use
0.3
Agriculture
4.4
Waste
0.2
Natural disasters
no data
Other
no data
0
2
4
6
8
10
12
14
PM10 emissions (kT)

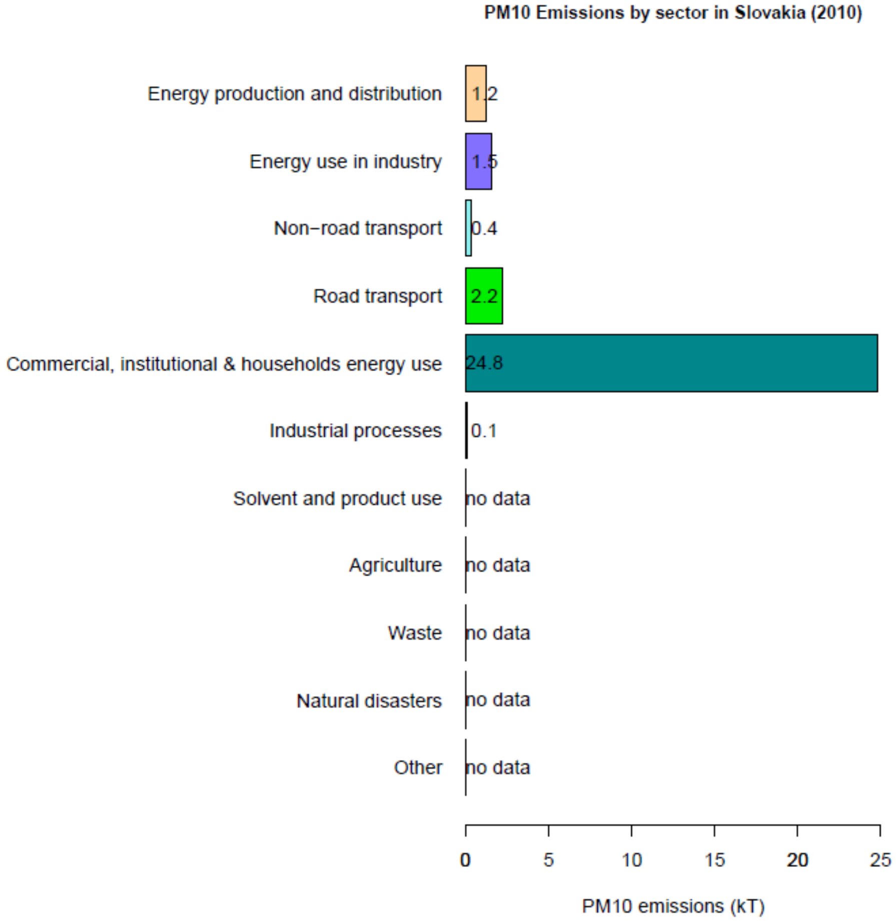
Slovenia (PM_{10})



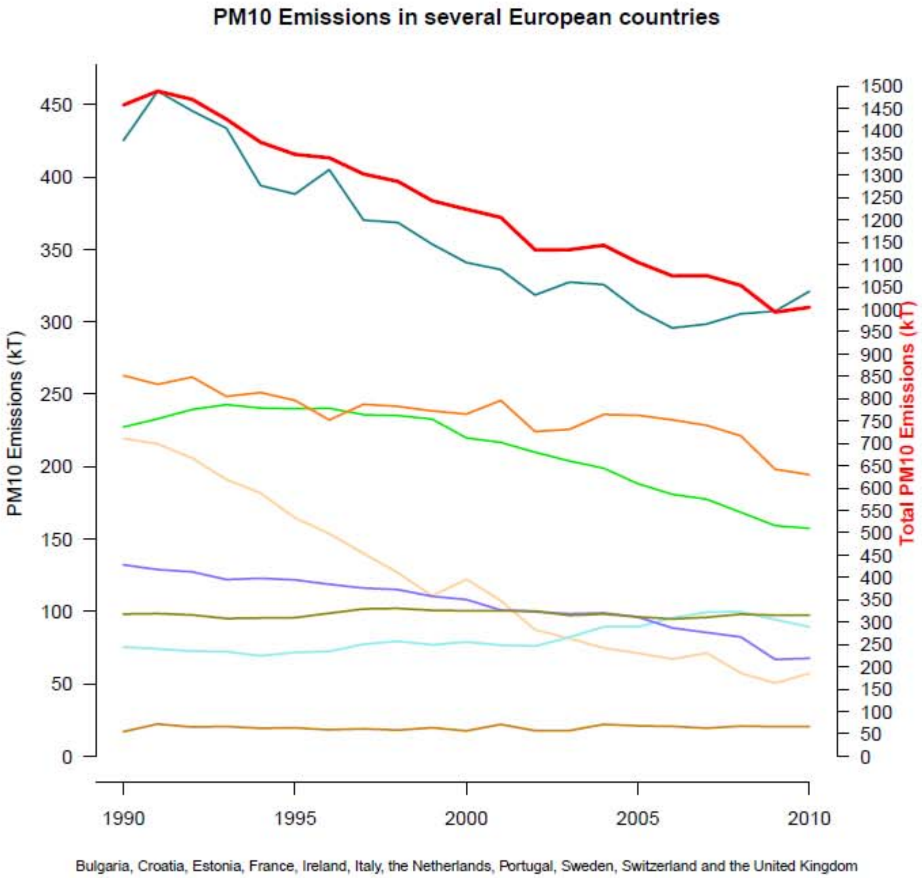


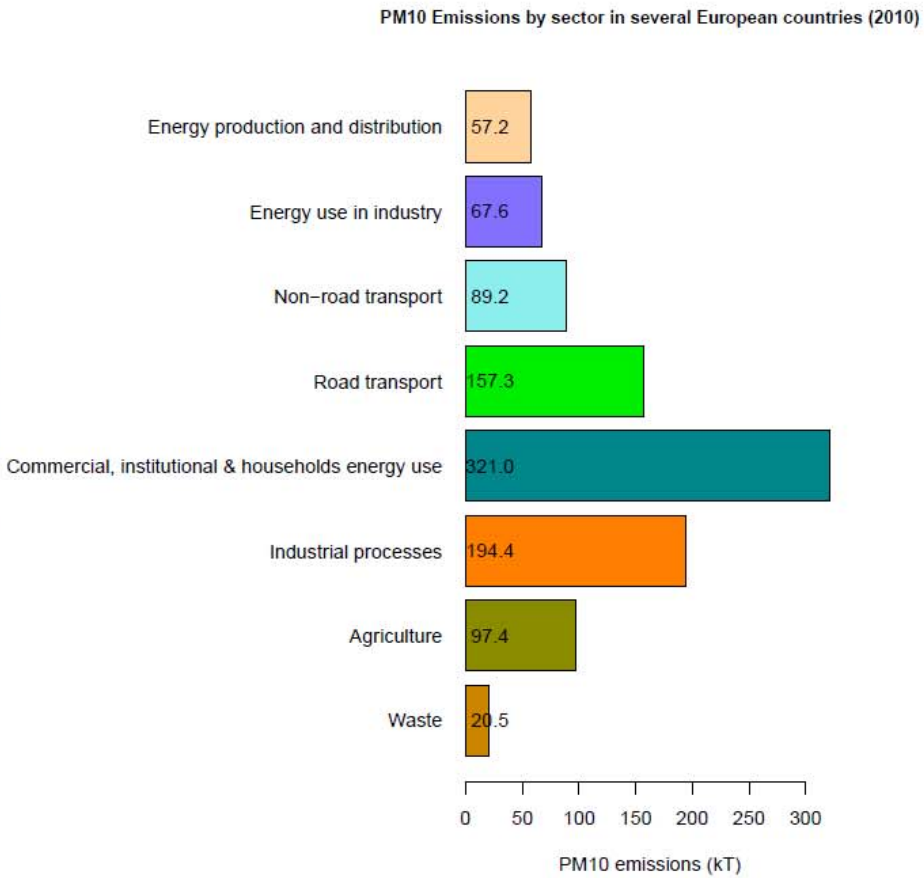
Slovakia (PM_{10})





Bulgaria, Croatia, Estonia, France, Ireland, Italy, the Netherlands, Portugal, Sweden, Switzerland and the United Kingdom (PM_{10})





II.3. NO_x

II.3.1. Data availability

Availability of NO_x data is described below:

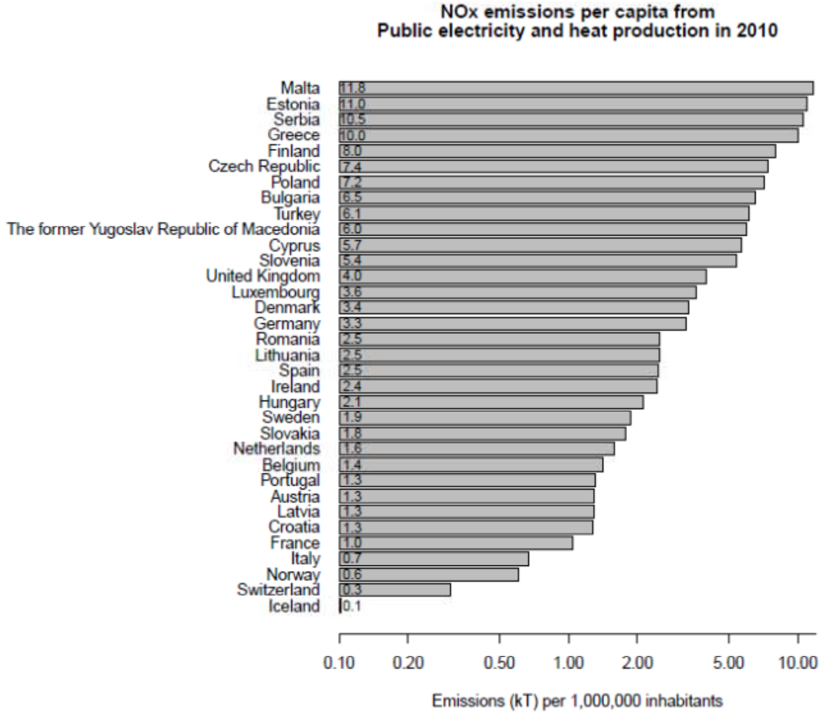
Country	From	To	Missing years	x = no data										
				EP	EI	NRT	RT	CIH	IP	S	A	W	N	O
Albania	1990	2009	-							x		x		x
Austria	1980	2010	-							x			x	
Belgium	1990	2010	1991-1999; 2001-2004							x	x		x	x
Bulgaria	1990	2010	-							x			x	x
Croatia	1990	2010	-							x				
Cyprus	1990	2010	-						x	x		x	x	
Czech Republic	2010	2010	-							x	x		x	
Denmark	1985	2010	-										x	x
Estonia	1990	2010	-										x	x
Finland	1980	2010	-										x	x
France	1980	2010	-											
Germany	1990	2010	-							x				x
Greece	1990	2010	-							x		x	x	x
Hungary	2010	2010	-										x	x
Iceland	1990	2010	-							x	x		x	
Ireland	1987	2010	1988-1989						x	x	x		x	
Italy	1980	2010	-							x				x
Latvia	1990	2010	-							x				
Lithuania	2008	2010	-							x	x	x	x	x
Luxembourg	1990	2010	-						x	x		x	x	x

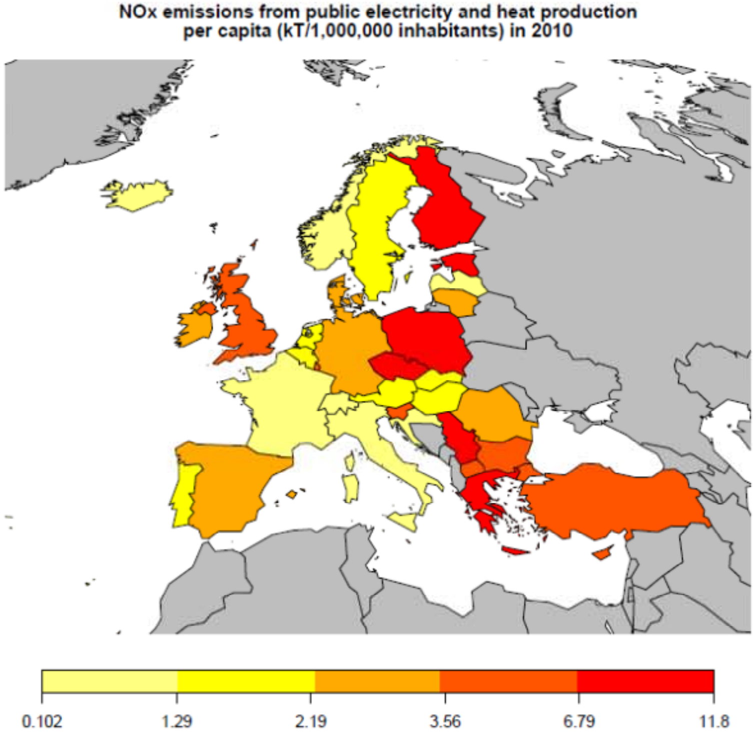
Country	From	To	Missing years	x = no data										
				EP	EI	NRT	RT	CIH	IP	S	A	W	N	O
FYROM	2010	2010	-						x					x
Malta	2000	2010	-						x	x	x		x	
Montenegro	1990	2009	-							x		x	x	x
Netherlands	1990	2010	-							x				
Norway	1980	2010	1981-1986; 1988										x	
Poland	2005	2010	2006-2008							x	x		x	x
Portugal	1990	2010	-							x				x
Romania	2005	2010	-							x			x	x
Serbia	2000	2010	-											x
Slovakia	2000	2010	-							x	x	x		x
Slovenia	1980	2010	-							x	x		x	x
Spain	1990	2010	-							x				x
Sweden	1990	2010	-							x	x		x	x
Switzerland	1990	2010	-											
Turkey	2010	2010	-							x	x		x	x
United Kingdom	1980	2010	-							x	x			

Abbreviation list:

EP: energy production and distribution
NRT: non-road transport
CIH: commercial, Institutional and Household energy use
S: solvent and product use
W: waste
O: other emissions
EI: energy use in industry
RT: road transport
IP: industrial processes
A: agriculture
N: natural emissions

II.3.2. NO_x emissions per capita from public electricity and heat production sector in 2010





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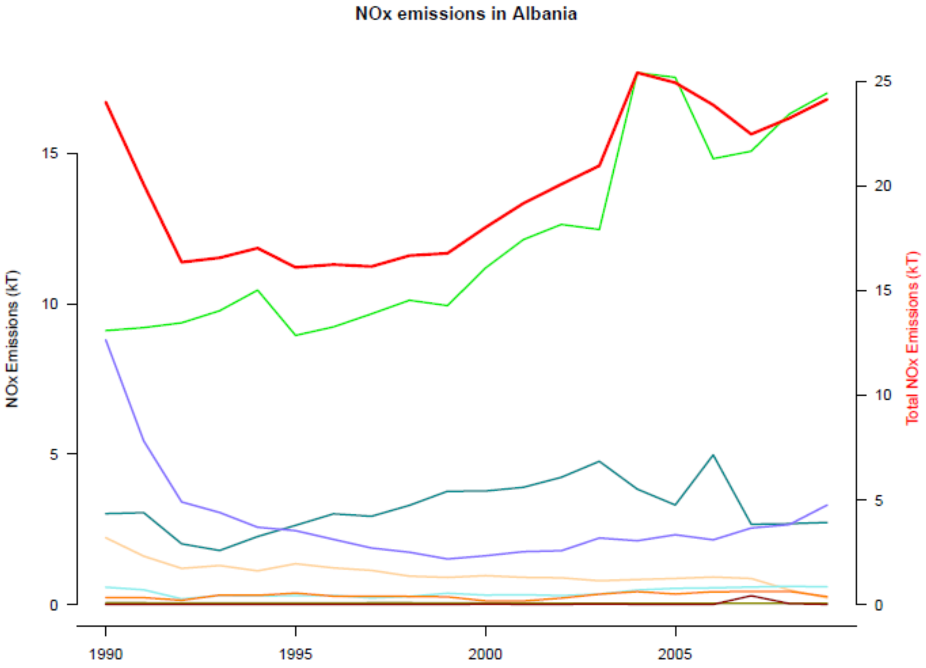
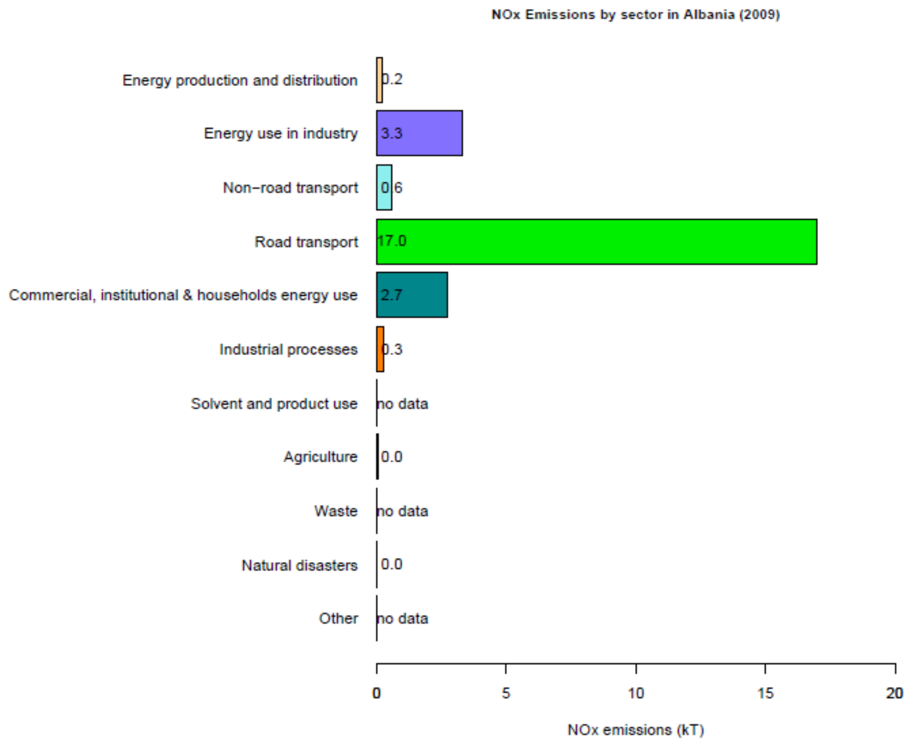
II.3.3. NO_x emissions trends by sector (left) and distribution of emissions for the last available year (right)

Albania (NO_x)





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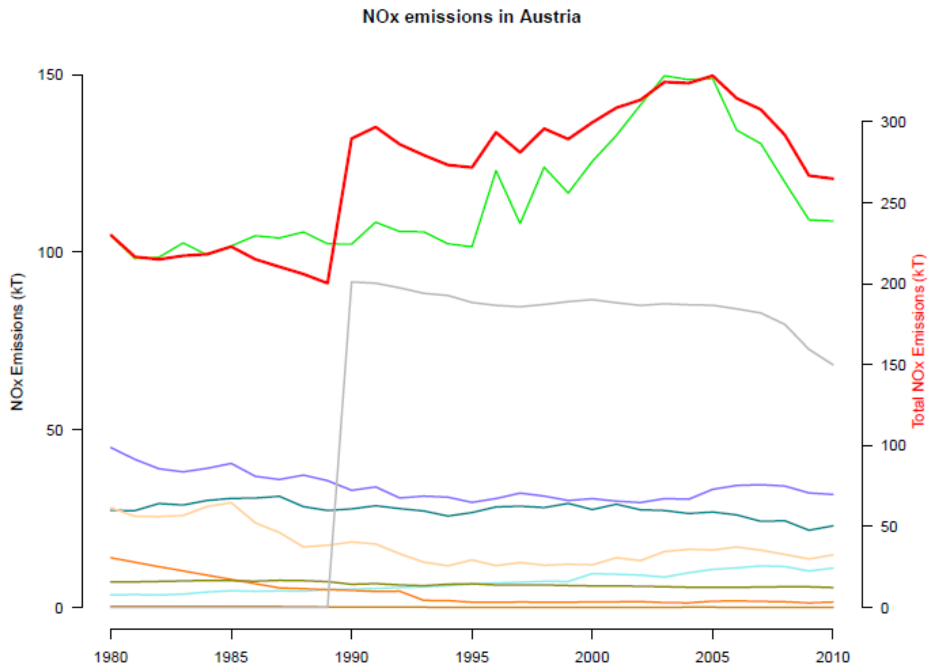
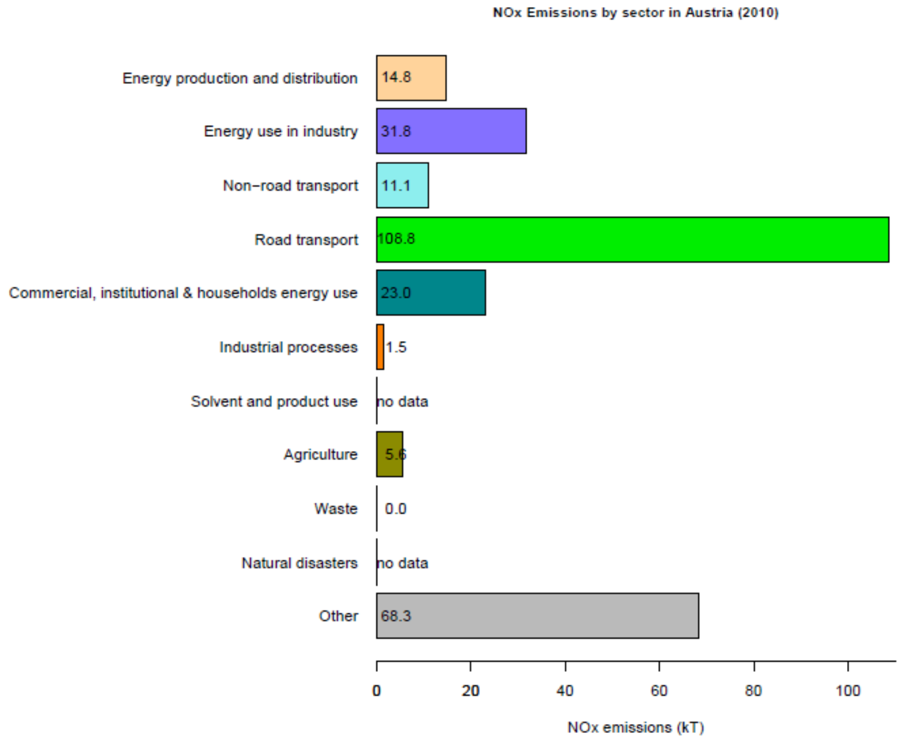
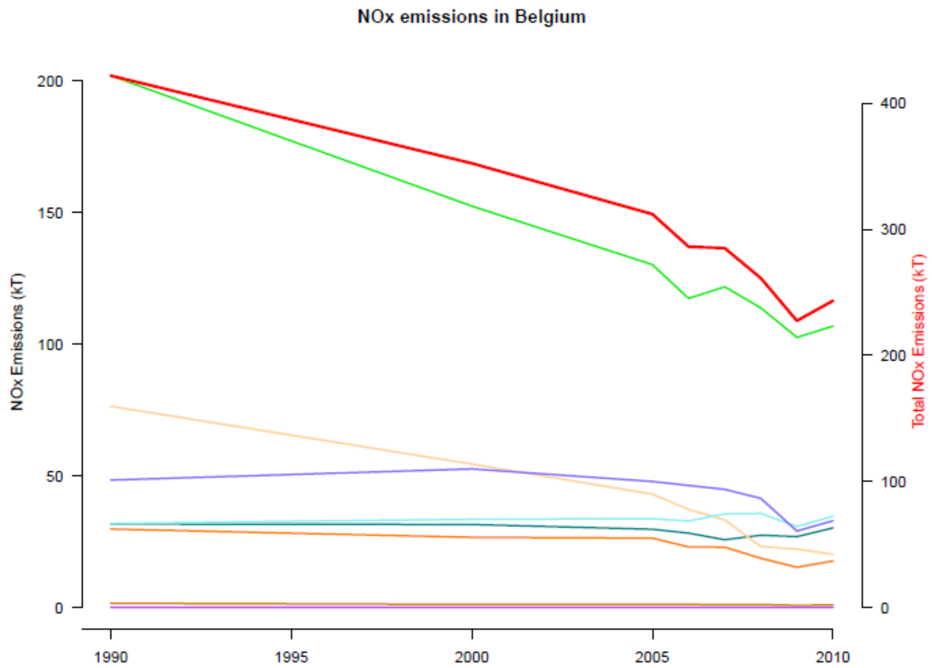
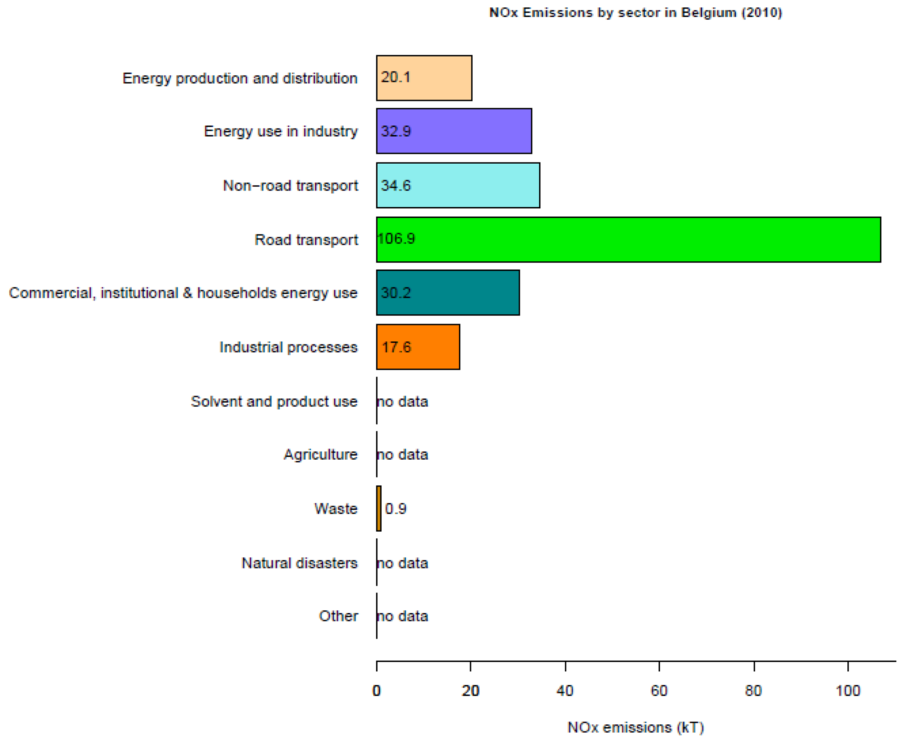
Austria (NO_x)



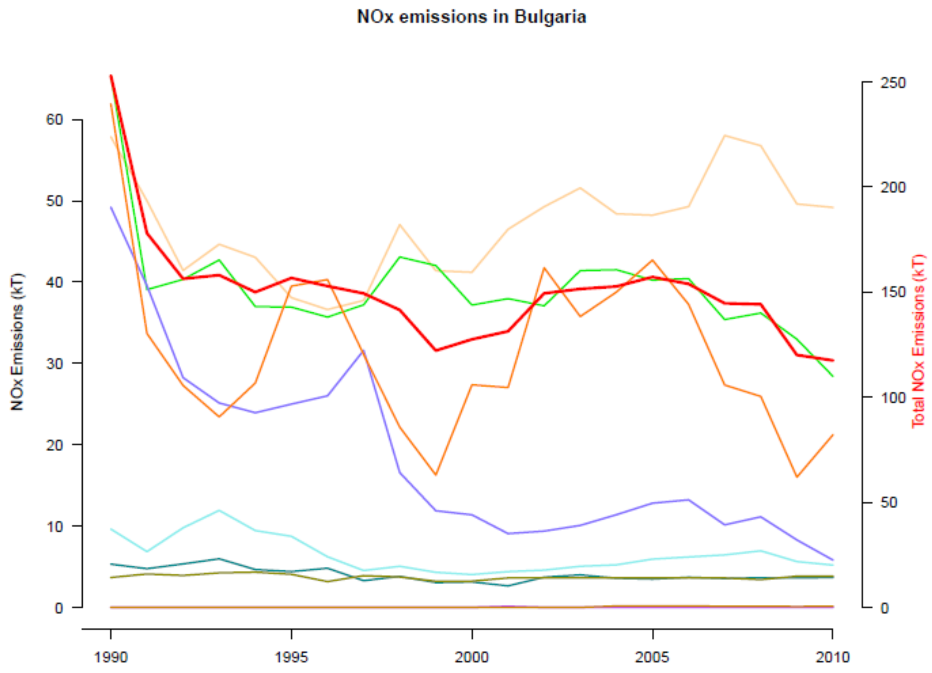


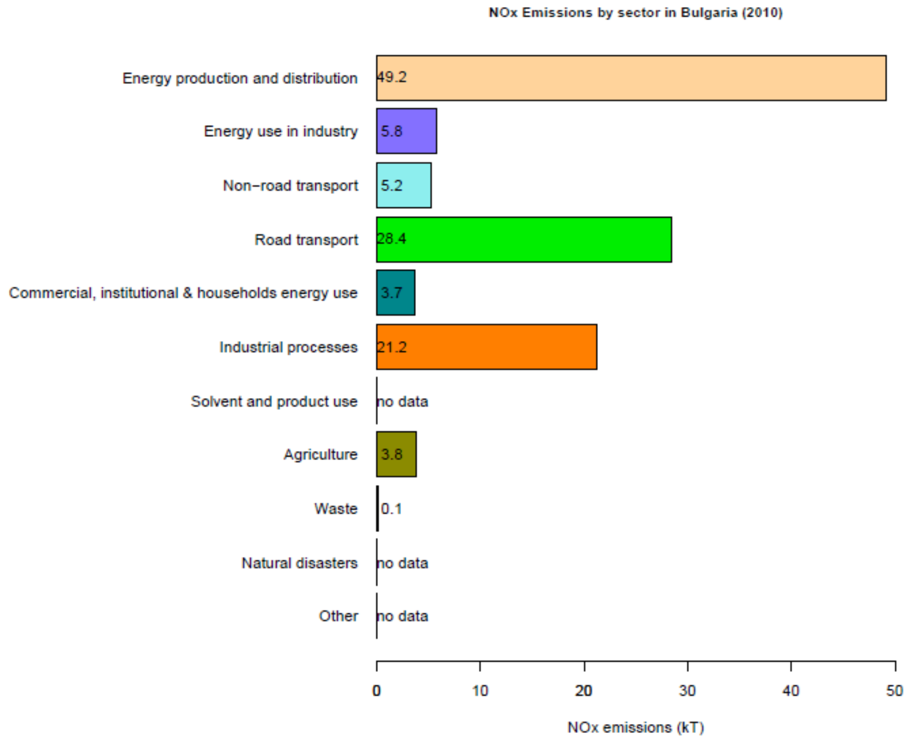
Belgium (NO_x)


NOx emissions in Belgium
NOx Emissions (kT)
Total NOx Emissions (kT)
200
150
100
50
0
400
300
200
100
0
1990
1995
2000
2005
2010

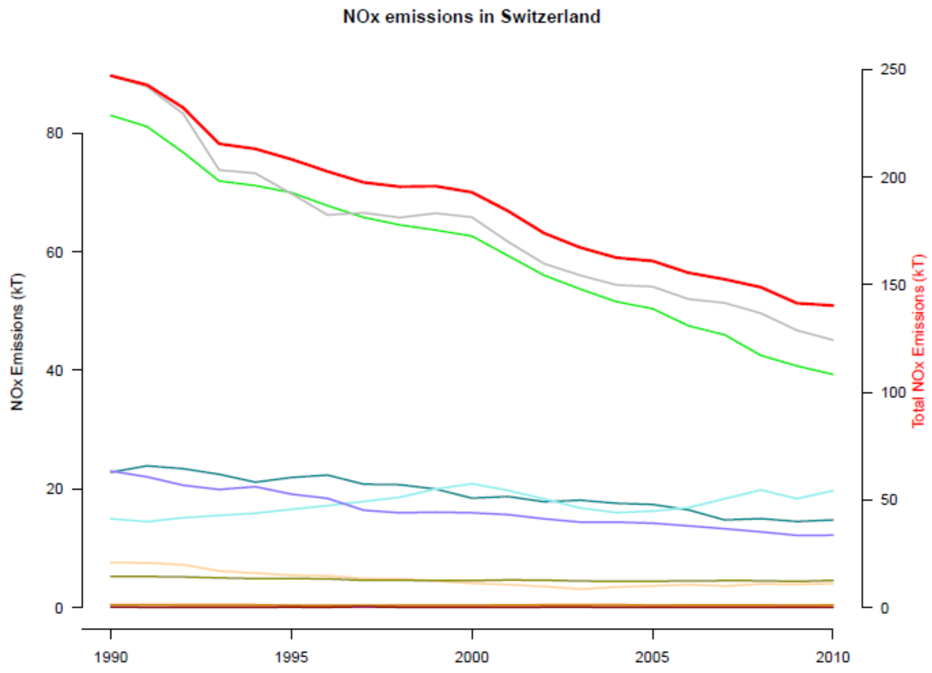

NOx Emissions by sector in Belgium (2010)
Energy production and distribution
20.1
Energy use in industry
32.9
Non−road transport
34.6
Road transport
106.9
Commercial, institutional & households energy use
30.2
Industrial processes
17.6
Solvent and product use
no data
Agriculture
no data
Waste
0.9
Natural disasters
no data
Other
no data
0
20
40
60
80
100
NOx emissions (kT)

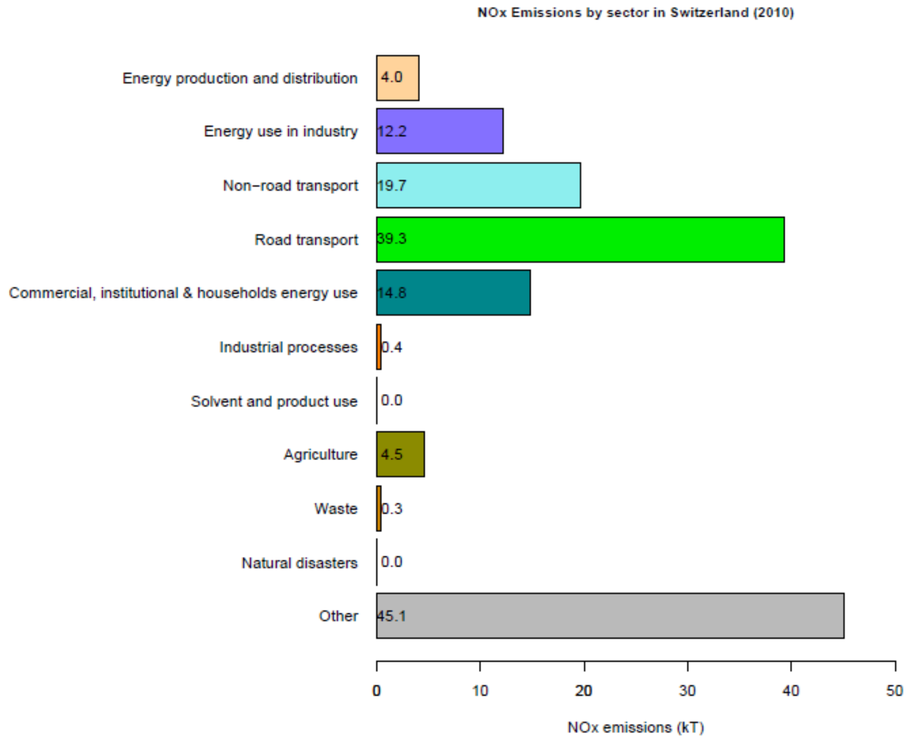
Bulgaria (NO_x)





Switzerland (NO_x)





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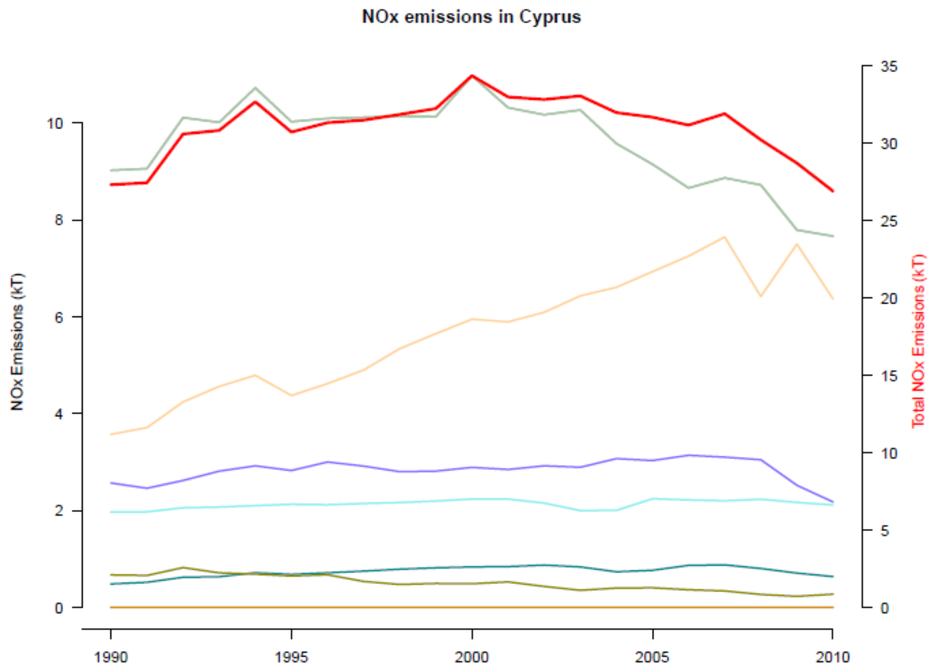
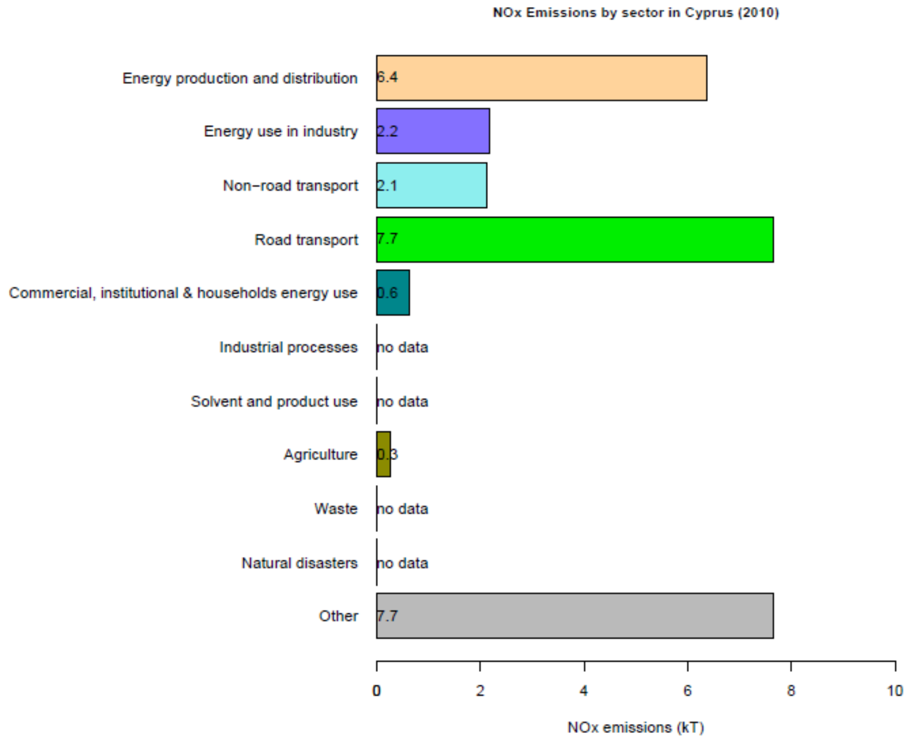
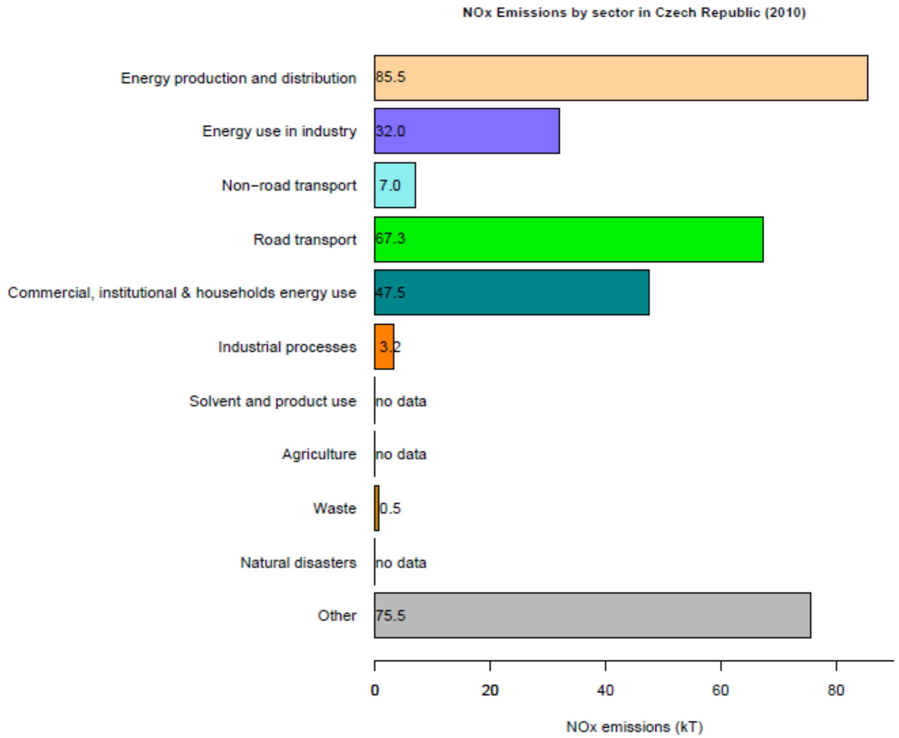
Cyprus (NO_x)

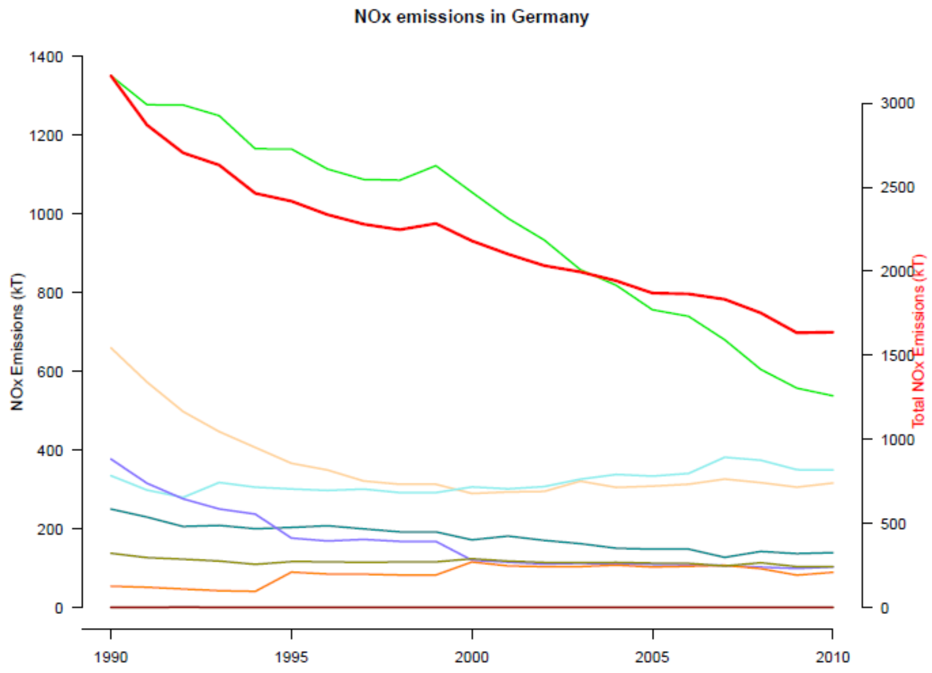




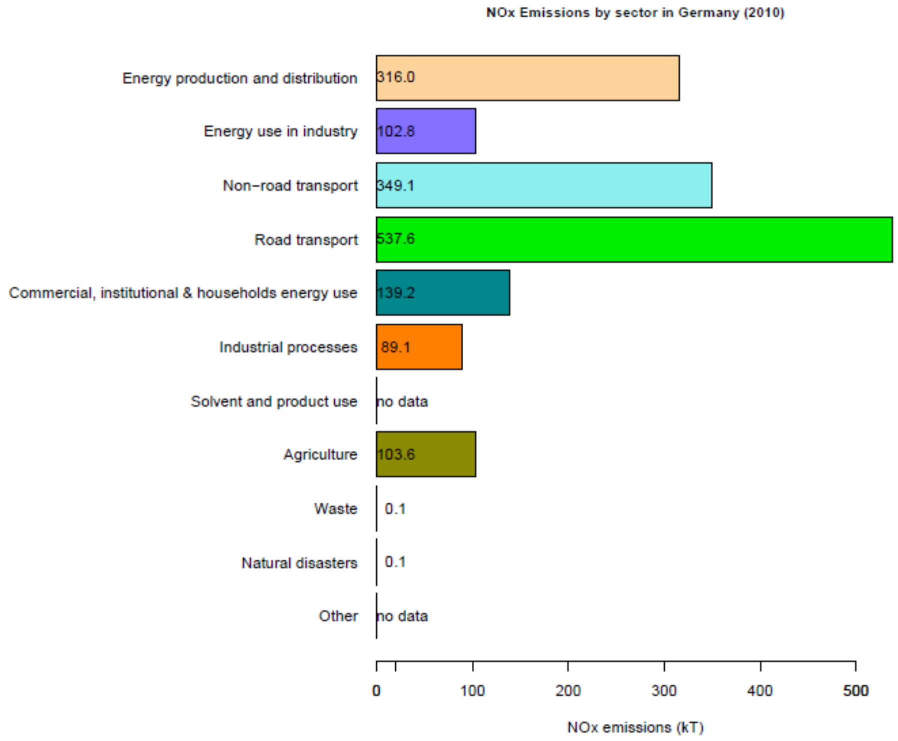
Czech Republic (NO_x)



Germany (NO_x)

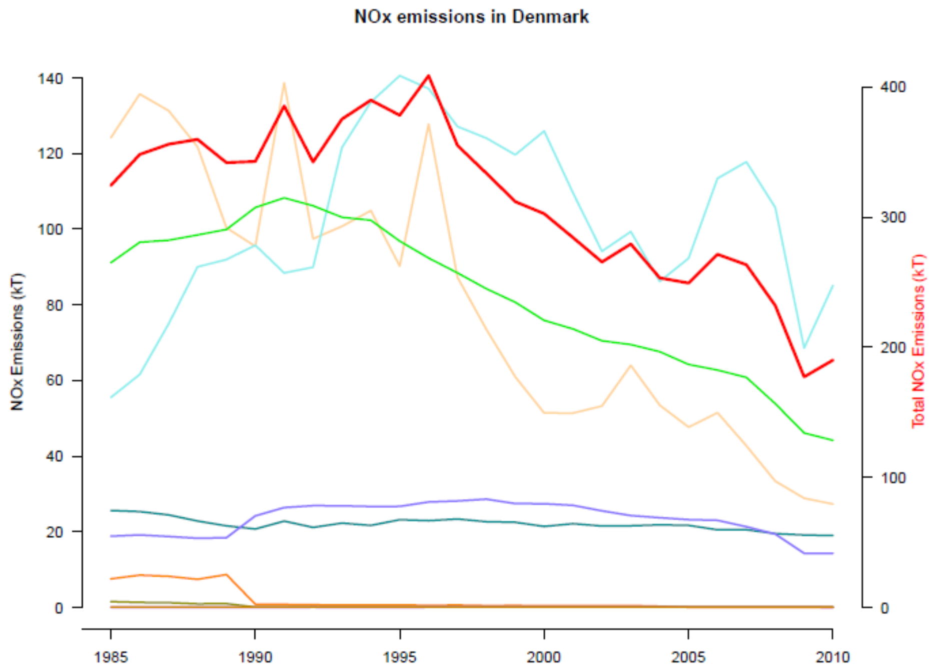


NOx emissions in Germany
NOx Emissions (kT)
Total NOx Emissions (kT)
1400
1200
1000
800
600
400
200
0
3000
2500
2000
1500
1000
500
0
1990
1995
2000
2005
2010

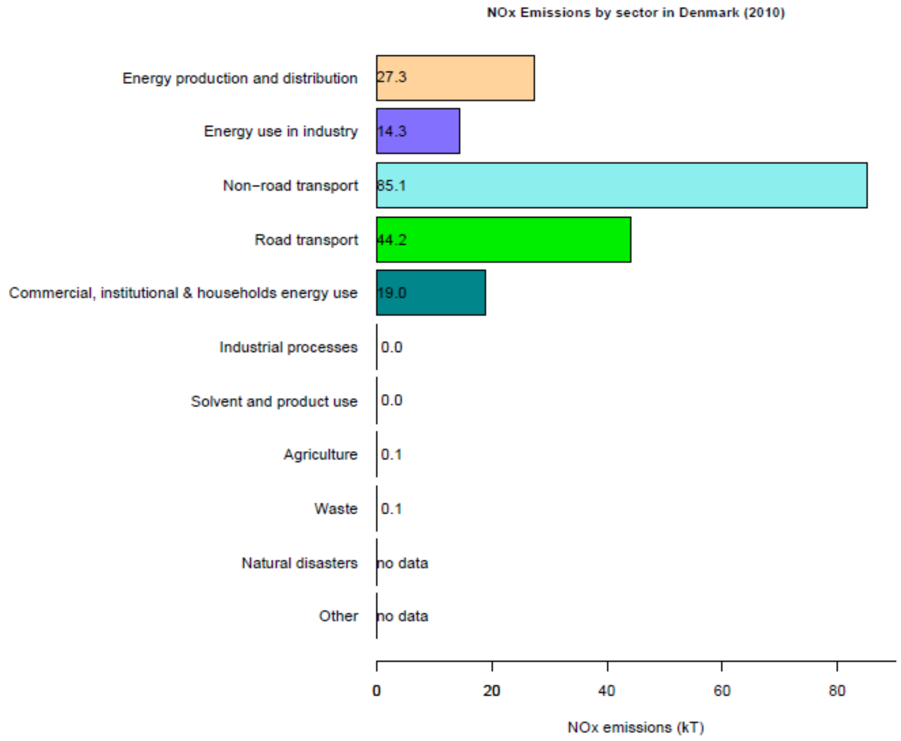


NOx Emissions by sector in Germany (2010)
Energy production and distribution
316.0
Energy use in industry
102.8
Non−road transport
349.1
Road transport
537.6
Commercial, institutional & households energy use
139.2
Industrial processes
89.1
Solvent and product use
no data
Agriculture
103.6
Waste
0.1
Natural disasters
0.1
Other
no data
0
100
200
300
400
500
NOx emissions (kT)

Denmark (NO_x)

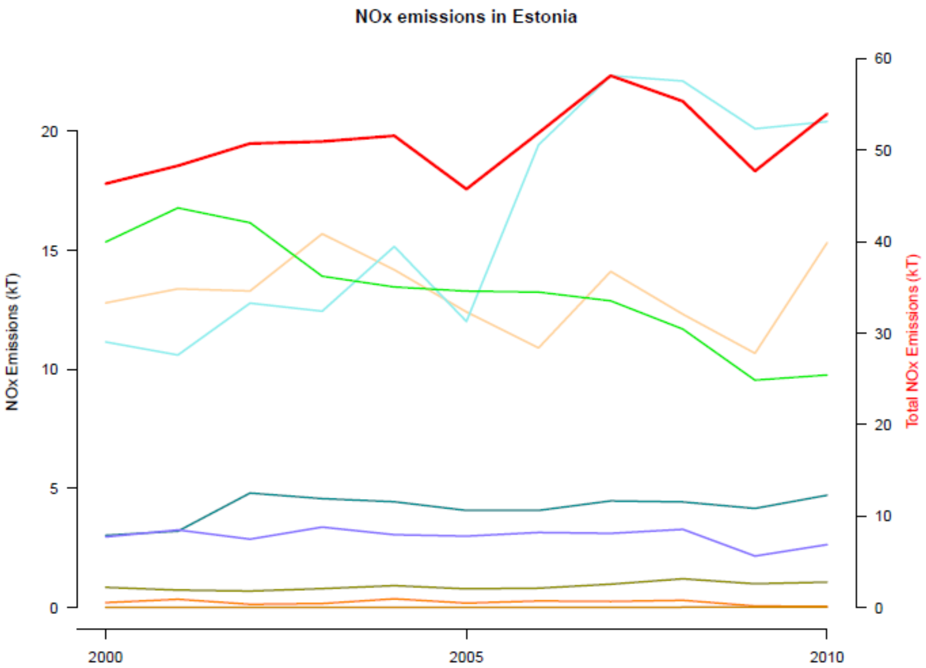


NOx emissions in Denmark
NOx Emissions (kT)
Total NOx Emissions (kT)
140
120
100
80
60
40
20
0
400
300
200
100
0
1985
1990
1995
2000
2005
2010

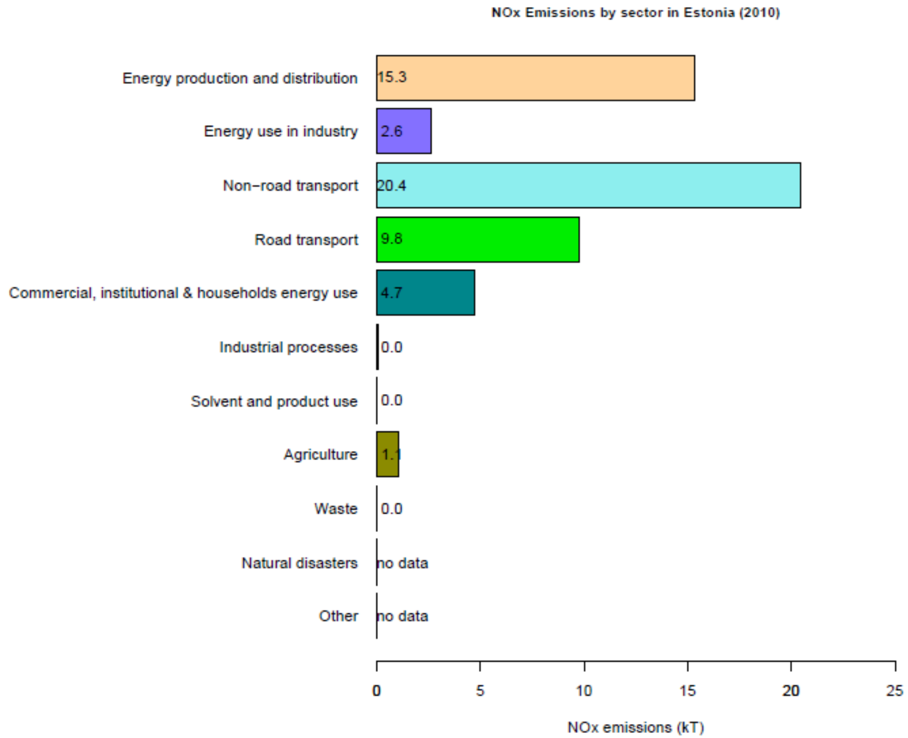


NOx Emissions by sector in Denmark (2010)
Energy production and distribution
27.3
Energy use in industry
14.3
Non−road transport
85.1
Road transport
44.2
Commercial, institutional & households energy use
19.0
Industrial processes
0.0
Solvent and product use
0.0
Agriculture
0.1
Waste
0.1
Natural disasters
no data
Other
no data
0
20
40
60
80
NOx emissions (kT)

Estonia (NO_x)

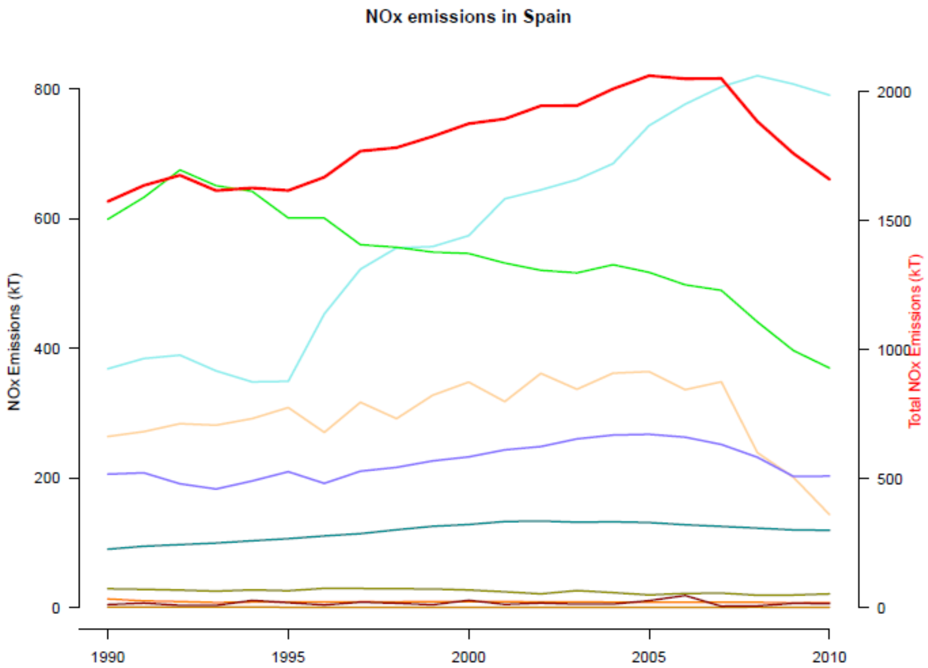


NOx emissions in Estonia
NOx Emissions (kT)
Total NOx Emissions (kT)
0
5
10
15
20
0
10
20
30
40
50
60
2000
2005
2010

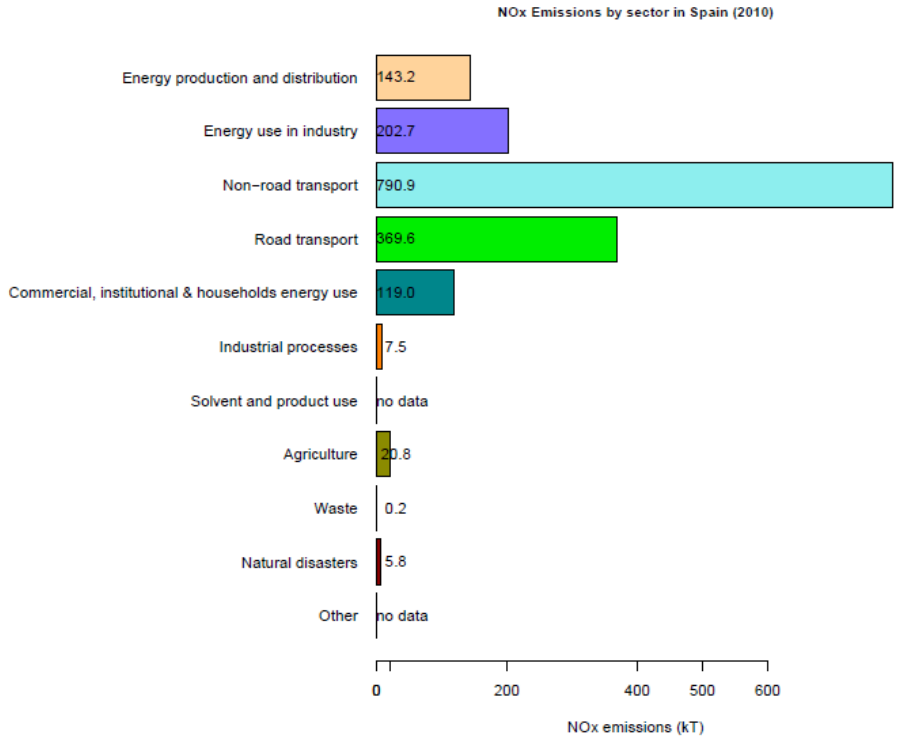


NOx Emissions by sector in Estonia (2010)
Energy production and distribution
15.3
Energy use in industry
2.6
Non−road transport
20.4
Road transport
9.8
Commercial, institutional & households energy use
4.7
Industrial processes
0.0
Solvent and product use
0.0
Agriculture
1.1
Waste
0.0
Natural disasters
no data
Other
no data
0
5
10
15
20
25
NOx emissions (kT)

Spain (NO_x)

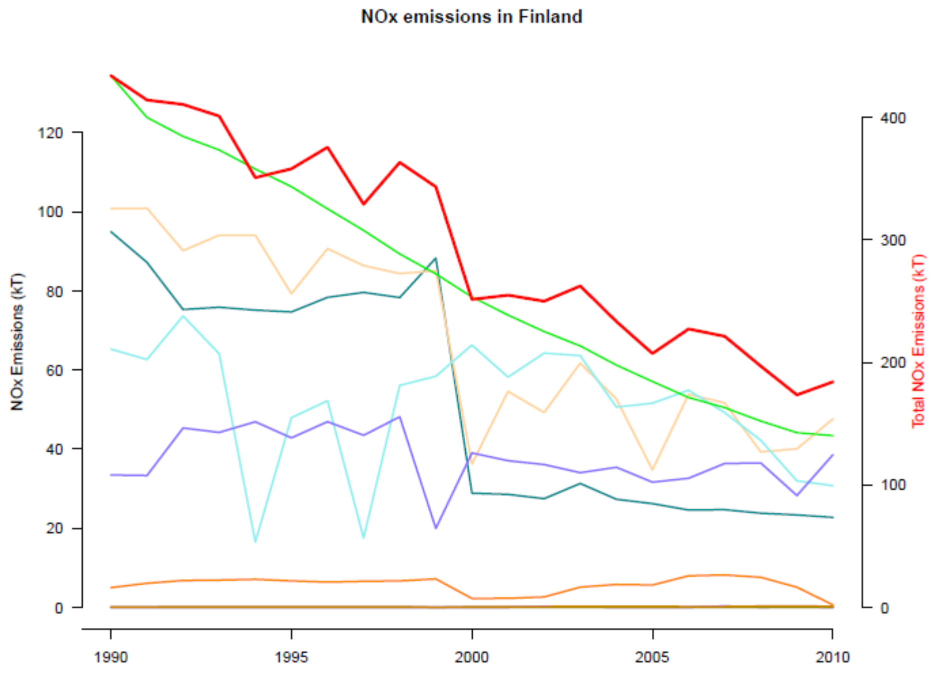


NOx emissions in Spain
NOx Emissions (kT)
Total NOx Emissions (kT)
0
200
400
600
800
0
500
1000
1500
2000
1990
1995
2000
2005
2010

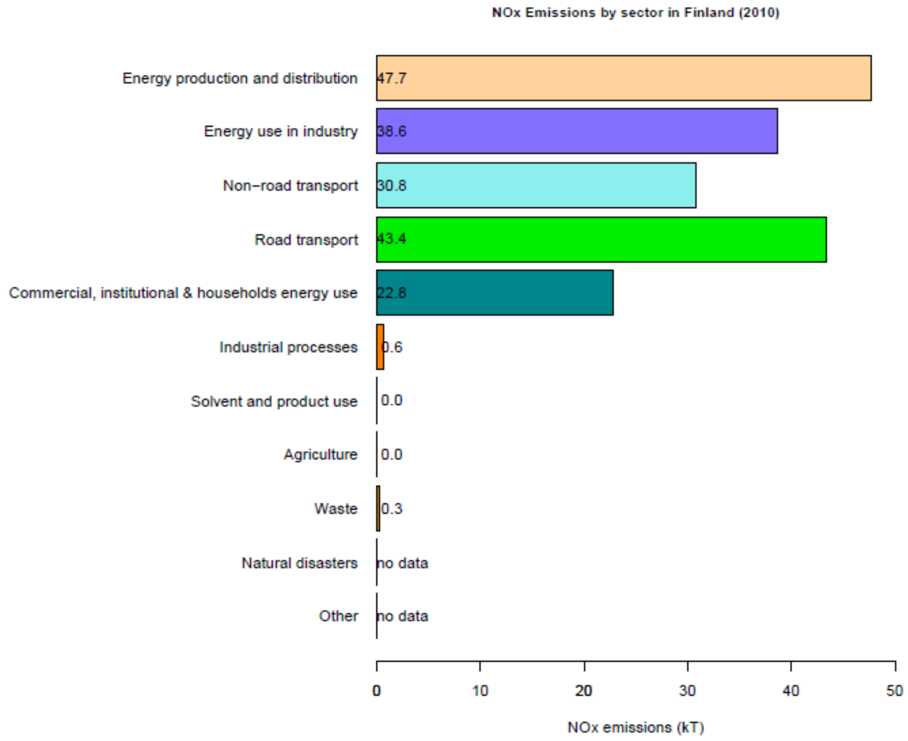


NOx Emissions by sector in Spain (2010)
Energy production and distribution
143.2
Energy use in industry
202.7
Non−road transport
790.9
Road transport
369.6
Commercial, institutional & households energy use
119.0
Industrial processes
7.5
Solvent and product use
no data
Agriculture
20.8
Waste
0.2
Natural disasters
5.8
Other
no data
0
200
400
500
600
NOx emissions (kT)

Finland (NO_x)



NOx emissions in Finland
NOx Emissions (kT)
Total NOx Emissions (kT)
0
20
40
60
80
100
120
0
100
200
300
400
1990
1995
2000
2005
2010



NOx Emissions by sector in Finland (2010)
Energy production and distribution
47.7
Energy use in industry
38.6
Non−road transport
30.8
Road transport
43.4
Commercial, institutional & households energy use
22.8
Industrial processes
0.6
Solvent and product use
0.0
Agriculture
0.0
Waste
0.3
Natural disasters
no data
Other
no data
0
10
20
30
40
50
NOx emissions (kT)

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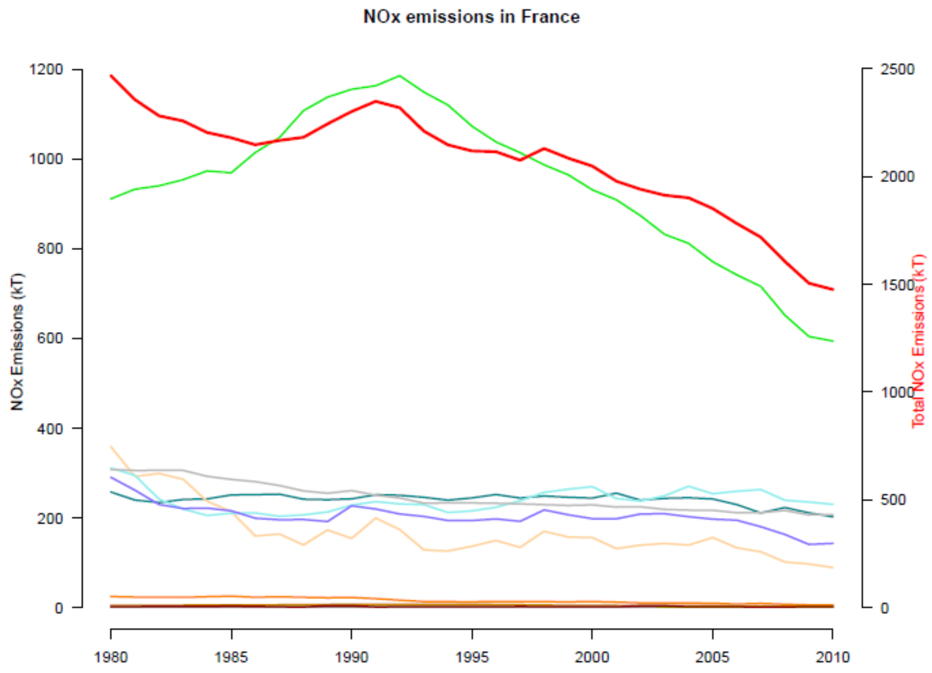
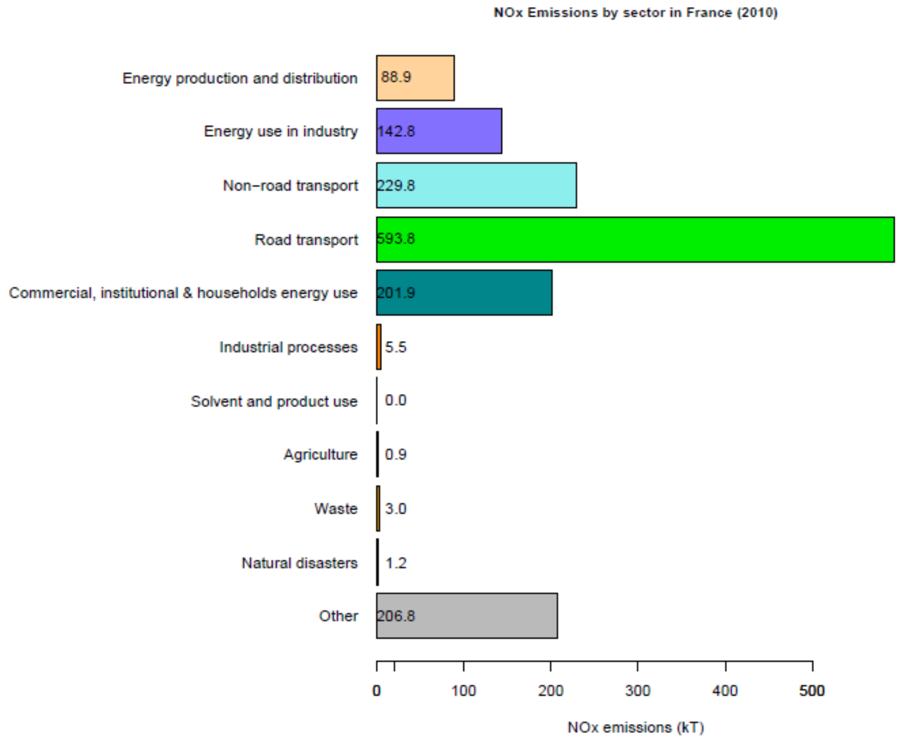
France (NO_x)





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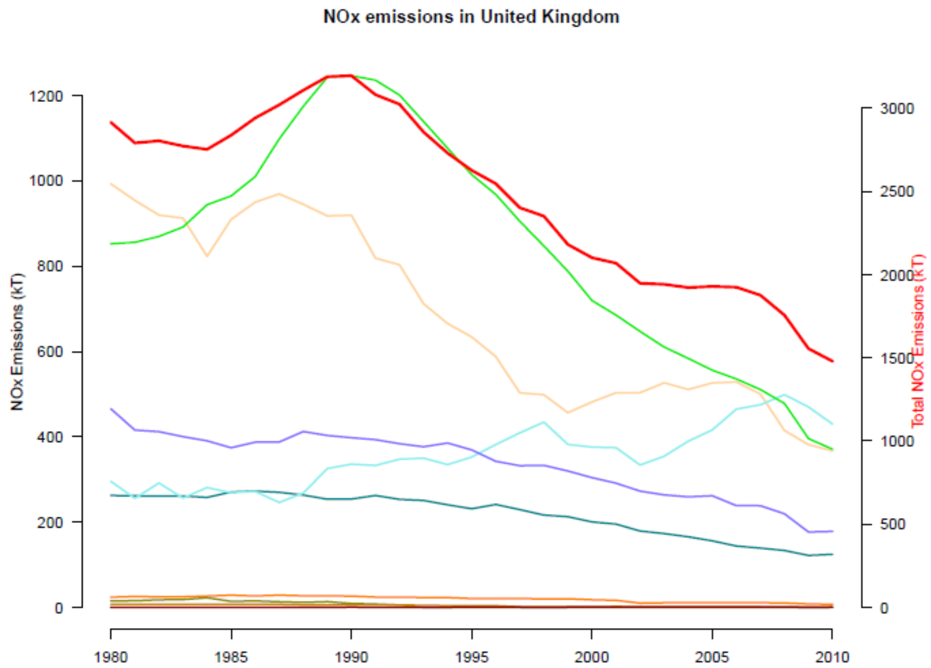
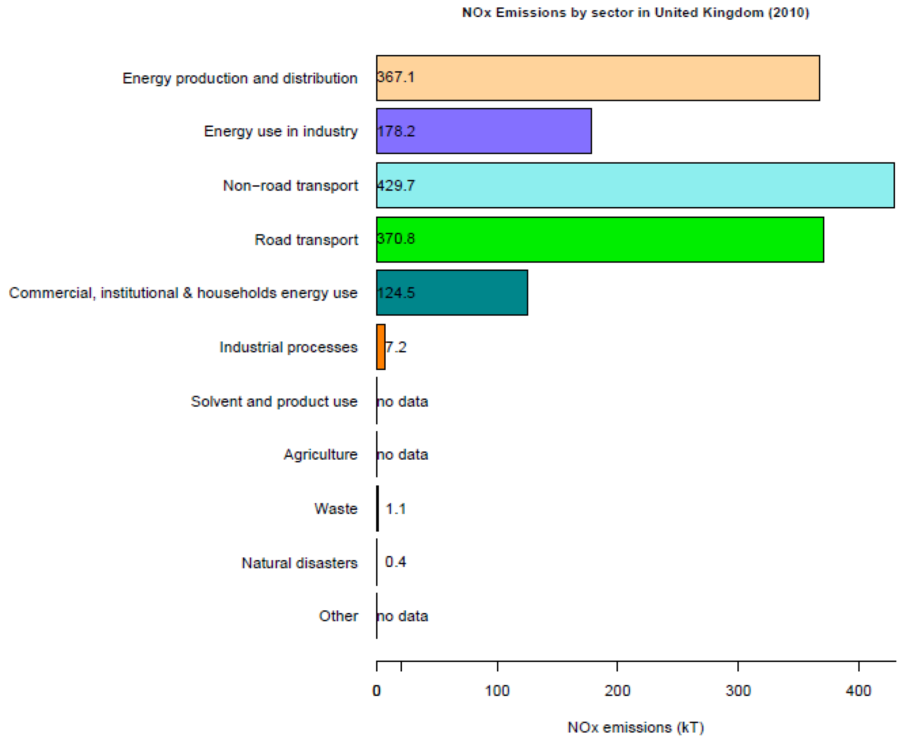
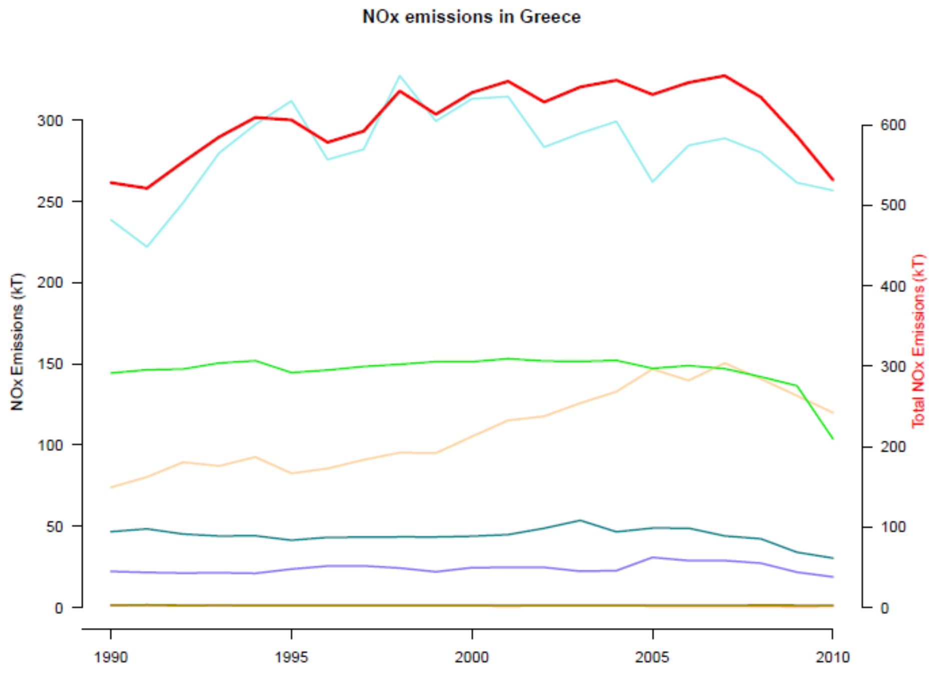
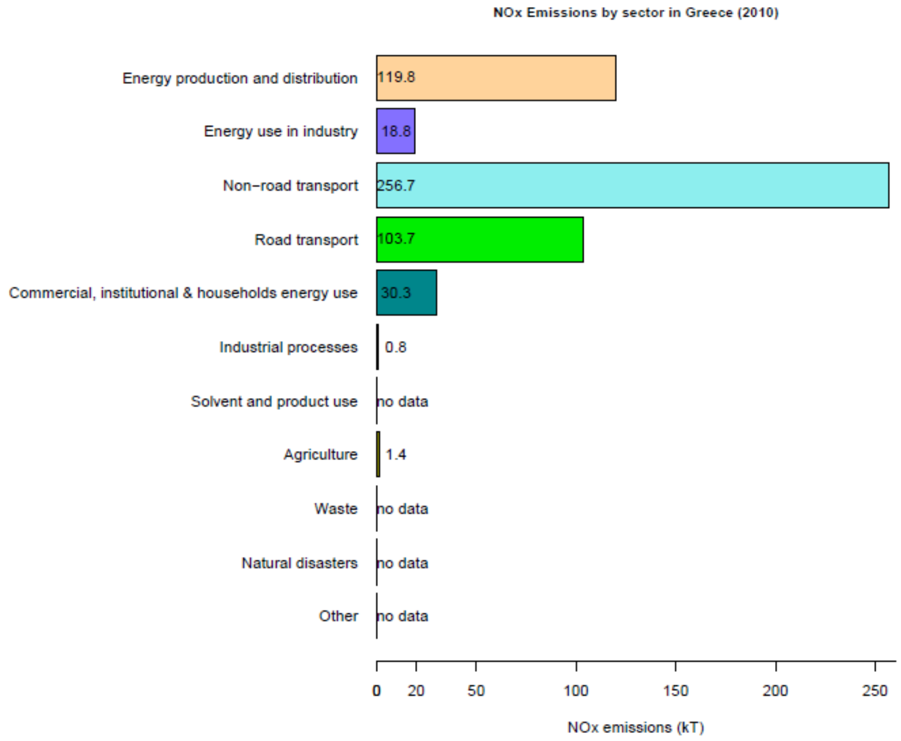
The United Kingdom (NO_x)



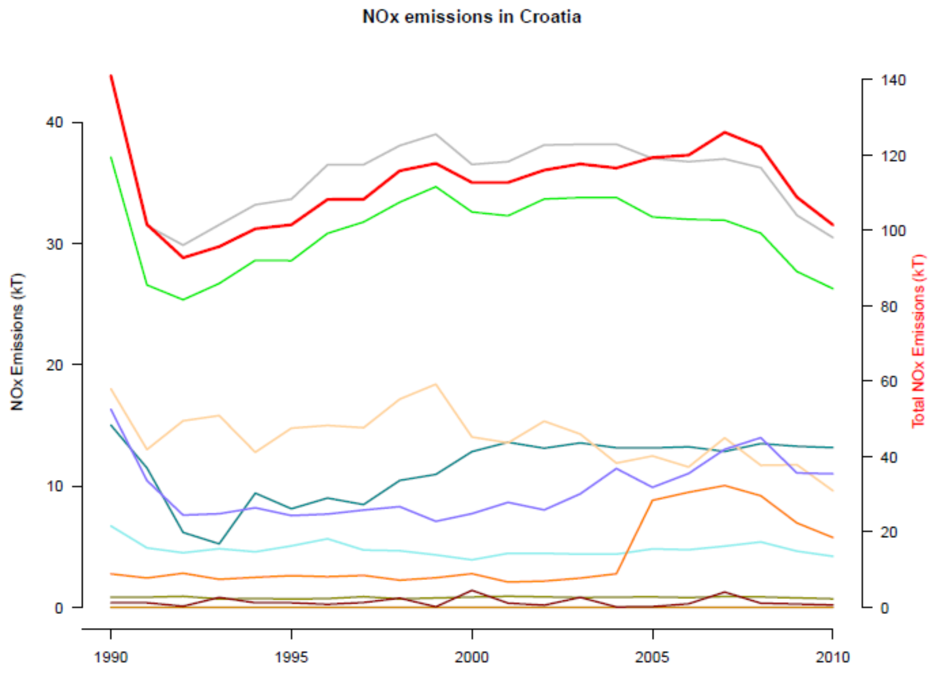


Greece (NO_x)

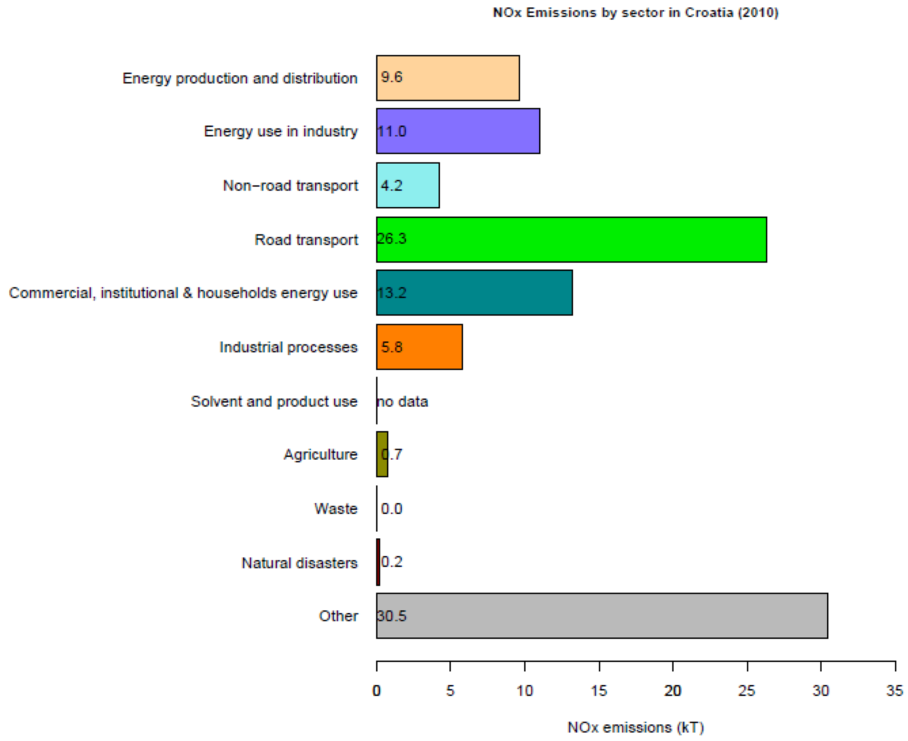

NOx emissions in Greece
NOx Emissions (kT)
Total NOx Emissions (kT)
300
250
200
150
100
50
0
600
500
400
300
200
100
0
1990
1995
2000
2005
2010


NOx Emissions by sector in Greece (2010)
Energy production and distribution
119.8
Energy use in industry
18.8
Non−road transport
256.7
Road transport
103.7
Commercial, institutional & households energy use
30.3
Industrial processes
0.8
Solvent and product use
no data
Agriculture
1.4
Waste
no data
Natural disasters
no data
Other
no data
0
20
50
100
150
200
250
NOx emissions (kT)

Croatia (NO_x)

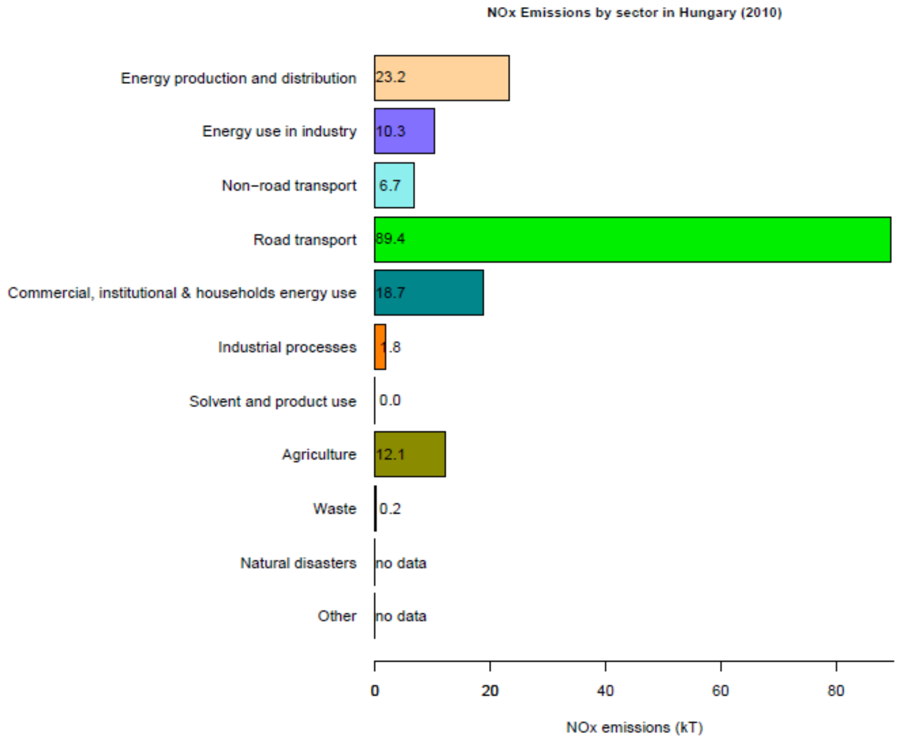


NOx emissions in Croatia
NOx Emissions (kT)
Total NOx Emissions (kT)
1990
1995
2000
2005
2010

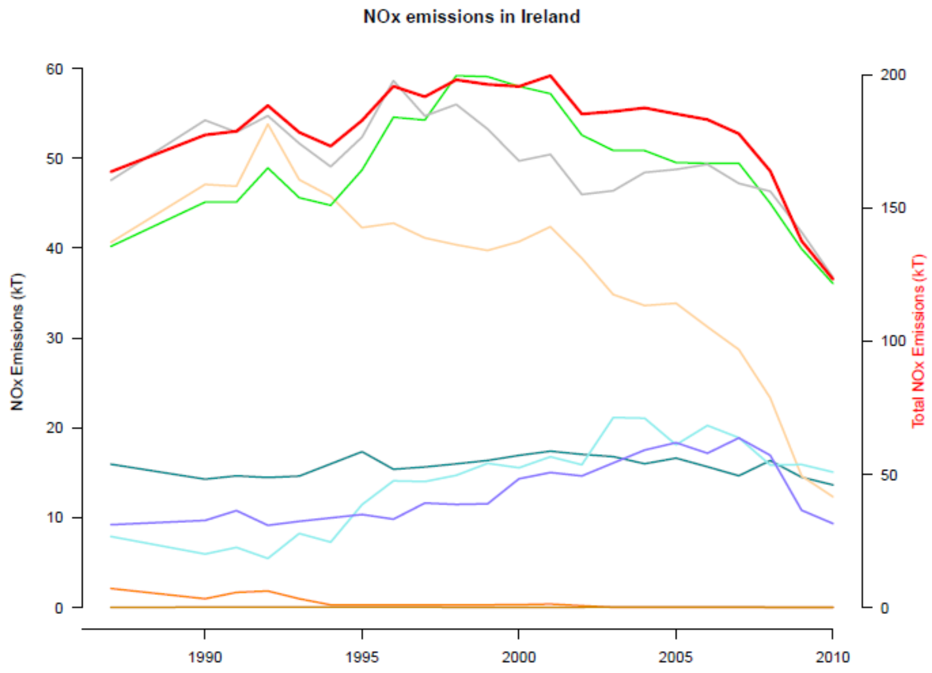


NOx Emissions by sector in Croatia (2010)
Energy production and distribution 9.6
Energy use in industry 11.0
Non−road transport 4.2
Road transport 26.3
Commercial, institutional & households energy use 13.2
Industrial processes 5.8
Solvent and product use no data
Agriculture 0.7
Waste 0.0
Natural disasters 0.2
Other 30.5
NOx emissions (kT)

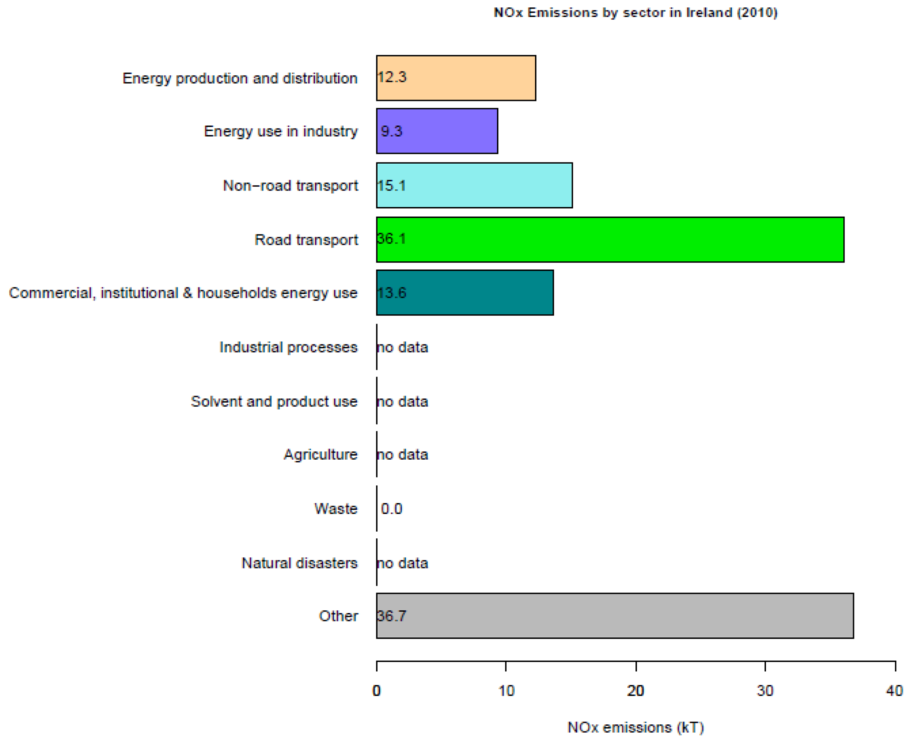
Hungary (NO_x)


NOx Emissions by sector in Hungary (2010)
Energy production and distribution
Energy use in industry
Non-road transport
Road transport
Commercial, institutional & households energy use
Industrial processes
Solvent and product use
Agriculture
Waste
Natural disasters
Other
23.2
10.3
6.7
89.4
18.7
1.8
0.0
12.1
0.2
no data
no data
0
20
40
60
80
NOx emissions (kT)

Ireland (NO_x)



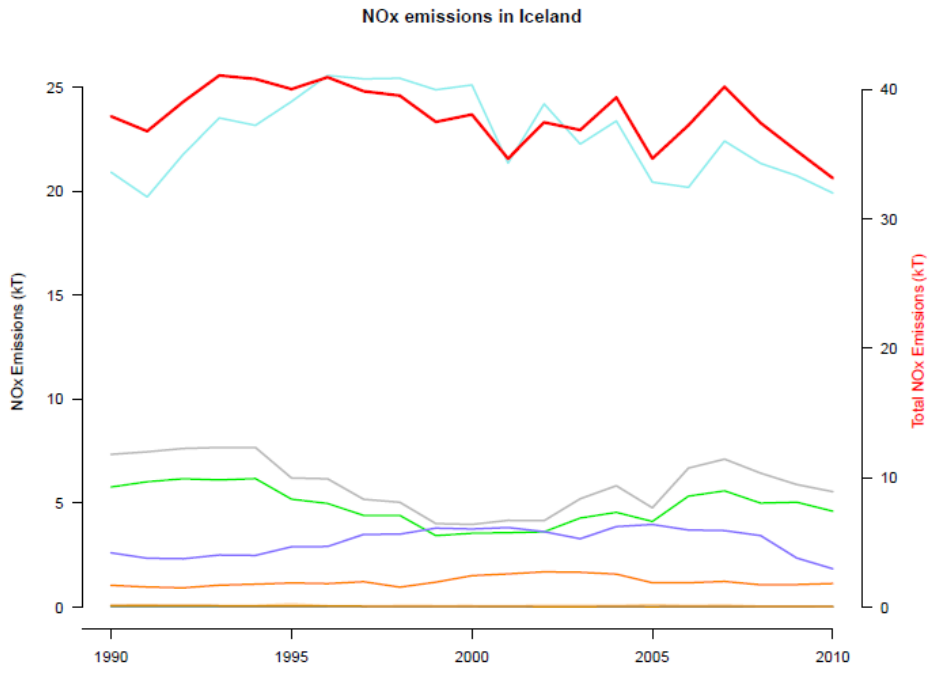
NOx emissions in Ireland
NOx Emissions (kT)
Total NOx Emissions (kT)
60
50
40
30
20
10
0
200
150
100
50
0
1990
1995
2000
2005
2010

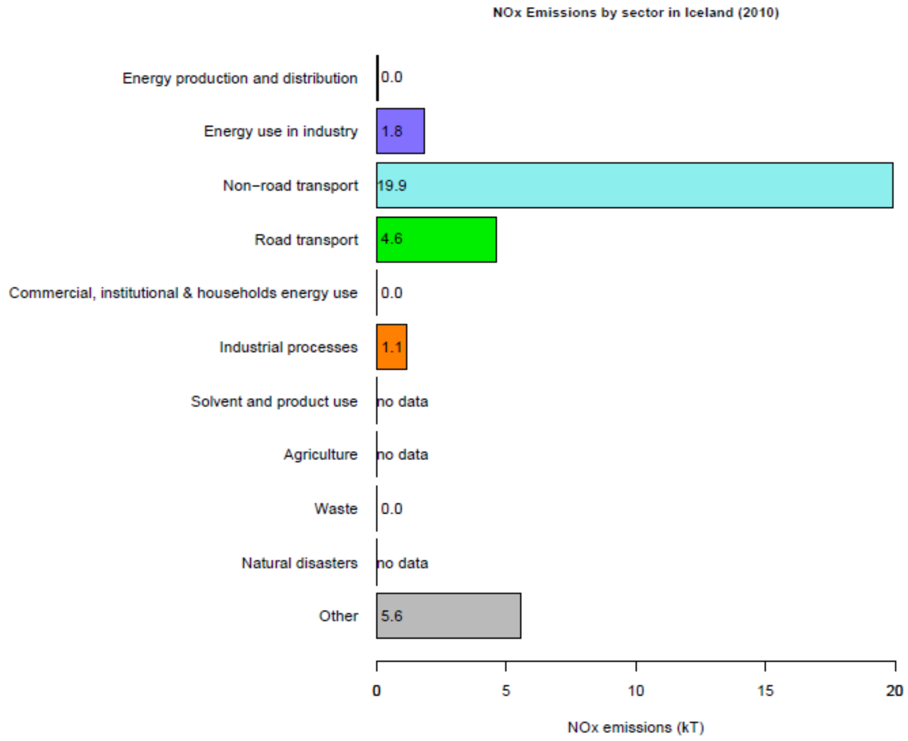


NOx Emissions by sector in Ireland (2010)
Energy production and distribution
12.3
Energy use in industry
9.3
Non−road transport
15.1
Road transport
36.1
Commercial, institutional & households energy use
13.6
Industrial processes
no data
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
no data
Other
36.7
0
10
20
30
40
NOx emissions (kT)

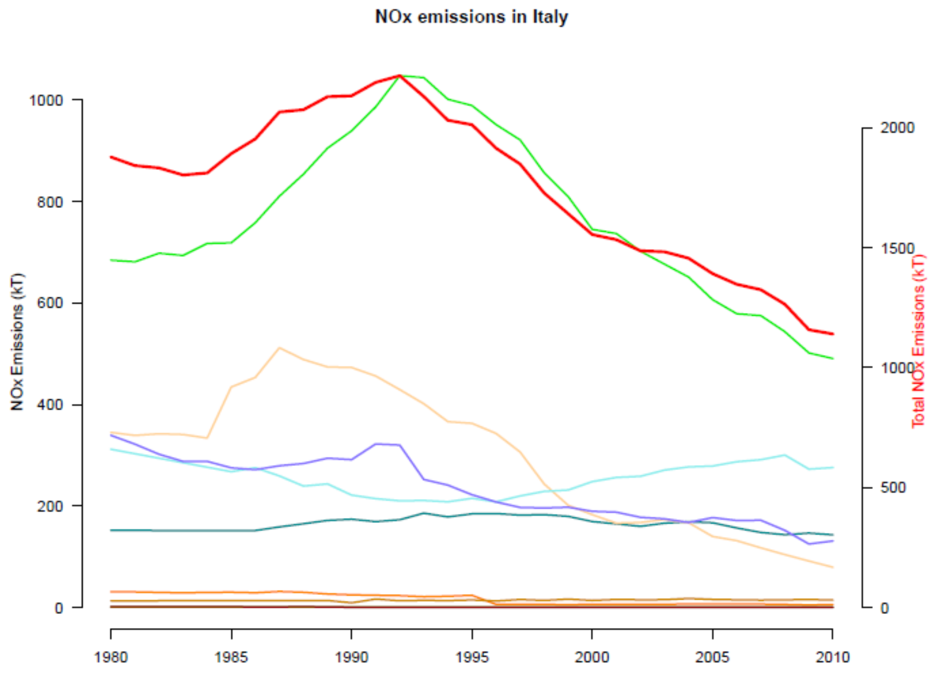
International Prevention Research Institute

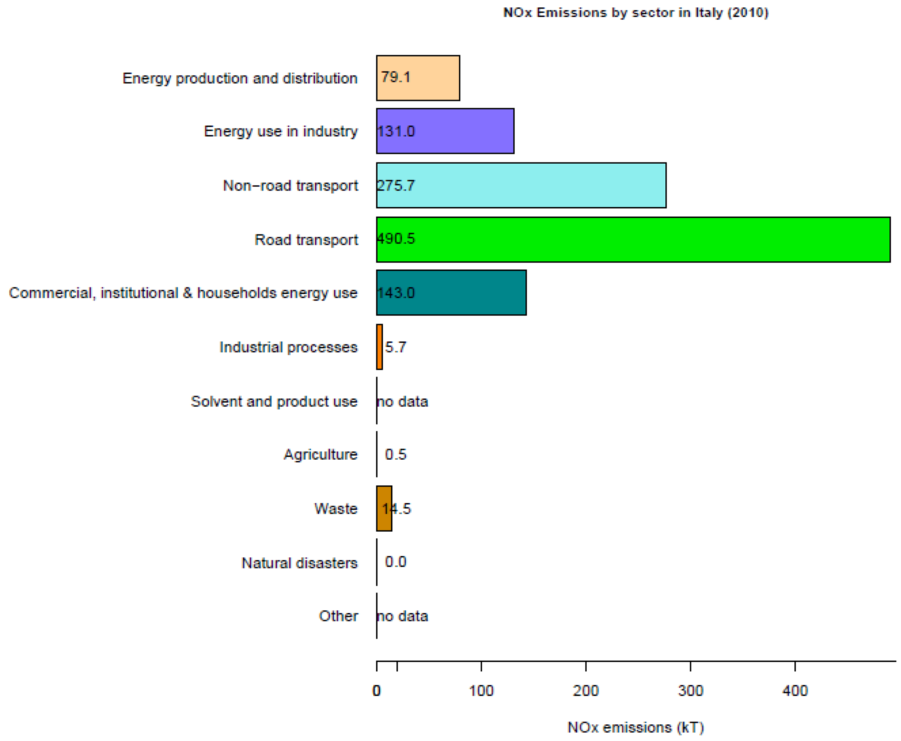
Iceland (NO_x)





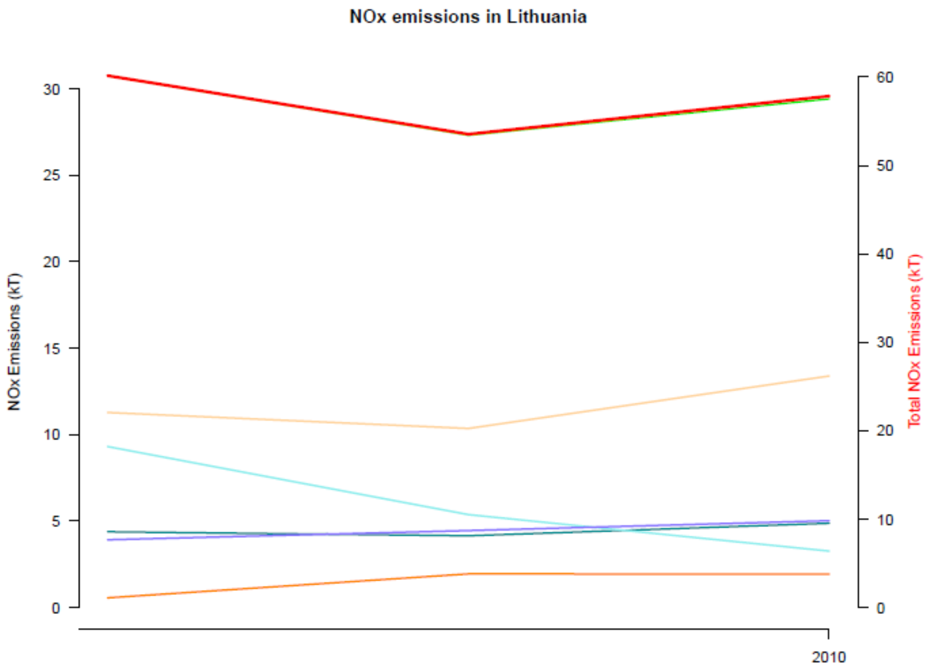
Italy (NO_x)

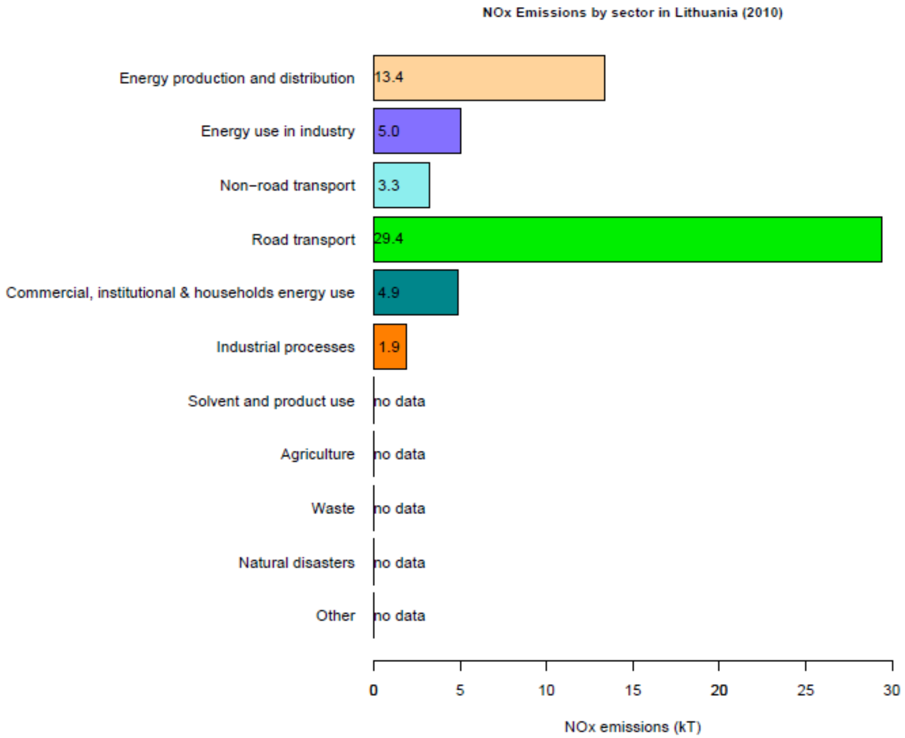

NOx emissions in Italy
NOx Emissions (kT)
Total NOx Emissions (kT)
1000
800
600
400
200
0
2000
1500
1000
500
0
1980
1985
1990
1995
2000
2005
2010


NOx Emissions by sector in Italy (2010)
Energy production and distribution
79.1
Energy use in industry
131.0
Non−road transport
275.7
Road transport
490.5
Commercial, institutional & households energy use
143.0
Industrial processes
5.7
Solvent and product use
no data
Agriculture
0.5
Waste
14.5
Natural disasters
0.0
Other
no data
0
100
200
300
400
NOx emissions (kT)

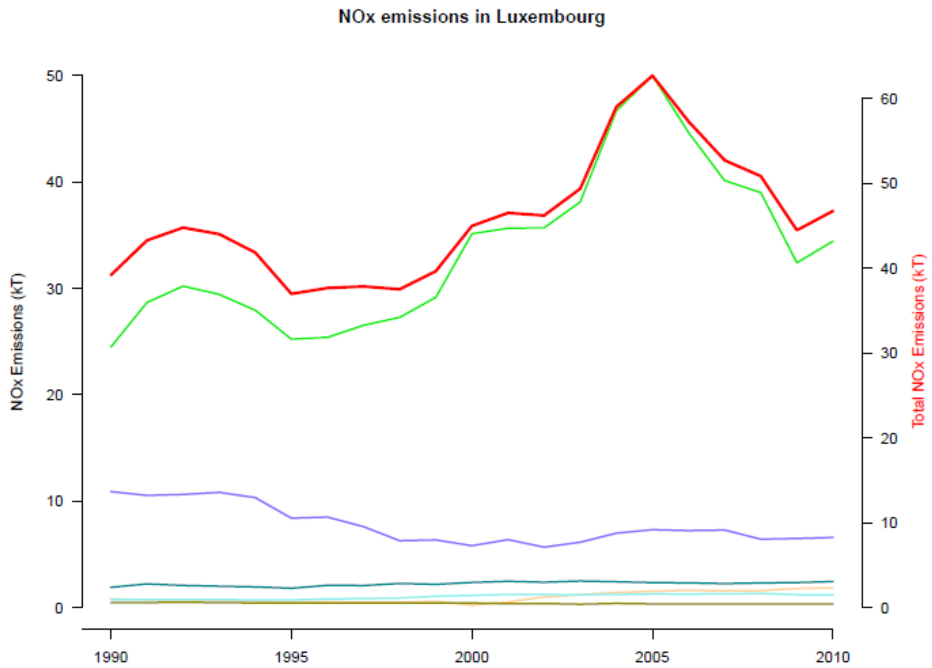
International Prevention Research Institute

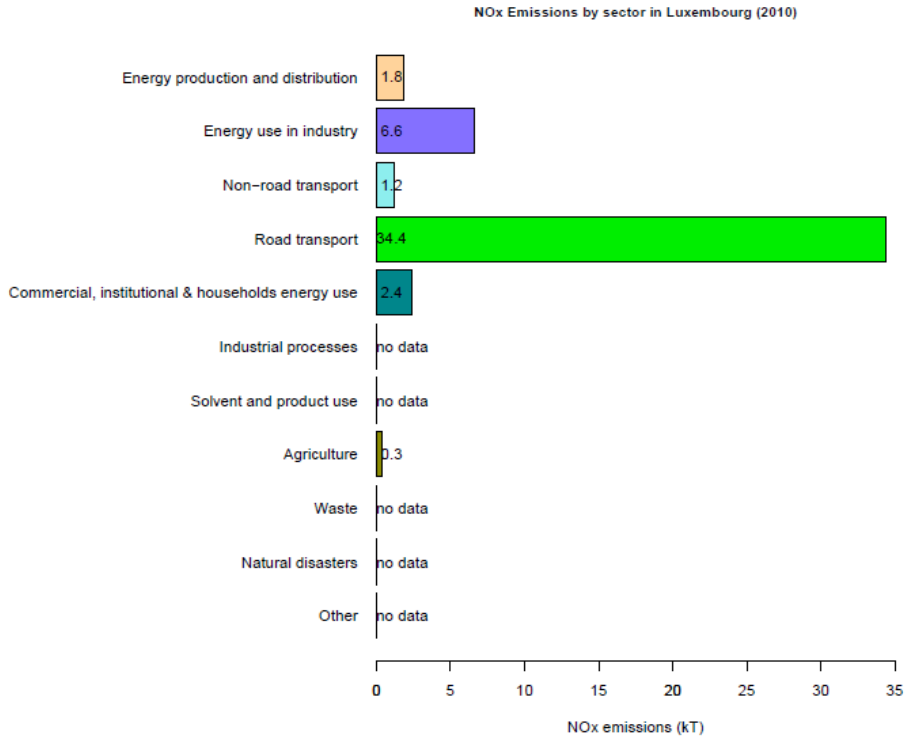
Lithuania (NO_x)



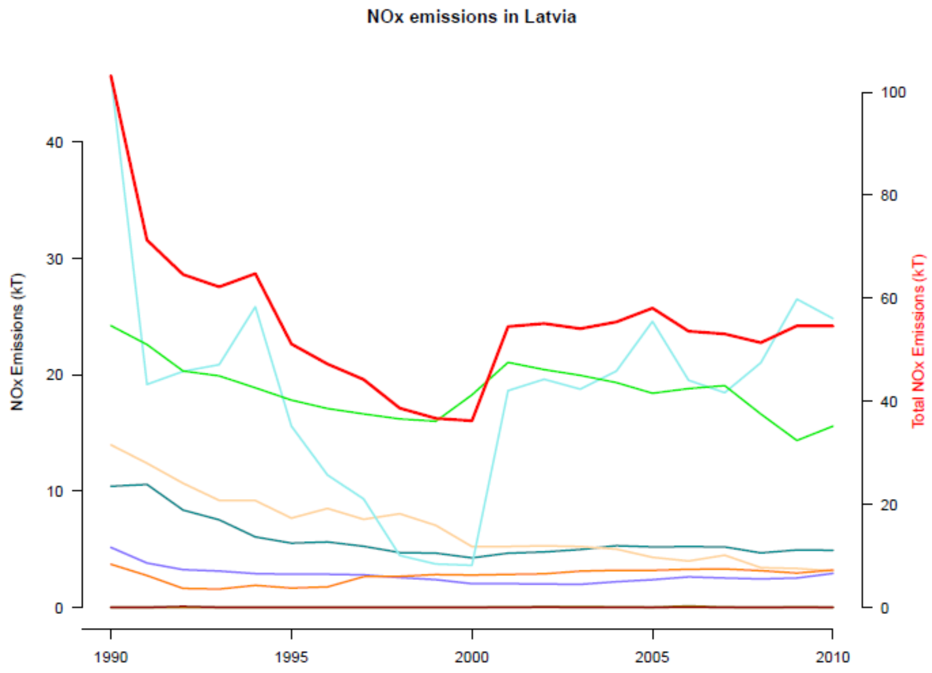


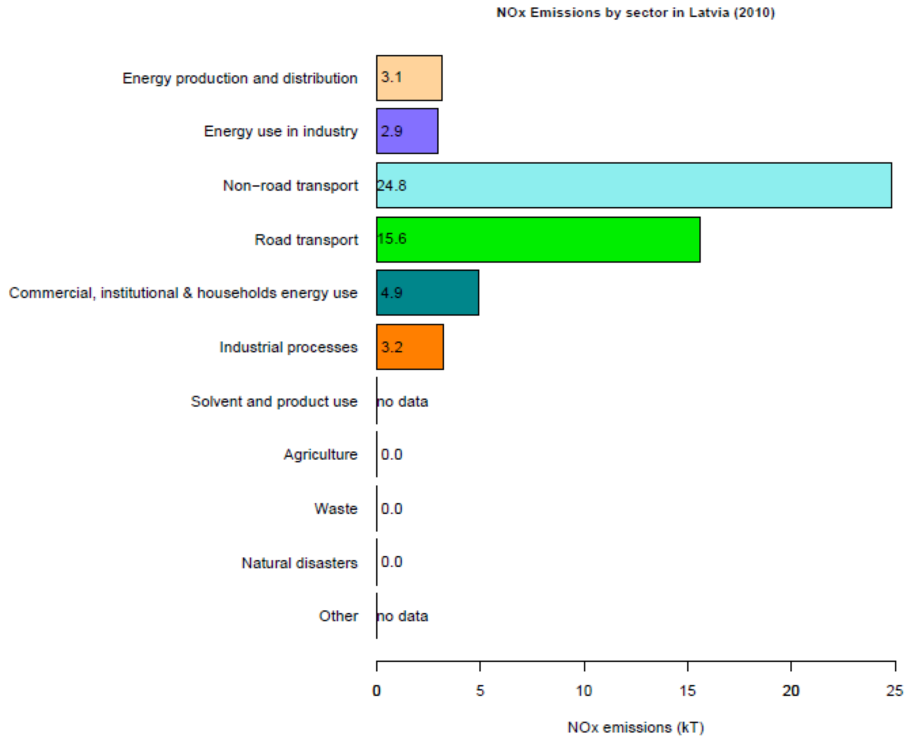
Luxembourg (NO_x)



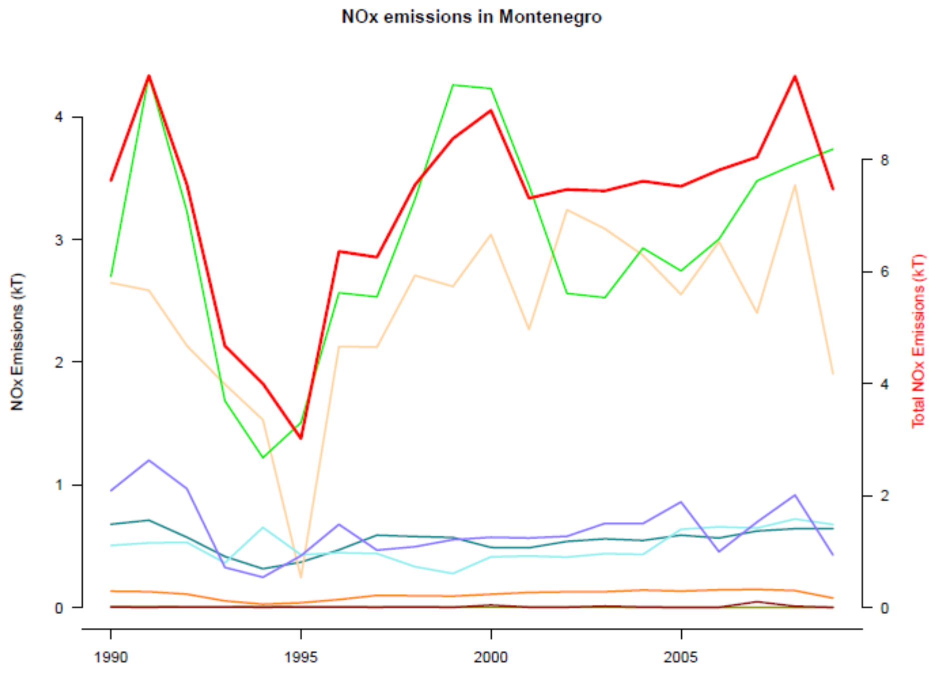


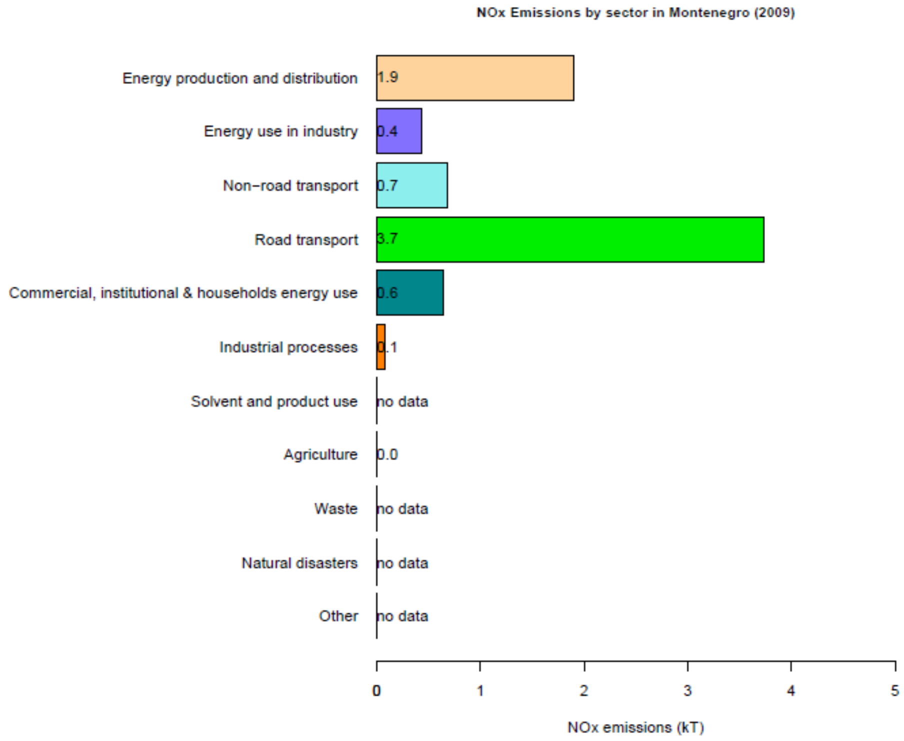
Latvia (NO_x)


NOx emissions in Latvia
NOx Emissions (kT)
Total NOx Emissions (kT)
0
10
20
30
40
0
20
40
60
80
100
1990
1995
2000
2005
2010

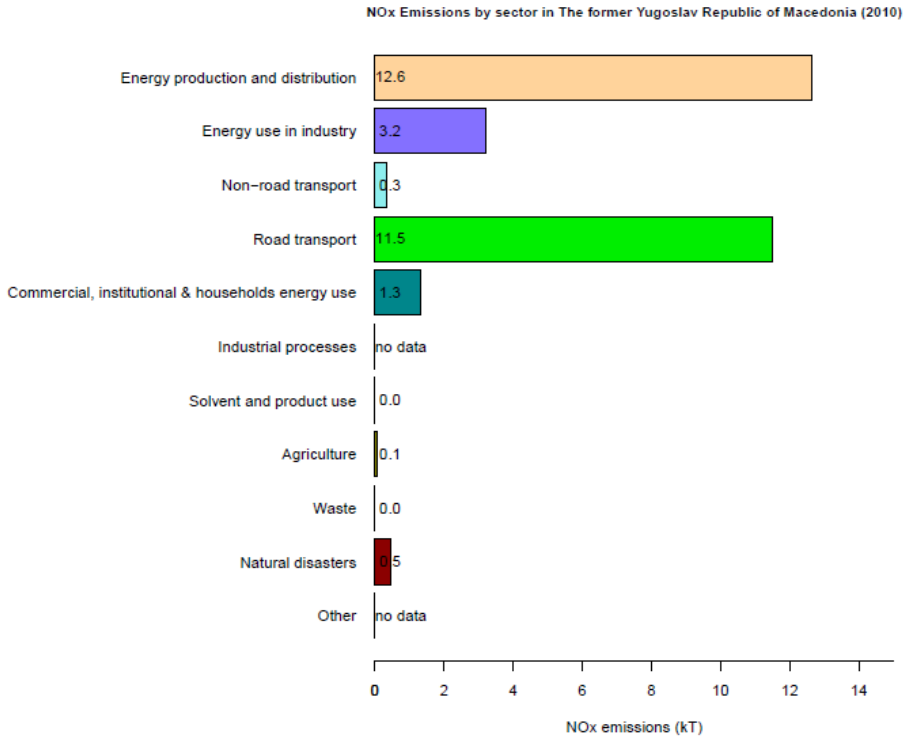

NOx Emissions by sector in Latvia (2010)
Energy production and distribution
3.1
Energy use in industry
2.9
Non−road transport
24.8
Road transport
15.6
Commercial, institutional & households energy use
4.9
Industrial processes
3.2
Solvent and product use
no data
Agriculture
0.0
Waste
0.0
Natural disasters
0.0
Other
no data
0
5
10
15
20
25
NOx emissions (kT)

Montenegro (NO_x)

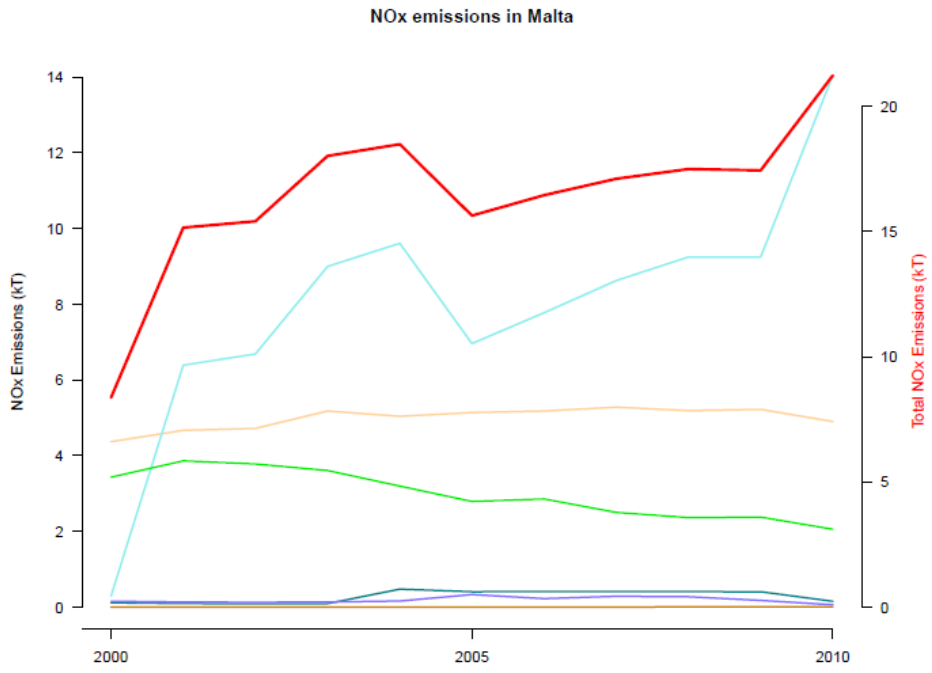




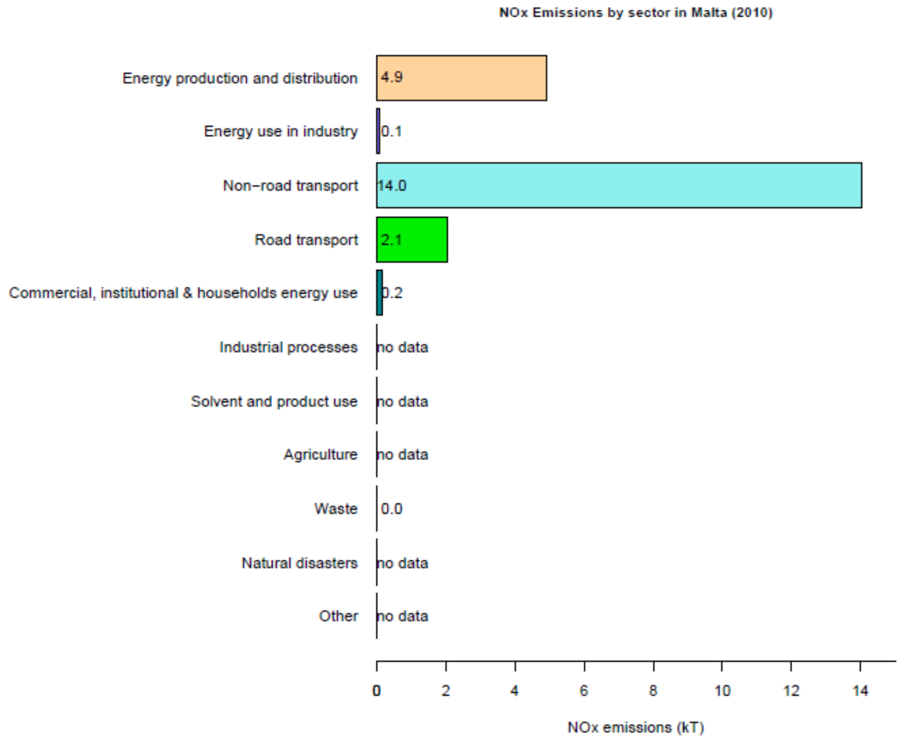
The Former Yugoslav Republic of Macedonia (NO_x)



Malta (NO_x)

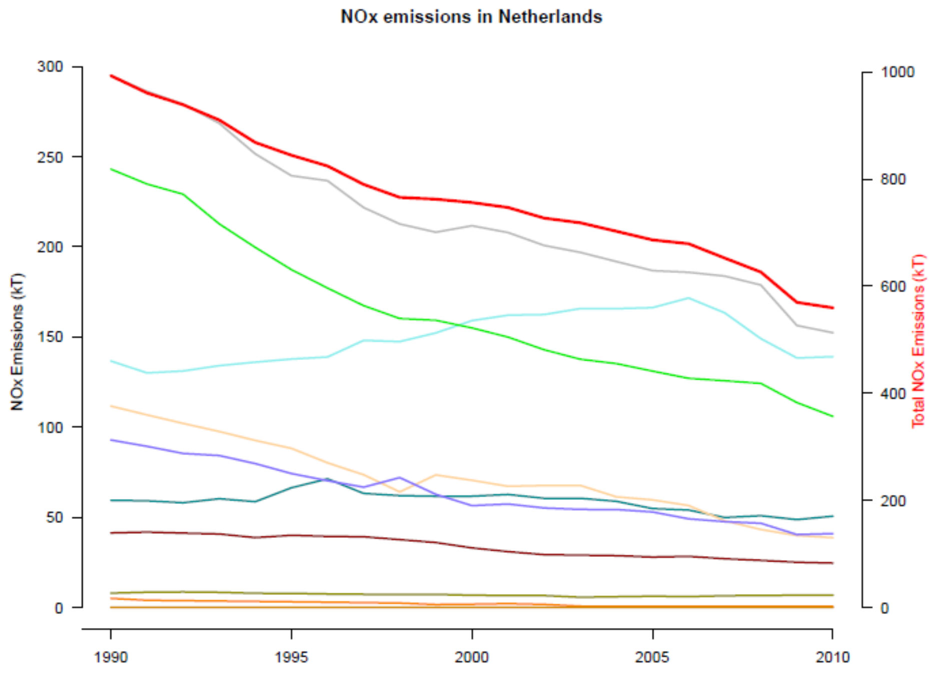


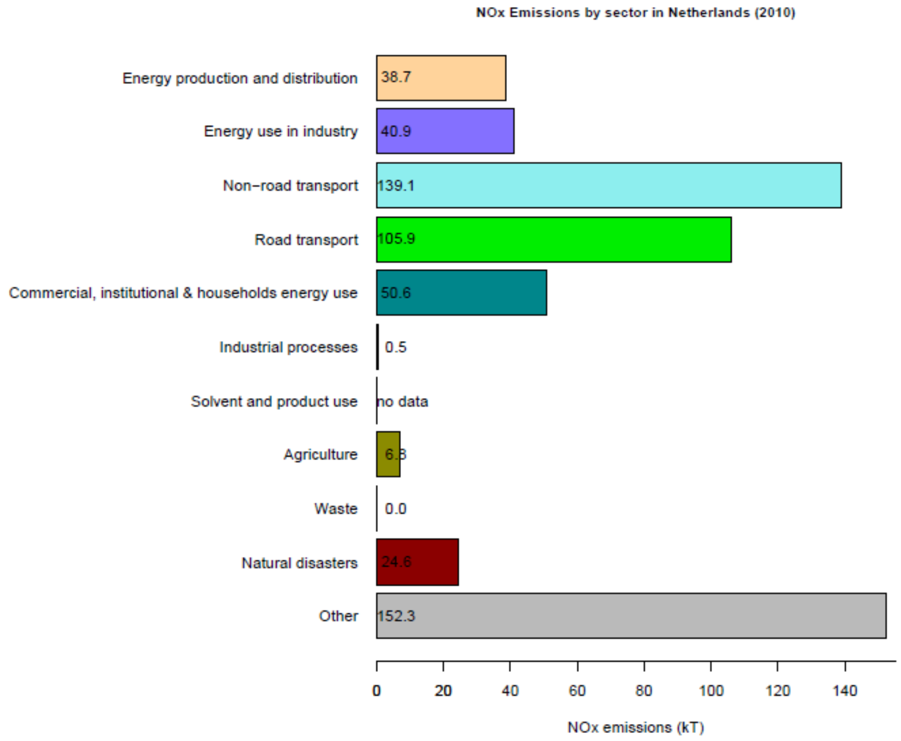
NOx emissions in Malta
NOx Emissions (kT)
Total NOx Emissions (kT)
0
2
4
6
8
10
12
14
5
15
20
2000
2005
2010



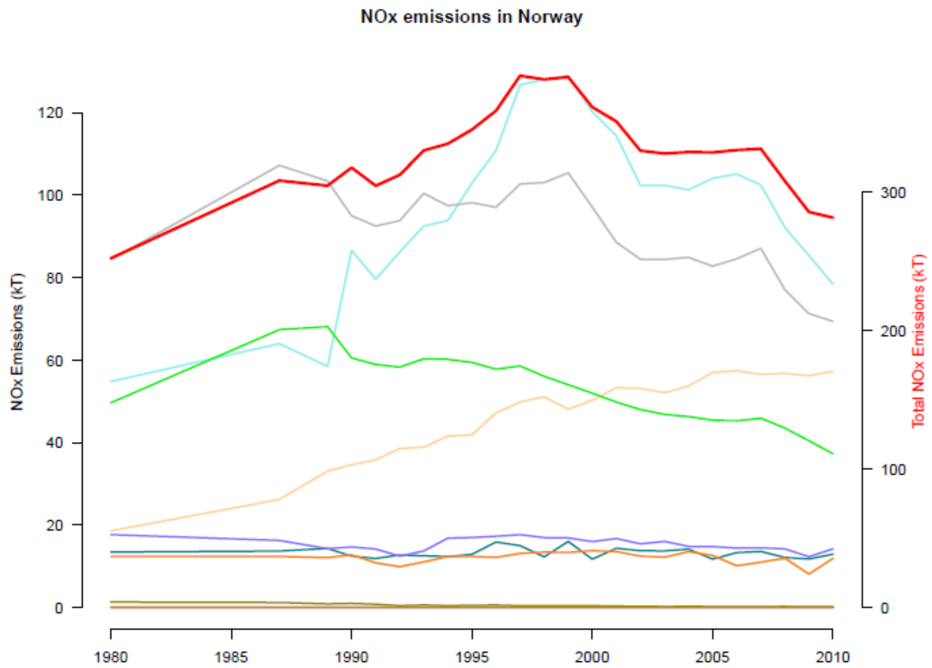
NOx Emissions by sector in Malta (2010)
Energy production and distribution
4.9
Energy use in industry
0.1
Non−road transport
14.0
Road transport
2.1
Commercial, institutional & households energy use
0.2
Industrial processes
no data
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
no data
Other
no data
0
2
4
6
8
10
12
14
NOx emissions (kT)

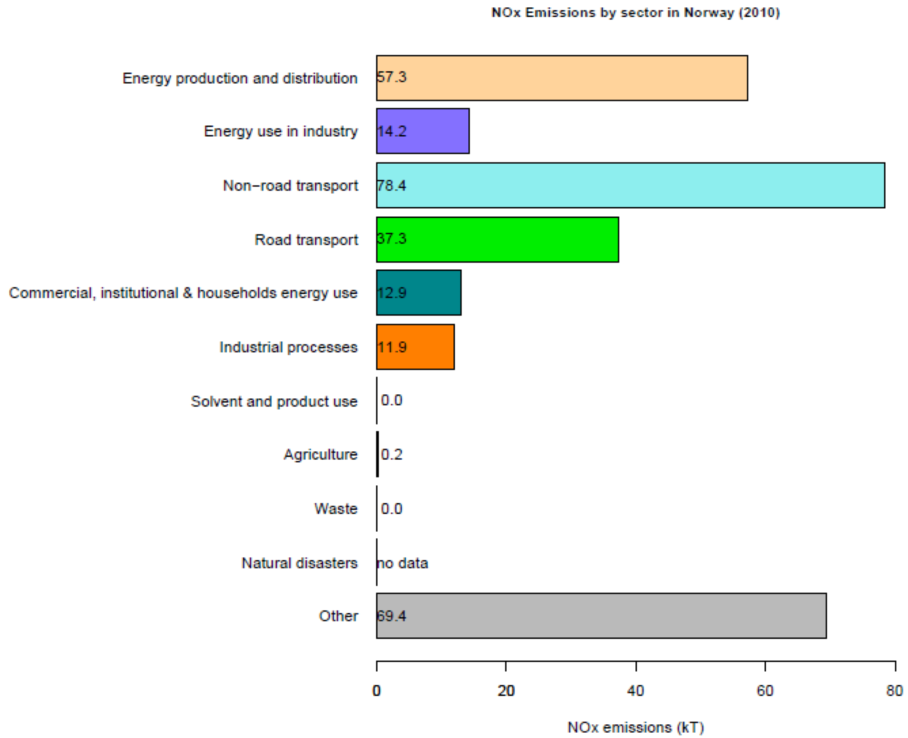
The Netherlands (NO_x)



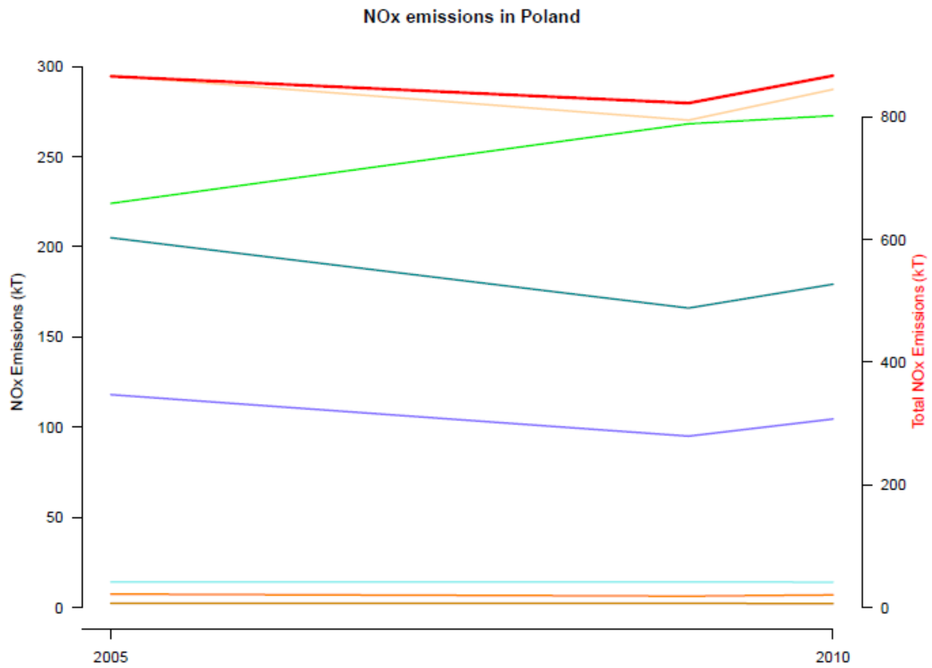


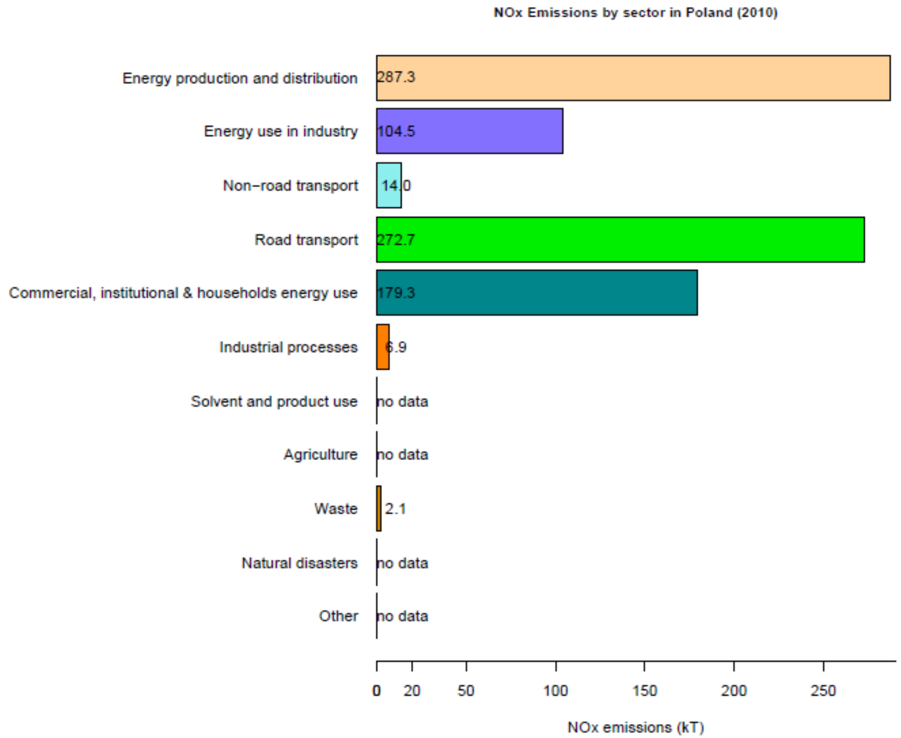
Norway (NO_x)


NOx emissions in Norway
NOx Emissions (kT)
Total NOx Emissions (kT)
120
100
80
60
40
20
0
300
200
100
0
1980
1985
1990
1995
2000
2005
2010

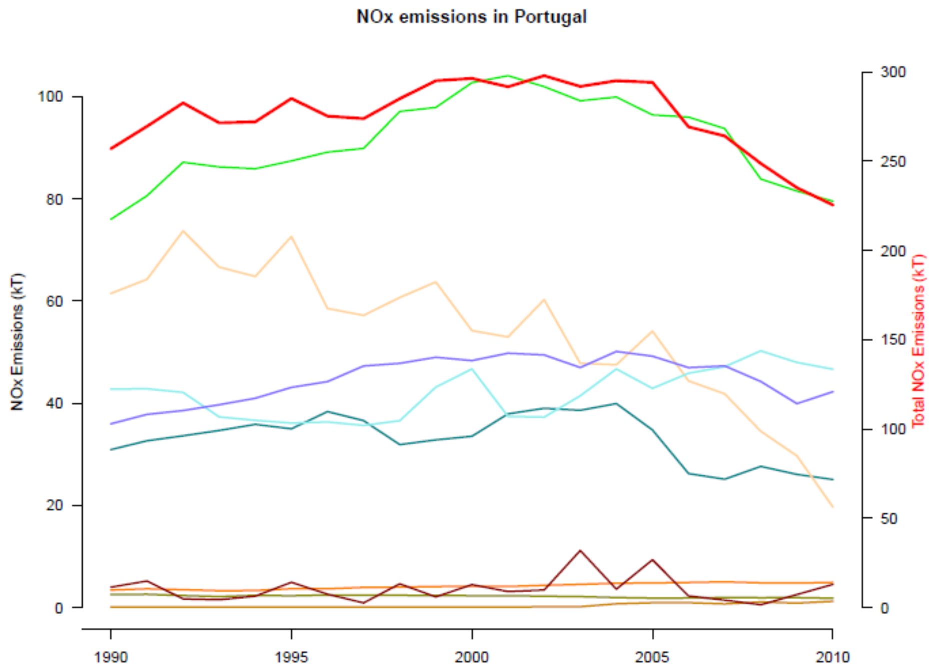

NOx Emissions by sector in Norway (2010)
Energy production and distribution
57.3
Energy use in industry
14.2
Non−road transport
78.4
Road transport
37.3
Commercial, institutional & households energy use
12.9
Industrial processes
11.9
Solvent and product use
0.0
Agriculture
0.2
Waste
0.0
Natural disasters
no data
Other
69.4
0
20
40
60
80
NOx emissions (kT)

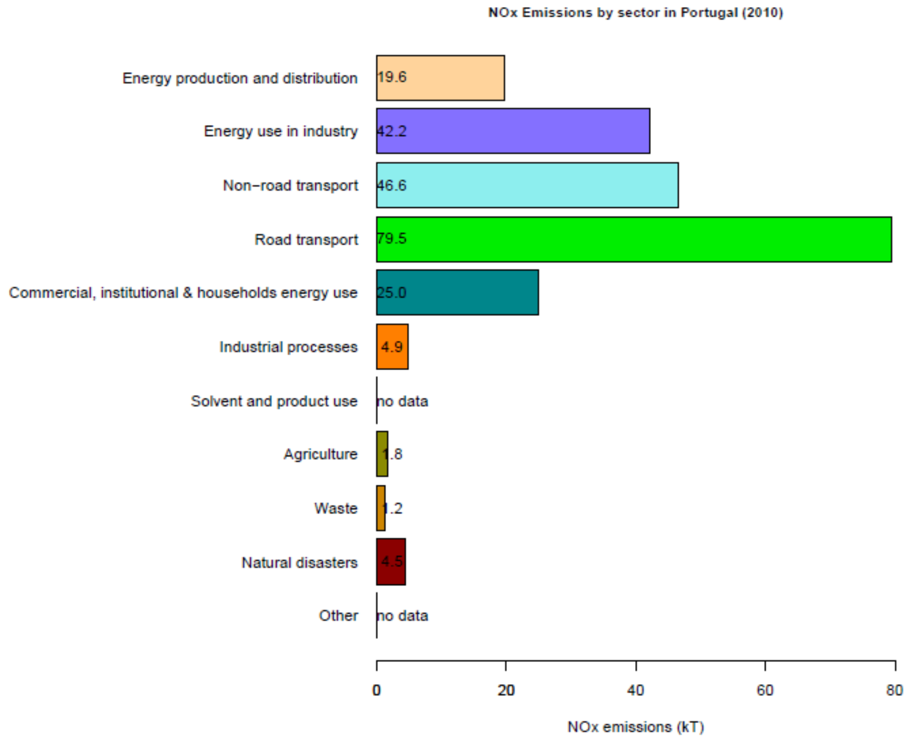
Poland (NO_x)


NOx emissions in Poland
NOx Emissions (kT)
Total NOx Emissions (kT)
300
250
200
150
100
50
0
800
600
400
200
0
2005
2010

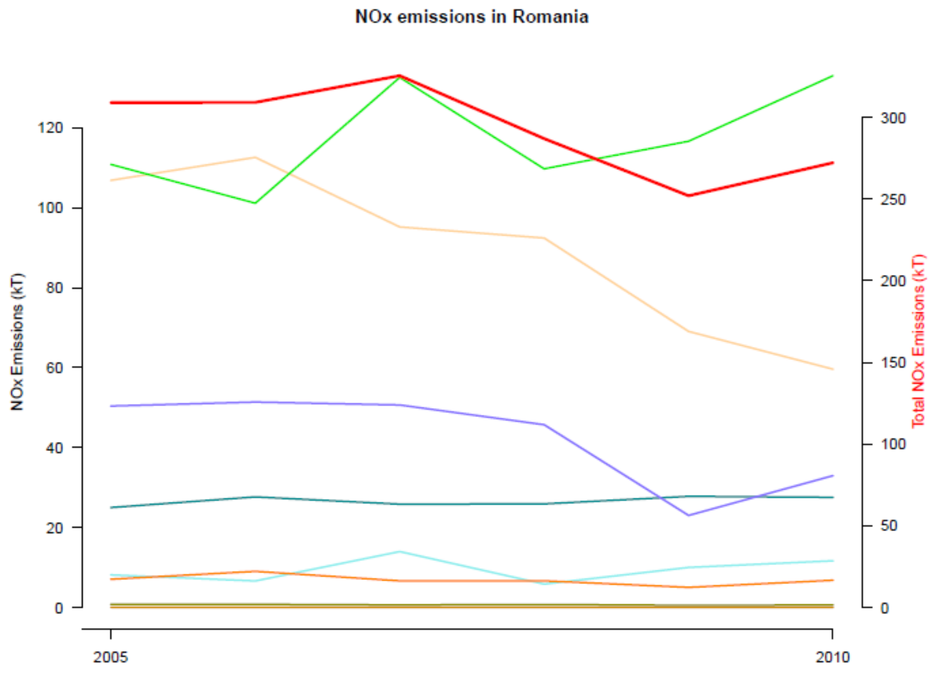

NOx Emissions by sector in Poland (2010)
Energy production and distribution
287.3
Energy use in industry
104.5
Non−road transport
14.0
Road transport
272.7
Commercial, institutional & households energy use
179.3
Industrial processes
6.9
Solvent and product use
no data
Agriculture
no data
Waste
2.1
Natural disasters
no data
Other
no data
0
20
50
100
150
200
250
NOx emissions (kT)

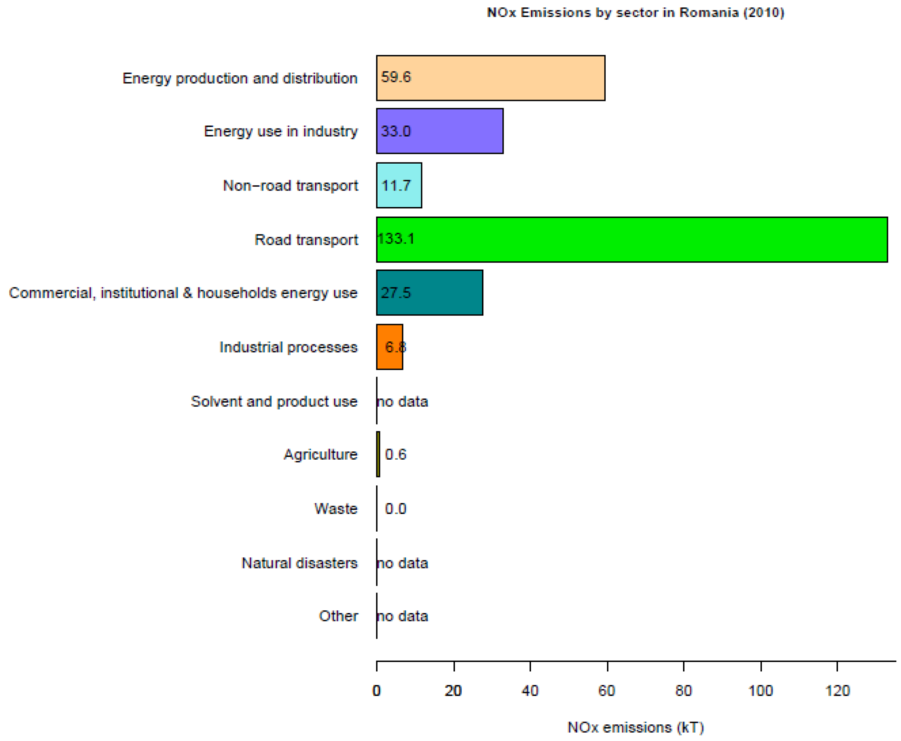
Portugal (NO_x)



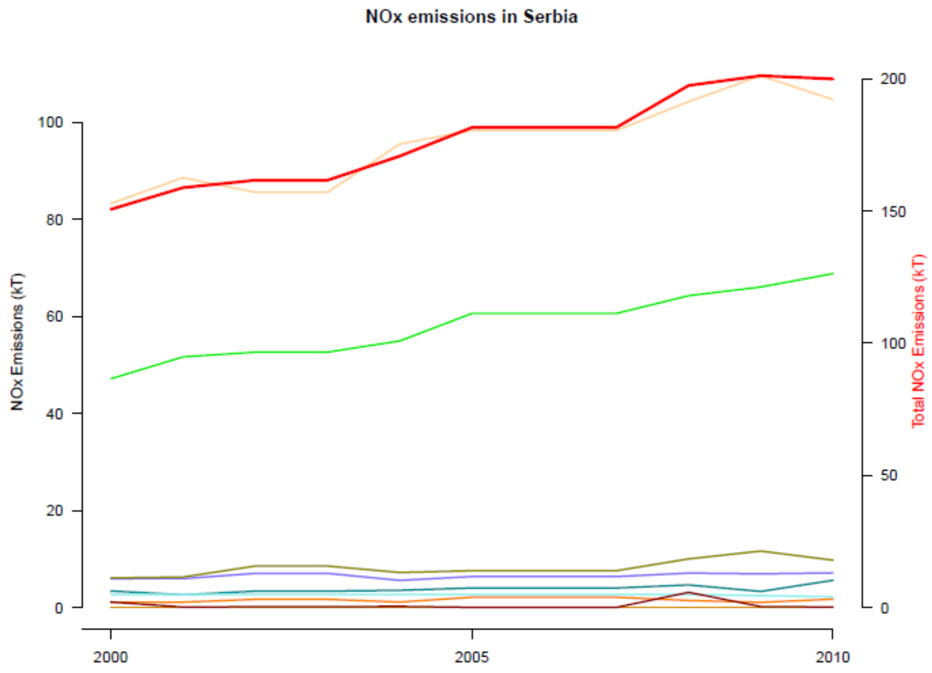


Romania (NO_x)

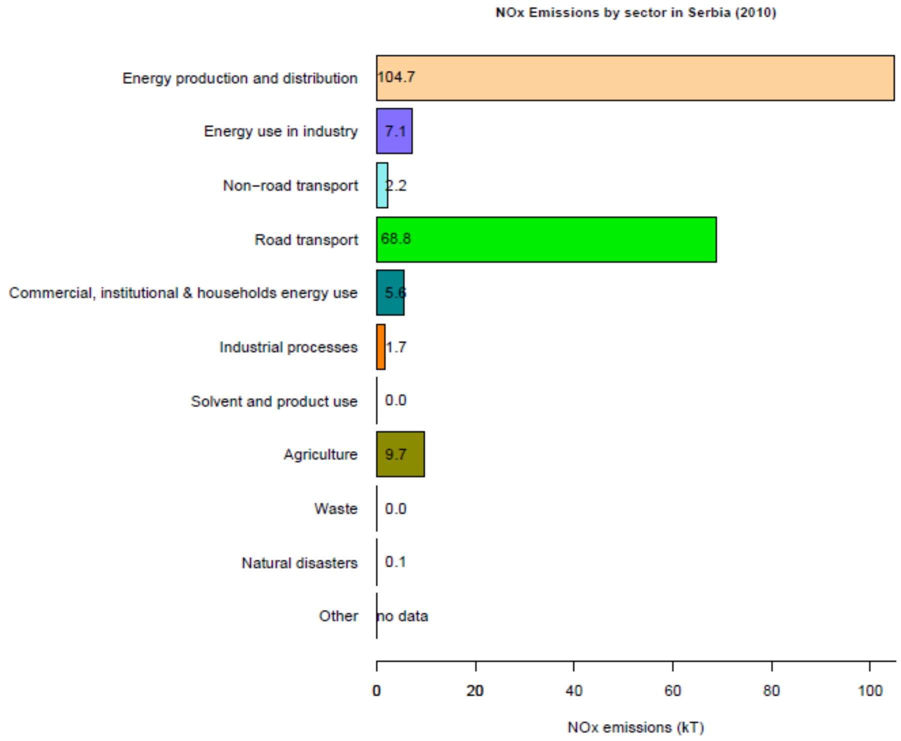

NOx emissions in Romania
NOx Emissions (kT)
Total NOx Emissions (kT)
120
100
80
60
40
20
0
300
250
200
150
100
50
0
2005
2010


NOx Emissions by sector in Romania (2010)
Energy production and distribution
59.6
Energy use in industry
33.0
Non−road transport
11.7
Road transport
133.1
Commercial, institutional & households energy use
27.5
Industrial processes
6.8
Solvent and product use
no data
Agriculture
0.6
Waste
0.0
Natural disasters
no data
Other
no data
0
20
40
60
80
100
120
NOx emissions (kT)

Serbia (NO_x)

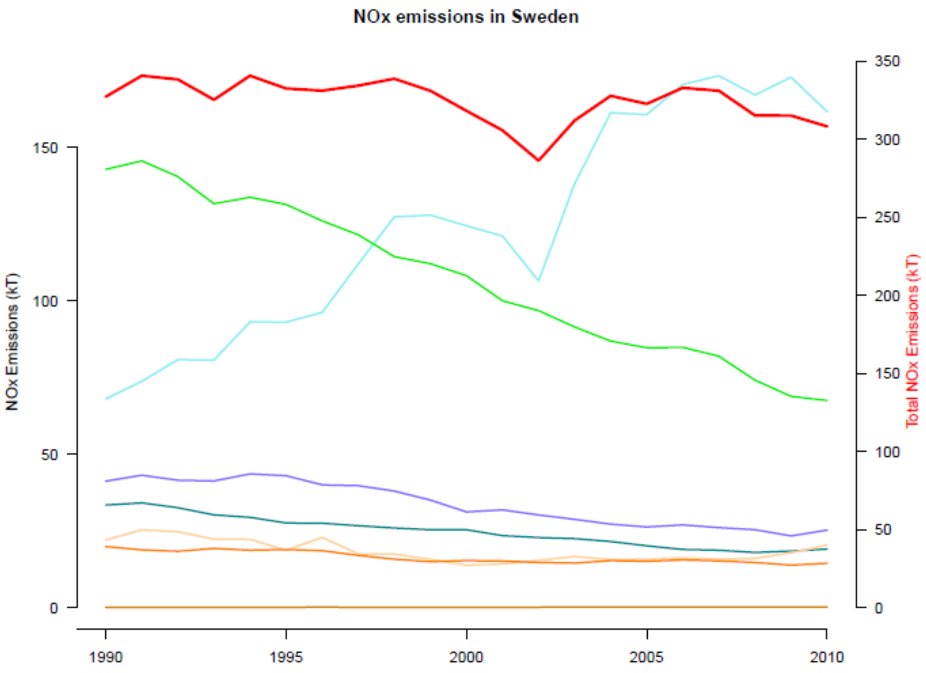


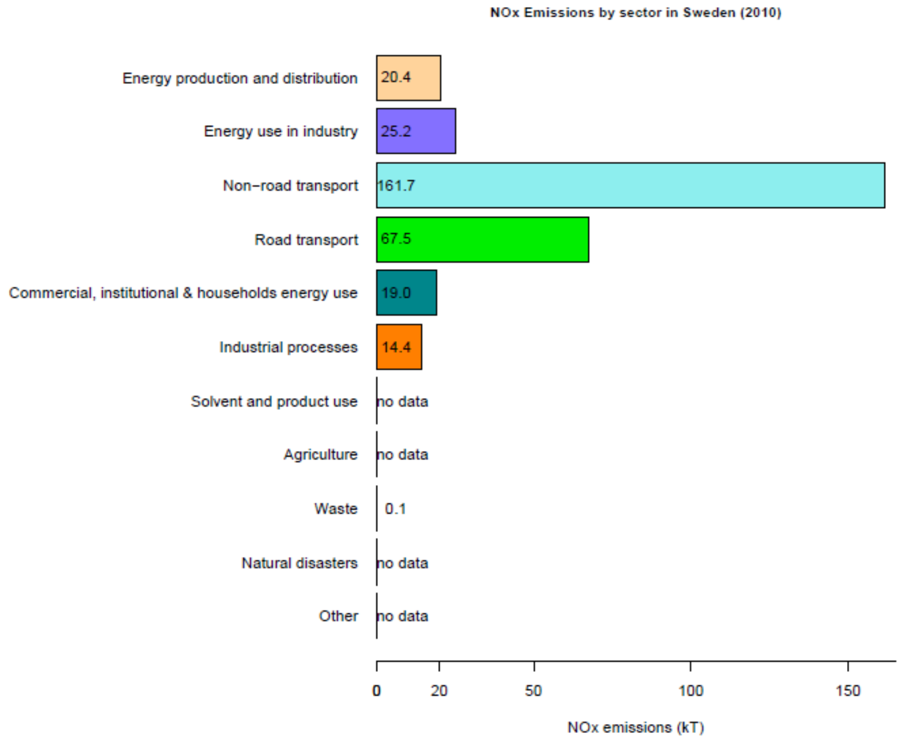
NOx emissions in Serbia
NOx Emissions (kT)
Total NOx Emissions (kT)
100
80
60
40
20
0
200
150
100
50
0
2000
2005
2010



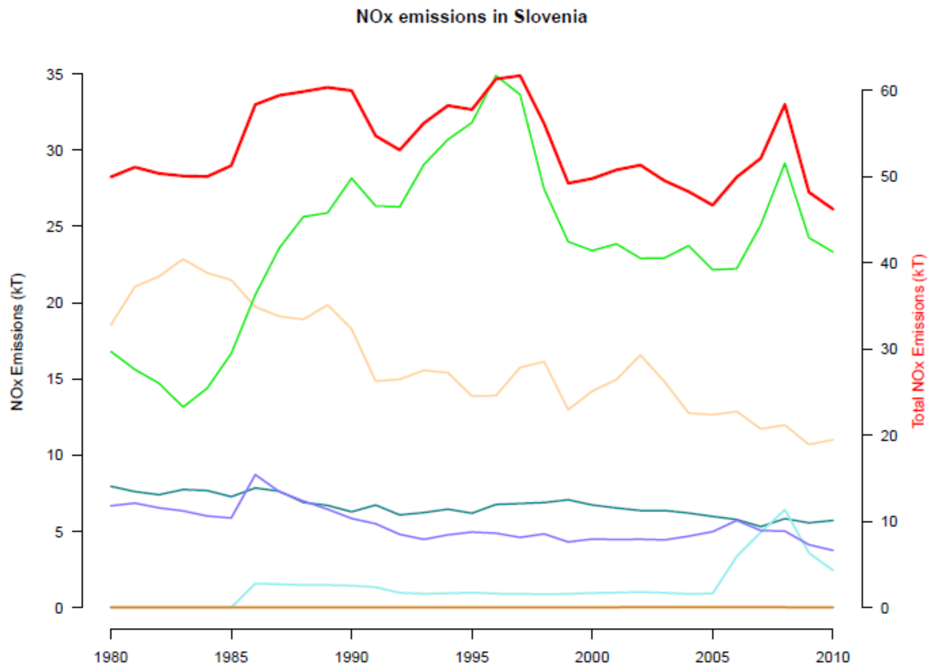
NOx Emissions by sector in Serbia (2010)
Energy production and distribution
104.7
Energy use in industry
7.1
Non−road transport
2.2
Road transport
68.8
Commercial, institutional & households energy use
5.6
Industrial processes
1.7
Solvent and product use
0.0
Agriculture
9.7
Waste
0.0
Natural disasters
0.1
Other
no data
0
20
40
60
80
100
NOx emissions (kT)

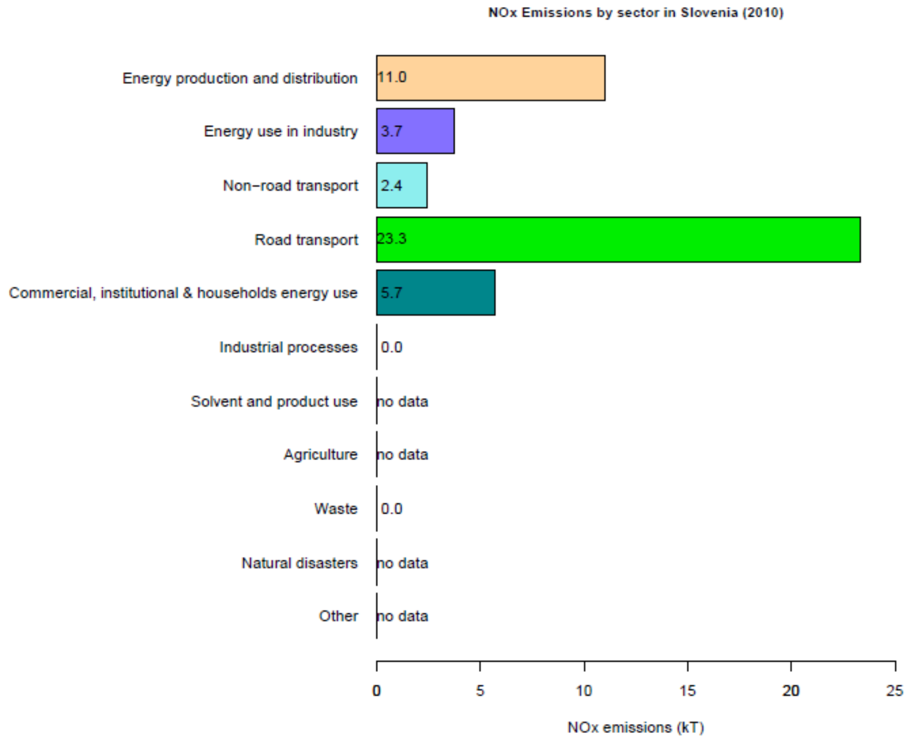
Sweden (NO_x)


NOx emissions in Sweden
NOx Emissions (kT)
Total NOx Emissions (kT)
0
50
100
150
0
50
100
150
200
250
300
350
1990
1995
2000
2005
2010

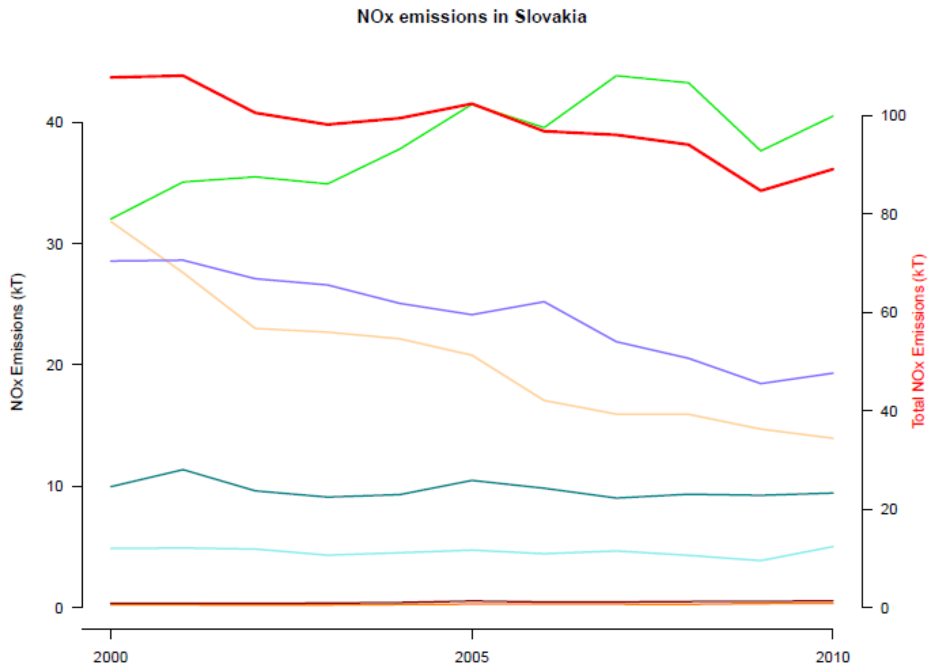

NOx Emissions by sector in Sweden (2010)
Energy production and distribution
20.4
Energy use in industry
25.2
Non−road transport
161.7
Road transport
67.5
Commercial, institutional & households energy use
19.0
Industrial processes
14.4
Solvent and product use
no data
Agriculture
no data
Waste
0.1
Natural disasters
no data
Other
no data
0
20
50
100
150
NOx emissions (kT)

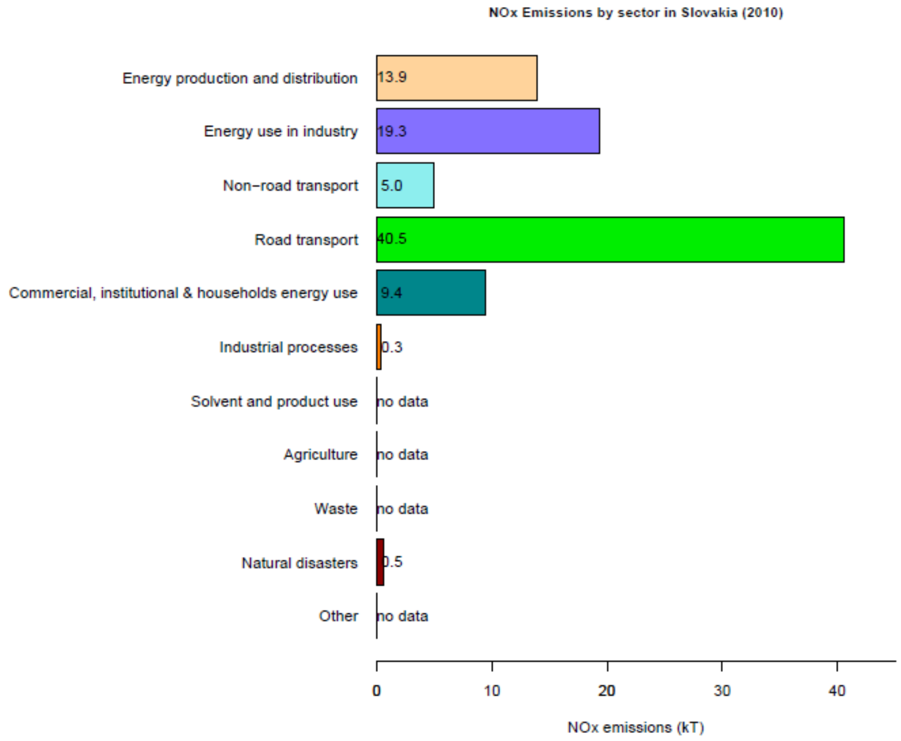
Slovenia (NO_x)



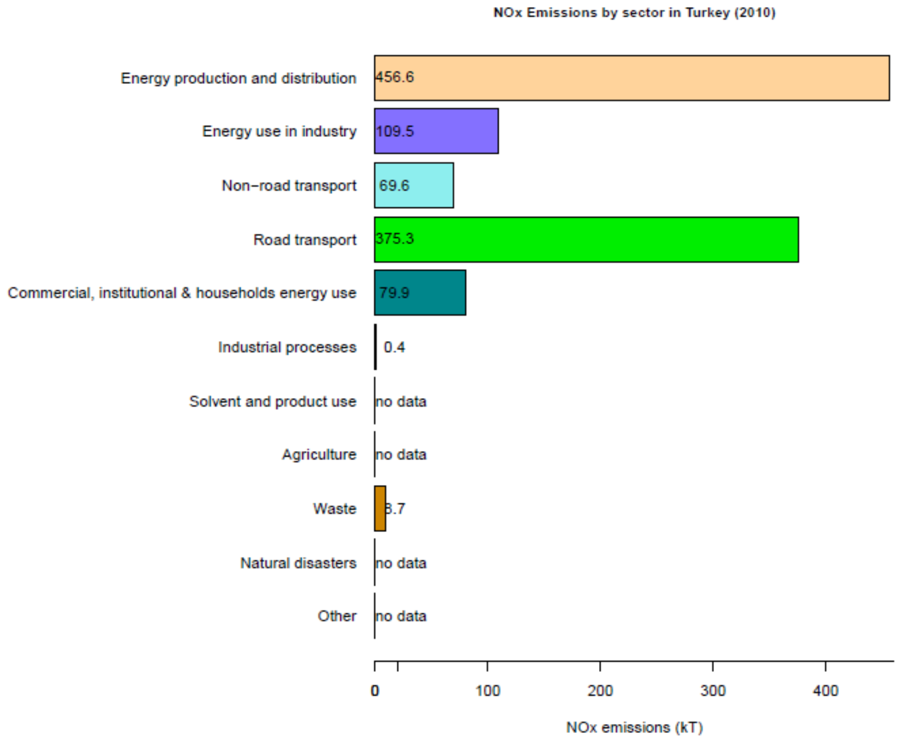


Slovakia (NO_x)



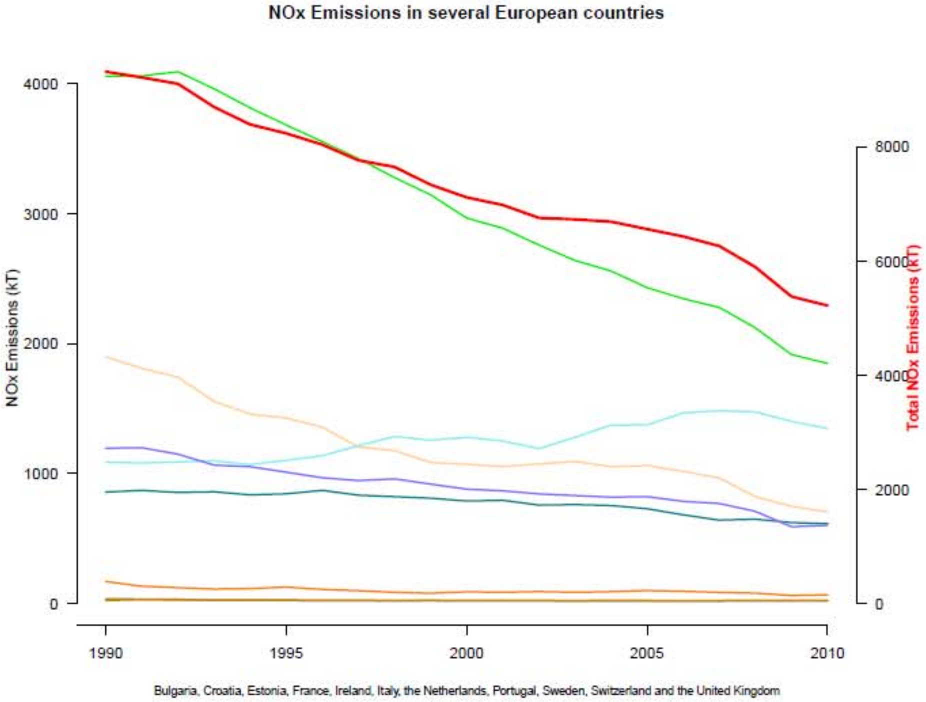


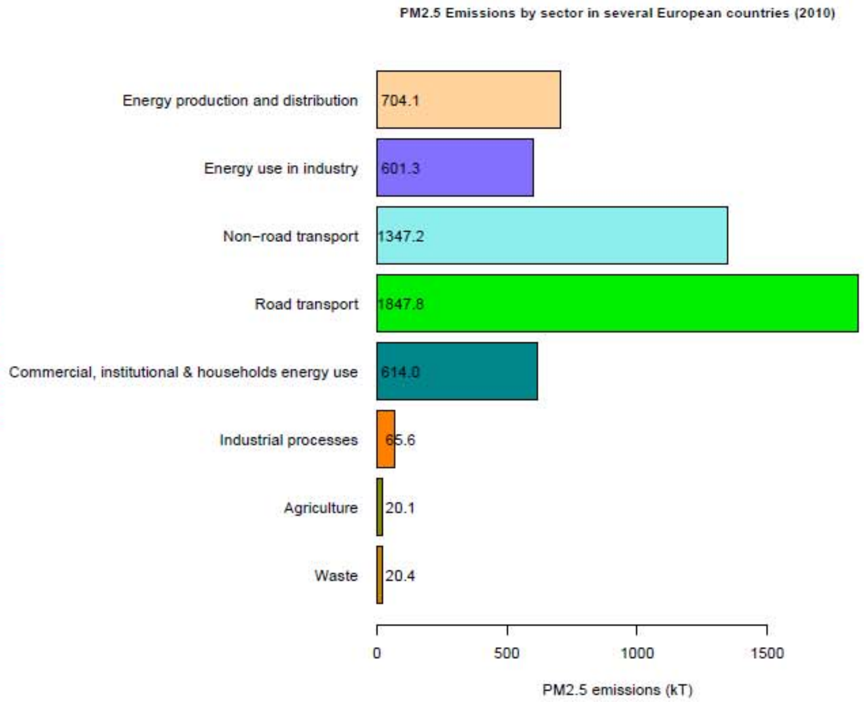
Turkey (NO_x)



NOx Emissions by sector in Turkey (2010)
Energy production and distribution
Energy use in industry
Non−road transport
Road transport
Commercial, institutional & households energy use
Industrial processes
Solvent and product use
Agriculture
Waste
Natural disasters
Other
456.6
109.5
69.6
375.3
79.9
0.4
no data
no data
8.7
no data
no data
0
100
200
300
400
NOx emissions (kT)

Bulgaria, Croatia, Estonia, France, Ireland, Italy, the Netherlands, Portugal, Sweden, Switzerland and the United Kingdom (NO_x)





II.4. SO_x

II.4.1 Data availability

Availability of NO_x data is described below:

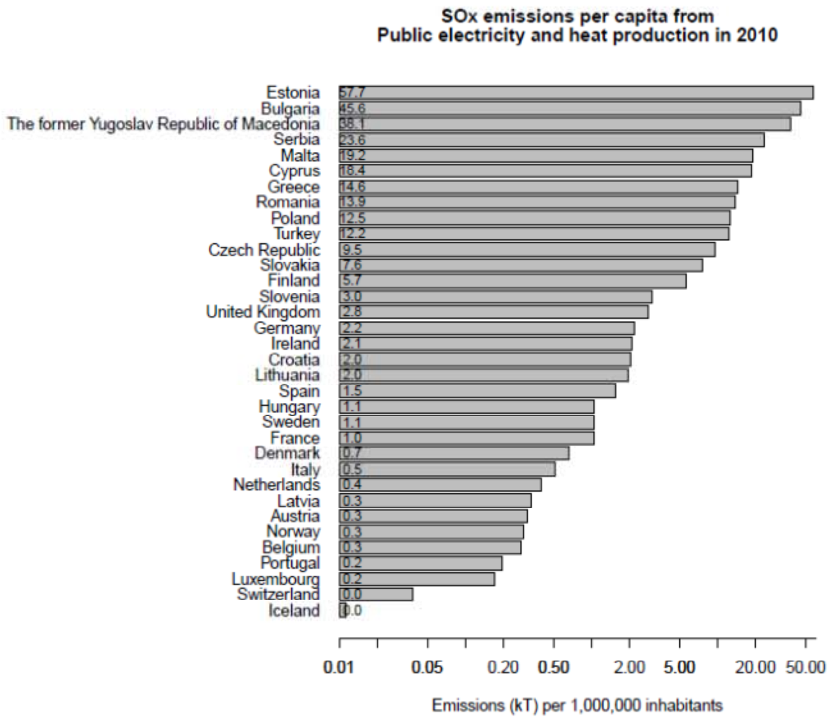
Country	From	To	Missing years	x = no data										
				EP	EI	NRT	RT	CIH	IP	S	A	W	N	O
Albania	1990	2009	-							x		x	x	x
Austria	1980	2010	-							x			x	
Belgium	1990	2010	1991-1999							x	x		x	x
Bulgaria	1990	2010	-							x	x		x	x
Croatia	1990	2010	-							x	x			
Cyprus	1990	2010	-						x	x		x	x	
Czech Republic	2010	2010	-							x	x		x	
Denmark	1980	2010	-										x	x
Estonia	1990	2010	-								x		x	x
Finland	1980	2010	-								x		x	x
France	1980	2010	-							x				x
Germany	1990	2010	-							x	x			x
Greece	1990	2010	-							x	x	x	x	x
Hungary	2010	2010	-							x			x	x
Iceland	1990	2010	-							x	x			
Ireland	1987	2010	1988-1989						x	x	x		x	
Italy	1980	2010	-							x	x			x
Latvia	1990	2010	-							x	x		x	x
Lithuania	2008	2010	-							x	x	x	x	x
Luxembourg	1990	2010	-						x	x	x	x	x	x
FYROM	2010	2010	-						x	x	x			x

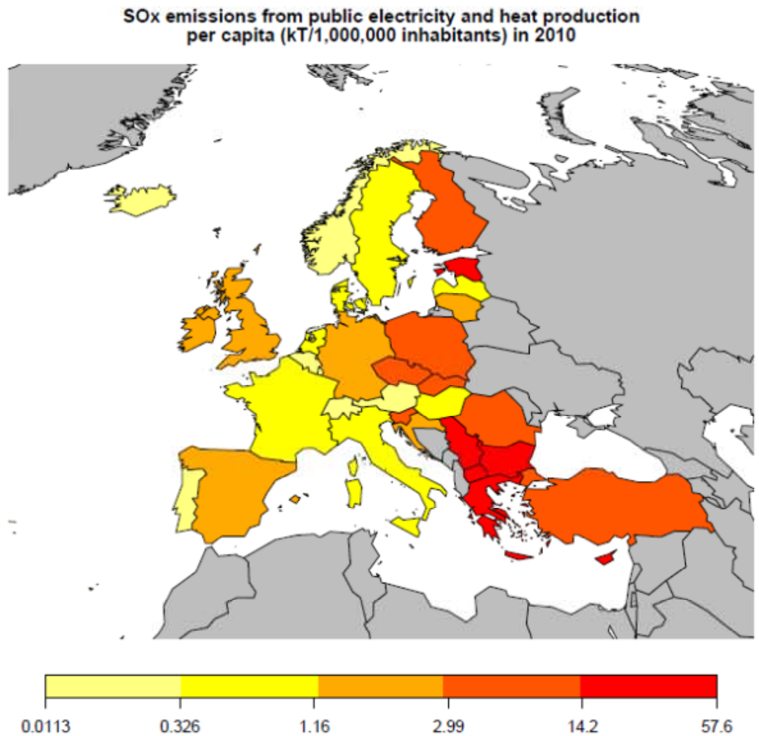
Country	From	To	Missing years	x = no data										
				EP	EI	NRT	RT	CIH	IP	S	A	W	N	O
Malta	2000	2010	-						x	x	x		x	
Montenegro	1990	2009	-							x	x	x	x	x
Netherlands	1990	2010	-							x	x		x	
Norway	1980	2010	1981-1986; 1988							x			x	
Poland	2005	2010	2006-2008							x	x		x	x
Portugal	1990	2010	-							x			x	x
Romania	2005	2010	-							x	x		x	x
Serbia	2000	2010	-							x	x			x
Slovakia	2000	2010	-							x	x	x	x	x
Slovenia	1980	2010	-							x	x		x	x
Spain	1990	2010	-							x				x
Sweden	1990	2010	-							x	x		x	x
Switzerland	1990	2010	-											
Turkey	2010	2010	-							x	x		x	x
United Kingdom	1980	2010	-							x	x		x	x

Abbreviation list:

EP: energy production and distribution
NRT: non-road transport
CIH: commercial, Institutional and Household energy use
S: solvent and product use
W: waste
O: other emissions
EI: energy use in industry
RT: road transport
IP: industrial processes
A: agriculture
N: natural emissions

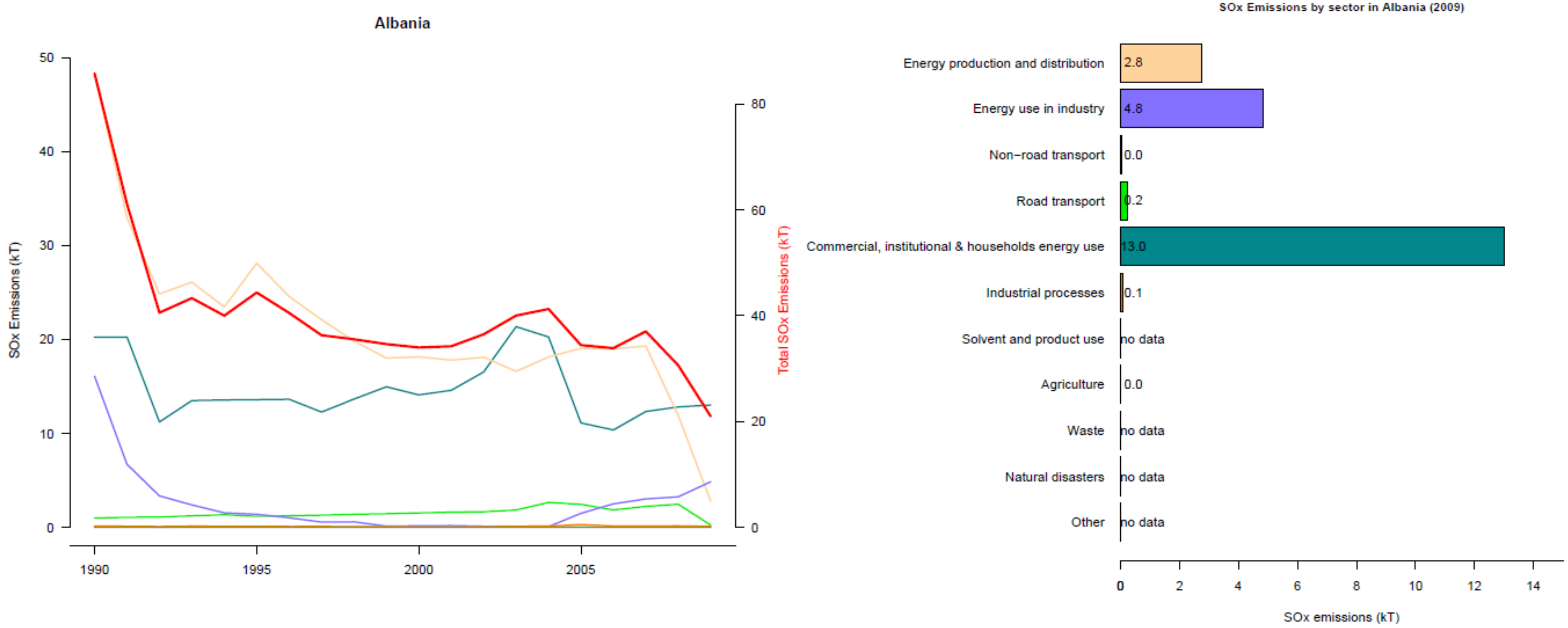
II.4.2. SO_x emissions per capita from Public electricity and heat production sector in 2010



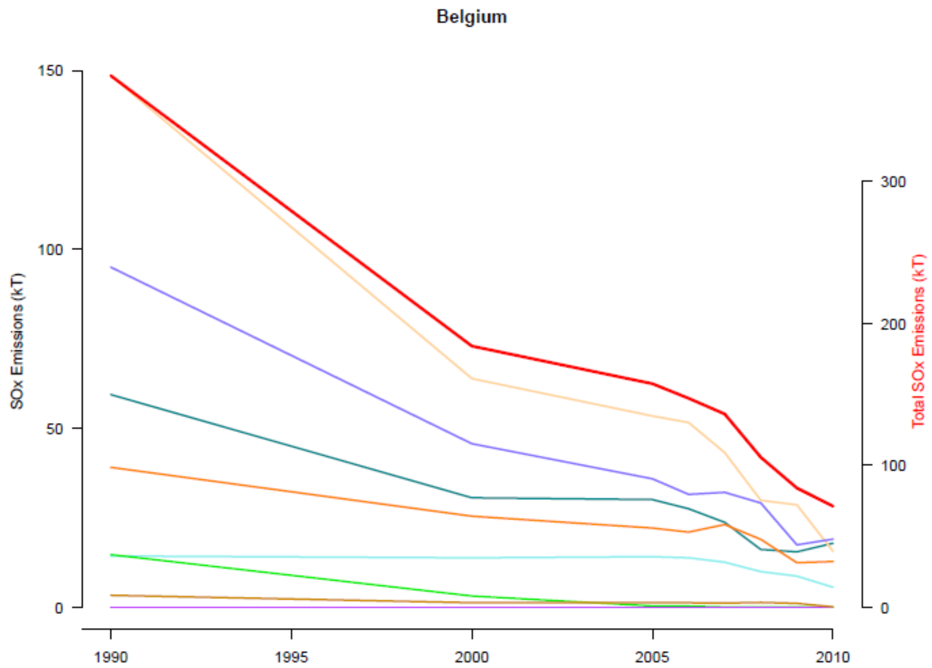


II.4.3. SO_x emission trends by sector (left) and distribution of emissions by sector for the last available year (right)

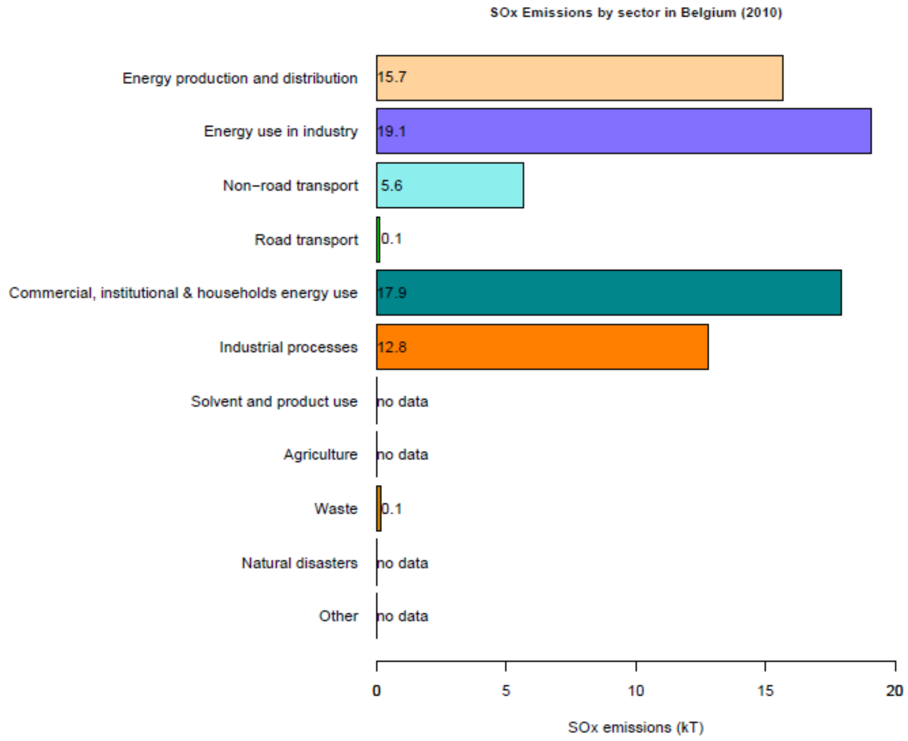
Albania (SO_x)



Belgium (SO_x)



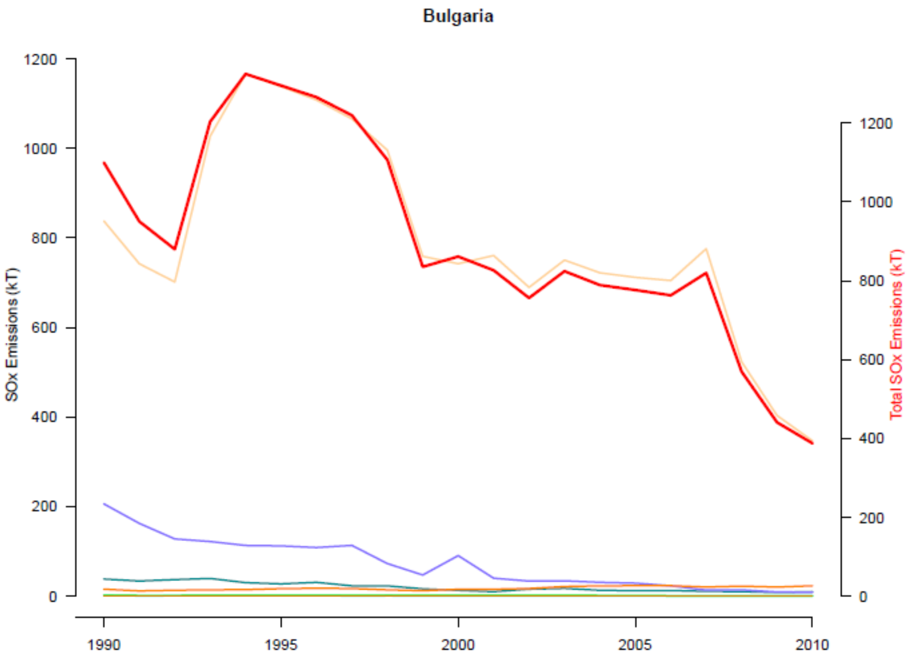
Belgium
150
100
50
0
SOx Emissions (kT)
300
200
100
0
Total SOx Emissions (kT)
1990
1995
2000
2005
2010

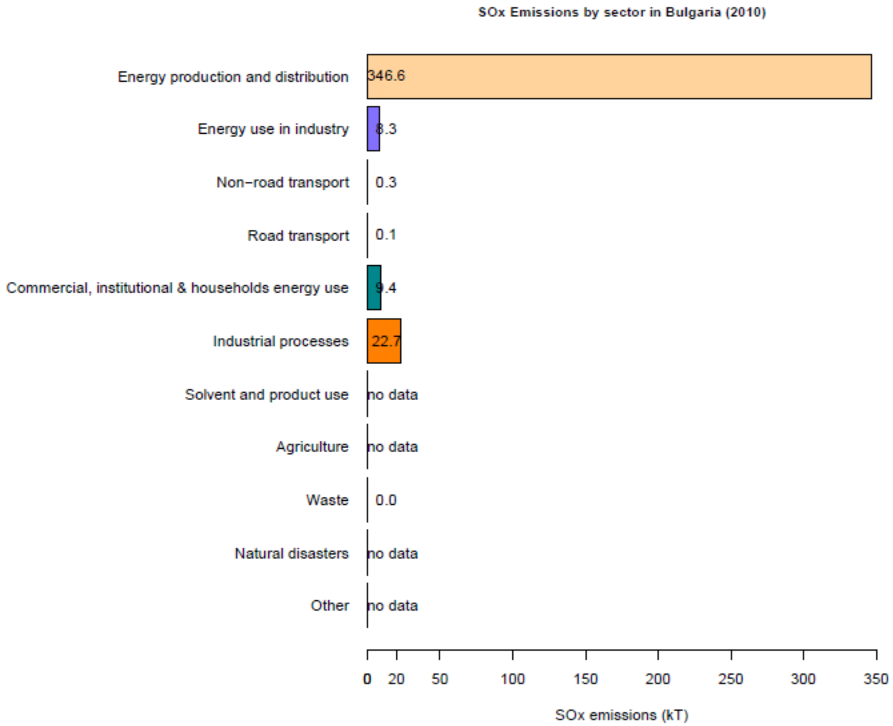


SOx Emissions by sector in Belgium (2010)
Energy production and distribution
15.7
Energy use in industry
19.1
Non−road transport
5.6
Road transport
0.1
Commercial, institutional & households energy use
17.9
Industrial processes
12.8
Solvent and product use
no data
Agriculture
no data
Waste
0.1
Natural disasters
no data
Other
no data
0
5
10
15
20
SOx emissions (kT)

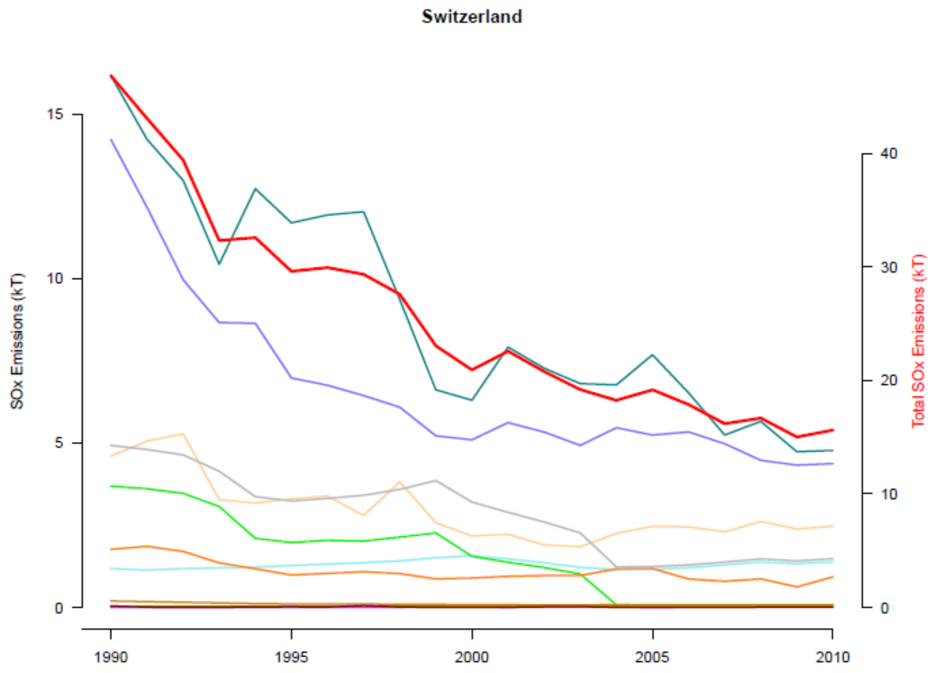
International Prevention Research Institute

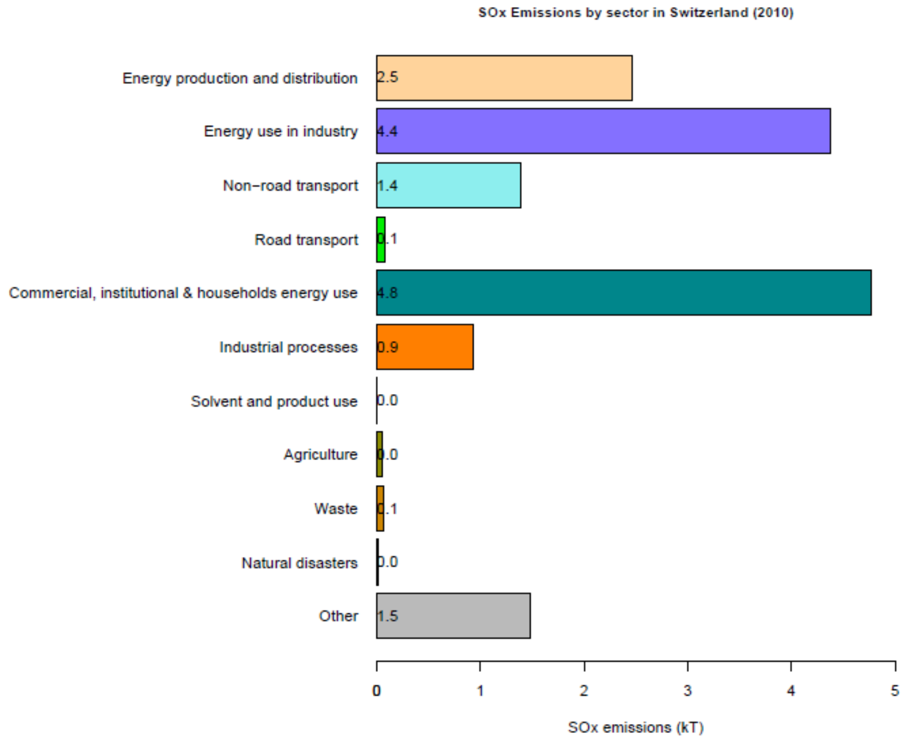
Bulgaria (SO_x)



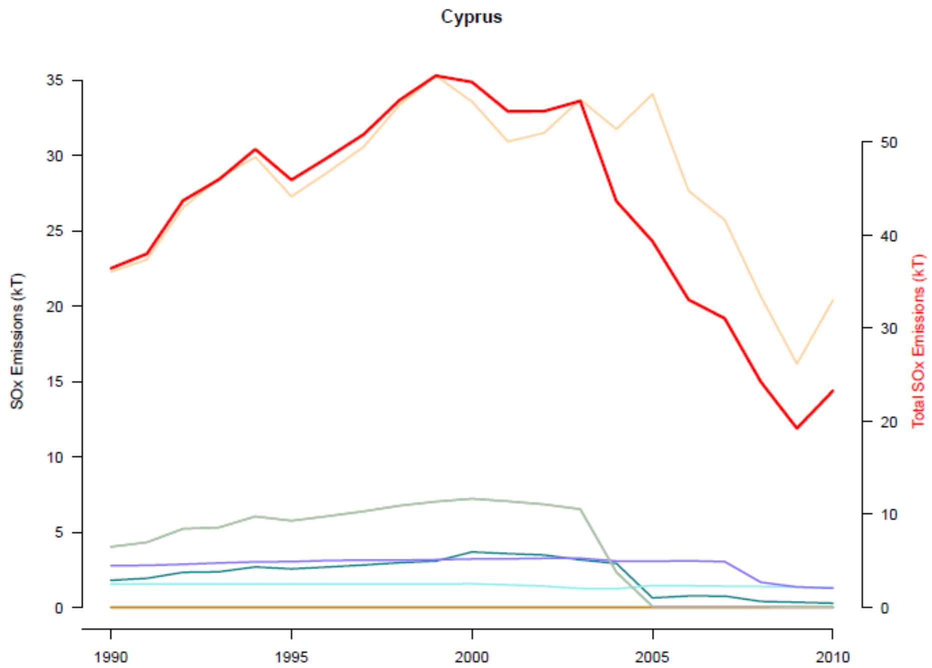


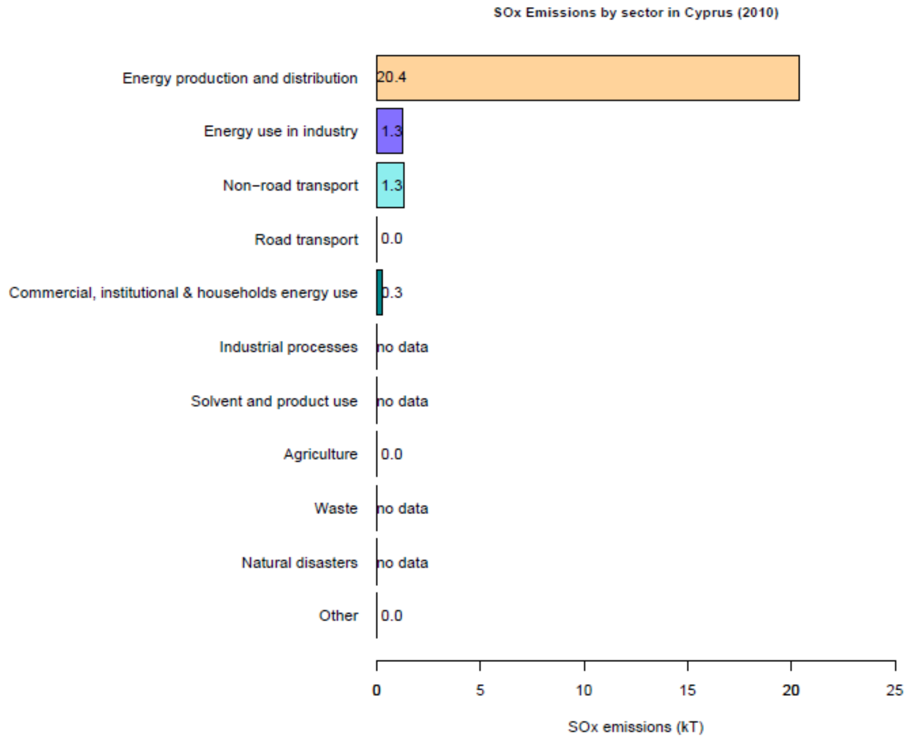
Switzerland (SO_x)



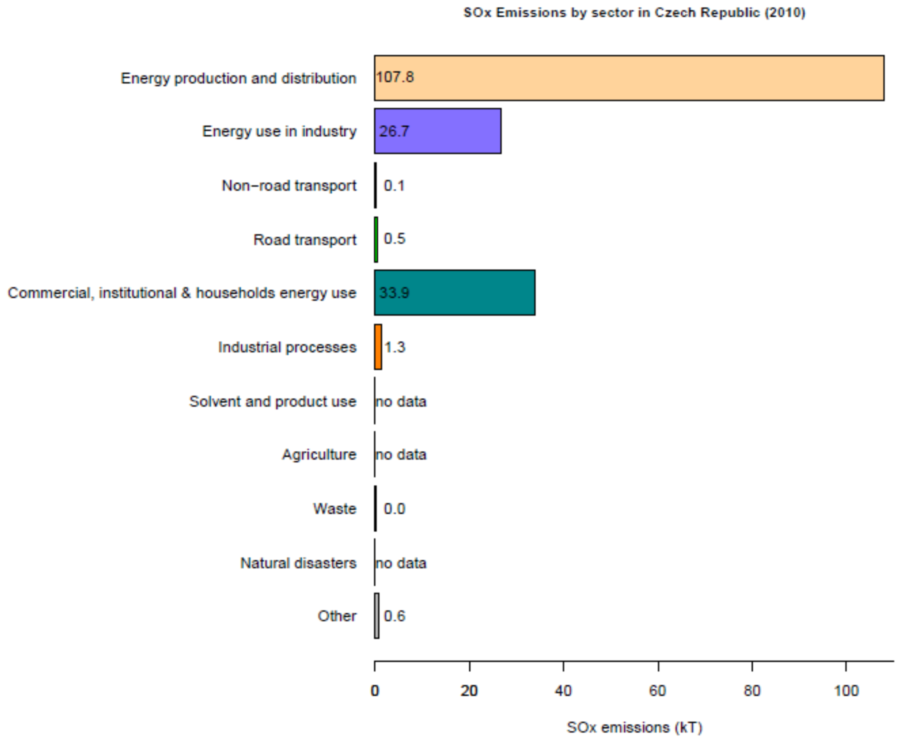


Cyprus (SO_x)

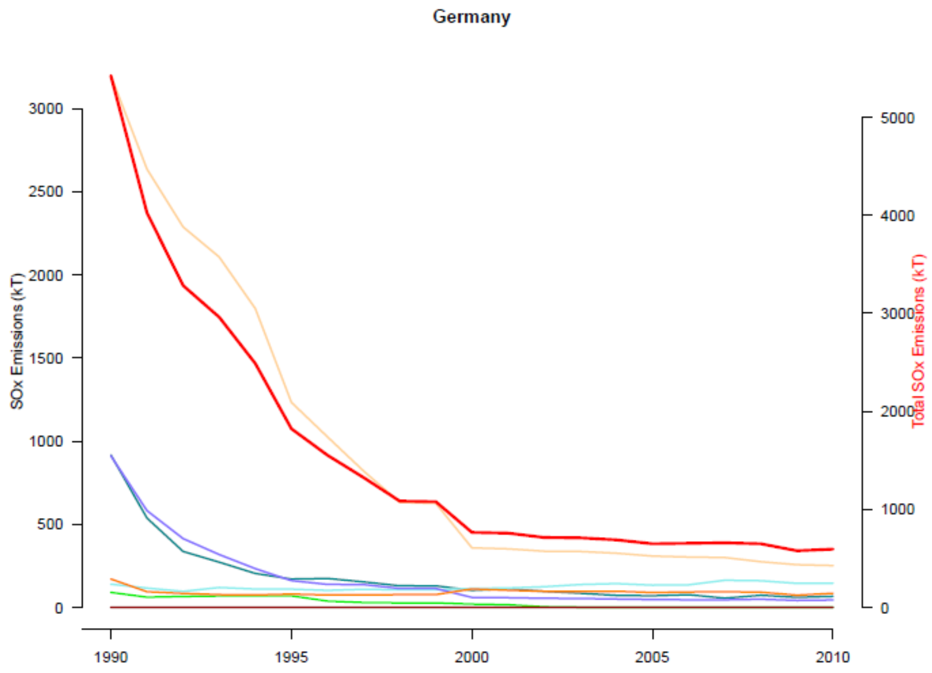

Cyprus
SOx Emissions (kT)
Total SOx Emissions (kT)
35
30
25
20
15
10
5
0
50
40
30
20
10
0
1990
1995
2000
2005
2010


SOx Emissions by sector in Cyprus (2010)
Energy production and distribution
20.4
Energy use in industry
1.3
Non−road transport
1.3
Road transport
0.0
Commercial, institutional & households energy use
0.3
Industrial processes
no data
Solvent and product use
no data
Agriculture
0.0
Waste
no data
Natural disasters
no data
Other
0.0
0
5
10
15
20
25
SOx emissions (kT)

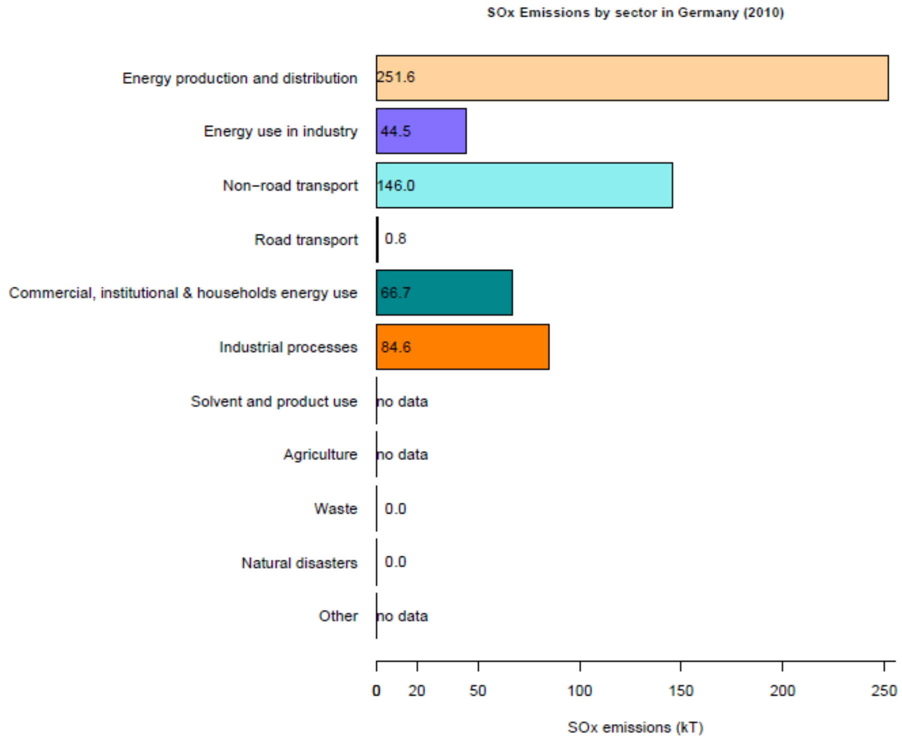
Czech Republic (SO_x)



Germany (SO_x)

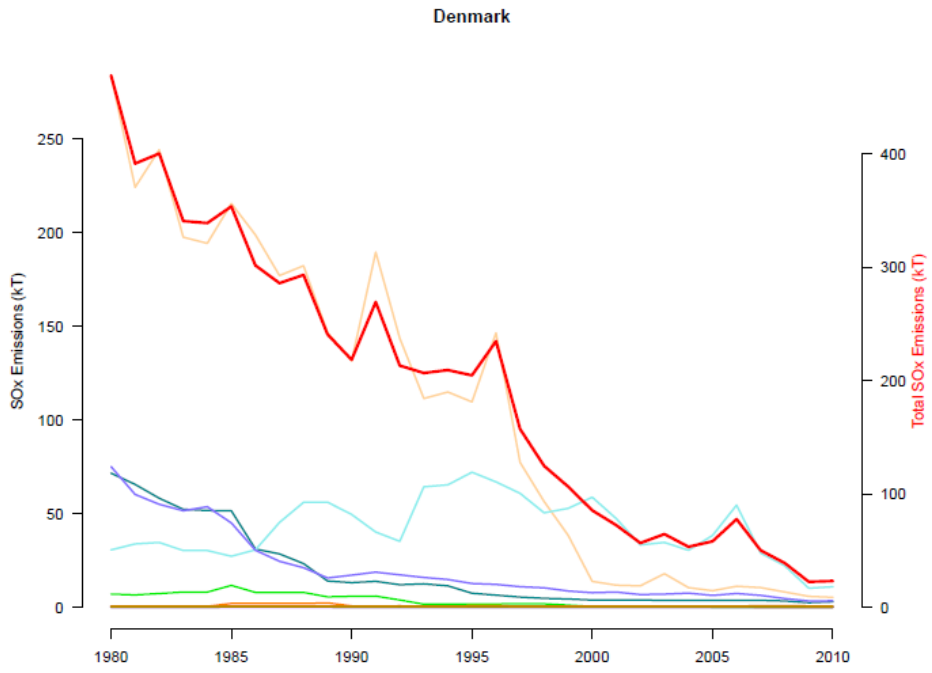


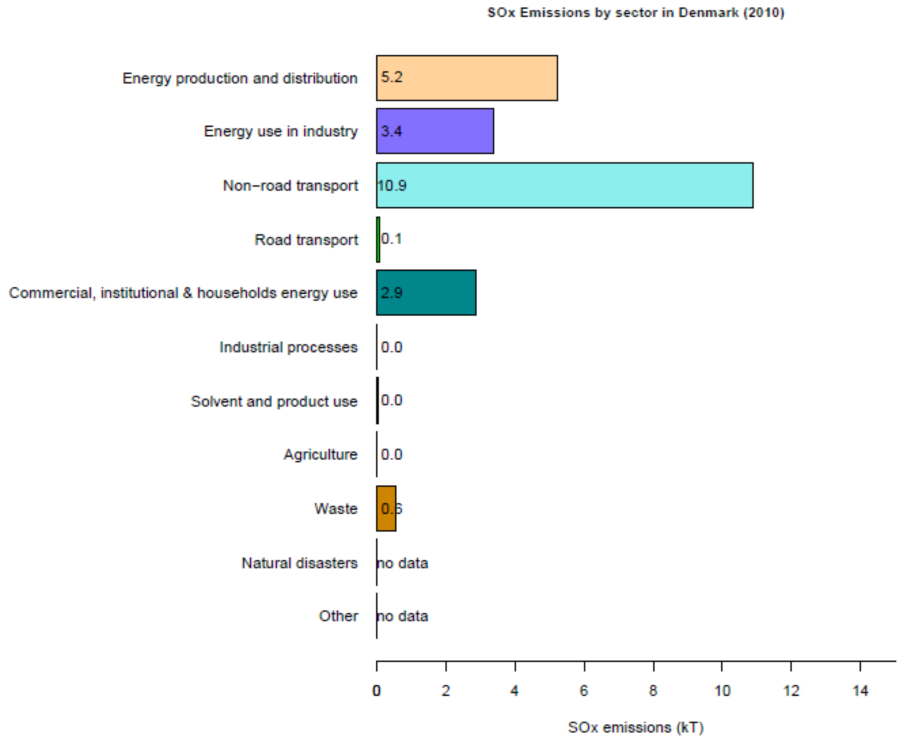
Germany
SOx Emissions (kT)
Total SOx Emissions (kT)
3000
2500
2000
1500
1000
500
0
5000
4000
3000
2000
1000
0
1990
1995
2000
2005
2010



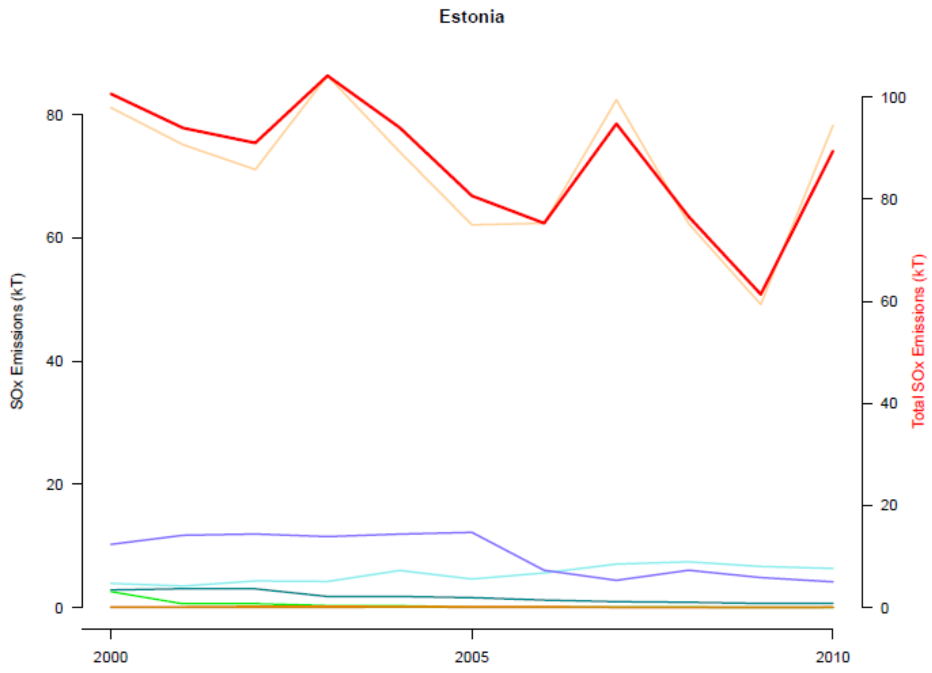
SOx Emissions by sector in Germany (2010)
Energy production and distribution
251.6
Energy use in industry
44.5
Non−road transport
146.0
Road transport
0.8
Commercial, institutional & households energy use
66.7
Industrial processes
84.6
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
0.0
Other
no data
0
20
50
100
150
200
250
SOx emissions (kT)

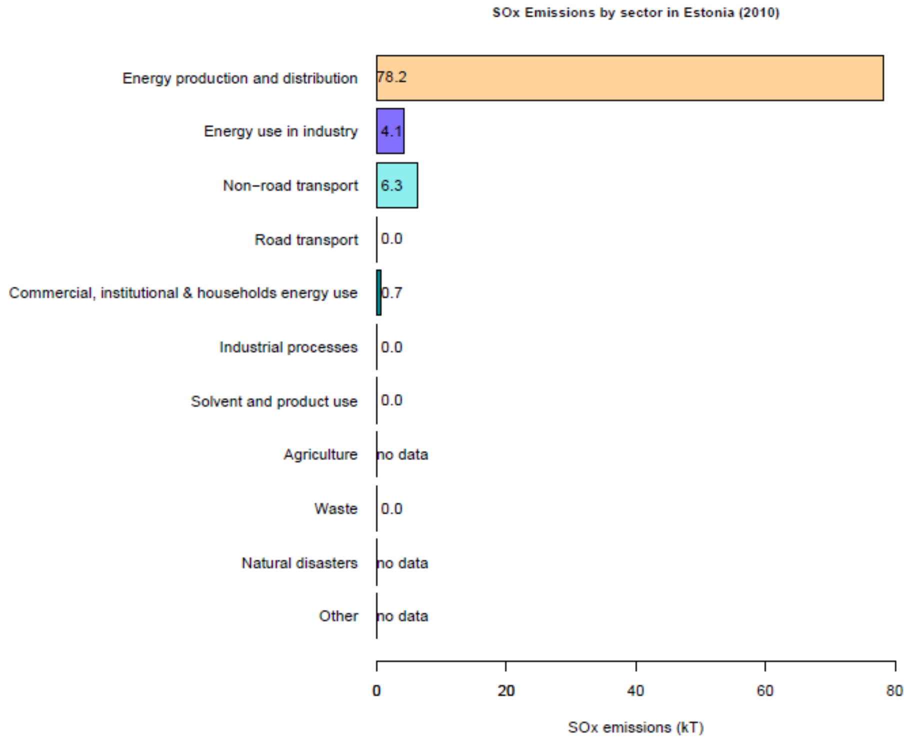
Denmark (SO_x)


Denmark
SOx Emissions (kT)
Total SOx Emissions (kT)
0
50
100
150
200
250
0
100
200
300
400
1980
1985
1990
1995
2000
2005
2010

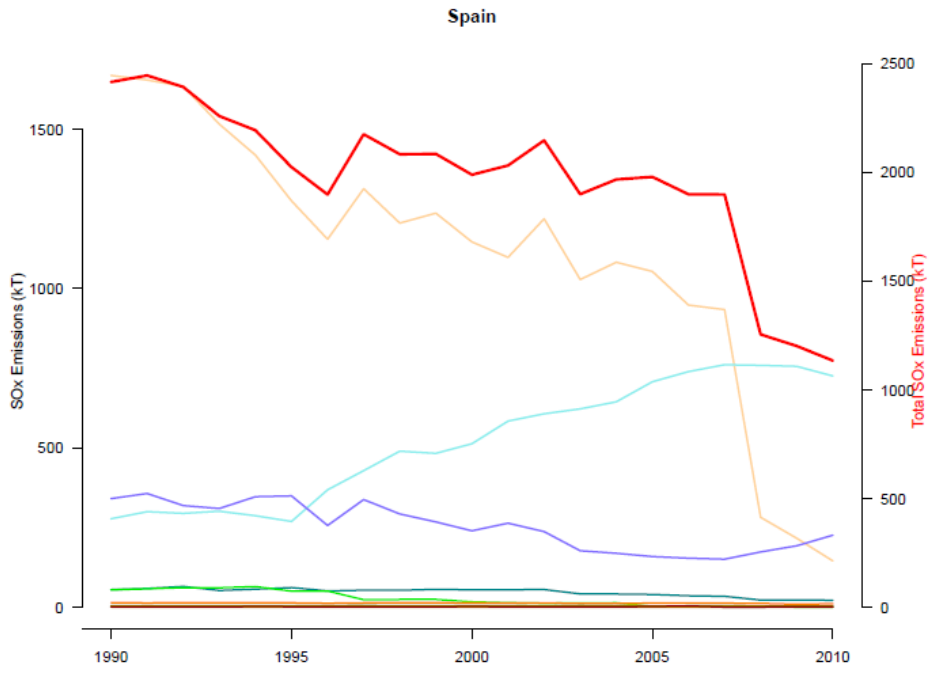

SOx Emissions by sector in Denmark (2010)
Energy production and distribution
5.2
Energy use in industry
3.4
Non−road transport
10.9
Road transport
0.1
Commercial, institutional & households energy use
2.9
Industrial processes
0.0
Solvent and product use
0.0
Agriculture
0.0
Waste
0.6
Natural disasters
no data
Other
no data
0
2
4
6
8
10
12
14
SOx emissions (kT)

Estonia (SO_x)

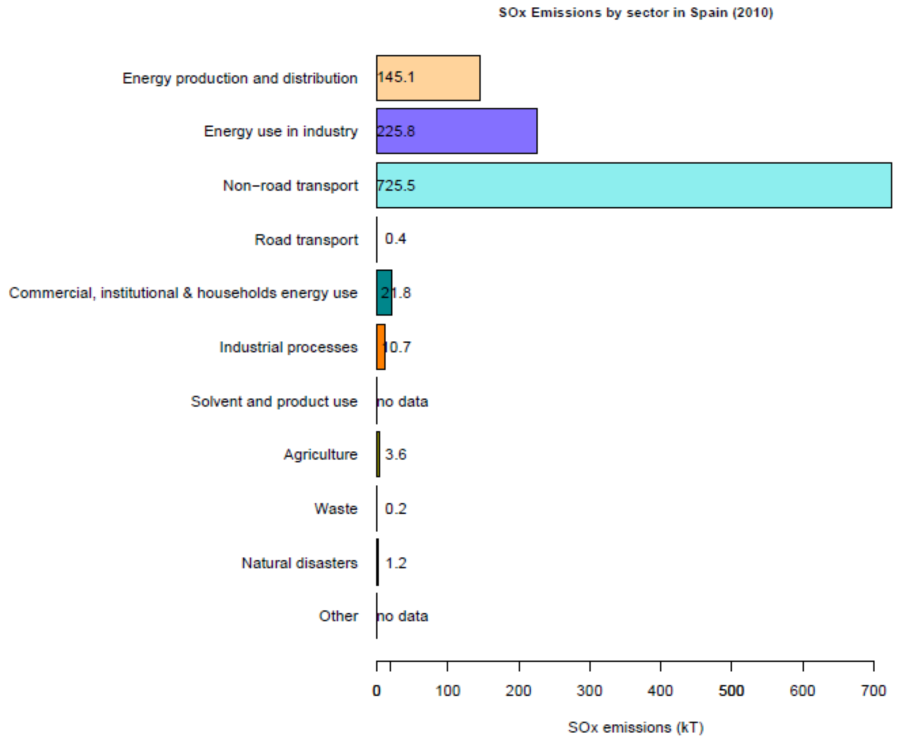

Estonia
SOx Emissions (kT)
Total SOx Emissions (kT)
0
20
40
60
80
100
2000
2005
2010


SOx Emissions by sector in Estonia (2010)
Energy production and distribution
78.2
Energy use in industry
4.1
Non−road transport
6.3
Road transport
0.0
Commercial, institutional & households energy use
0.7
Industrial processes
0.0
Solvent and product use
0.0
Agriculture
no data
Waste
0.0
Natural disasters
no data
Other
no data
0
20
40
60
80
SOx emissions (kT)

Spain (SO_x)

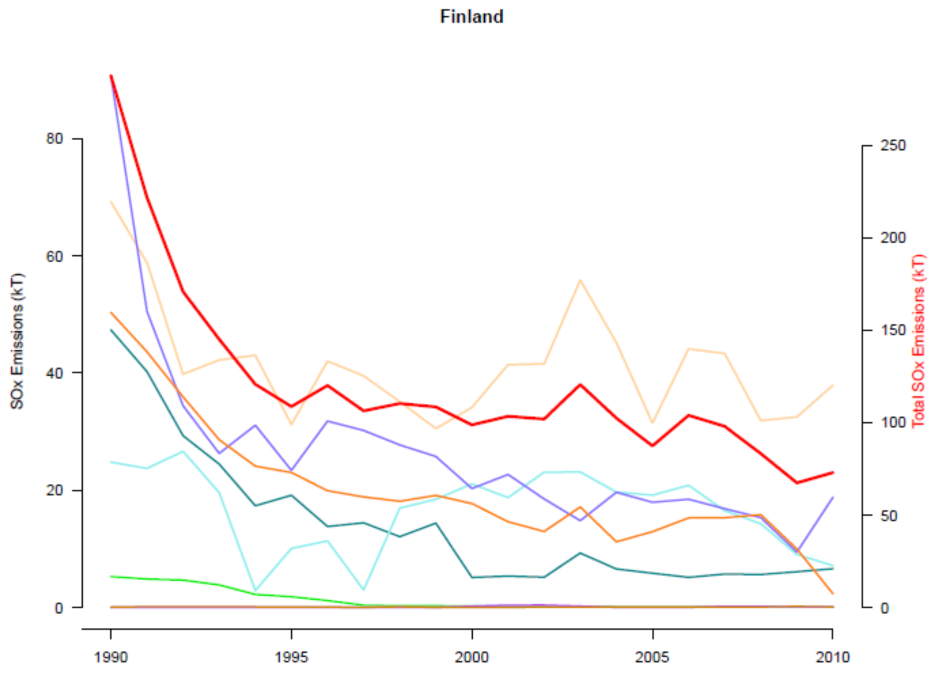


Spain
SOx Emissions (kT)
Total SOx Emissions (kT)
0
500
1000
1500
2000
2500
1990
1995
2000
2005
2010

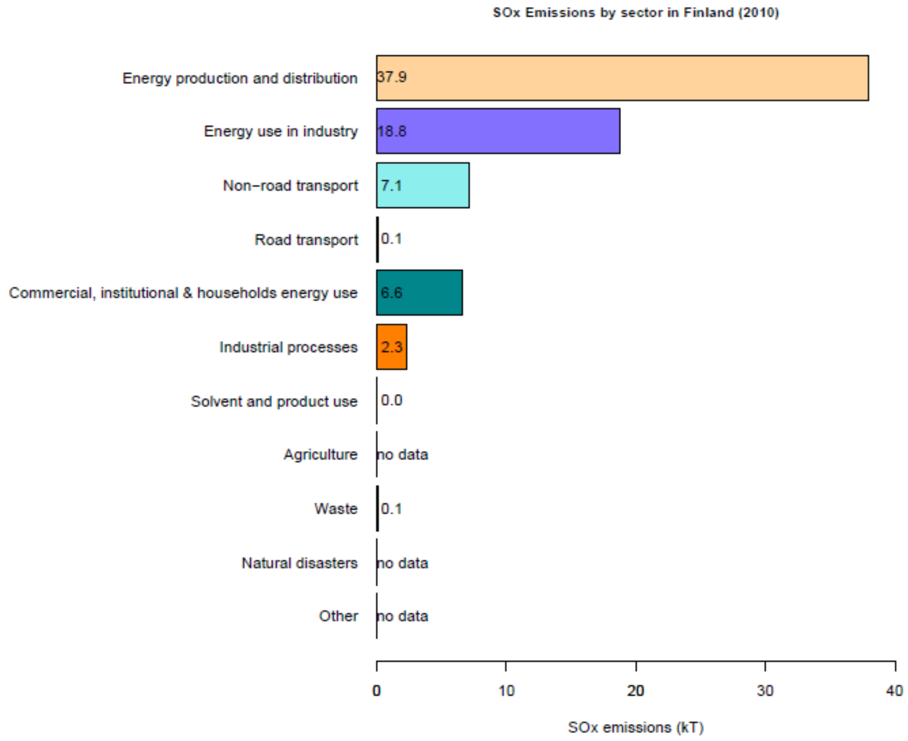


SOx Emissions by sector in Spain (2010)
Energy production and distribution
145.1
Energy use in industry
225.8
Non−road transport
725.5
Road transport
0.4
Commercial, institutional & households energy use
21.8
Industrial processes
10.7
Solvent and product use
no data
Agriculture
3.6
Waste
0.2
Natural disasters
1.2
Other
no data
0
100
200
300
400
500
600
700
SOx emissions (kT)

Finland (SO_x)

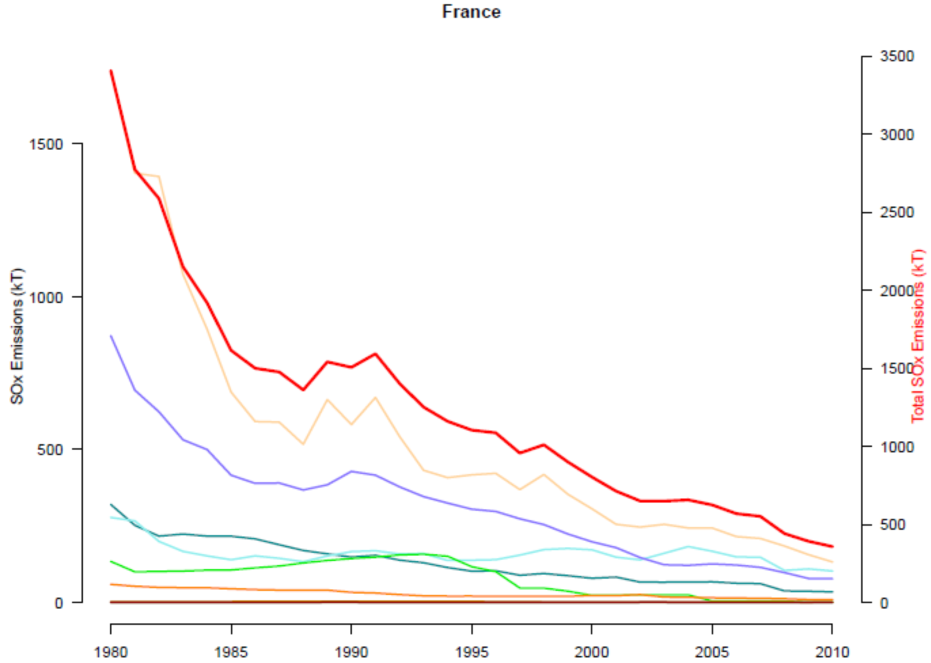


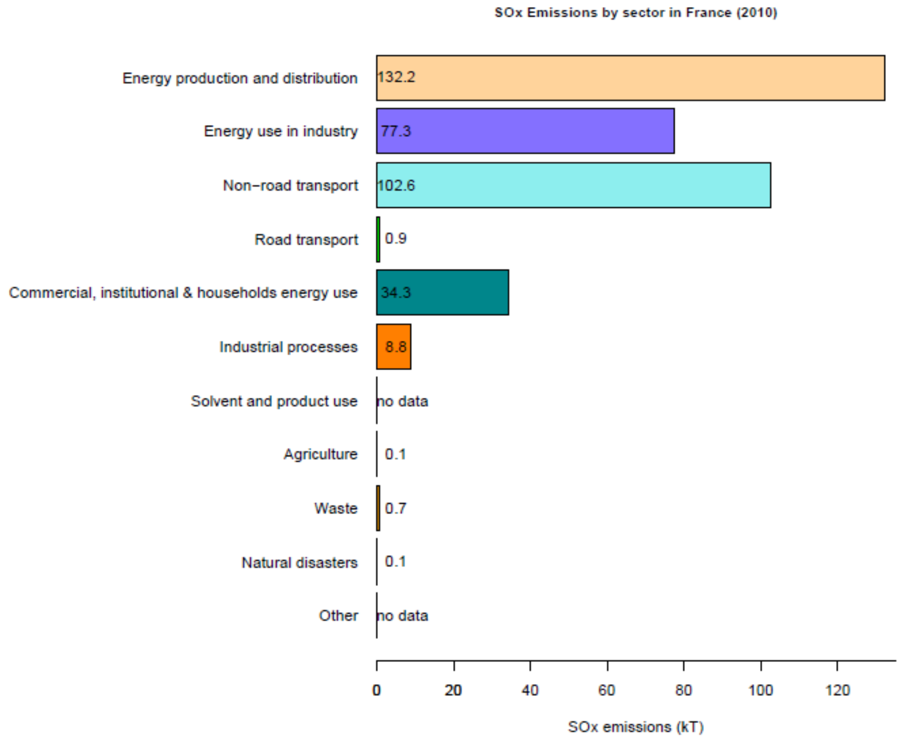
Finland
SOx Emissions (kT)
Total SOx Emissions (kT)
0
20
40
60
80
0
50
100
150
200
250
1990
1995
2000
2005
2010



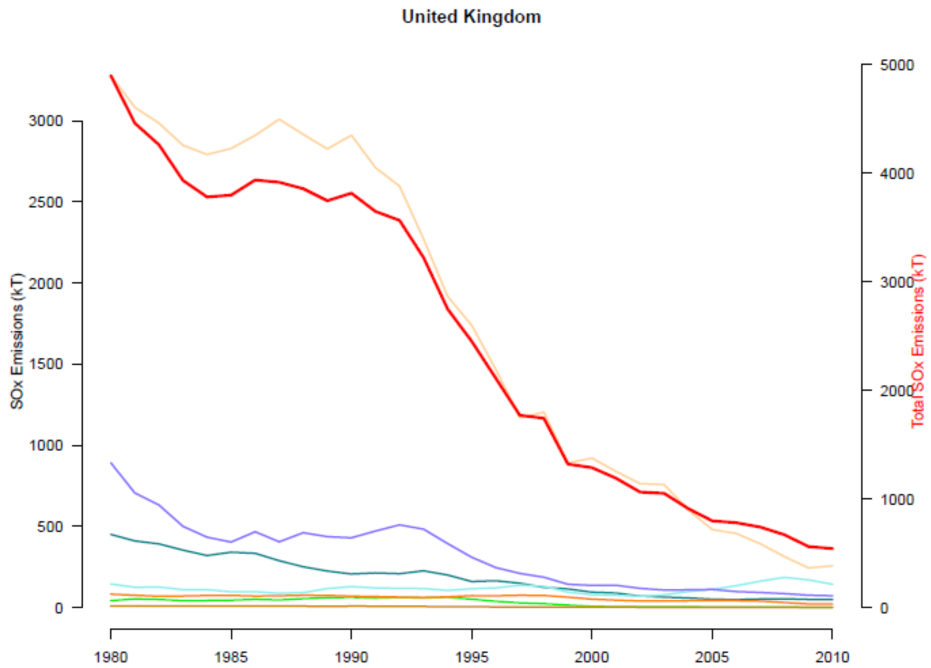
SOx Emissions by sector in Finland (2010)
Energy production and distribution
37.9
Energy use in industry
18.8
Non−road transport
7.1
Road transport
0.1
Commercial, institutional & households energy use
6.6
Industrial processes
2.3
Solvent and product use
0.0
Agriculture
no data
Waste
0.1
Natural disasters
no data
Other
no data
0
10
20
30
40
SOx emissions (kT)

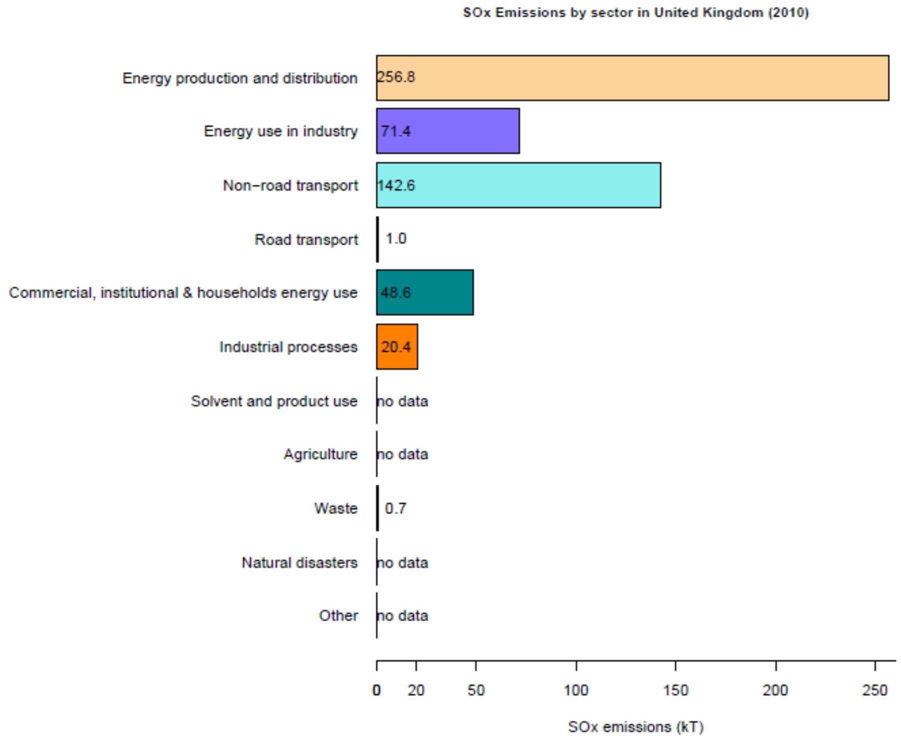
France (SO_x)


France
SOx Emissions (kT)
Total SOx Emissions (kT)
0
500
1000
1500
0
500
1000
1500
2000
2500
3000
3500
1980
1985
1990
1995
2000
2005
2010

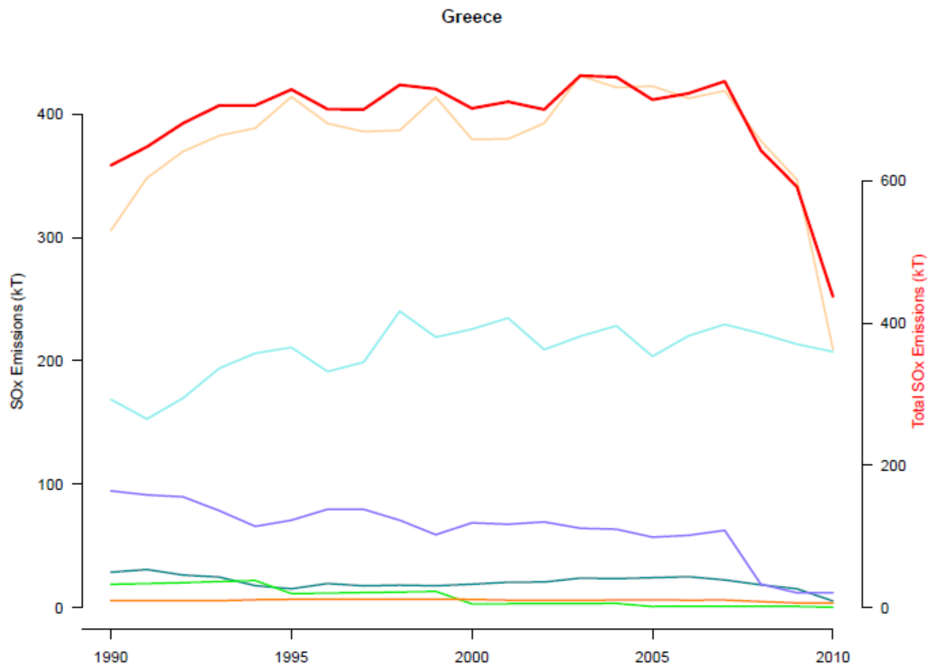

SOx Emissions by sector in France (2010)
Energy production and distribution
132.2
Energy use in industry
77.3
Non−road transport
102.6
Road transport
0.9
Commercial, institutional & households energy use
34.3
Industrial processes
8.8
Solvent and product use
no data
Agriculture
0.1
Waste
0.7
Natural disasters
0.1
Other
no data
0
20
40
60
80
100
120
SOx emissions (kT)

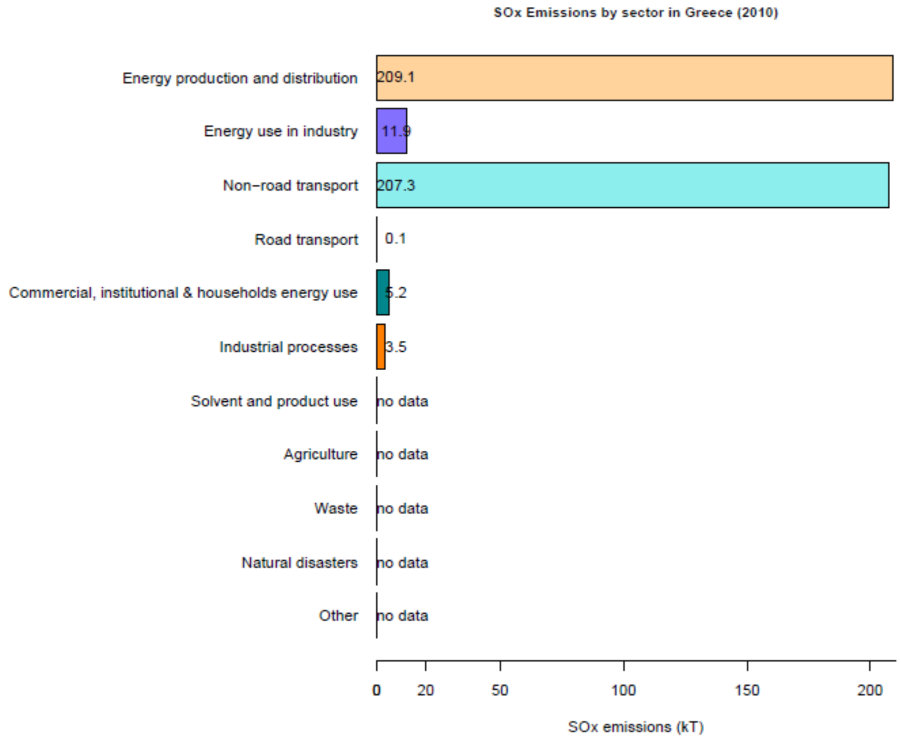
The United Kingdom (SO_x)



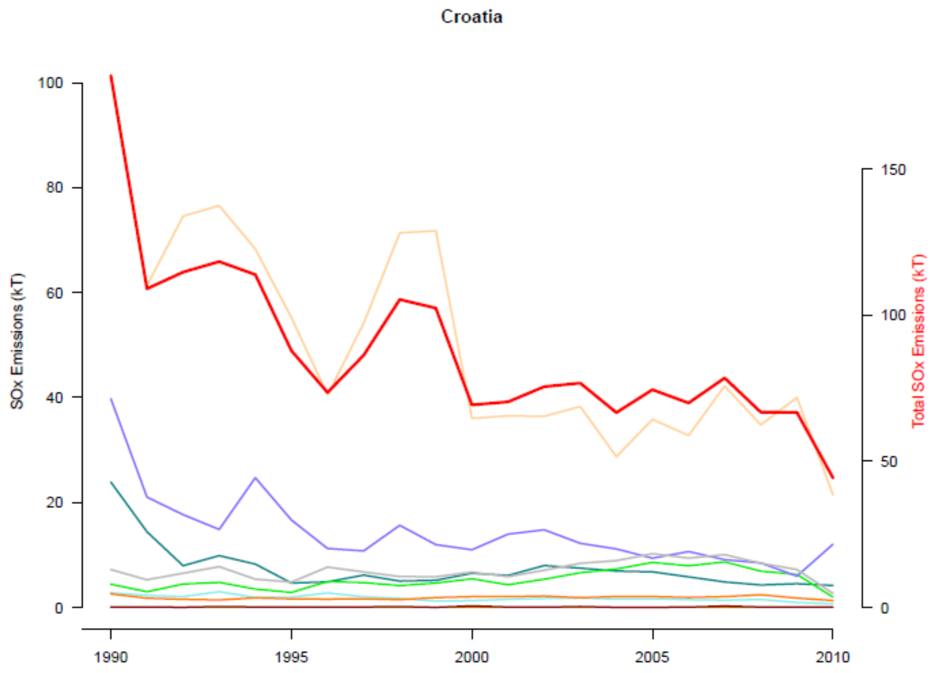


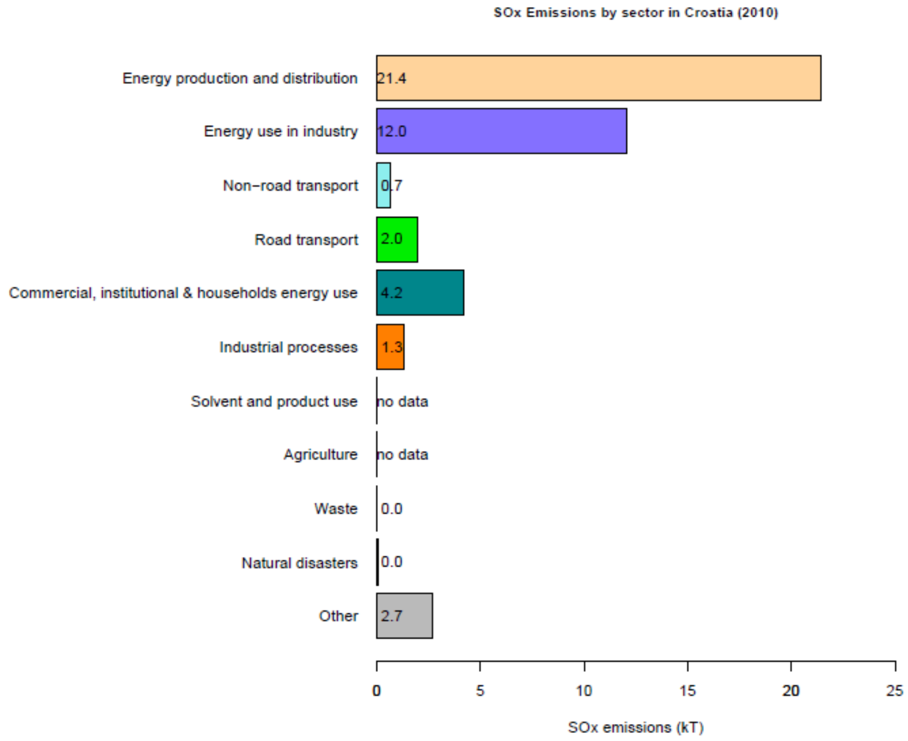
Greece (SO_x)


Greece
SOx Emissions (kT)
Total SOx Emissions (kT)
0
100
200
300
400
600
1990
1995
2000
2005
2010

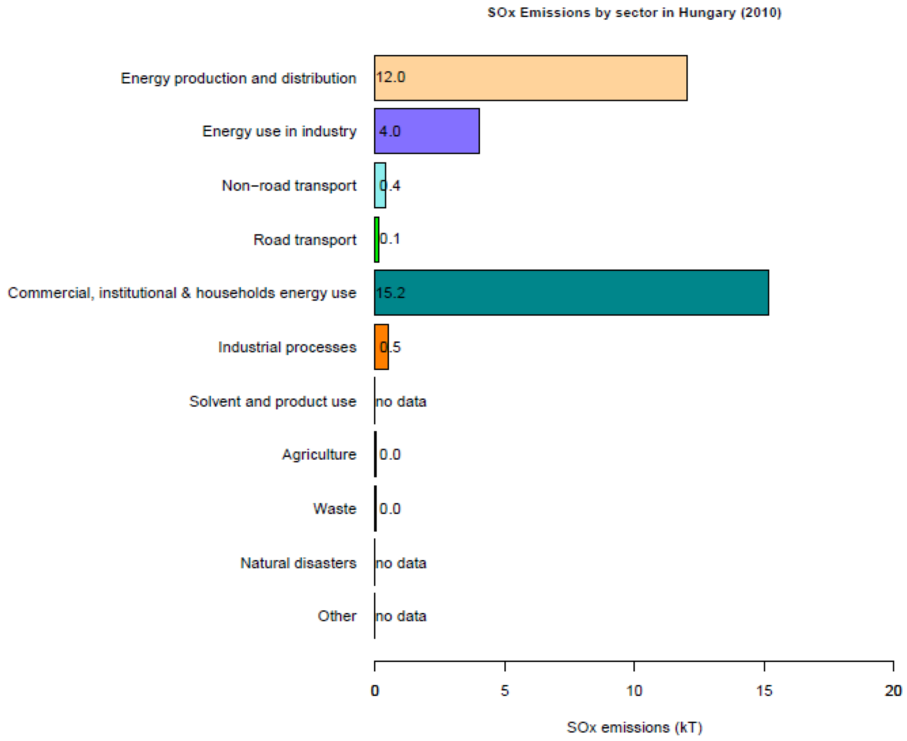

SOx Emissions by sector in Greece (2010)
Energy production and distribution
209.1
Energy use in industry
11.9
Non-road transport
207.3
Road transport
0.1
Commercial, institutional & households energy use
5.2
Industrial processes
3.5
Solvent and product use
no data
Agriculture
no data
Waste
no data
Natural disasters
no data
Other
no data
0
20
50
100
150
200
SOx emissions (kT)

Croatia (SO_x)

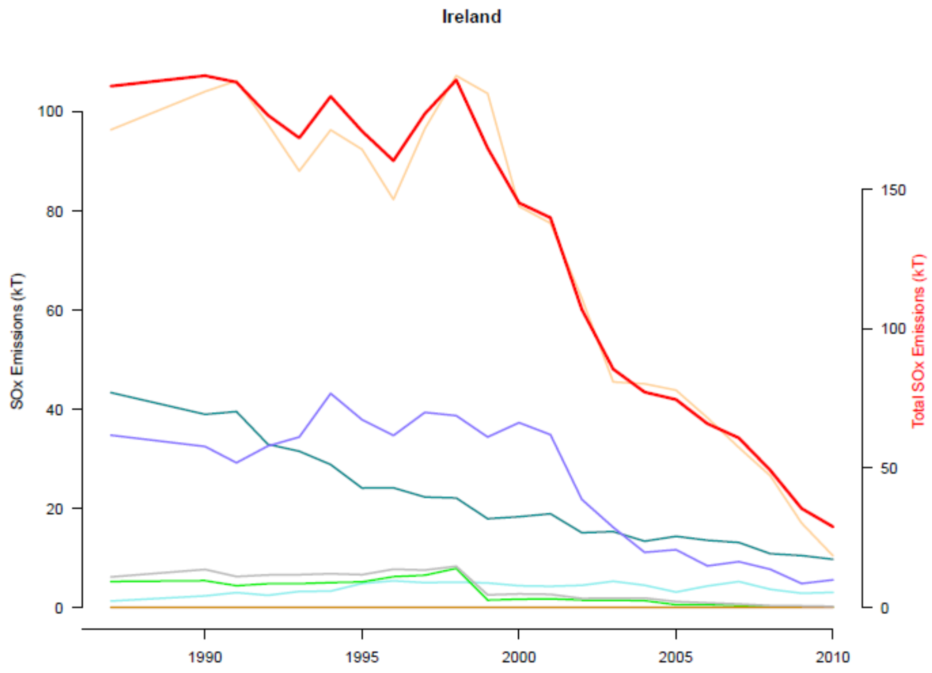

Croatia
SOx Emissions (kT)
Total SOx Emissions (kT)
0
20
40
60
80
100
0
50
100
150
1990
1995
2000
2005
2010


SOx Emissions by sector in Croatia (2010)
Energy production and distribution
21.4
Energy use in industry
12.0
Non−road transport
0.7
Road transport
2.0
Commercial, institutional & households energy use
4.2
Industrial processes
1.3
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
0.0
Other
2.7
0
5
10
15
20
25
SOx emissions (kT)

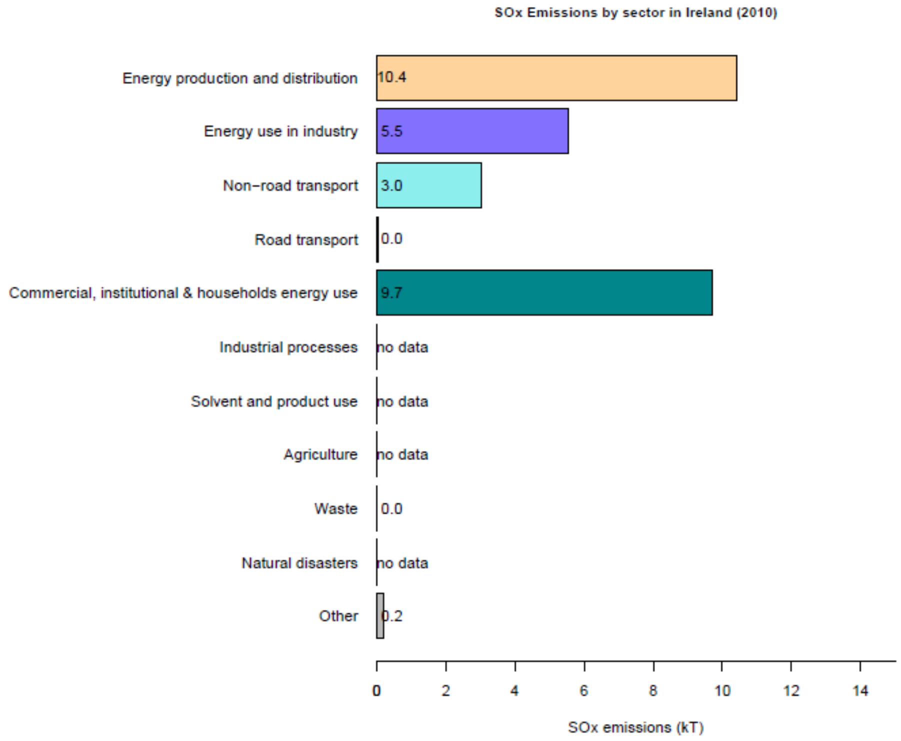
Hungary (SO_x)


SOx Emissions by sector in Hungary (2010)
Energy production and distribution
Energy use in industry
Non−road transport
Road transport
Commercial, institutional & households energy use
Industrial processes
Solvent and product use
Agriculture
Waste
Natural disasters
Other
12.0
4.0
0.4
0.1
15.2
0.5
no data
0.0
0.0
no data
no data
0
5
10
15
20
SOx emissions (kT)

Ireland (SO_x)



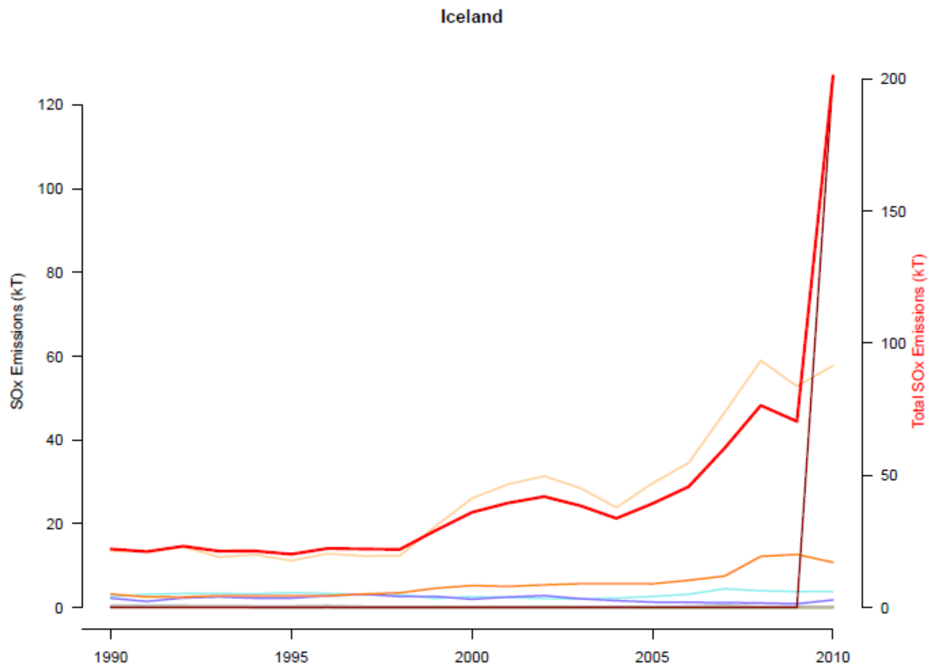
Ireland
SOx Emissions (kT)
Total SOx Emissions (kT)
0
20
40
60
80
100
0
50
100
150
1990
1995
2000
2005
2010

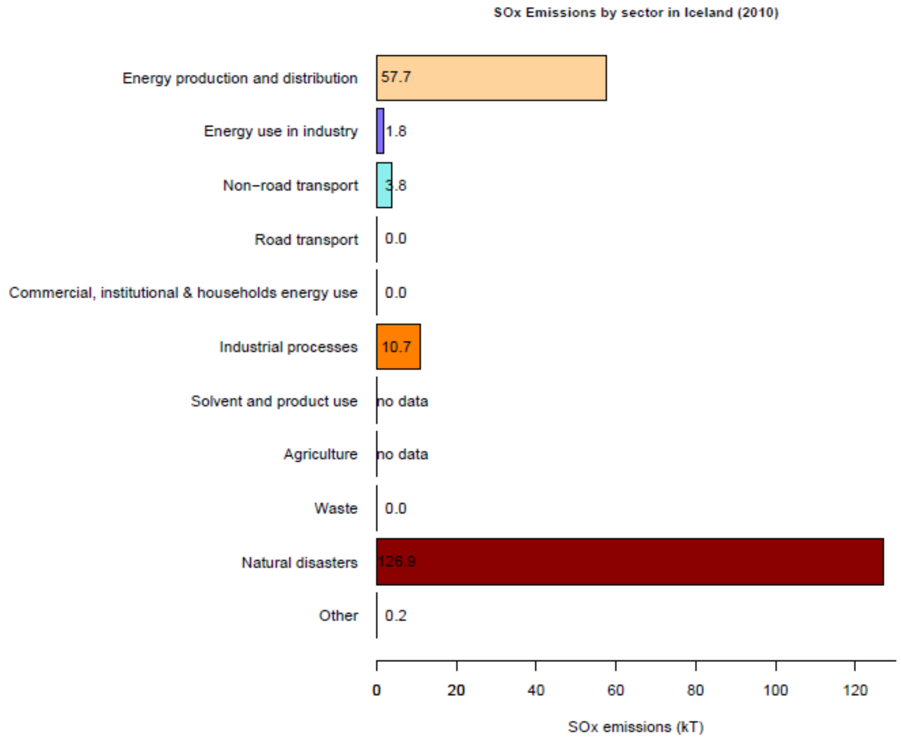


SOx Emissions by sector in Ireland (2010)
Energy production and distribution
10.4
Energy use in industry
5.5
Non−road transport
3.0
Road transport
0.0
Commercial, institutional & households energy use
9.7
Industrial processes
no data
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
no data
Other
0.2
0
2
4
6
8
10
12
14
SOx emissions (kT)

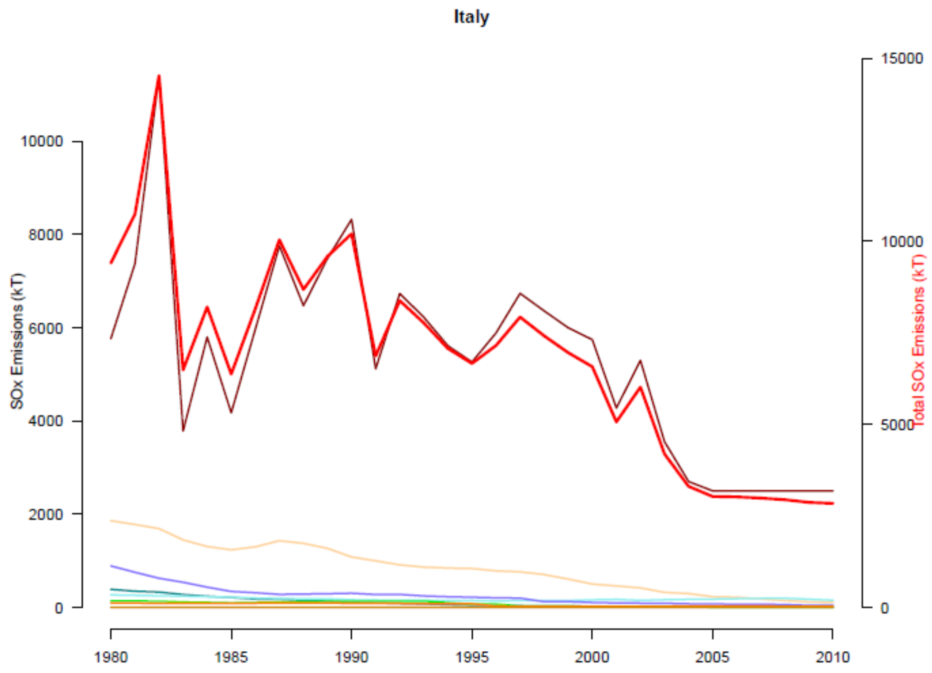
International Prevention Research Institute

Iceland (SO_x)

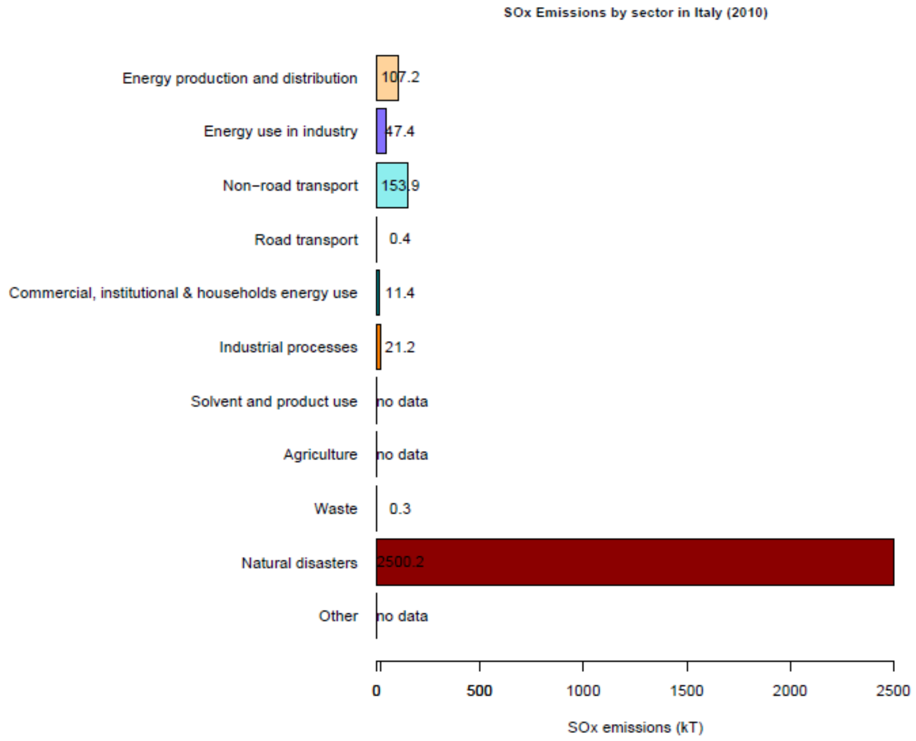




Italy (SO_x)

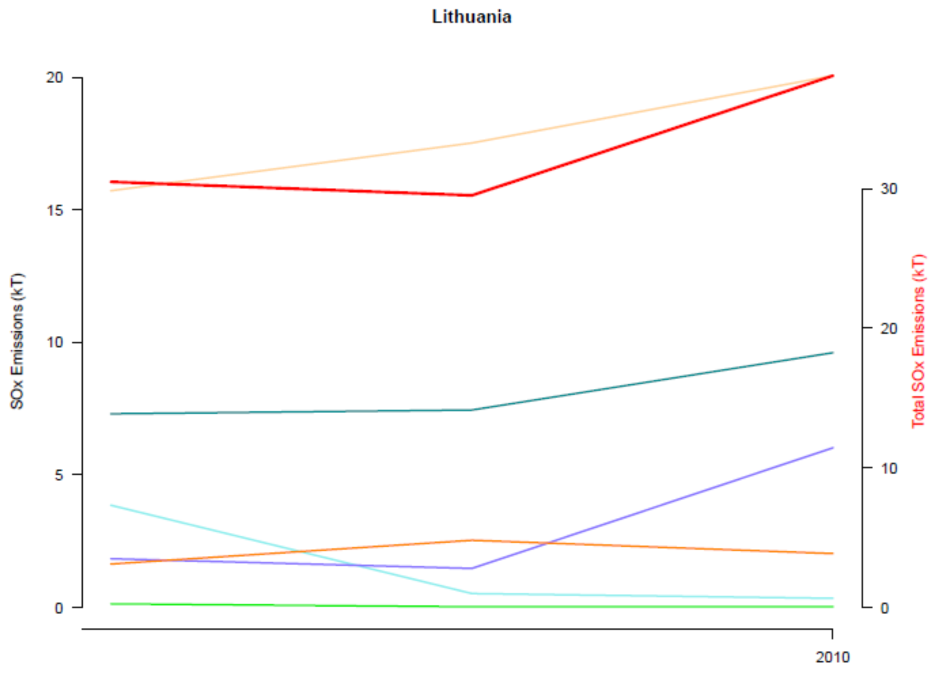


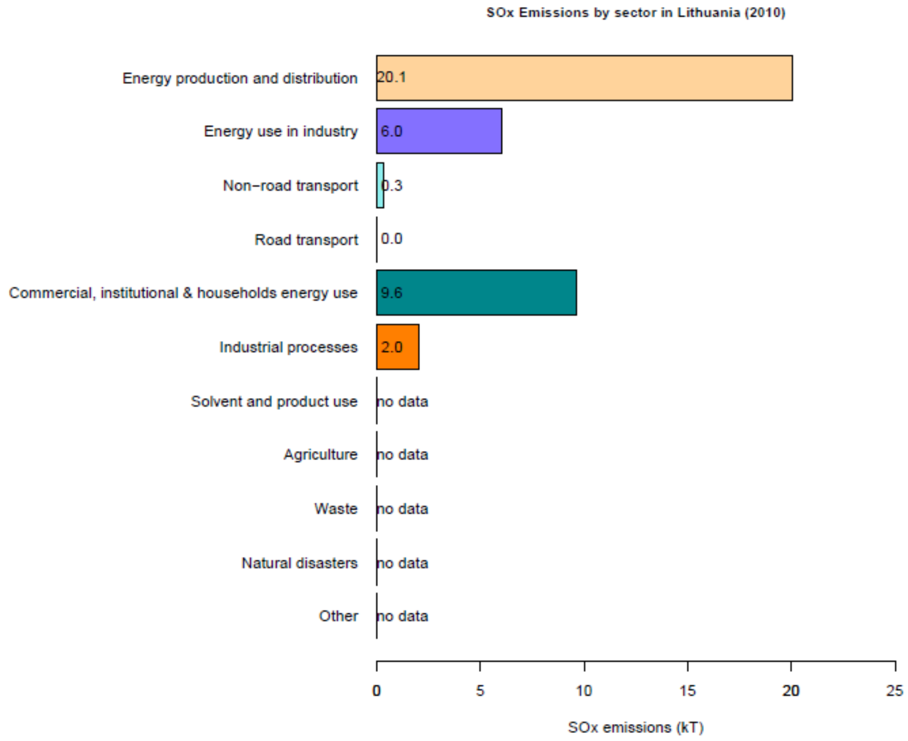
Italy
SOx Emissions (kT)
Total SOx Emissions (kT)
10000
8000
6000
4000
2000
0
15000
10000
5000
0
1980
1985
1990
1995
2000
2005
2010



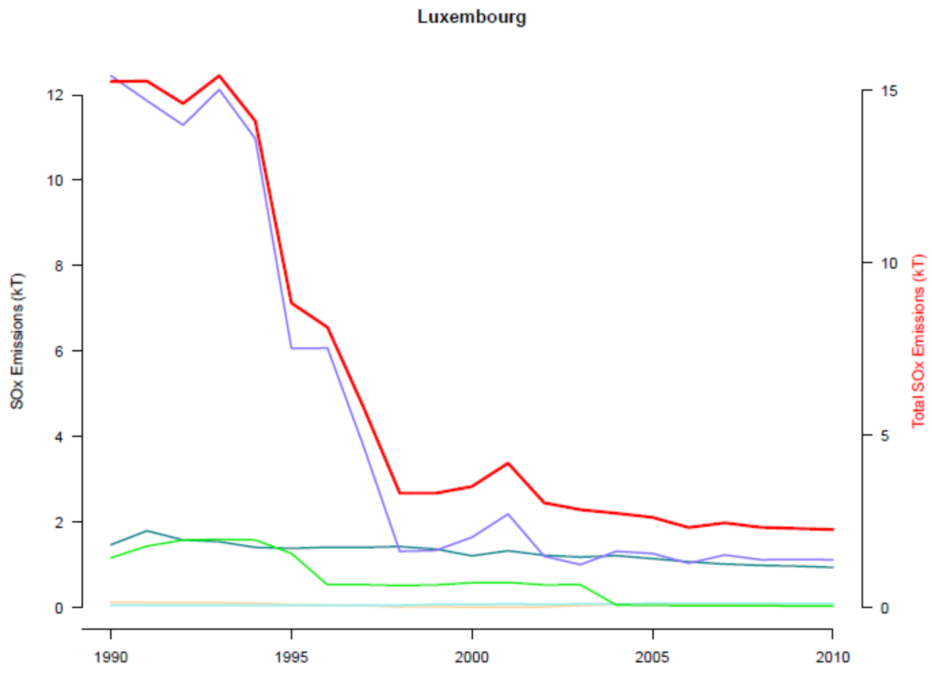
SOx Emissions by sector in Italy (2010)
Energy production and distribution
107.2
Energy use in industry
47.4
Non−road transport
153.9
Road transport
0.4
Commercial, institutional & households energy use
11.4
Industrial processes
21.2
Solvent and product use
no data
Agriculture
no data
Waste
0.3
Natural disasters
2500.2
Other
no data
0
500
1000
1500
2000
2500
SOx emissions (kT)

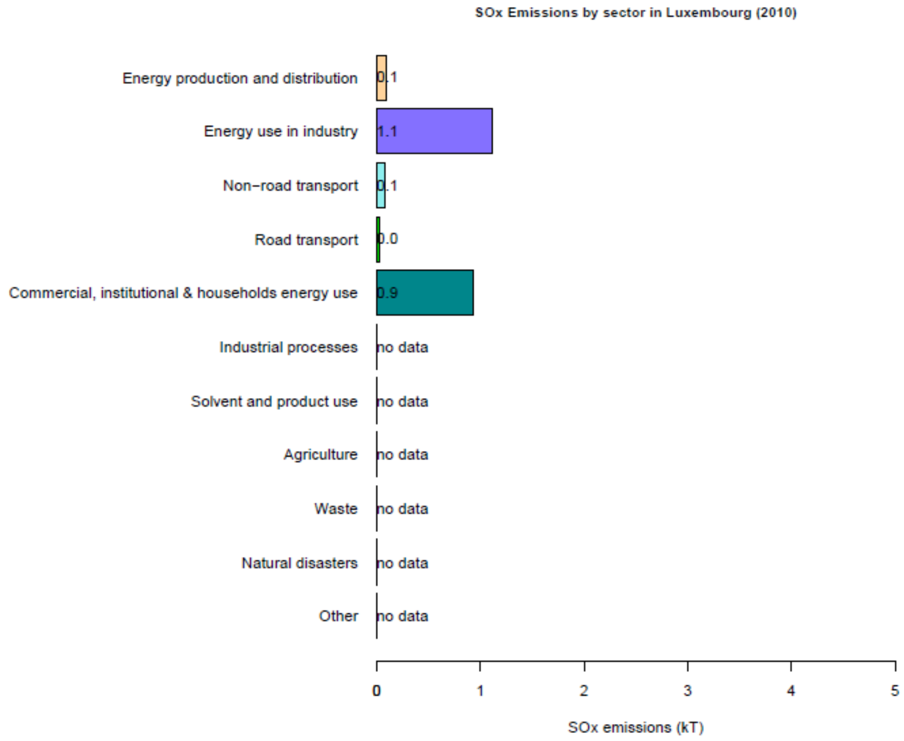
Lithuania (SO_x)



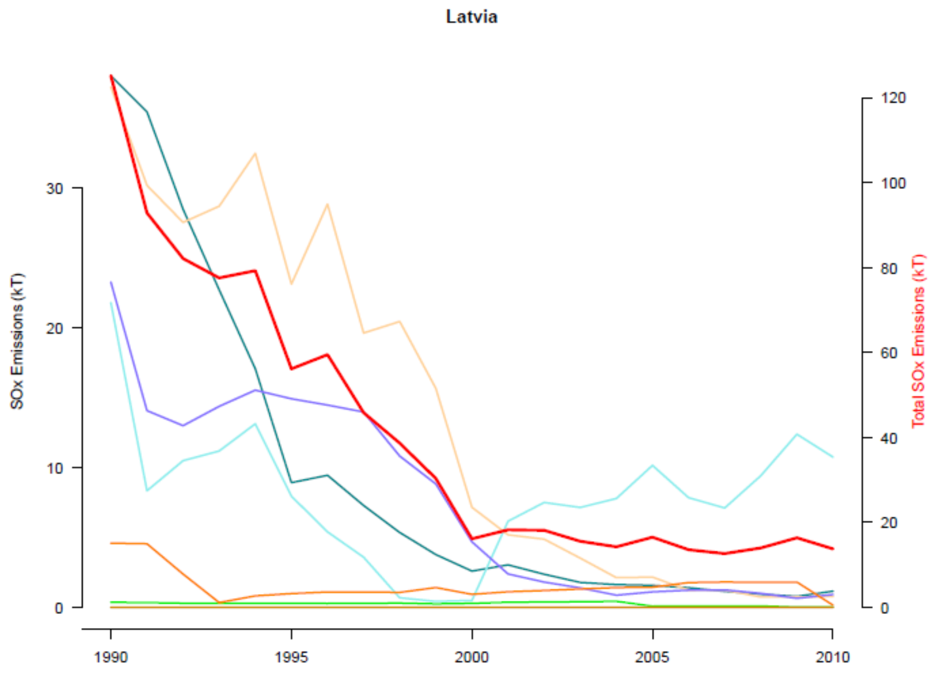


Luxembourg (SO_x)

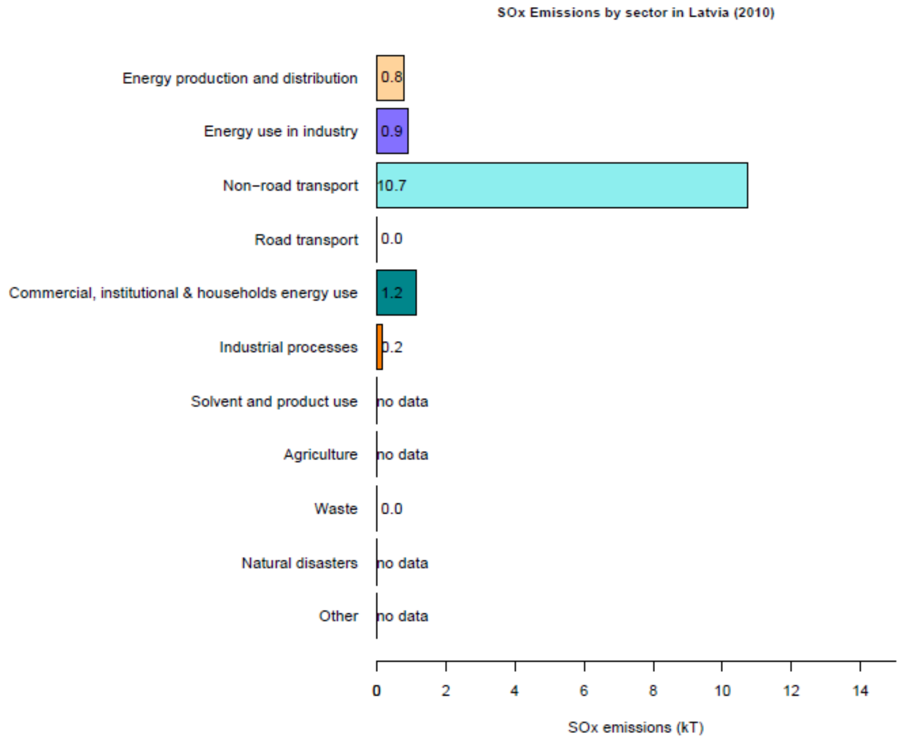




Latvia (SO_x)

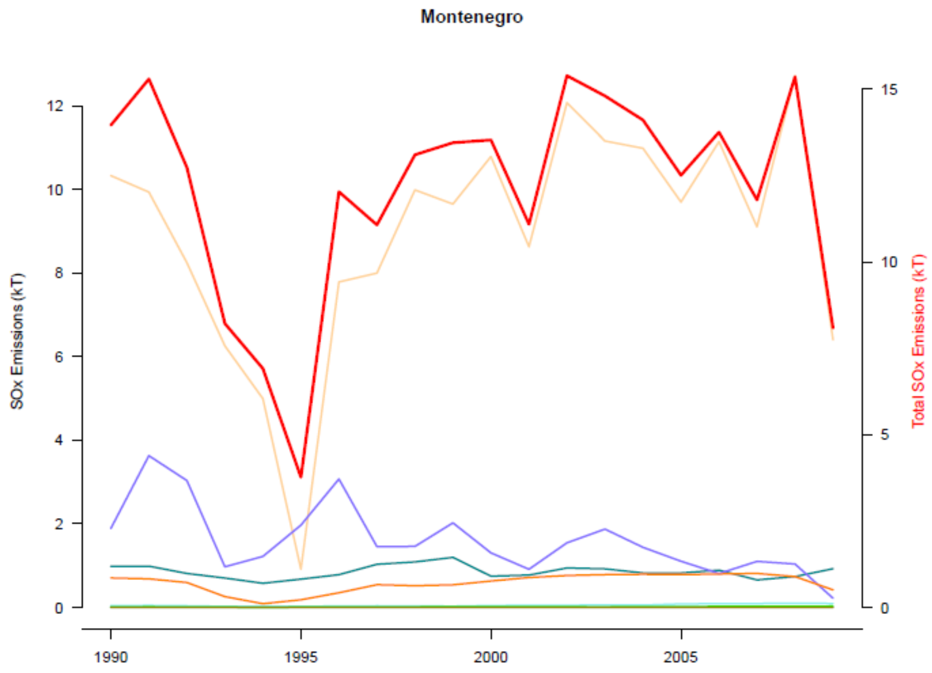


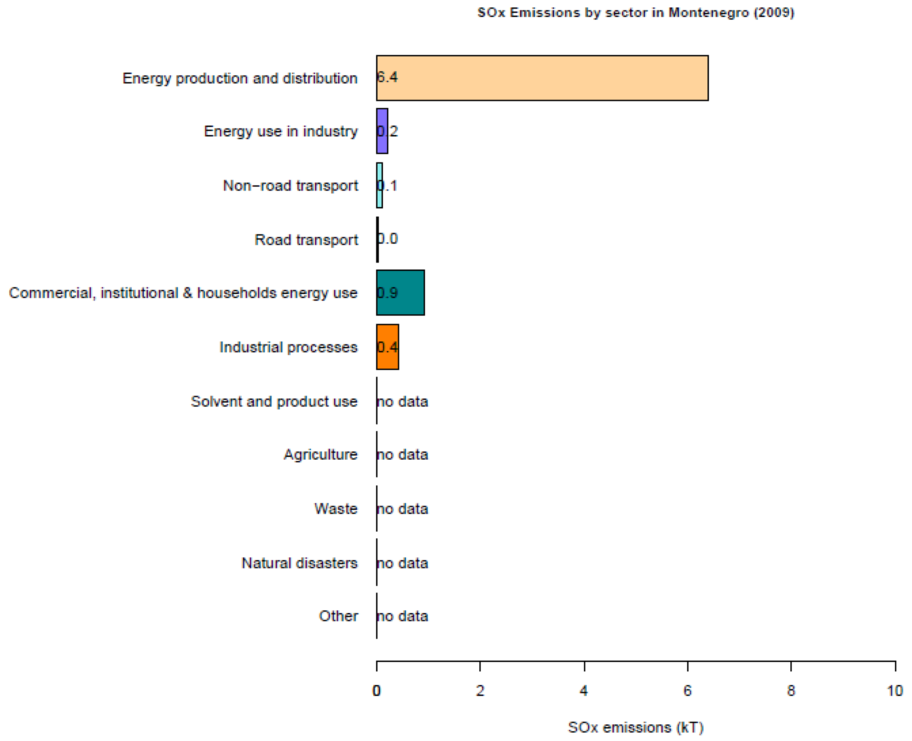
Latvia
SOx Emissions (kT)
Total SOx Emissions (kT)
30
20
10
0
120
100
80
60
40
20
0
1990
1995
2000
2005
2010



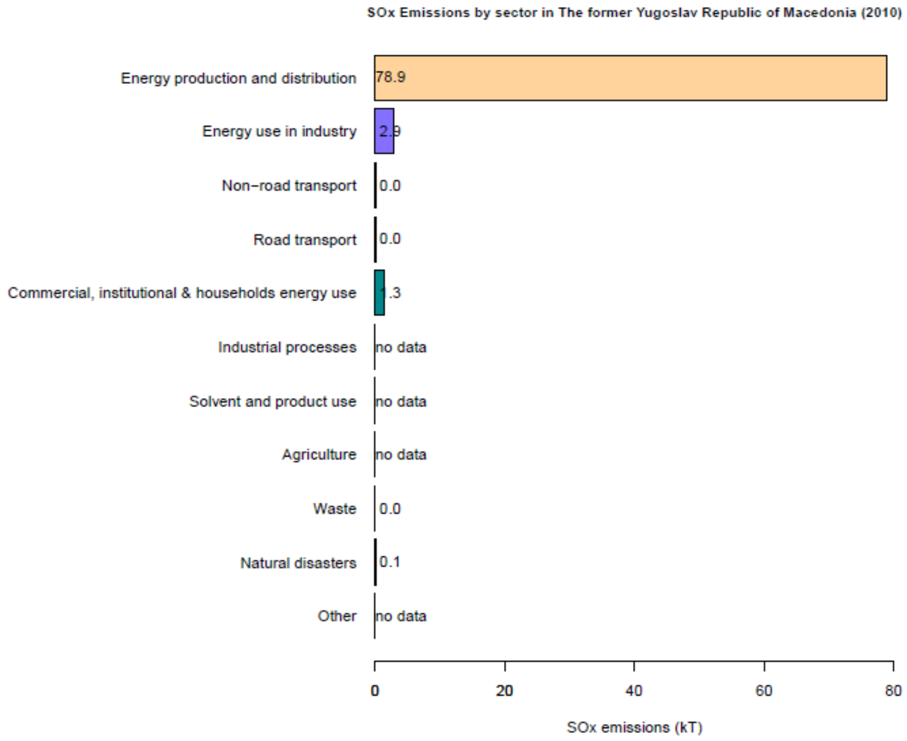
SOx Emissions by sector in Latvia (2010)
Energy production and distribution
0.8
Energy use in industry
0.9
Non−road transport
10.7
Road transport
0.0
Commercial, institutional & households energy use
1.2
Industrial processes
0.2
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
no data
Other
no data
0
2
4
6
8
10
12
14
SOx emissions (kT)

Montenegro (SO_x)

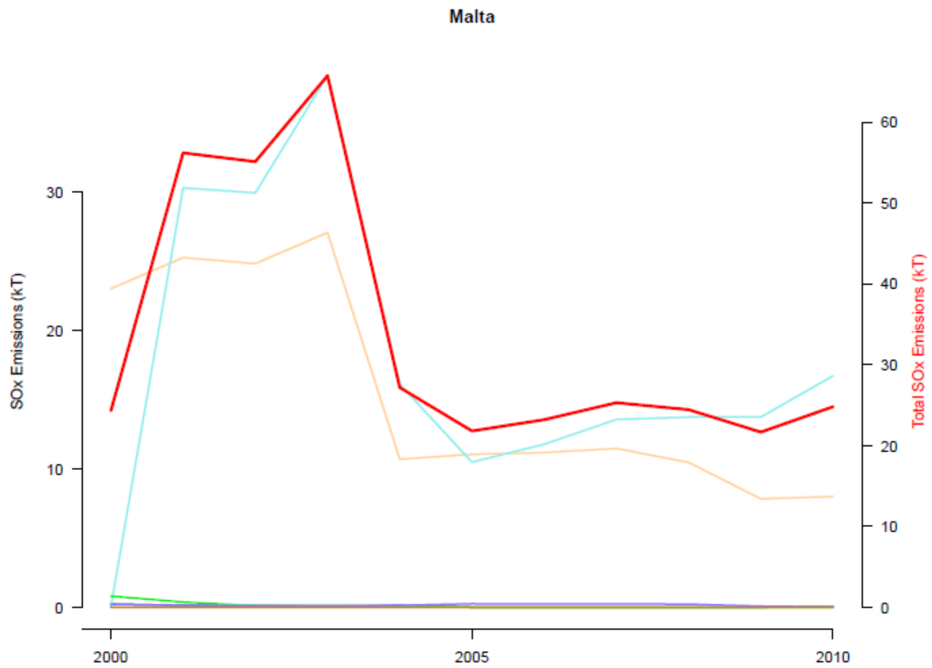


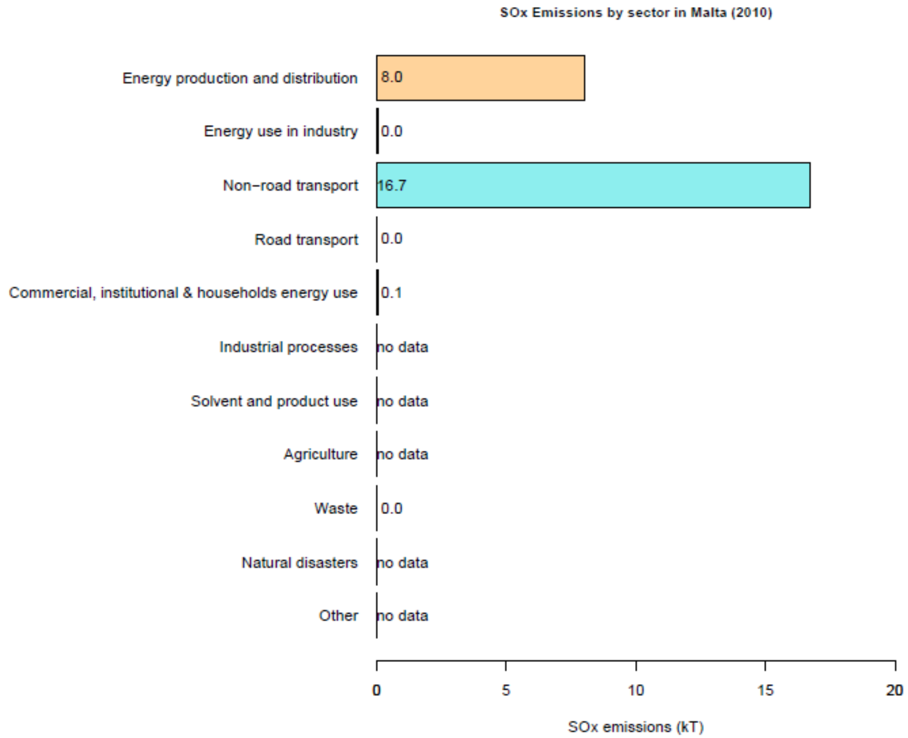


The Former Yugoslav Republic of Macedonia (SO_x)



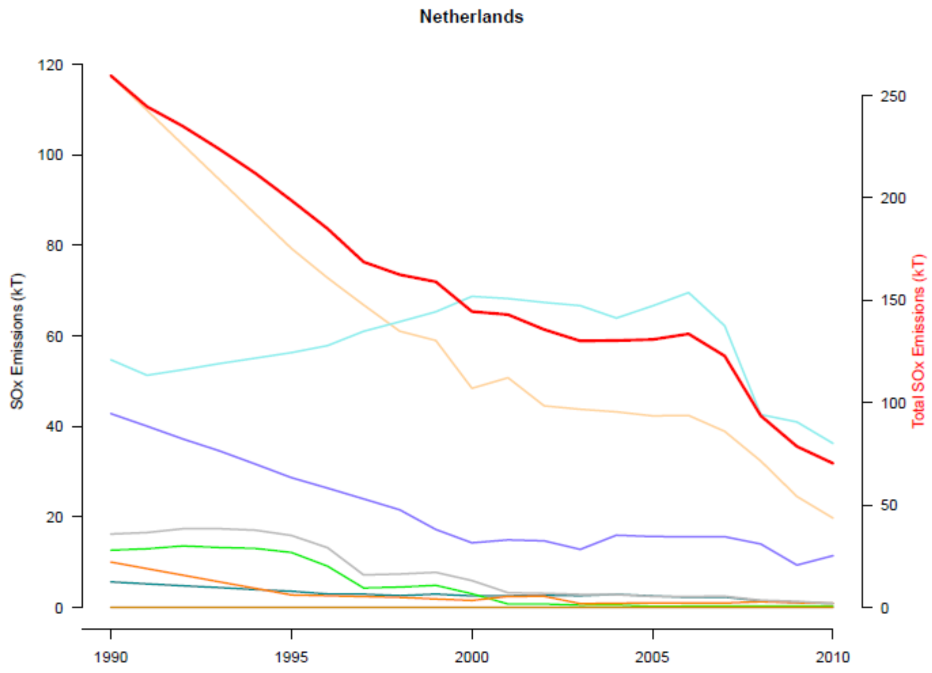
Malta (SO_x)

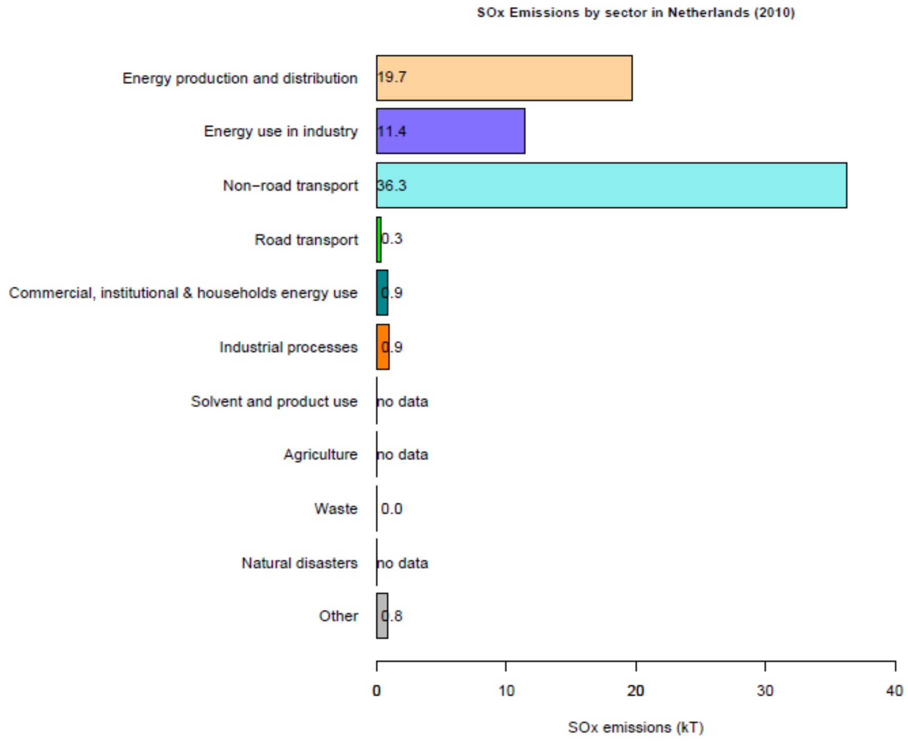

Malta
SOx Emissions (kT)
Total SOx Emissions (kT)
0
10
20
30
40
50
60
2000
2005
2010


SOx Emissions by sector in Malta (2010)
Energy production and distribution
8.0
Energy use in industry
0.0
Non−road transport
16.7
Road transport
0.0
Commercial, institutional & households energy use
0.1
Industrial processes
no data
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
no data
Other
no data
0
5
10
15
20
SOx emissions (kT)

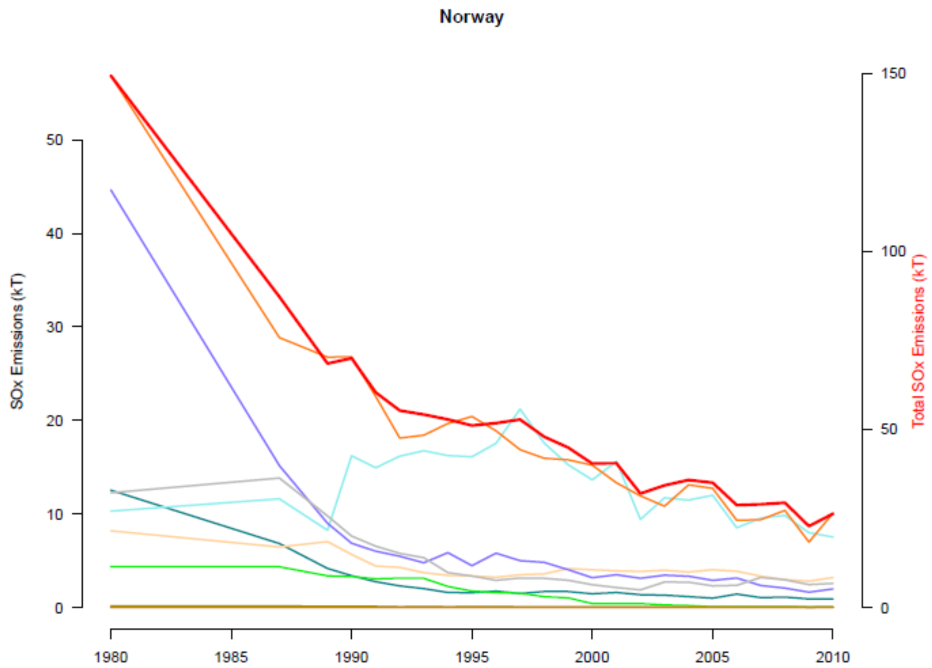
International Prevention Research Institute

The Netherlands (SO_x)

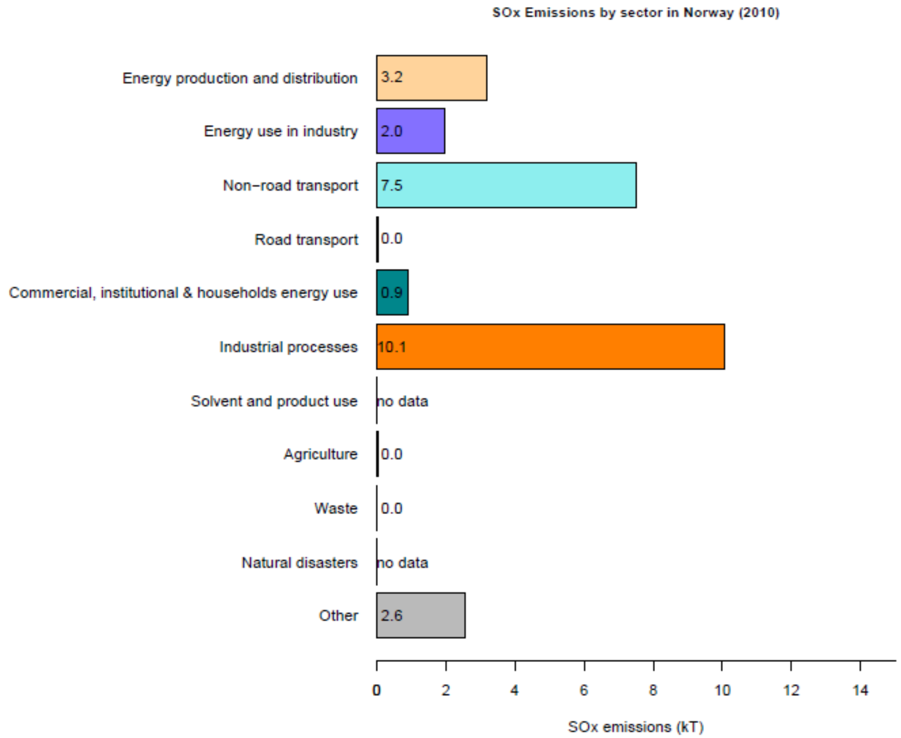




Norway (SO_x)

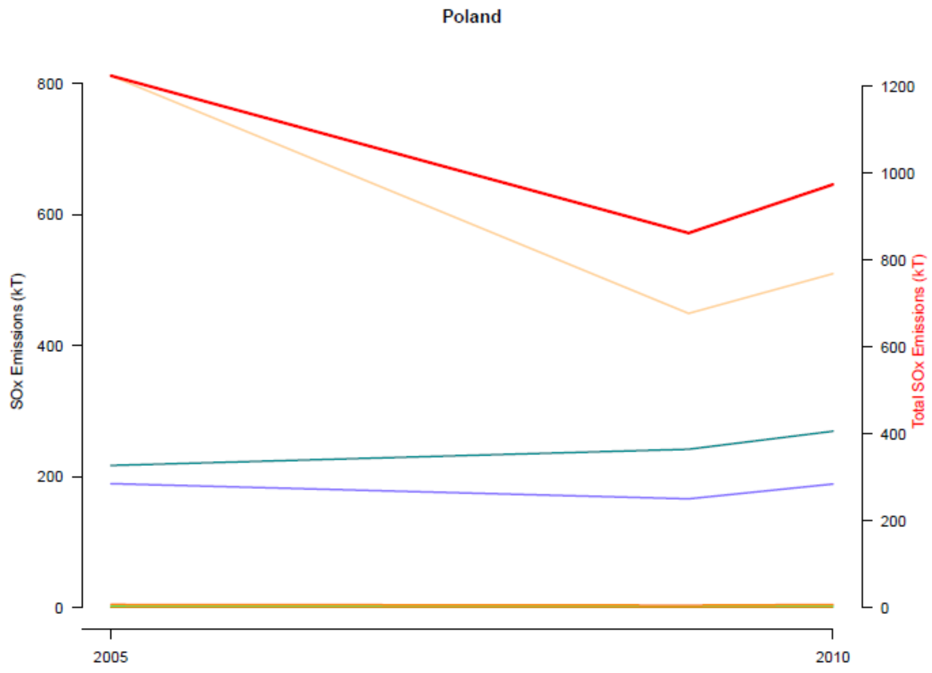


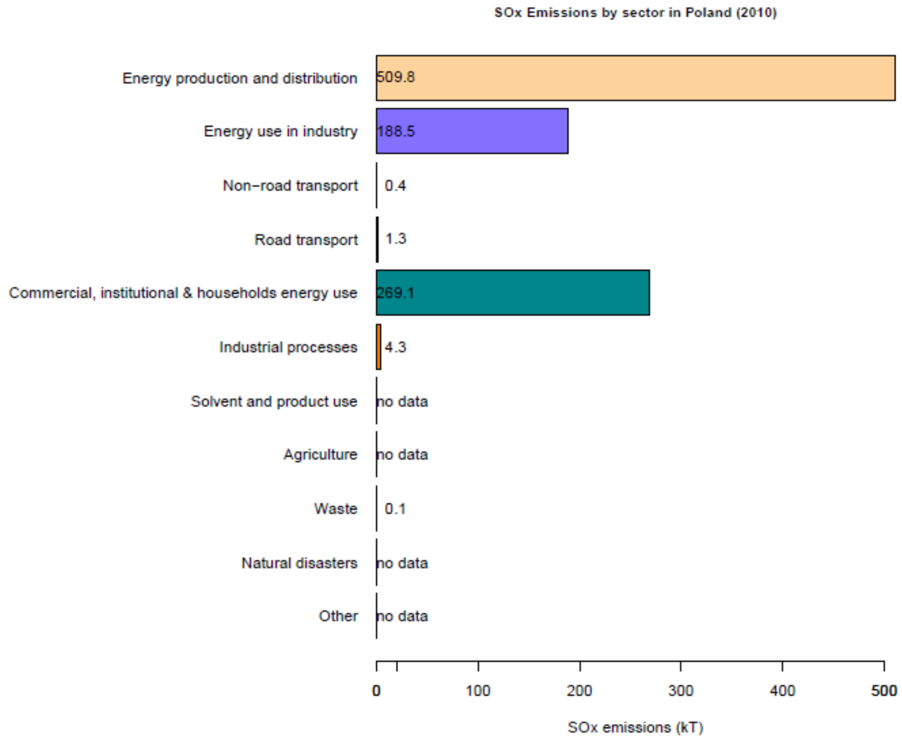
Norway
SOx Emissions (kT)
Total SOx Emissions (kT)
50
40
30
20
10
0
150
100
50
0
1980
1985
1990
1995
2000
2005
2010



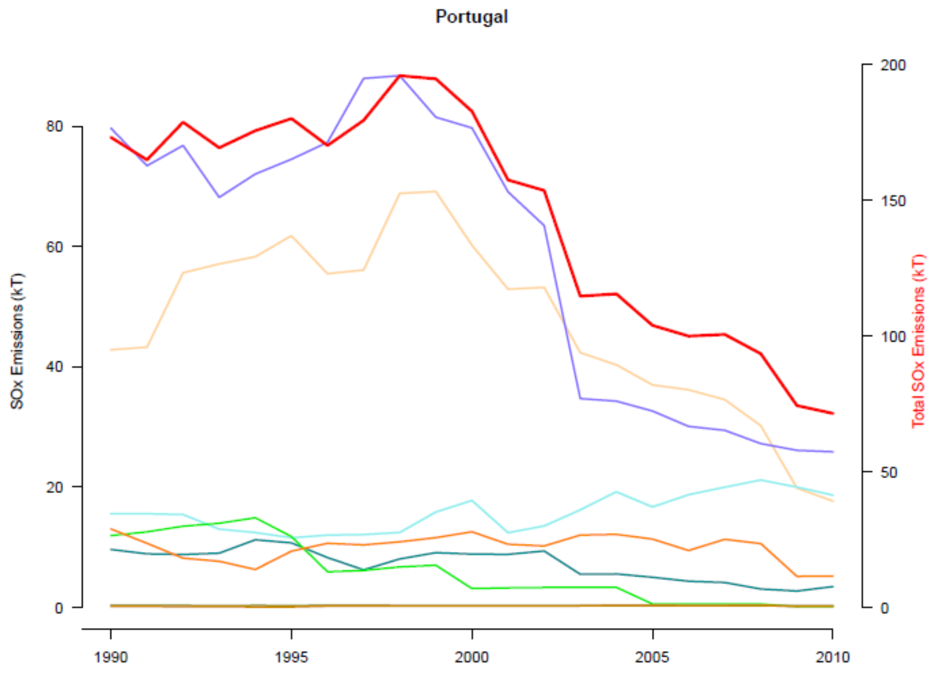
SOx Emissions by sector in Norway (2010)
Energy production and distribution
3.2
Energy use in industry
2.0
Non−road transport
7.5
Road transport
0.0
Commercial, institutional & households energy use
0.9
Industrial processes
10.1
Solvent and product use
no data
Agriculture
0.0
Waste
0.0
Natural disasters
no data
Other
2.6
0
2
4
6
8
10
12
14
SOx emissions (kT)

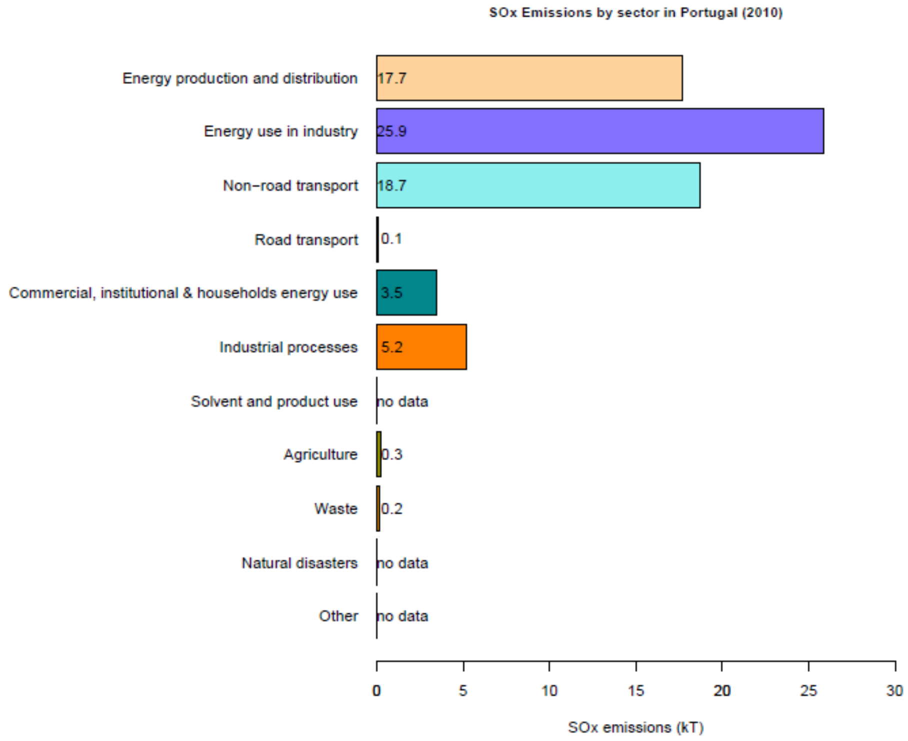
Poland (SO_x)


Poland
SOx Emissions (kT)
Total SOx Emissions (kT)
800
600
400
200
0
1200
1000
800
600
400
200
0
2005
2010

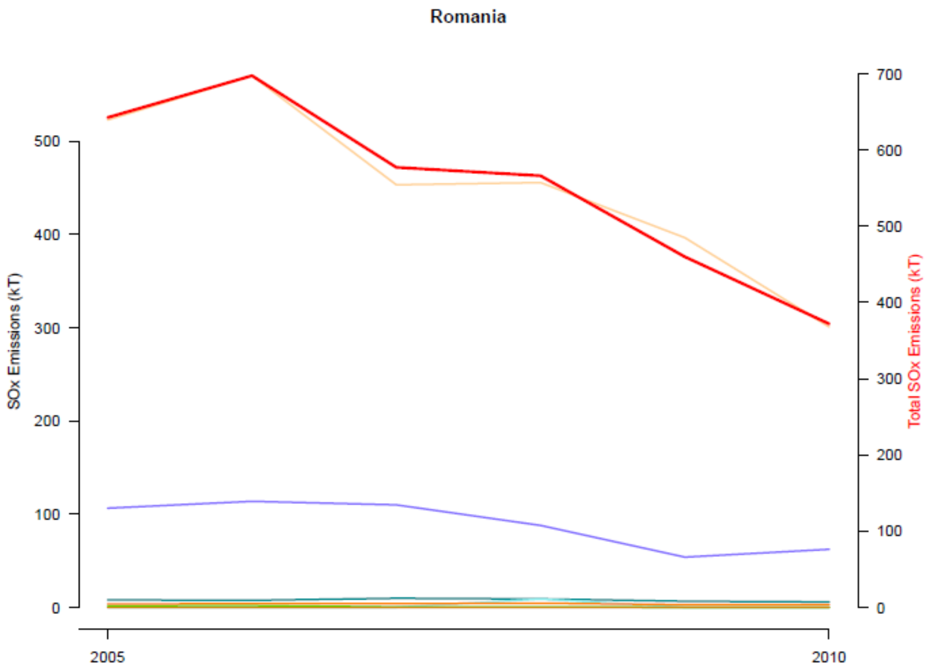

SOx Emissions by sector in Poland (2010)
Energy production and distribution
509.8
Energy use in industry
188.5
Non−road transport
0.4
Road transport
1.3
Commercial, institutional & households energy use
269.1
Industrial processes
4.3
Solvent and product use
no data
Agriculture
no data
Waste
0.1
Natural disasters
no data
Other
no data
0
100
200
300
400
500
SOx emissions (kT)

Portugal (SO_x)

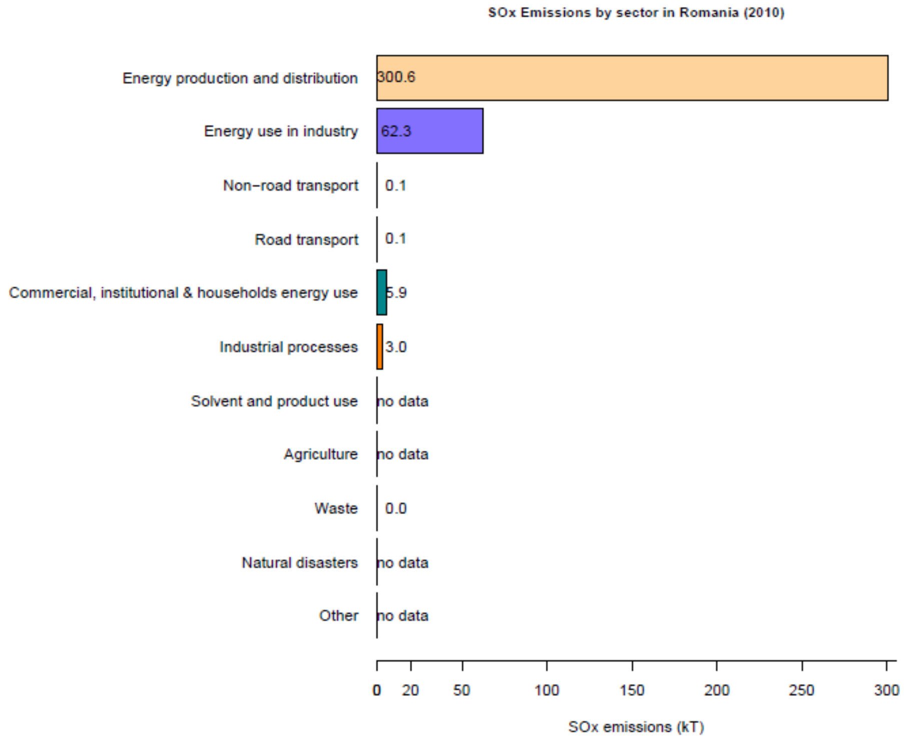




Romania (SO_x)

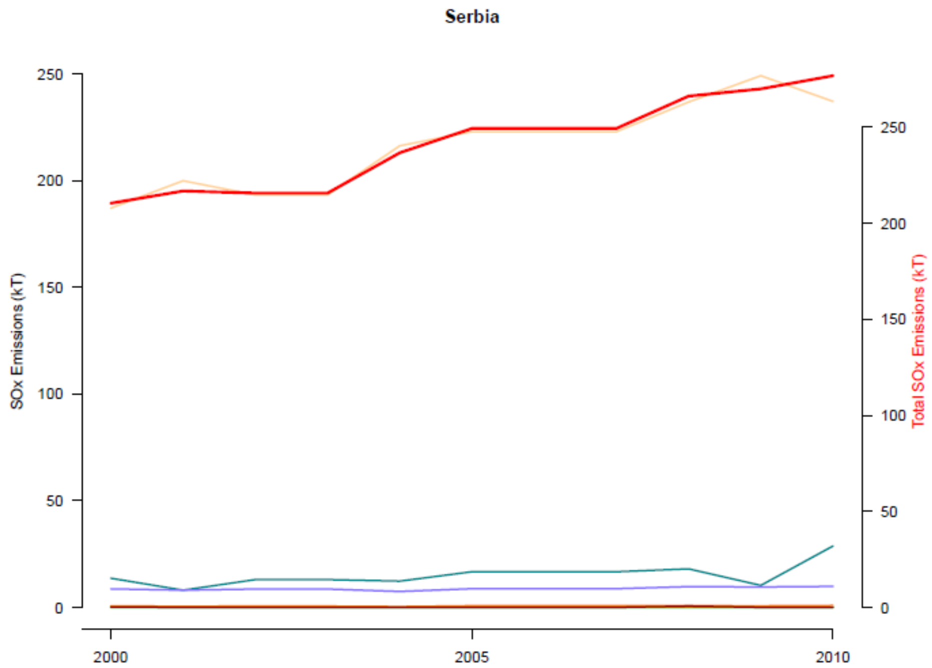


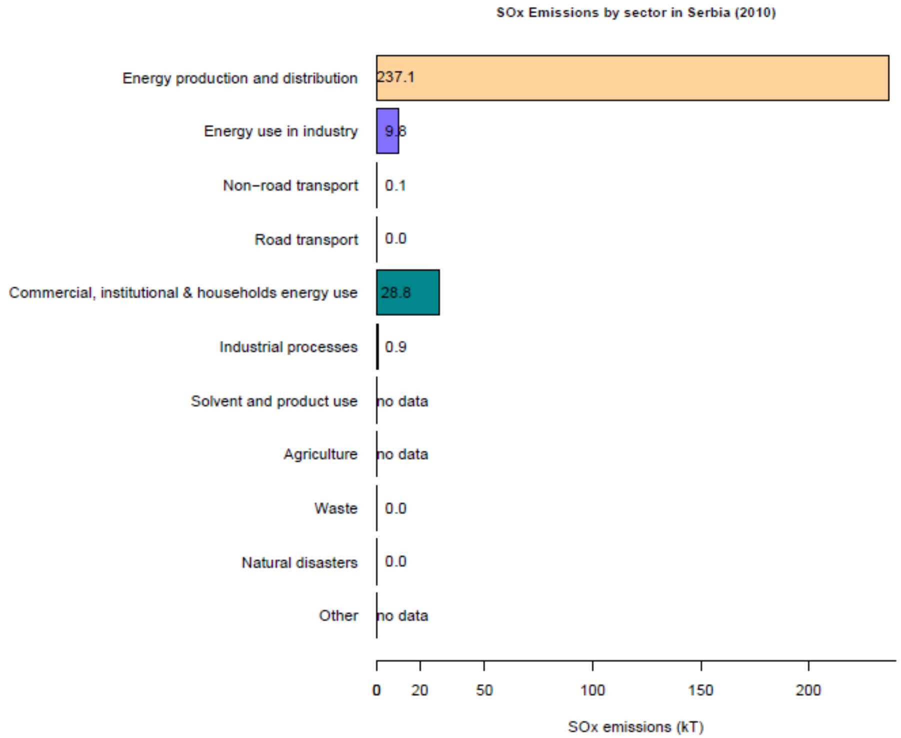
Romania
SOx Emissions (kT)
Total SOx Emissions (kT)
0
100
200
300
400
500
600
700
2005
2010



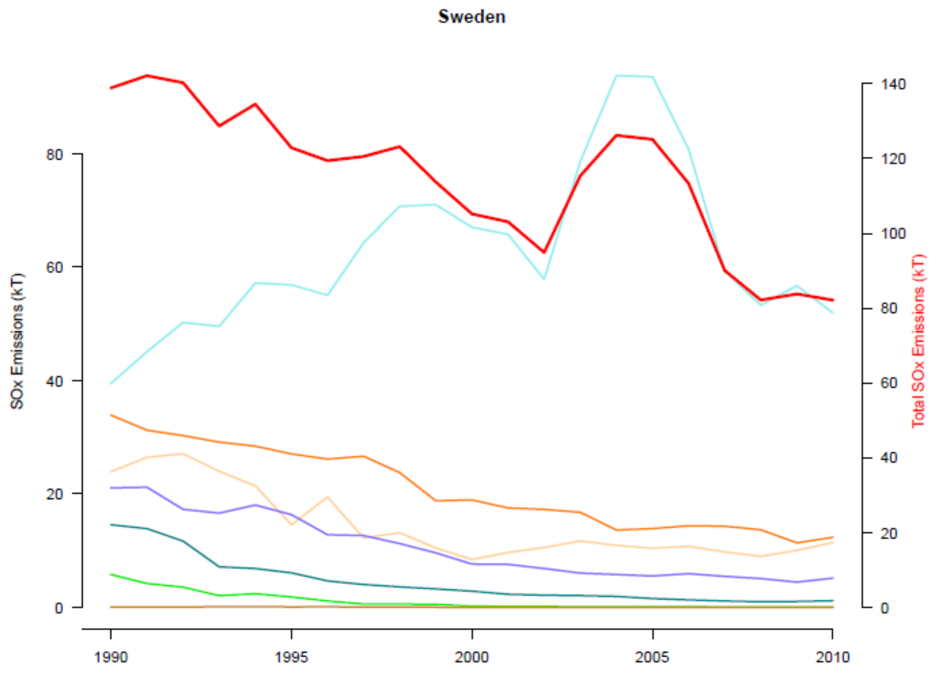
SOx Emissions by sector in Romania (2010)
Energy production and distribution
300.6
Energy use in industry
62.3
Non−road transport
0.1
Road transport
0.1
Commercial, institutional & households energy use
5.9
Industrial processes
3.0
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
no data
Other
no data
0
20
50
100
150
200
250
300
SOx emissions (kT)

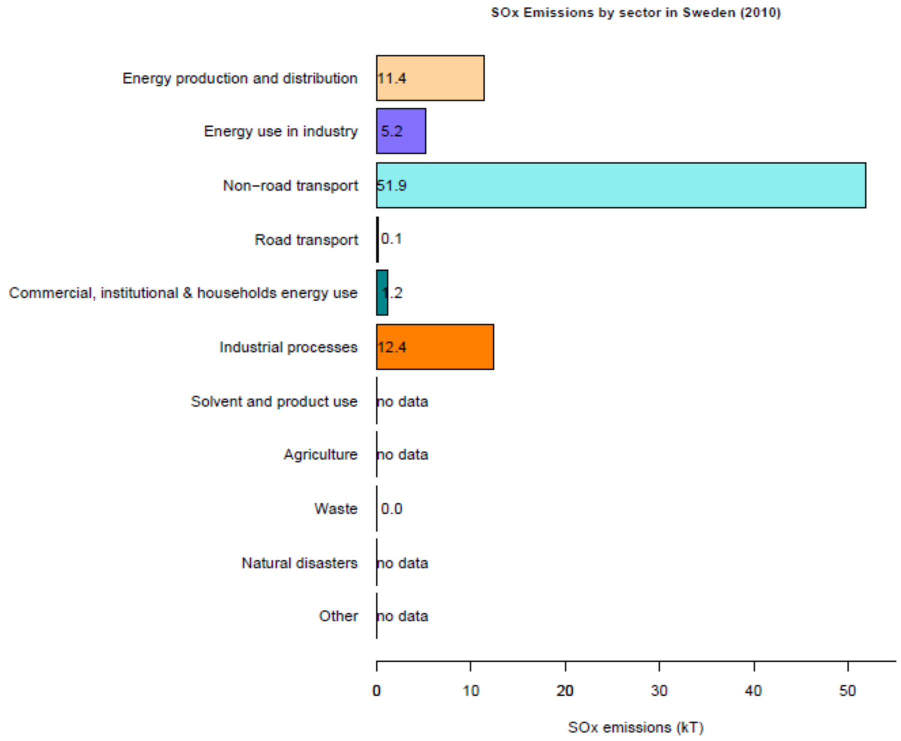
Serbia (SO_x)


Serbia
250
200
150
100
50
0
SOx Emissions (kT)
250
200
150
100
50
0
Total SOx Emissions (kT)
2000
2005
2010

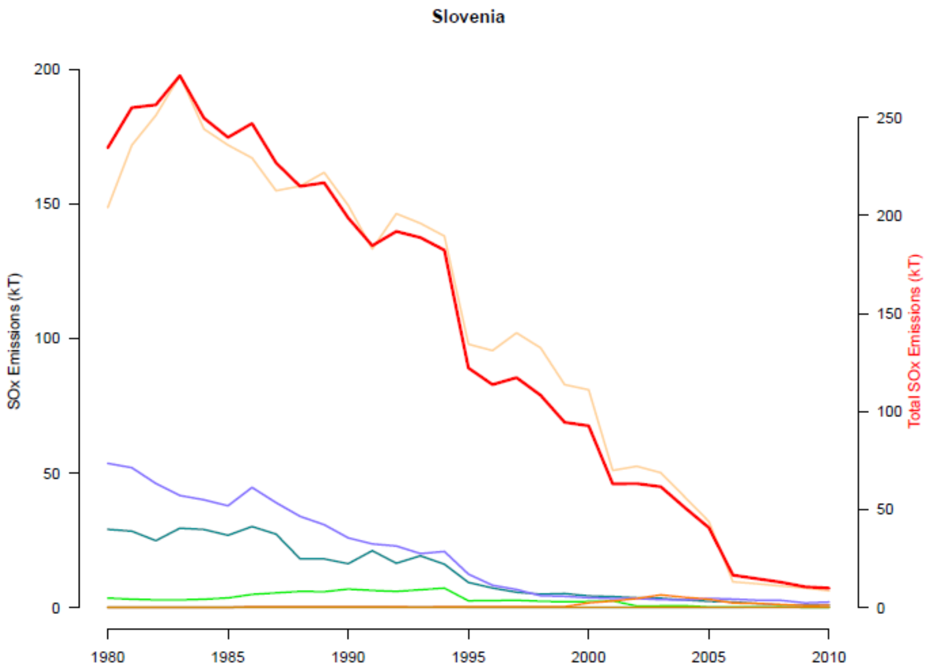

SOx Emissions by sector in Serbia (2010)
Energy production and distribution
237.1
Energy use in industry
9.8
Non−road transport
0.1
Road transport
0.0
Commercial, institutional & households energy use
28.8
Industrial processes
0.9
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
0.0
Other
no data
0
20
50
100
150
200
SOx emissions (kT)

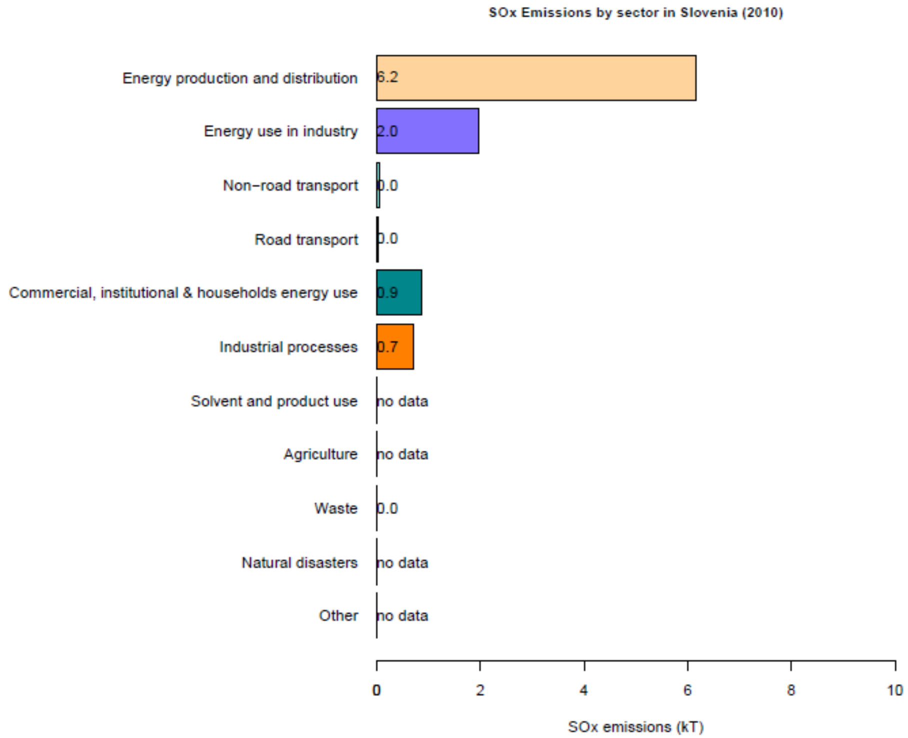
Sweden (SO_x)


Sweden
SOx Emissions (kT)
Total SOx Emissions (kT)
0
20
40
60
80
100
120
140
1990
1995
2000
2005
2010


SOx Emissions by sector in Sweden (2010)
Energy production and distribution
11.4
Energy use in industry
5.2
Non−road transport
51.9
Road transport
0.1
Commercial, institutional & households energy use
1.2
Industrial processes
12.4
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
no data
Other
no data
0
10
20
30
40
50
SOx emissions (kT)

Slovenia (SO_x)





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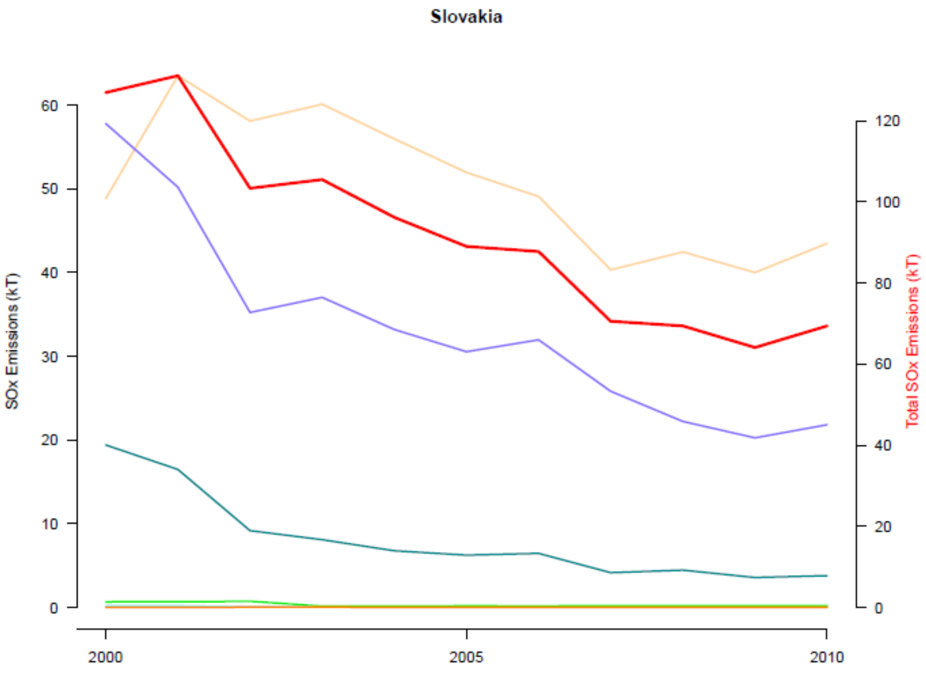
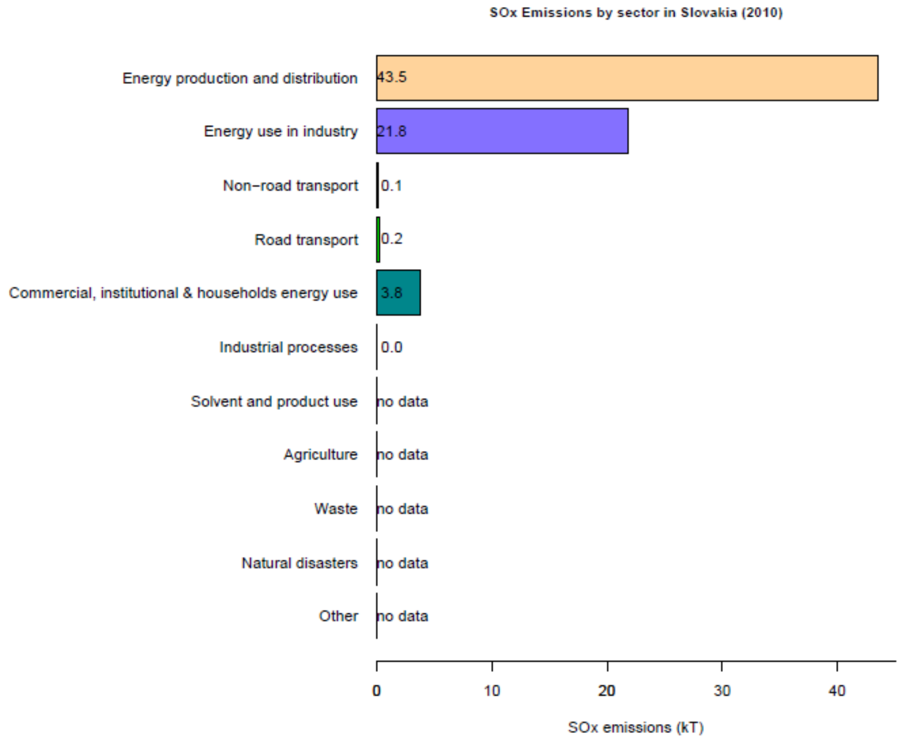
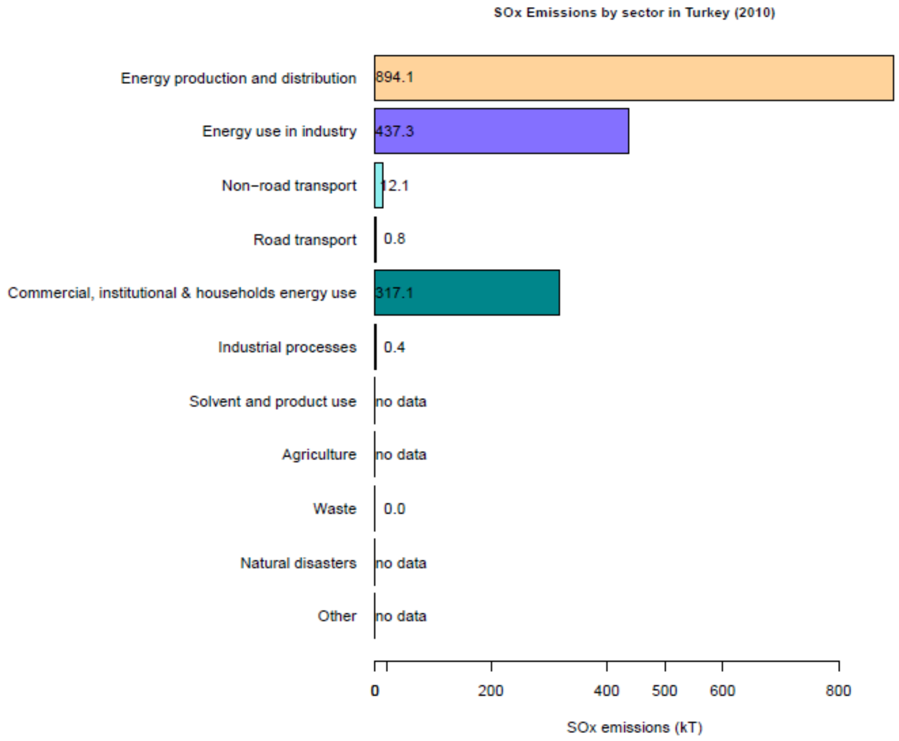
Slovakia (SO_x)





Turkey (SO_x)



SOx Emissions by sector in Turkey (2010)
Energy production and distribution
894.1
Energy use in industry
437.3
Non−road transport
12.1
Road transport
0.8
Commercial, institutional & households energy use
317.1
Industrial processes
0.4
Solvent and product use
no data
Agriculture
no data
Waste
0.0
Natural disasters
no data
Other
no data
0
200
400
500
600
800
SOx emissions (kT)

Bulgaria, Croatia, France, Ireland, Italy, the Netherlands, Portugal, Sweden, Switzerland and the United Kingdom (SO_x)