UNIVERSITY OF GOTHENBURG
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Master Thesis

A Synthetic Assessment of the Gothenburg Congestion Charge

Abstract

This study adopts a quasi-experimental framework using the synthetic control method to examine the impact of the 2013 congestion tax implemented in Gothenburg. The study constructs a counterfactual for Gothenburg to estimate what air pollution and health outcomes would have been in the absence of the congestion charge. It also provides a new perspective on measuring environmental and health economic effects using a more appropriate counterfactual. It specifically measures these effects by analyzing nitrogen dioxide (NO_2) levels and asthma & chronic obstructive pulmonary disease (COPD) hospitalization rates. The study finds no evidence that the policy has significantly reduced air pollution or the incidence of respiratory disease.

Keywords: Congestion charge, Air pollution, Traffic, Asthma, Synthetic Control Method.

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1. Introduction

Traffic congestion is one of the most crucial problems in urban cities, surpassing power, water supply, health, and safety issues (Bretzke, 2013). Traffic congestion affects the air quality due to the emissions from too many vehicles, industries, and human activity within crowded areas. Many countries and city centers have problems with smog and hazardous pollutants, leading to suboptimal air quality (EPA, 2015). United Nations Environment Program UNEP (2021) estimates that up to 7 million people die prematurely from air pollution globally each year, of which 4.2 million deaths are linked to outdoor air pollution. EEA (2023) estimates that around 1200 premature deaths for people under 18 are caused by outdoor air pollution in the European economic area. In the case of Sweden, air pollution is estimated to cause 6 700 premature deaths yearly (Gustafsson et al., 2022). However, regarding the effect of congestion on air pollution, the transport sector is one of the largest contributors. The European Commission (2023) provides statistics that transport-related air pollution contributes to more than 40% of the nitrogen oxide emissions in Europe, mainly from urban areas. The impact of congestion is not limited to air pollution but also the quality of life and traffic-related injuries and deaths. Traffic-related congestion polluting the air is an important topic in tackling the effects of global climate change, requiring an intervention to reduce the harm to people and the environment.

1.1. Policy Solutions

The problem of air pollution is a hot potato in society; there is an issue of free-riding regarding who is responsible for the emissions because there is no incentive for actors to pay the total cost of the damage from emissions. This market failure leads to consumption and production beyond the socially optimal level, resulting in excessive emissions because those responsible do not bear the costs of emitting pollutants. For actors to take the total social cost into account and fix the market failure, there is a need for a governing body to intervene. To create a policy that makes polluters internalize the social cost and reduces the congestion and pollution externality.

Several attempts to improve air pollution have been made, including technical and emission standards, such as fuel or engine standards (EPA, 2015). Other examples are investments in public transport, electric vehicle subsidies, charging infrastructure, and road infrastructure (EEA, 2023). Another policy implemented in many cities, including Bogotá, is restricting cars from entering the city center depending on the digits on the license plate (Brigard Urrutia, 2020). Furthermore, high-occupancy vehicle lanes, car-sharing programs, and education

campaigns are examples of trying to induce behavioral changes in individuals' driving patterns to reduce congestion-related air pollution.

1.2. Congestion Charge

One solution to change driving patterns is a congestion charge, taxing vehicles that drive in city centers to address the problem of traffic congestion by placing a fee on the use of specific transportation during peak congestion hours. Congestion charges are constructed to internalize the externality associated with congestion, reduce environmental impact, and raise tax revenue for the local government (Skatteverket, n.d.). Leape (2006) describes that a congestion charge has been discussed in London since the 1960s. He argues that for many years, there was no implementation of a tax on congestion because of the difficulties with estimating an optimal tax rate resembling Pigouvian taxation, where the marginal social cost should equal the marginal tax to internalize the externalities to induce a behavioral change. In addition, Leape (2006) discusses that the practical cost of implementing the logistical framework of the congestion tax was too high. With technological improvements, however, the logistical aspect of implementing the taxation is simplified for registering vehicles that enter the congestion charge tolls. Reducing bureaucratic costs makes the tax scheme more accessible today than in the 1960s.

1.3. Implementations of Congestion Charges

The earliest form of a congestion charge policy was Electronic Road Pricing (ERP), implemented in 1998 in Singapore (NLB, 2023). One of the main goals was to increase the average speed of vehicles to decrease congestion (LTA, 2023). The London congestion charge was implemented in 2003 with a similar goal: to reduce congestion and improve air quality, with a £5 fee per day for vehicles driving within the congestion charge tolls (Leape, 2006). Globally, not many cities have implemented a congestion charge; some examples are Durham, Milano, Rome, Valletta, and, in the Swedish context, Stockholm and Gothenburg. The Gothenburg congestion charge was introduced to reduce traffic congestion, improve air quality, and fund infrastructure investments (Trafikverket, 2023). Unlike Stockholm, Gothenburg implemented the congestion charge without a trial in January 2013. The charge varies dynamically throughout the day, with higher fees during peak hours when traffic is heavy and lower fees during off-peak hours when the city center is less congested. This policy aims to change commuters' behavior, thereby reducing traffic congestion and air pollution to improve

overall societal well-being. This thesis will investigate the following research questions to determine whether the policy achieves its goals.

1.4. Research Questions

The overarching aim of the thesis is to investigate the effects of the congestion charge in Gothenburg on air pollution and respiratory health. The specific research objectives are:

1. Did the congestion charge in Gothenburg reduce air pollution (NO_2)?
2. Did the congestion charge in Gothenburg reduce respiratory health in terms of incidence of asthma and chronic obstructive pulmonary disease (COPD) hospitalization rates?

Our analysis reveals that the congestion charge has not resulted in a statistically significant reduction in NO_2 levels or improved respiratory health outcomes in Gothenburg. Robustness tests, including placebo (in-space and in-time) tests, an in-time validation, and a leave-one-out analysis, confirm the reliability of these findings.

The following sections of this thesis are organized as follows: Section 2 provides the background of the congestion charge in Gothenburg. Section 3 presents the theoretical framework explaining the mechanisms through which the congestion charge induces behavioral change. Section 4 discusses the most relevant literature on the topic. Section 5 describes the data used in the analysis. Section 6 details the econometric method employed to evaluate the policy. Section 7 presents the results and the robustness checks of our findings. Section 8 discusses the implications of the results. Finally, Section 9 concludes the thesis with final remarks and suggestions for future research.

2. Background

This section covers general information about the congestion charge in Gothenburg, outlining some details regarding the area covered and dynamic pricing (see Figure 2.1). The congestion pricing scheme is dynamic over the day, taxing vehicles more during peak hours. Börjesson & Kristoffersson (2015) found that the congestion charge in Gothenburg effectively reduces traffic volumes with a 12% reduction in the congestion charge zone during the charged hours. The scheme does not charge vehicles on weekends, holidays, the day before the holidays, and during July. The maximum fee a vehicle can be charged daily is 60 SEK (≈ €5), regardless of how many times one passes the toll in a day. Assuming that there are, on average, 20 days the congestion charge is active per month, the maximum fee is, on average, 1200 SEK (≈ €100).

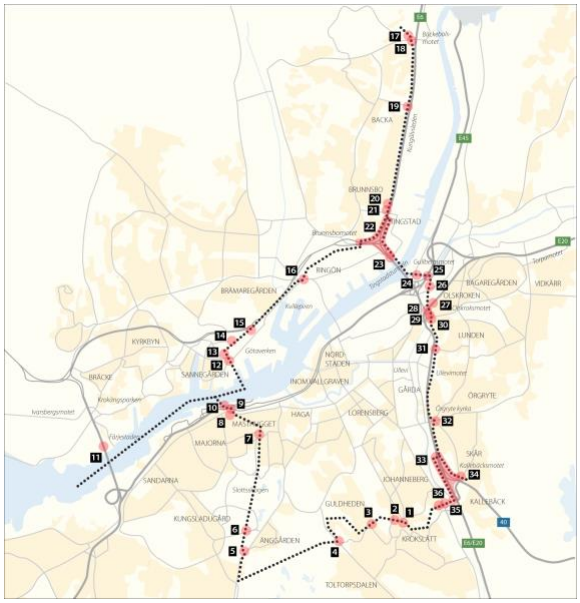
However, the average tax fee is far smaller, at around 150 SEK (≈€13) monthly (Radio Sweden, 2013).

West & Börjesson (2020) conducted a cost-benefit analysis of the Gothenburg congestion charge, concluding that it generates a net surplus of €20 million annually. Additionally, the investment cost was repaid in just over a year. Even though 57% of Gothenburg's citizens voted against the implementation of a congestion charge in a referendum, Andersson and Nässén (2016) conducted a pre-post analysis of the Gothenburg congestion charge, finding that after implementing the congestion charge, people's attitude towards it became more positive over time.

Figure 2.1: Map of congestion charge area and dynamic pricing

Gothenburg

Time	Fee
06:00–06:29	9 SEK
06:30–06:59	16 SEK
07:00–07:59	22 SEK
08:00–08:29	16 SEK
08:30–14:59	9 SEK
15:00–15:29	16 SEK
15:30–16:59	22 SEK
17:00–17:59	16 SEK
18:00–18:29	9 SEK
18:30–05:59	0 SEK



Note: The left panel shows the price dynamics of the congestion charge in Gothenburg. The right panel shows the map of the area where the congestion charge is implemented. (Source: Urban access regulations, n.d.)

3. Theoretical Framework

This section will cover the theoretical framework explaining the behavioral effect of taxation in section 3.1, linking the discussion on taxation to congestion charges in section 3.2, and briefly covering the effect of air pollution on health in section 3.3. The theoretical framework is founded on empirical evidence of policy interventions' effects on reducing air pollution. In addition, epidemiology research links the effects of air pollution on respiratory health

outcomes. We are attempting to establish the causal link if the congestion charges affect air pollution and respiratory health.

3.1. Pigouvian Taxation

A Pigouvian tax should address negative externalities like environmental pollution or traffic congestion. The idea is to internalize the external costs associated with certain harmful activities by imposing a tax to discourage individuals or businesses from those activities. The congestion charge should be set to an optimal level to internalize the social cost of air pollution entirely and reduce the negative externality by making harmful activities more expensive. In an ideal scenario, through an environmental damage function, the fee entirely offsets the environmental damage.

3.2. Congestion Tax

Kacker et al. (2023) estimate the relationship between pollution and traffic congestion and conclude that implementing a congestion pricing system improves air quality. Implementing a tax on peak hours of congestion can incentivize commuters to adjust their travel times and encourage them to explore alternative transportation options, ultimately reducing congestion. While, at the same time, raising revenue for the government to fund public transport and improve air quality, in line with the theory of Pigouvian taxation. Gibson and Carnovale (2015) conclude that road pricing policies and public transportation are substitutes; introducing the cost of driving in the cities makes people switch to public transport.

The congestion charge should theoretically make it sufficiently costly to incentivize people to use alternative modes of transportation, explaining the effect a congestion charge has on traffic. Prud'homme and Bocarejo (2005) discuss an economic appraisal of the London congestion tax, analyzing the cost-benefit of the tax scheme. They estimate the supply and demand for road usage and argue that the taxation is suboptimal in targeting congestion. They argue that the social cost is three times higher than the tax, linking the issue of setting an optimal Pigouvian tax to induce behavioral change.

3.3. Effect on Health

We need to consult research that provides causal evidence on how ambient air pollution impacts respiratory health, to substantiate the theoretical framework linked to our second research question. Veras et al. (2023) outlined the adverse consequences of short- and long-term

exposure to air pollutants like O_3, NO_2, PM_{10}, and $PM_{2.5}$,[1] which are associated with decreased lung function, increased risk of asthma development, and hospitalization. Furthermore, they point out that these effects of exposure are not only limited to the respiratory system but are also associated with other health-related issues such as obesity, autism, and increased blood pressure. Similarly, Achakulwisut et al. (2019) found a significant correlation between respiratory illnesses like asthma and traffic congestion.

4. Literature Review

In this section, we cover relevant literature focusing on the outcomes of implementing a congestion charge policy to address the problems associated with traffic congestion in sections 4.1-4.3. While several empirical studies have estimated the impact of the congestion charge on various factors, our focus remains on its effect on traffic flows, air pollution, and health. Section 4.4 covers the gaps in the literature.

4.1. Effects on Traffic

Congestion charges have been found to significantly alter commuters' behavior. A comprehensive body of literature suggests that congestion charges reduce traffic flows; Leape (2006) highlights the case of London; prior to the congestion tax implementation, private cars accounted for 50% of London's traffic share. Post-implementation, this proportion decreased to one-third, a reduction of 34%. Beevers and Carslaw (2005) highlight a significant reduction in traffic flows measured in London after implementing the London congestion charging scheme. Following the congestion charge in Milan, Gibson and Carnovale (2015) observed a substantial reduction in traffic volume by 14.5%. Furthermore, Coria et al. (2015) analyze the Stockholm congestion charge for non-exempt traffic in three different time windows: morning (06:30–09:00), off-peak period (09:00–15:30), and afternoon (15:30–18:30) and find a reduction of 25.2%, 32.5%, and 37.2% respectively in overall average traffic between the years 2005-2010.

Empirical studies prove that congestion charge policy has produced significant results in the congestion zone, particularly in city centers, where the issue is most prevalent. Leape (2006) states that London experienced a 15% decrease in traffic circulation within the zone. Johansson et al. (2009) analyzed the impact of the Stockholm congestion tax on traffic patterns. Their study included an analysis of vehicle counts and road usage, along with quantifying the distance

[1] O_3 = Ozone, NO_2 = Nitrogen dioxide, PM_{10} = Particulate matter with diameter of 10 micrometer or less, and $PM_{2.5}$ = Particlaute matter with diameter of 2.5 micrometer or less.

traveled by vehicles. The findings indicate a significant reduction in vehicles by 22% within the congestion zone and a 15% decrease in road usage.

4.2. Effects on Air Pollution

The question remains whether the congestion charge is an effective policy to reduce air pollution in urban cities. A broad body of literature evaluates the effects of these congestion taxes on air quality in different cities. It concludes that implementing congestion policies reduces air pollutants. Green et al. (2020) reexamined the effect of the London congestion charge on air pollutants using a difference-in-differences setup. They estimate a reduction of 20% in NO and PM_{10} and a 6-9% reduction in CO concentration compared to pre-treatment means for the congestion zone. However, Green et al. (2020) also concluded that the concentration level NO increased in the air, possibly due to the substitution effect since commuters have preferred using public transport that is primarily diesel-driven. Gibson and Carnovale (2015) found that in the absence of a congestion charge, concentrations of CO increased by 6%, and PM_{10} levels increased by 17% in Milano. Johansson et al. (2009) found a reduction of 8.5% in NO_X and 13% in PM_{10} after implementing the Stockholm congestion tax. Simeonova et al. (2019) analyzed ambient air pollution from traffic in Stockholm using NO_2 and PM_{10} as proxies for air pollution. Their results show that the congestion charge decreases air pollution by 10–20%.[2]

4.3. Effects on Health

The literature concerning the impact of congestion charges on health outcomes is not extensive; therefore, the subject remains a critical area of research. Currie and Walker (2011) suggested with their results that policies explicitly designed to reduce traffic congestion can positively influence the health of the population residing within congestion zones. Simeonova et al. (2021) investigate the relationship between traffic congestion and acute asthma attacks in children under five years of age. They investigate the effect of a congestion charge implemented in Stockholm. First as a trial in 2006 and then permanently in 2007. Simeonova et al. (2021) build on epidemiology research showing evidence that air pollution primarily affects respiratory ability through asthma. Children are more susceptible to air pollution since their lungs are still developing, and the damage can be permanent. However, they find that the policy's effect is lagged, taking time for it to reach full effect.

[2] NO = Nitric oxide, CO = Carbon monoxide, and NO_x = Nitrogen oxides.

4.4. Gaps in Literature

This thesis addresses the existing literature gaps by focusing on the following: First, the relationship between congestion tax and respiratory health in Gothenburg has not been previously examined. Existing studies, such as Simeonova et al. (2019), have previously focused on Stockholm. Second, the number of studies evaluating the impact of the congestion charge in Gothenburg with respect to air pollution is very limited. Thus, we will provide additional evidence on the effect of congestion charges on respiratory health and the impact on air pollution. Third, this paper will be unique in applying the synthetic control method to investigate a congestion charge and specifically evaluate Gothenburg's congestion charge.

5. Data

This thesis examines the effects of Gothenburg's congestion charge during 2010-2015. The study involves ten Swedish municipalities, with Gothenburg as the treated unit and the others forming the donor pool.[3] Section 5.1 covers the selection of the donor pool. Section 5.2 describes the outcome variables, and section 5.3 describes the predictor variables. Section 5.4 shows graphical relationships and the trend for the outcome variables for each municipality.

5.1. Selection of Control Municipalities

Selecting a donor pool is an essential step in this analysis and is crucial for the precision of the synthetic control method (SCM). Out of Sweden's 290 municipalities, Stockholm and Gothenburg have introduced congestion charges, providing a wide potential donor pool. The choice of municipalities impacts the accuracy of the SCM. The study will analyze hourly NO_2 rates, but such detailed data is scarce across Swedish municipalities, influencing donor selection. Gathering the municipal register medical data via individual emails to each county adds to the complexity, being a time-intensive process. Therefore, constraints on NO_2 data availability and the health data collection process limit the available municipalities in this master thesis.

5.2. Outcome Variables

Air pollution (nitrogen dioxide (NO_2)) is gathered from the Swedish Meteorological and Hydrological Institute (SMHI) (SMHI, n.d.). All municipalities oversee the air quality of their respective territory, but SMHI is responsible for hosting and making the data available to the

[3] Municipalities in the donor pool: Borås, Botkyrka Helsingborg, Landskrona, Lund, Malmö, Mölndal, Norrtälje, and Uppsala

public (Naturvårdsverket, 2023). The proxy for air pollution, nitrogen dioxide (NO_2), is appropriate for this analysis for two reasons: First, NO_2 is historically Sweden's most frequently and accurately measured air pollutant. Thus, NO_2 will ensure we obtain the least missing values. Second, NO_2 is the primary pollutant from combustion engines and has been shown to affect human respiratory health (Costa et al., 2014), thus making it suitable for investigating the link between traffic congestion and asthma & COPD.

Coria et al. (2015) reveal a complex interaction between traffic volumes and NO_2 concentrations across different cities. Despite traffic significantly contributing to NO_2 levels, the correlation is not direct and varies throughout the day. In the mornings, NO_2 levels rise sharply following traffic peaks, indicating a rapid response. The afternoon traffic, despite often being higher and more congested, correlates with a slower and lower increase in NO_2 levels. Coria et al. (2015) demonstrate the morning-to-afternoon NO_2 ratios observed in Santiago, London, and Stockholm, underscoring the complex dynamics influencing urban air quality. Therefore, it is essential to use hourly data to account for such variation.

The proxy for respiratory health outcomes is the hospitalization rate for asthma & COPD, collected from county records in Sweden and available to the public upon request. Since this register data contains potentially sensitive information, it is not directly accessible online. The observation rate is based on monthly registrations of asthma & COPD cases for open and closed specialized care, categorized by the patients' municipality of residence.

5.3. Predictor Variables

The predictors for the model specification include weather, traffic, the number of private vehicles, simulated traffic, and socio-economic and geographical factors, such as education levels and the percentage of the urban population.

Weather factors will be the most crucial explanatory variables for the level of NO_2 from traffic. Windspeed is the most important because it disburses air pollution effectively (Hansen-Lewis, 2018). Demuzere et al. (2009) state that weather conditions significantly influence pollutant levels in the atmosphere. High temperatures can enhance the photochemical reactions that produce NO_2, leading to higher concentrations, especially in urban areas with heavy traffic. On the other hand, windy conditions can disperse NO_2, reducing its concentration. With little wind,

NO_2 can accumulate during stable periods, as there is less atmospheric mixing to dilute the pollutant. Rainfall can also reduce NO_2 levels by clearing it out of the atmosphere.

Weather data for this study is sourced from visualcrossing.com (n.d.) due to its comprehensive use of meteorological forecasts and measurements from various stations, ensuring a more precise depiction of weather conditions. This private provider is selected over SMHI because it does not offer the same level of detail or frequency, often recording data only once or twice a day for most municipalities. *Visualcrossing* incorporates data from multiple stations to create forecasts for individual municipalities, providing the necessary, hourly granularity and historical accuracy for this analysis.

Traffic, defined as the hourly number of vehicles on the roads, is sporadically recorded by Trafikverket, making comprehensive historical data scarce. To address this, we use the methodology of Coria et al. (2015) namely to predict traffic flow. We create a traffic model based on samples of 500-1000 hourly observations from central roads within each municipality. An ordinary least squares (OLS) regression predicts traffic flow, factoring in NO_2, windspeed, windspeed squared, temperature, precipitation, and visibility. The model achieves an 89% correlation with observed traffic data, providing a good foundation for the synthetic counterfactual construction.

The predictor variables, covering socio-economic factors and geography, are transformed from monthly or yearly rates to hourly rates. These predictors are essential for modeling the factors affecting NO_2 emissions, encapsulating economic activities linked to pollution, and the geographic potential for pollution dispersion or concentration. This approach allows the synthetic control to closely replicate the attributes of Gothenburg, the treated unit, enabling a more precise estimate of the congestion charge's impact on NO_2 levels. Data for these variables are sourced from Statistics Sweden (n.d.).

The study utilizes z-score normalization to standardize the air pollution and respiratory health variables to construct the synthetic counterfactual.

$$Z = \frac{x - \mu}{\sigma}$$

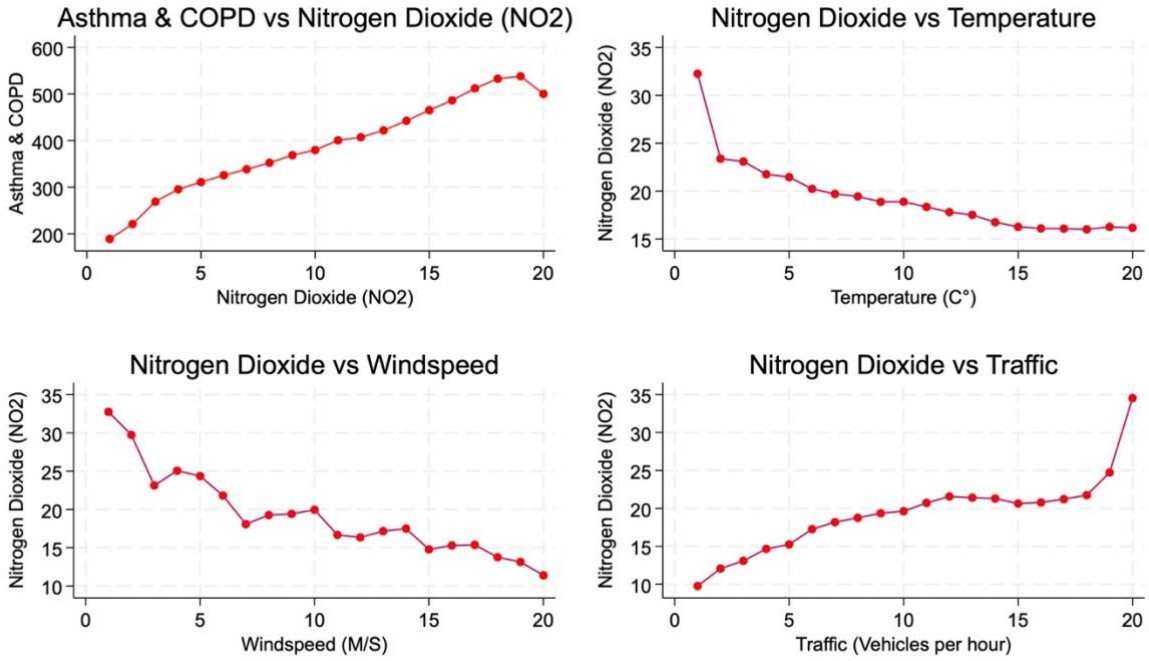
This method involves adjusting the variables to have a mean of zero and a standard deviation of one by subtracting the mean (μ) from (x) and dividing by the standard deviation (σ). The

standardized values are interpreted as "z" standard deviations away from the mean. This standardization helps to compare scales and achieve a good pre-treatment match.

5.4. Graphical Relationships

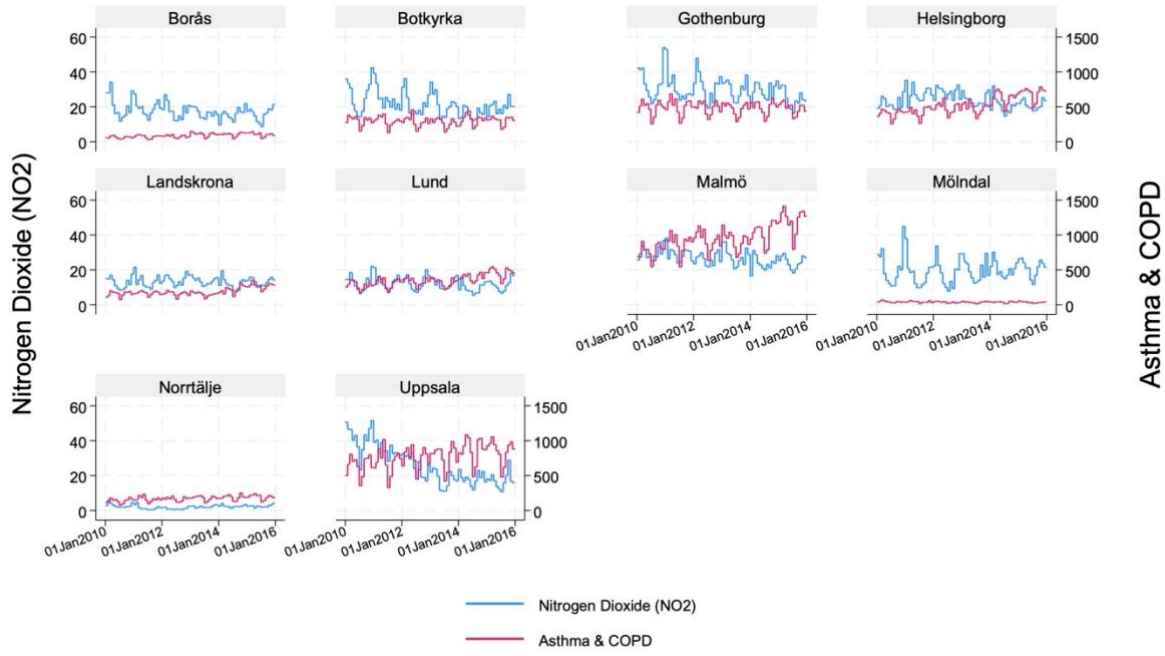
Figure 5.4.1 illustrates scatterplots examining the correlations between NO_2 levels and variables such as asthma & COPD, temperature, windspeed, and traffic, categorized into ventils representing 5% of the distribution. Asthma & COPD rates generally increase with higher NO_2 levels, although the relationship decreases for the highest level of NO_2. The reason is that Mölndal has high levels of air pollution but low cases of asthma & COPD, as shown in Figure 5.4.2. Therefore, Mölndal will drive up the mean asthma & COPD levels in the very highest levels of NO_2, hence the decrease in the relationship at the end of the distribution. This is supported by excluding Mölndal and plotting the relationship, shown in Appendix 1.1, where we do not see a decrease towards the higher levels. There is a negative quadratic relationship between temperature and NO_2, indicating that higher pollution is correlated to lower temperatures. There is a negative relationship between windspeed and NO_2, with less windy conditions associated with more pollution. Traffic is positively linked to NO_2 and is non-linear; an initial concave increase in pollution with higher traffic is observed in the lower ventils, shifting to a convex increase in the upper ventils. These findings reflect the complex interplay between environmental factors and air pollution. The relationship between NO_2 and asthma & COPD seems relatively linear.

Figure 5.4.1: Scatterplots of main variables



Note: The graph shows the calculated ventils of asthma & COPD, temperature, windspeed, traffic, and the average nitrogen dioxide (NO_2) per ventile. The data points are divided into 20 groups of equal size, with each group representing 5% of the total distribution, to make the relationship between variables easier to observe.

Figure 5.4.2: Nitrogen Dioxide (NO_2) and Asthma & COPD trends by municipality.



Note: The graph shows the average emission level of Nitrogen Dioxide (NO_2) and asthma & COPD cases for all ten municipalities from 2010 to 2015. The average NO_2 level for Gothenburg is 30 $\mu g/m^3$, and the average asthma & COPD is 488 cases.

Figure 5.4.2 shows the emission trends for NO_2 alongside asthma & COPD cases for the municipalities from 2010 to 2015. Gothenburg's data reflects a downward NO_2 trend with an average value of 30 $\mu g/m^3$.[4] Asthma & COPD rates remain steady, at an average of 488 hospitalizations per month. Mölndal exhibits a similar NO_2 trend to Gothenburg, possibly due to their proximity and shared socio-economic dynamics, including many people living in Mölndal commute to Gothenburg for work. As a result, the sources of NO_2 emissions, specifically traffic, are likely to be comparable in both Mölndal and Gothenburg. Furthermore, air pollutants like NO_2 can travel short distances depending on windspeed, wind-direction, and other atmospheric conditions. Therefore, emissions released in one area can affect nearby regions, contributing to similarities in air quality between Mölndal and Gothenburg. This phenomenon is known as local dispersion, where pollutants disperse and mix within the atmosphere. Other municipalities, like Borås, Malmö, and Uppsala, show a decline in NO_2 emissions, while asthma & COPD cases rise. It is worth noting that Malmö exhibited a reduction in NO_2 post-2009, likely due to targeted environmental policies such as the introduction of the environmental program 2009-2020 (Malmö stad, 2009).[5]

Despite a decrease in NO_2, Malmö experiences an uptick in respiratory disease, see Figure 5.4.2, this makes us question whether air pollution alone is to blame for these respiratory problems or whether this could be because of non-environmental factors such as socio-economic changes, housing conditions, and demographic shifts due to immigration, as outlined by Oudin et al. (2015). This observation is crucial to report and could affect our results, as our method relies on weighted averages of municipalities for constructing a synthetic counterfactual. We must stress that the increase in respiratory problems in Malmö depends not only on environmental factors, as reported by Oudin et al. (2015). Many immigrants who have moved to Malmö faced adverse housing conditions, such as overcrowding and dampness. These living situations are known to worsen respiratory problems, including asthma. Therefore, it could be one of the possibilities that these factors are contributing to the rise of asthma cases in Malmö.

[4] $\mu g/m^3$ = Micrograms per cubic metre

[5] At the end of 2009, the city of Malmö adopted the Environmental action program incorporating two objectives.
1. Adaptation to climate change such as changes in temperature, rise in sea level and increased precipitation.
2. Green and blue aspects should be developed aiming to expand and protect green areas and the aquatic environment.

6. Methodology

In this section, we will discuss the methodology employed in this thesis. Difference-in-differences (DiD) is a common choice among researchers for comparative case studies; subsection 6.1 will explain why a synthetic control method (SCM) is preferable. Then, we will discuss the SCM in section 6.2, finishing with a description of our robustness tests in section 6.3.

6.1. Why the Synthetic Control Method?

One of the main challenges in quasi-experiments is to estimate how the outcome of interest would have evolved for the affected unit in the absence of the treatment. In other words, the issue of causal inference is to assess and measure an appropriate counterfactual.

The application of the DiD approach in evaluating policy interventions relies on the assumption of parallel trends to calculate the difference of the treated minus the difference of the counterfactual if the counterfactual scenario represents the outcome of what the treated group would have been in the absence of the intervention. While evaluating a policy through DiD comparing two cities, even if the assumption of parallel trends holds, it might be insufficient to assume they are entirely comparable. For instance, Gothenburg is the treated municipality, and using Malmö as a counterfactual, based on the parallel trends in the pre-treatment period, can be challenging because both cities exhibit several different factors, such as Gothenburg has a larger industrial sector or higher levels of traffic congestion.

Abadie et al. (2012) argue that combining different untreated units can offer a better comparison for the treated unit. The synthetic control method mitigates reliance on the assumption of parallel trends by constructing a synthetic control unit from a weighted combination of untreated units to closely match the treated unit's pre-treatment characteristics and outcomes. For instance, Abadie and Gardeazabal (2003), while evaluating the economic impact of terrorism in a Spanish region (Basque Country), use a combination of two Spanish regions to closely mimic the economic growth that the Basque Country would have experienced in the absence of terrorism. Furthermore, the SCM is advantageous when there are few treated units (Abadie, 2021). This fits the scenario where only Gothenburg and Stockholm have implemented a congestion charge in Sweden. Additionally, Andersson (2019) explains that an advantage of the SCM is its capacity to account for unobserved factors that could influence the outcome, thus reducing bias in the estimation. Abadie (2021) further argues that the SCM

safeguards against p-hacking through transparency in constructing the counterfactual and robustness checks.

Despite the growing body of literature demonstrating that the synthetic control method is a robust and effective tool for assessing causal effects, it has not yet been applied to evaluate the impact of environmental congestion charges. This thesis is the first to assess the effects of a congestion charge using a synthetic counterfactual. Furthermore, we utilize granular hourly data, which, to our knowledge, has not been done previously in the context of SCM.

6.2. Synthetic Control Method

This subsection will cover the econometric model; we construct a synthetic Gothenburg using weights from the optimization process, multiplied with the values from other Swedish municipalities so that the resulting synthetic Gothenburg reproduces air pollution (NO_2) and respiratory health outcomes (asthma & COPD) in pre-treatment period and calculating the difference between Gothenburg and the synthetic counterfactual in the post-treatment period.

Using the setting explained by Abadie and Gardeazabal (2003); Abadie et al. (2010); Abadie et al. (2015); and Abadie (2021), we obtain $J+1$ Swedish municipalities, with $j=1$ being the treated unit, Gothenburg. Whereas $j=2,\dots,J+1$ is a collection of untreated Swedish municipalities called the donor pool. We will use the weighted average of $j=2,\dots,J+1$ to construct the synthetic Gothenburg, represented by a vector of weights. The units are observed from 2010 to the end of 2015, denoted $t=1,\dots,T$. Let the treatment indicator, T_0, be equal to the 1st of January 2013, when the congestion charge was implemented $1<T_0\leq T$. Defining the "pre-treatment" $\equiv t\in\{t<T_0\}$ and the "post-treatment" $\equiv t\in\{t\geq T_0\}$.

Furthermore, let $\boldsymbol{Y}_{jt}$ be a $(T\times J+1)$ matrix describing the outcome variable NO_2 for the municipalities, j at time, t. We further decompose $\boldsymbol{Y}_{jt}$ into treated and untreated municipalities' outcomes. The treated outcome becomes a $(T\times 1)$ vector denoted by $\boldsymbol{Y}_{1t}^{T}$, and the untreated outcome becomes a $(T\times j)$ matrix denoted by $\boldsymbol{Y}_{jt}^{C}$.

Abadie (2021) explains that to evaluate a policy, one of the main challenges is to estimate how the outcome of interest would have evolved for the affected unit in the absence of the treatment. In other words, the issue of causal inference in quasi-experiments is to create or measure an

appropriate counterfactual. Before the treatment, the treated and untreated units should be similar in terms of the outcome variable $\boldsymbol{Y}_{jt}^{T} = \boldsymbol{Y}_{jt}^{C}$. Thus, the exogenous variation of the congestion charge should impose the treatment effect between the treatment and counterfactual. Therefore, for the post-treatment, the congestion charge effect is estimated by:

$$\tau_{1t} = \boldsymbol{Y}_{1t}^{T} - \boldsymbol{Y}_{1t}^{C} \tag{1}$$

The counterfactual outcome $\boldsymbol{Y}_{1t}^{C}$, mentioned above is not directly observable and, therefore, needs to be estimated. Abadie (2021) suggests using the weighted average of the outcomes from the donor pool, which is SCM's most essential aspect, to construct a synthetic counterfactual to closely resemble Gothenburg before the treatment was implemented. Hence, the estimated effect of congestion charge is the difference between the observed values of NO_2 in Gothenburg and the estimated unobserved outcome.

$$\hat{\tau}_{1t} = \boldsymbol{Y}_{1t}^{T} - \widehat{\boldsymbol{Y}}_{1t}^{C} \tag{2}$$

To construct the weights, we match the characteristics of the treated municipality, Gothenburg, to mimic the levels of NO_2 using a set of predictor variables represented by the $\boldsymbol{X}_1$ a $(K \times 1)$ column vector. For each unit, j, there is a set of K predictors. The $\boldsymbol{X}_0$ a $(K \times j)$ matrix contains the set of predictors, K, for units $j = 2, \ldots., J+1$, the municipalities in the donor pool. The optimization process to select the weights, $\mathbf{W}$, minimize $\|\boldsymbol{X}_1 - \boldsymbol{X}_0\boldsymbol{W}\|$, focuses on reducing the squared difference between the treated unit's characteristics and those of the synthetic control. The weights must be non-negative and sum to 1 to avoid extrapolation (Abadie, 2021).

$$\|\boldsymbol{X}_1 - \boldsymbol{X}_0\boldsymbol{W}\| = \sqrt{\left(\sum_{h=1}^{K} v_h\left(X_{h1} - w_2 X_{h2} -, \ldots, -W_{J+1}X_{hJ+1}\right)^2\right)} \tag{3}$$

In eq(3), we observe the positive constants $v_1, \ldots, v_k$, which represent a specific combination of weights assigned to predictor variables, where each weight represents the relative importance of a predictor variable in the synthetic control. Abadie (2021) suggests that by using constrained quadratic optimization, we can minimize eq (3) to find the optimal set of weights that best replicate the values of the predictor variables for the treated unit. Each choice of $\boldsymbol{V} = (v_1, \ldots, v_K)$, we produce a synthetic control $\boldsymbol{W}(\boldsymbol{V}) = \left(w_2(\boldsymbol{V}), \ldots, w_{J+1}(\boldsymbol{V})\right)$. Abadie (2021) further argues that an optimal way of jointly choosing $\boldsymbol{V}$ and $\boldsymbol{W}$, is to minimize the mean squared prediction error (MSPE) between the treated outcome, $\boldsymbol{Y}_{1t}^{T}$, and the synthetic control $\boldsymbol{Y}_{1t}^{C}$. Mathematically, the objective is to select $\boldsymbol{W}^{*}$ that minimizes the MSPE between

Gothenburg and the synthetic control in the pre-treatment period for both the predictors and the outcome:

$$\boldsymbol{W}^* = \min_{\boldsymbol{W}} \|\boldsymbol{X}_1 - \boldsymbol{X}_0\boldsymbol{W}\| = \sqrt{(\boldsymbol{X}_1 - \boldsymbol{X}_0\boldsymbol{W})'\boldsymbol{V}(\boldsymbol{X}_1 - \boldsymbol{X}_0\boldsymbol{W})}$$
$$s.t. 0 \leq w_j \leq 1 \; and \sum_{j=2}^{J+1} w_j = 1 \tag{4}$$

Substituting the optimal weights, w_J^*, into the treatment effect estimation eq(2) is:

$$\hat{\tau}_{1t} = Y_{1t}^T - \sum_{j=2}^{J+1} w_j^* Y_{jt}^C = \boldsymbol{Y}_{1t}^T - \boldsymbol{Y}_{jt}^C\boldsymbol{W}^* \rightarrow \boldsymbol{Y}_{1t}^T - \widehat{\boldsymbol{Y}}_{1t}^C \tag{5}$$

Using the *synth* command in R, we use numerical optimization methods, called the Nelder-Mead and the Broyden-Fletcher-Goldfarb-Shanno method, to minimize the MSPE over the optimization period (Hainmueller et al., 2023). The weights in Table 7.1.1 show the weights that minimize the MSPE over the pre-treatment period to mimic Gothenburg's NO_2 values.

To obtain the values for the synthetic counterfactual, we use the weights from the optimization problem. We multiply the subsequent NO_2 level for each point in time for each municipality in the donor pool by its weight. Formally as: $\sum_{j=2}^{J+1} w_j^* Y_{jt}^C = \hat{Y}_{1,t}^C$. Thus, we theoretically construct the appropriate counterfactual for Gothenburg. Furthermore, we apply the weights derived in the synthetic counterfactual for Gothenburg in terms of NO_2 to the variable asthma & COPD, to estimate the effect of the congestion charge on emissions and respiratory health using the same counterfactual for both outcomes. This approach allows us to explore the impact of changes in air quality due to the congestion charge and corresponding shifts in respiratory health outcomes.

6.3. Robustness Tests

We conduct robustness tests to assess the statistical significance and causality of the results. These tests include two placebo tests (in-space and in-time), an in-time validation, and a leave-one-out analysis. The in-space and in-time placebo tests help determine a significant treatment effect and ensure that the observed effects are not due to random variation. The in-time validation checks the consistency of the results over different pre-treatment periods. Finally, the leave-one-out analysis tests the robustness of the results by sequentially excluding each municipality in the donor pool from the analysis.

In the in-space placebo test, we will assign the treatment to each municipality in the donor pool, with a non-zero weight, where a congestion charge was not implemented, and compare the placebo results to the real treatment result for Gothenburg. This helps determine if the treatment effect observed in Gothenburg is unique and stands out. The placebo effects should not generate a treatment effect; otherwise, the real results could be due to randomness. For the in-time placebo test, we will assign the treatment to Gothenburg at another time when the treatment did not exist, from 2013 to 2012 and then to 2011. To verify whether the treatment effect is more likely to be a real treatment effect or is due to randomness. The pre-treatment emission and asthma & COPD trends must remain consistent to validate the credibility of our post-treatment analysis.

For the in-time validation test, we check whether the results are sensitive to the pre-treatment period specification, to be transparent and to check if there is a substantial divergence in the quality of the model when we change the pre-treatment years from three years, 2010-2013, to two years, 2011-2013 and then to only one year 2012-2013. The leave-one-out analysis involves discarding one municipality at a time, creating a synthetic counterfactual of Gothenburg for each iteration where a municipality in the donor pool with a non-zero weight is left out. This test enables us to assess how much our results depend on any single control municipality (Abadie et al. 2015). Thus, we will create four synthetic counterfactuals for Gothenburg, leaving one municipality out at a time.

To assess the robustness tests, we use MSPE ratios of the pre-and post-treatment to make a ratio that is easy to compare.

$$MSPE = \frac{1}{N}\sum_{i=1}^{N}(y_i - \hat{y}_i)^2 \rightarrow MSPE\ ratio = \frac{MSPE_{post}}{MSPE_{pre}}$$

If the ratio is around one, the $MSPE_{pre}$ and the $MSPE_{post}$ are approximately equal, indicating no substantial treatment effect. If the ratio is small, the denominator, $MSPE_{pre}$, is larger than the numerator, $MSPE_{post}$. Meaning that the counterfactual mimics the observed values in post-treatment more closely than in the pre-treatment period. Indicating a suboptimal synthetic fit, with an insubstantial treatment effect. If the ratio is large, the $MSPE_{pre}$ is smaller than the $MSPE_{post}$, and a large deviation in the post-treatment period from a close synthetic fit in the pre-treatment period indicates a potential treatment effect. We also assess the ratios by ranking them; for example, for an in-space placebo test, if the MSPE ratio for the treated unit is substantially larger than the placebo-treated donor pool, we are more confident that the

treatment effect is unique and not due to randomness, possibly indicating a statistically significant effect (Andersson, 2019).

7. Results

This section presents the outcomes of the synthetic control method, followed by a robustness assessment to ascertain causality. Initially, we detail the optimization process, determining the weights and synthetic values and creating Gothenburg's counterfactual in section 7.1. Next, the synthetic fit for NO_2 and asthma & chronic obstructive pulmonary disease (COPD) is presented in section 7.2. Finally, the robustness is tested through four methods: in-time validation, in-space placebo, in-time placebo, and a leave-one-out analysis in section 7.3. We end with a summary conclusion.

7.1. Construction of the Synthetic Counterfactual

For a detailed explanation of the derivation of weights and their critical role in the synthetic control method (SCM), see Section 5.2. Table 7.1.1 displays the weights, summing to 100%, constituting the synthetic counterfactual for Gothenburg.

Table 7.1.1: Weights for synthetic counterfactual of Gothenburg

Municipality	Weights
Borås	0,168
Botkyrka	0,000
Helsingborg	0,000
Landskrona	0,000
Lund	0,000
Malmö	0,121
Mölndal	0,570
Norrtälje	0,000
Uppsala	0,140
Sum	1,000

Note: To avoid extrapolation, all weights are between $0 \leq w_j \leq 1$ and $\sum_{j=2}^{J+1} w_j = 1$. The weights are rounded to three decimal places, indicating that four municipalities from the donor pool significantly contribute to the counterfactual. Specifically, Mölndal contributes the largest share at 57%, followed by Borås at 17%, Uppsala at approximately 14%, and Malmö at around 12%. Collectively, these weights sum to 100%.

The weights are essential to estimating the counterfactual and key to determining synthetic values for NO_2 and Asthma & COPD. As an example, for a specific time *t*, the level of NO_2 for each respective municipality is Malmö = 30 µg/m³, Mölndal = 25 µg/m³, Borås = 20 µg/m³,

and Uppsala = 20 µg/m³.[6] Multiplying the weights associated with each municipality to the NO_2 level and summing the products gives a synthetic NO_2 value of $24\ \mu g/m^3$ for Gothenburg at time *t*. $30 * 0.121 + 25 * 0.570 + 20 * 0.168 + 20 * 0.140 = 24.04 \approx 24\ \mu g/m^3$

Our analysis seeks to determine the congestion charge's effect on air pollution and the impact of air pollution on respiratory health. By applying the weights derived in the synthetic counterfactual for Gothenburg in terms of NO_2 to the variable for asthma & COPD, we can estimate the respiratory health had the congestion charge not been introduced. This approach allows us to explore the impact of changes in air quality due to the congestion charge and corresponding shifts in respiratory health outcomes, which would otherwise have been difficult to assess.

Table 7.1.2 outlines the observed average values for Gothenburg against the estimated synthetic averages and the donor pool averages across predictor variables. The model's performance exhibits variability; notably, the estimate for Gothenburg's synthetic population size is 116 600, compared to the actual population of 517 102. This difference is anticipated, given that Stockholm is excluded because the congestion charge was already implemented there in 2007, making it unsuitable for the donor pool. Moreover, other larger municipalities are excluded from the donor pool due to missing data for NO_2 and non-responses from the Swedish counties, resulting in a sample average of 120 839.

The model achieves a closer approximation for the other variables. The synthetic average temperature is approximately 7°C, compared to Gothenburg's actual average temperature of around 8°C. Concerning air quality, the "NO_2 > 90 µg/m³" variable reveals that Gothenburg experiences approximately 14% of days with one or more hours surpassing the 90 $\mu g/m^3$ threshold, which is deemed to be particularly hazardous for human health, as reported by Coria et al. (2015). This figure contrasts with the synthetic estimate of 10% and the sample mean of 6%, suggesting that Gothenburg encounters a higher frequency of days with critical NO_2 levels relative to the municipalities in the donor pool.

[6] µg/m³ = Micrograms per cubic metre

Table 7.1.2: Nitrogen dioxide (NO_2) predictor means.

Predictors	Unit of measurement	Gothenburg	Synthetic Gothenburg	Sample Average
Temperature	Celsius (°C)	8,026	7,188	7,094
Precipitation	Millimeters (mm)	0,096	0,093	0,075
Snow	Centimeters (cm)	0,007	0,008	0,009
Windspeed	Kilometers per hour (km/h)	10,549	11,654	11,701
Wind-direction	Degrees (°)	172,767	183,248	194,566
Cloud cover	Percentage (%)	68,553	66,333	63,699
Visibility	Kilometers (km)	24,019	22,761	22,512
Feels like	Celsius (°C)	6,772	5,556	5,448
Dew	Celsius (°C)	4,121	3,799	3,934
Humidity	Percentage (%)	78,281	80,912	82,078
Snow depth	Centimeters (cm)	3,002	3,899	2,936
Sea Level Pressure	Millibars (mbar)	1012,567	1012,493	1012,705
Municipality Area	Square kilometers (km^2)	448	561	724
Income	SEK (per capita, thousands)	0,479	3,217	2,766
Post-secondary education, 3 years or more	Percentage of population (%)	0,191	0,165	0,149
Post-secondary education, less than 3 years	Percentage of population (%)	0,126	0,110	0,107
Postgraduate education	Percentage of population (%)	0,012	0,017	0,014
Pre-gymnasial education shorter than 9 years	Percentage of population (%)	0,038	0,039	0,046
Pre-gymnasial education, 9 (10) years	Percentage of population (%)	0,095	0,100	0,105
Gymnasial education, 3 years	Percentage of population (%)	0,159	0,157	0,157
Gymnasial education, up to 2 years	Percentage of population (%)	0,126	0,136	0,149
Information on level of education is missing	Percentage of population (%)	0,018	0,015	0,020
Population Size	Count	517102	116600	120839
Number of Vehicles	Count (per capita)	0,343	0,429	0,421
Unemployment Rate	Count (per capita)	0,048	0,036	0,043
NO_2 > 90 µg/m3	Binary dummy variable	0,143	0,095	0,059
Simulated Traffic	Cars per hour (cars/h)	826	641	595

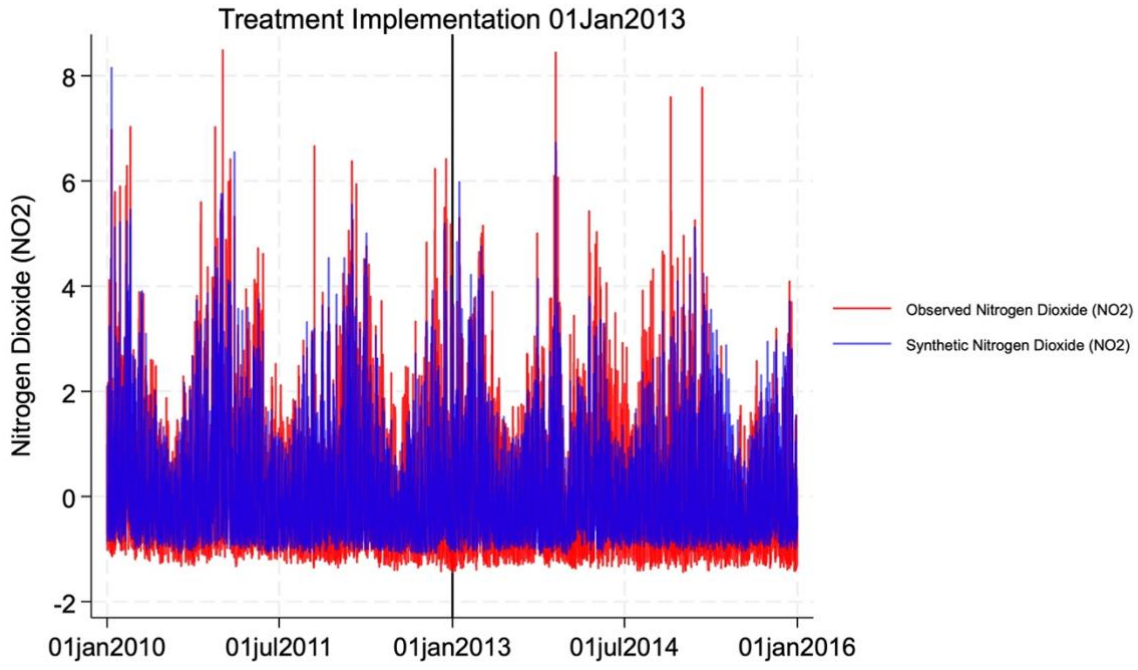
Note: The first column represents Gothenburg X_1, the second column represents Synthetic Gothenburg, X_0W^* and the third column represents the sample mean X_j of the municipalities in the donor pool. All variables are averaged for the 2010-2015 period. Missing values of NO_2 are filled by calculating the average NO_2 level by municipality, year, month, day and hour.

7.2. Synthetic Fit

A deeper discussion on the importance of a good synthetic fit for identification can be read in section 6.2. Assessing the synthetic fit in Figure 7.2.1 illustrates that our model captures most of the observed variation in terms of NO_2. However, it is less effective at mimicking the pollutant's peaks during certain hours of the day. This limitation is likely due to the detailed granularity of our data, using hourly observations over several years. The challenge arises when no municipality in the donor pool exhibits similar extreme spikes in air pollution, making it difficult to mimic Gothenburg's most severe air pollution episodes. Furthermore, the graphical representation in Figure 7.2.1 does not conclusively demonstrate a shift in NO_2 levels in the

post-treatment period. Therefore, we aggregate the observational rate to a monthly rate to more easily determine how well we mimic the air pollution with the synthetic counterfactual.

Figure 7.2.1: Synthetic fit of Nitrogen dioxide (NO_2), hourly rate.



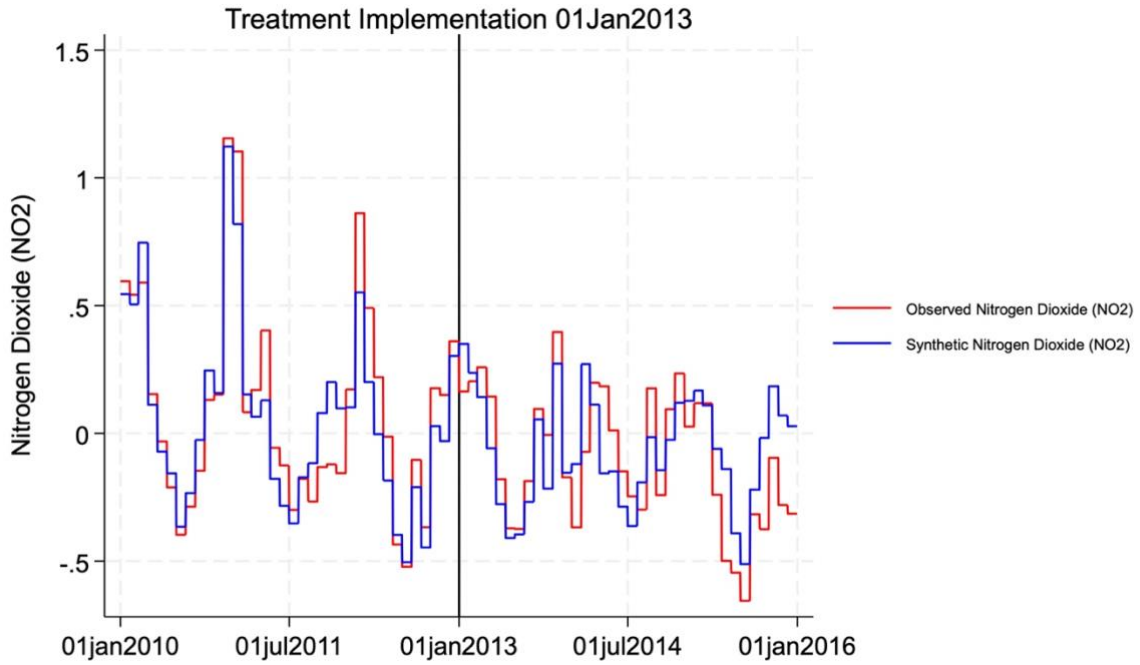
Note: The graph shows pre- and post-treatment synthetic fit with an hourly rate, with the red area in the graph showing the observed NO_2 emissions in Gothenburg and the blue area showing the synthetic emission levels mimicking the observed values.

Aggregating the data to a lower level of granularity, the monthly observational rate for NO_2 offers a clearer understanding of the synthetic fit compared to the hourly rate. Figure 7.2.2 illustrates a more solid pre-treatment fit than the hourly rate. There is a slight deviation around the end of 2011. Otherwise, we are able to accurately mimic the NO_2 trend and level in the pre-treatment period. The mean difference between the observed and synthetic values for hourly and monthly rates is -0.034 in the post-treatment period. This means that the observed, treated values are lower than the synthetic counterfactual values.

We apply the weights derived in the synthetic counterfactual for Gothenburg in terms of NO_2, see Table 7.1.1, to the variable for asthma & COPD, to estimate the effect on respiratory health from the congestion charge, through a change in NO_2. Observing Figure 7.2.3, the synthetic asthma & COPD effectively mimic most of the variation of the observed counterpart. However, we are unable to mimic the "peaks" and the "valleys" of the observed asthma & COPD rates. The mean difference between the observed and synthetic values in the post-treatment period is

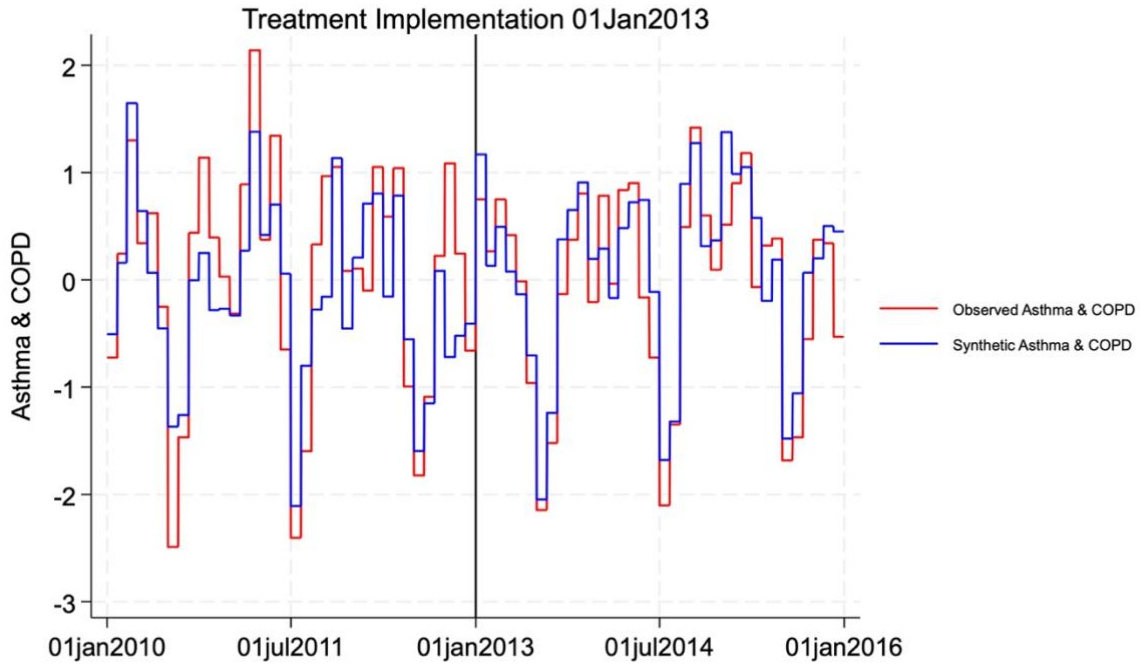
-0.153, which means that the observed treated values are lower than the synthetic counterfactual values.

Figure 7.2.2: Synthetic fit of Nitrogen dioxide (NO_2), monthly rate.



Note: The graph shows pre- and post-treatment synthetic fit using a monthly rate, with the red area in the graph showing the observed NO_2 emissions in Gothenburg and the blue area showing the synthetic emission levels.

Figure 7.2.3: Synthetic fit of Asthma & COPD



Note: The graph shows the pre-and post-treatment synthetic fit for asthma & COPD hospitalizations. The synthetic control for asthma & COPD is constructed using the same weights for creating Synthetic NO_2.

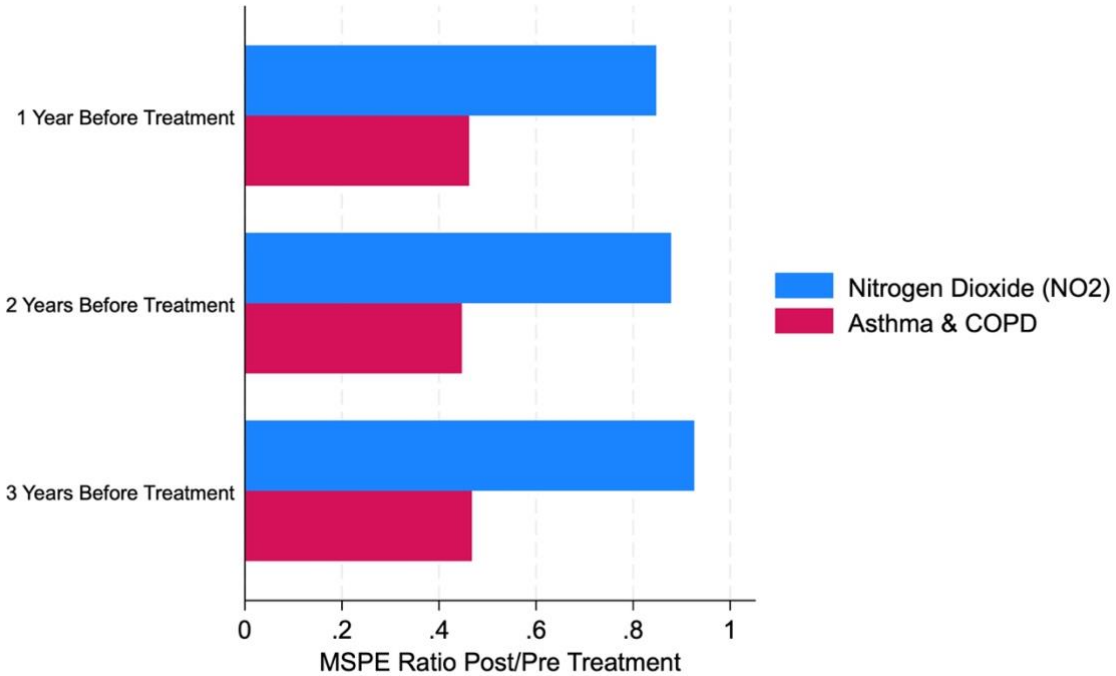
7.3. Robustness Tests

In this section, we will scrutinize the test results and understand how they change as we alter the parameters of the treatment intervention. We will conduct four robustness tests on the municipalities with non-zero weight, discussed in section 6.3.

7.3.1. In-Time Validation Test

In this robustness test, we examine how the pre-treatment fit and post-treatment effect change as we adjust the years included in the pre-treatment specification. Looking at the ratios for NO_2, represented by the blue bars in Figure 7.3.1, we find that the MSPE ratios for all three specifications are similar, hovering around 0.8. However, there is a slight increase as the treatment period extends. This suggests no significant deviation in the post-treatment period compared to the pre-treatment fit when altering the pre-treatment period. Regarding asthma & COPD, the MSPE ratios remain consistent across all treatment specifications, approximately at 0.45. In summary, our results appear robust regarding changes in the treatment period. This consistency is further evidenced in Appendix 1.2, 1.3, 1.4, and 1.5, where the synthetic fit for different specifications yields similar outcomes in terms of both pre-treatment fit and post-treatment results.

Figure 7.3.1: Robustness test: In-time validation

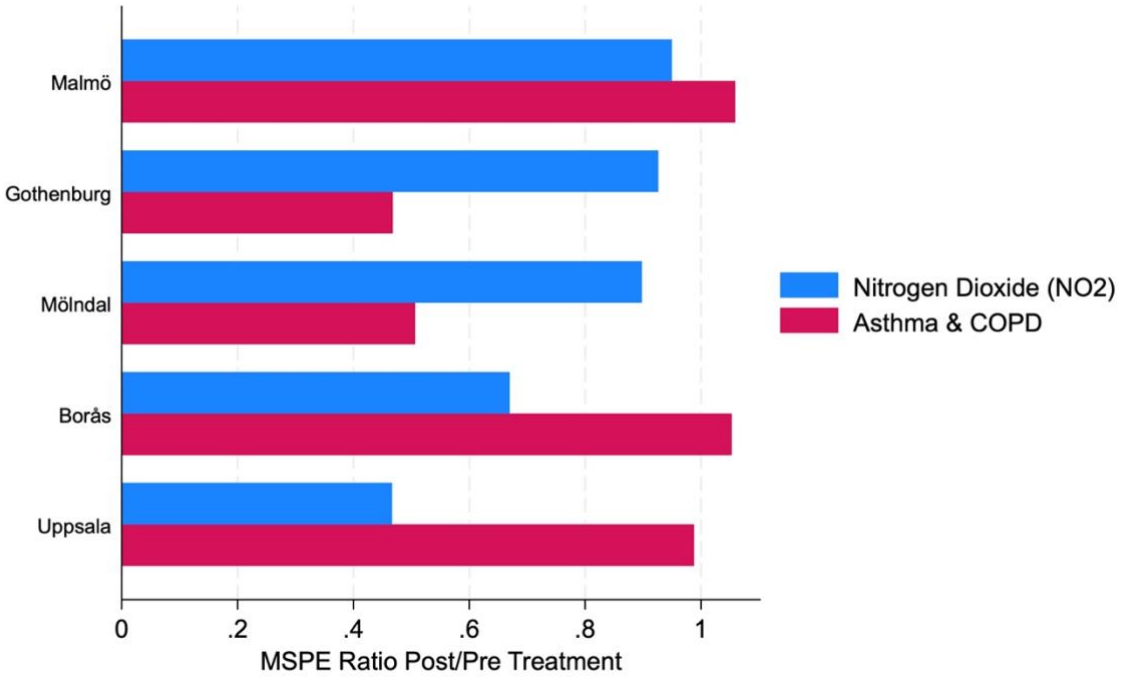


Note: The blue bars represent MSPE ratios for NO_2 across different time specifications, similar to around 0.8, with slight increases as the treatment period expands. For asthma & COPD, MSPE ratios represented by red bars remain stable at approximately 0.45. Overall, the results are robust to changes in the pre-treatment period.

7.3.2. In-Space Placebo Test

In this robustness test, we assign a placebo treatment of implementing a congestion charge, as in Gothenburg, to each municipality in the donor pool with non-zero weight. The motivation is that if a municipality in the donor pool receives a placebo treatment, it should not exhibit any placebo treatment effect. Identifying a placebo treatment effect would suggest that any treatment effect observed in Gothenburg could be non-unique or due to random variation.

Figure 7.3.2: Robustness test: In-space placebo



Note: For NO_2 (blue bars), Gothenburg's ratio is about 0.9, and Malmö's is around 0.95, both under one, indicating minimal deviation post-treatment. Gothenburg's ratios do not outrank the placebo treatments. For asthma & COPD (red bars), Gothenburg's ratio is low at around 0.45, compared with placebo effects, showing no unique treatment effect.

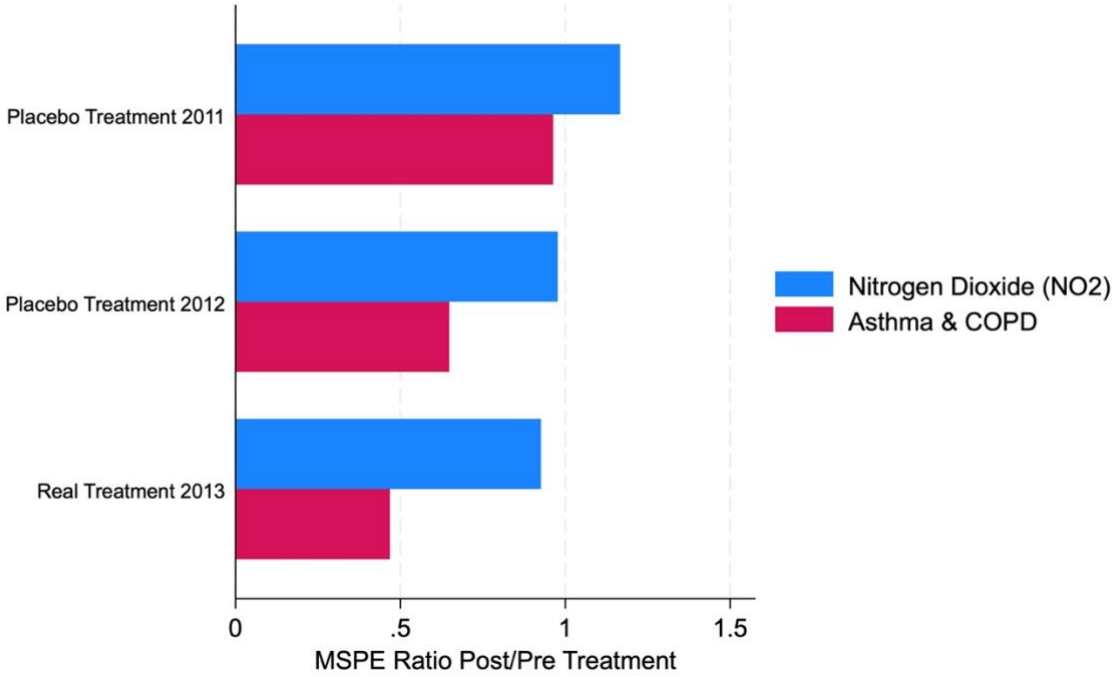
The original synthetic MSPE ratios for Gothenburg are included as a reference for comparison. Beginning with NO_2, indicated by the blue bars in Figure 7.3.2, Gothenburg's ratio is approximately 0.9, surpassed only by Malmö at approximately 0.95. No MSPE ratio exceeds 1, indicating no substantial treatment effect. Gothenburg is not ranked the highest nor unique compared to the placebo treatments for the municipalities in the donor pool. Turning to asthma & COPD, the ratio for Gothenburg is ranked the lowest at around 0.45, which means that the result is neither unique nor outranking the placebo treatment effects for the municipalities in the donor pool. In Appendix 1.6 and 1.7, we show the synthetic fit of the placebo treatments. What becomes evident is that Gothenburg is not unique in getting a good pre-treatment fit

compared to the donor pool. Nor is the real treatment generating enough variation in the post-treatment period to stand out amongst the placebo treatments.

7.3.3. In-Time Placebo Test

In the in-time placebo test, we assigned a placebo treatment to Gothenburg, shifting the real treatment from January 2013 to January 2012 and January 2011, with a one-year pre-treatment period and three years afterward. This analysis helps us determine whether the real treatment implementation effect is unique or more likely attributable to random variation.

Figure 7.3.3: Robustness test: In-time placebo



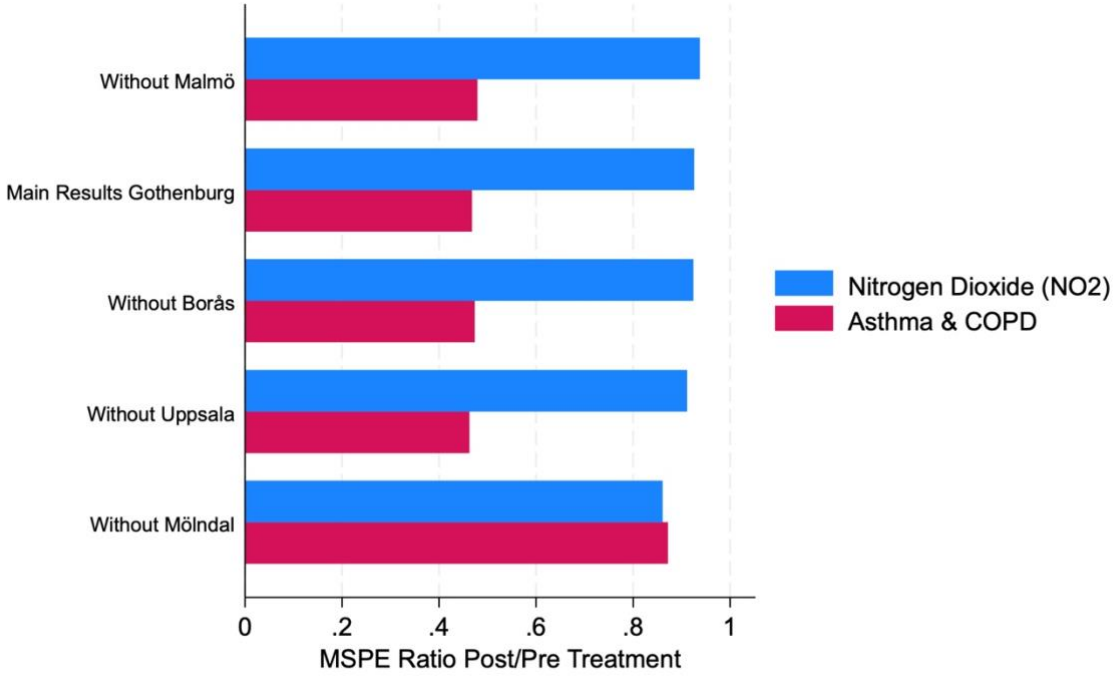
Note: Examining the MSPE ratios for NO_2 in the figure, we see that placebo treatments slightly outperform the real treatment. Thus, the real effect appears non-unique and possibly random compared to the placebo treatments. For asthma & COPD, ratios for placebo treatments in 2012 and 2011 exceeded the real treatment ratio of 0.45. This suggests that the real treatment effect may not be unique.

Analyzing the MSPE ratios in Figure 7.3.3, the in-time placebo treatments for NO_2 are slightly higher than the real treatment. Thus, comparing the placebo treatments to the real ones in 2013, the real effect does not seem unique since the placebo treatments perform better than the real treatment. Regarding asthma & COPD, the ratio for the real treatment is roughly equal to 0.45, whereas the ratio for placebo treatment in 2012 is roughly 0.6. Moving to placebo treatment in 2011, the ratio is approximately one. Indicating that the real treatment effect is not unique; since the placebo treatments have an equal or even larger effect than the real one, we cannot exclude that the effect is due to random variation.

7.3.4. Leave-One-Out Test

This robustness analysis scrutinizes the synthetic control by excluding each donor municipality with a non-zero weight, one at a time.

Figure 7.3.4: Robustness test: Leave-one-out



Note: Excluding the municipalities generally produces MSPE ratios consistent with the main counterfactual. However, excluding Mölndal slightly reduces the MSPE ratio from 0.9 to 0.85. Conversely, the counterfactual for asthma & COPD is sensitive to excluding Mölndal, as its absence increases Malmö's weight, raising the MSPE ratio from 0.45 to 0.85.

From Figure 7.3.4, the MSPE ratios closely aligned with the main counterfactual. However, when Mölndal is excluded, there is a slight decrease in the MSPE ratio from 0.9 to 0.85, suggesting some sensitivity towards Mölndal for NO_2 levels; from Appendix 1.8, the synthetic fit without Mölndal is solid. Asthma & COPD indicate a similar robust relationship for leaving out each municipality except excluding Mölndal, leading to a notable increase in the MSPE ratio from 0.45 to 0.85. This increase primarily stems from Mölndal's absence, the optimization process assigns more weight to Malmö in the synthetic control. Given Malmö's rising asthma & COPD rates, potentially linked to factors such as increased immigration and poor living conditions, driving up the rates of respiratory ailments, as discussed in section 5.4. Analyzing the trend of asthma & COPD when excluding Mölndal in Appendix 1.9, the synthetic control poorly matches the pre-treatment asthma & COPD levels and overestimates the post-treatment period.

In conclusion, we attain a satisfactory pre-treatment fit for NO_2 and a reasonable fit for asthma & COPD to identify the treatment effect. The mean difference, indicating the treatment effect for NO_2 in section 7.2, is -0.034 standard deviations from the mean of $30\ \mu g/m^3$. Put in real, non-standardized terms, about -0.7 μg/m³ reduction in NO_2. Similarly, for asthma & COPD, the treatment effect, or mean difference, is -0.153 standard deviations from the mean of 488 monthly cases. Put in real, non-standardized terms, about 14 fewer monthly hospitalizations for asthma & COPD. The robustness tests show the results are robust in changing the included pre-treatment period. From the in-space placebo test, we observe that the real treatment for Gothenburg does not seem to generate enough variation to differentiate it from random variation in either NO_2 or asthma & COPD. The in-time placebo test shows that the real treatment for Gothenburg is outperformed by the in-time placebo treatments when we change the treatment timing from 2013 to 2012 and 2011. Lastly, the leave-one-out test indicates that the result for Gothenburg is robust to leaving any one municipality out of the donor pool, with the caveat that leaving Mölndal out, gives a considerable weight to Malmö which confounds the results for asthma & COPD, as indicated by Appendix 1.9, since Malmö has an increasing rate of respiratory illness, possibly due to an increase in immigrants and poor indoor air pollution as discussed in section 5.4.

In summary, based on the comprehensive evidence presented in this thesis, there does not appear to be a significant impact of the congestion charge in Gothenburg on air pollution or respiratory health.

8. Discussion

Concerning the first research question, we are unable to accurately identify a treatment effect of the congestion charge on air pollution levels. We obtain a satisfactory pre-treatment fit but are unable to mimic the pollutant's peaks during certain hours of the day. However, we can show a better pre-treatment fit using aggregated monthly rates, mimicking the air pollution trend and absolute levels of Gothenburg very closely. We observe a slight decrease in air pollution in the post-treatment period. Regarding the second research question, for asthma & COPD we can show a reasonable pre-treatment fit and a divergence in the post-treatment period. We observe a decrease in the rate of respiratory disease in the post-treatment period.
The robustness tests show that the results are robust to change the pre-treatment period. The in-space and in-times placebo tests show that the treatment for Gothenburg does not seem unique and differentiates itself from the placebo treatments. The leave-one-out test indicates that the

result for Gothenburg is robust to leaving any one municipality out of the donor pool except Mölndal, which confounds the results for asthma & COPD. In essence, the treatment for Gothenburg is not generating enough variation in the post-treatment period to differentiate itself from the donor pool placebo treatments. The results we find for air pollution are insignificant and do not seem to indicate a substantial reduction in NO_2 from the congestion charge. Furthermore, the results for asthma & COPD do not seem unique to Gothenburg, and we cannot link the decrease to a change in air pollution. Possibly showing that the asthma & COPD rates are explained by other unaccounted factors than the congestion charge.

We fail to detect a treatment effect for a few reasons. We use nine municipalities in the donor pool due to a lack of data for other municipalities. Because accessing the register data from the counties is time-consuming and air pollution is only accurately measured for a few municipalities. Thus, we are leaving nearly 280 out of the 290 possible municipalities we could have used in the donor pool to improve the synthetic fit. Furthermore, the SCM typically favors aggregate-level data, whereas analyzing air pollution necessitates granularity, often observed in hourly rates to capture volatile fluctuations. Therefore, our study adopts hourly observations to accommodate the dynamic nature of air pollution, particularly NO_2, influenced by traffic patterns and weather variations throughout the day. Another aspect is that the lack of identification of a treatment effect can be linked to the discussion on Pigouvian taxation in section 3.1, namely that the tax is set too low, with an average fee of approximately 150 SEK per month, to effectively discourage people from driving at peak congestion hours. Thus, the tax rate does not internalize the social cost of polluting, which is the essence of a Pigouvian tax, to connect a sufficient cost to the negative externality.

Other policies might influence the results than an exact vehicle taxation scheme. Since the tax in Gothenburg is so low, there could be similar effects in deterring people from driving into the city from parking fees. For example, parking in Gothenburg at 30 SEK/h is far more expensive than driving during peak congestion at 22 SEK. The model and analysis do not include these aspects and alternative policies that discourage driving in the donor pool municipalities. This could affect the results, as the underpriced congestion tax is equally or less effective than other policies, which leads to not finding unique results for the Gothenburg congestion charge.

9. Conclusion

This thesis employs the synthetic control method (SCM) to explore the impact of the congestion charge in Gothenburg on air pollution and respiratory health outcomes, specifically focusing on nitrogen dioxide (NO_2) levels and the incidence of asthma & chronic obstructive pulmonary disorder (COPD). The study investigates whether the policy effectively reduced air pollution and improved public health.

The results indicate that while there is a measurable reduction in NO_2 levels during the post-intervention period, this decrease is not statistically significant, suggesting that the congestion charge has not substantially reduced NO_2 levels. This could be due to the set congestion tax rate not being high enough to discourage individuals from driving during peak hours of congestion. Thereby failing to align with the theoretical framework of Pigouvian taxation, which advocates for a tax rate that internalizes the social costs of pollution. Regarding health outcomes, the thesis does not find a significant treatment effect on the rate of asthma & COPD hospitalizations.

However, what is the actual social cost associated with the environmental damages from traffic congestion? One of the main challenges is to set the tax at an optimal level. Thus, calculating the social cost of driving in Gothenburg would be very beneficial since we could adjust the tax accordingly to improve the effectiveness of the congestion charge.

An important consideration is that the congestion tax, aimed at reducing traffic at certain hours through a uniform charge for all drivers, does not differentiate based on income. While its primary goal is to discourage driving to alleviate traffic and pollution, it imposes the same financial burden on every motorist. According to the findings of this thesis, if the congestion tax fails to achieve its intended environmental objectives, it essentially serves only to generate tax revenue. Consequently, this uniform tax disproportionately impacts lower-income drivers, making it costlier for them to drive and, thus, less redistributive. A more equitable approach might be to adopt a progressive tax based on income, which could be based on the driver's ability to pay, ensuring that the tax burden is shared more fairly and aligning better with principles of equality.

Another way to improve tax equality would be to use a similar setting to the Manhattan congestion charge in New York City. Where people with an annual income of less than $50,000

will pay half the price of the congestion tax after their first ten trips. Another notable approach is the method employed in Melbourne, Australia, where public transport is free of charge within the city center. This strategy highlights the role of public transportation as a substitute for road pricing policies. Implementing a similar policy in Gothenburg could encourage people to substitute driving for public transportation, thereby reducing congestion and pollution.

Given the limited data set and the granularity required to assess air pollution, we recommend further research involving more municipalities and possibly other cities in the Nordic countries since they are similar to Gothenburg in terms of characteristics. Considering that while evaluating a policy using SCM, it is hard to estimate inference with a small donor pool. Furthermore, air pollution data from 2019 onward, captured through satellite imagery, is available from the European Union and NASA and opens the door to a substantially enriched dataset for using SCM in another setting for environmental policy evaluation.

10. References

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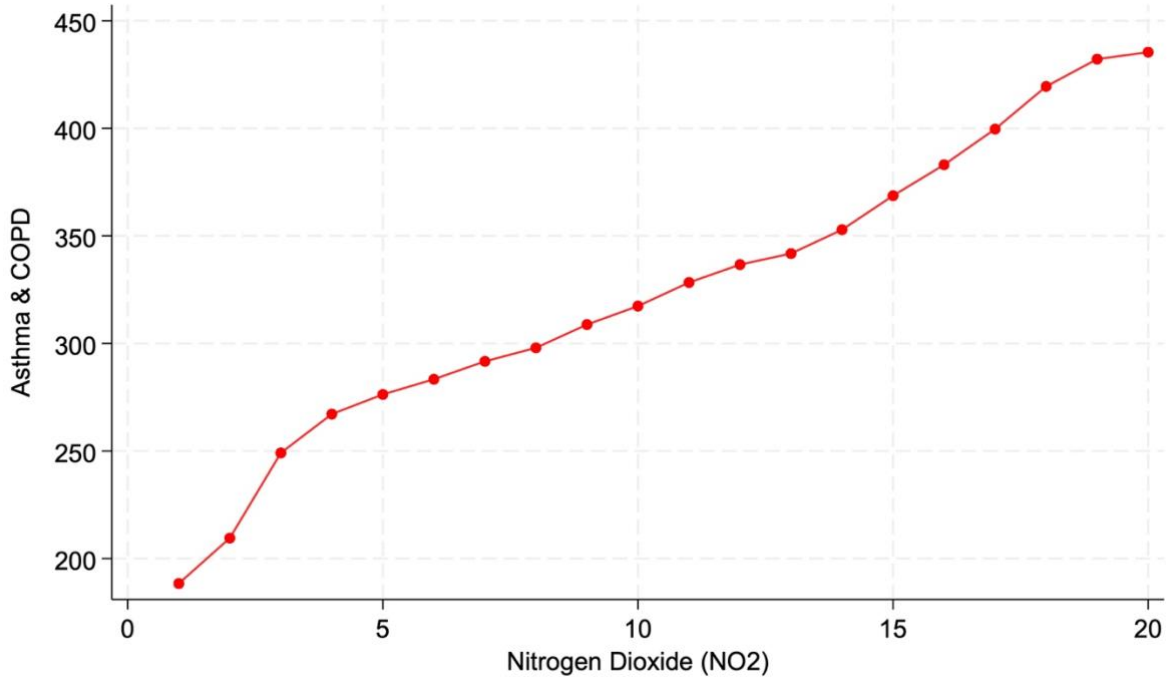
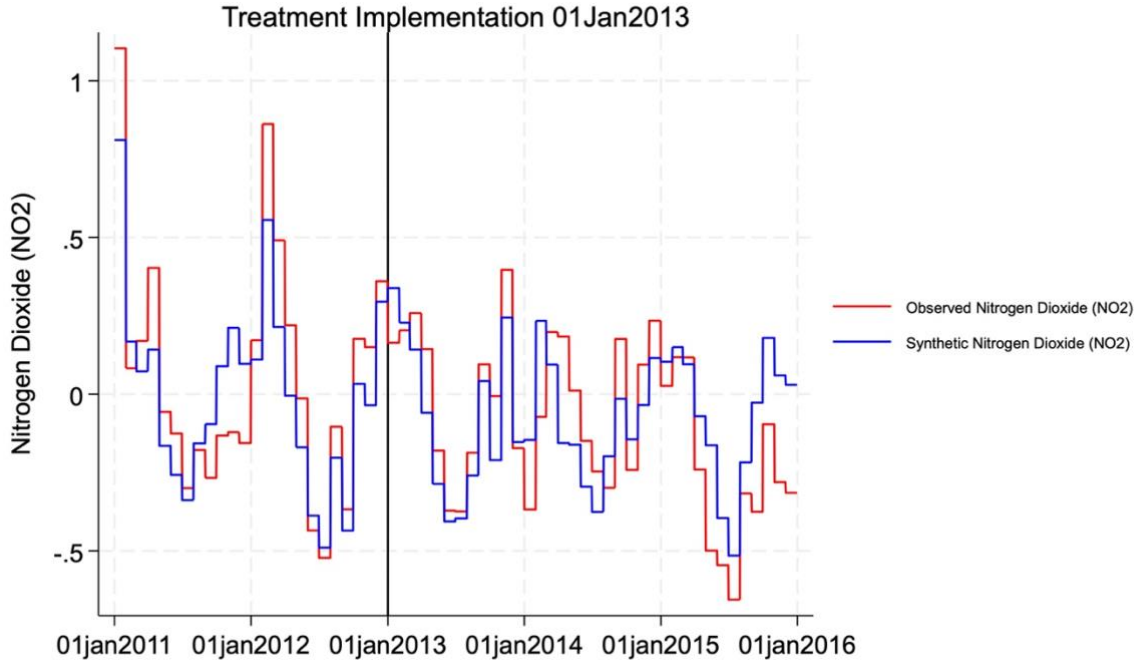
11. Appendix

Appendix 1.1: Scatterplots of NO_2 and asthma & COPD, Without Mölndal



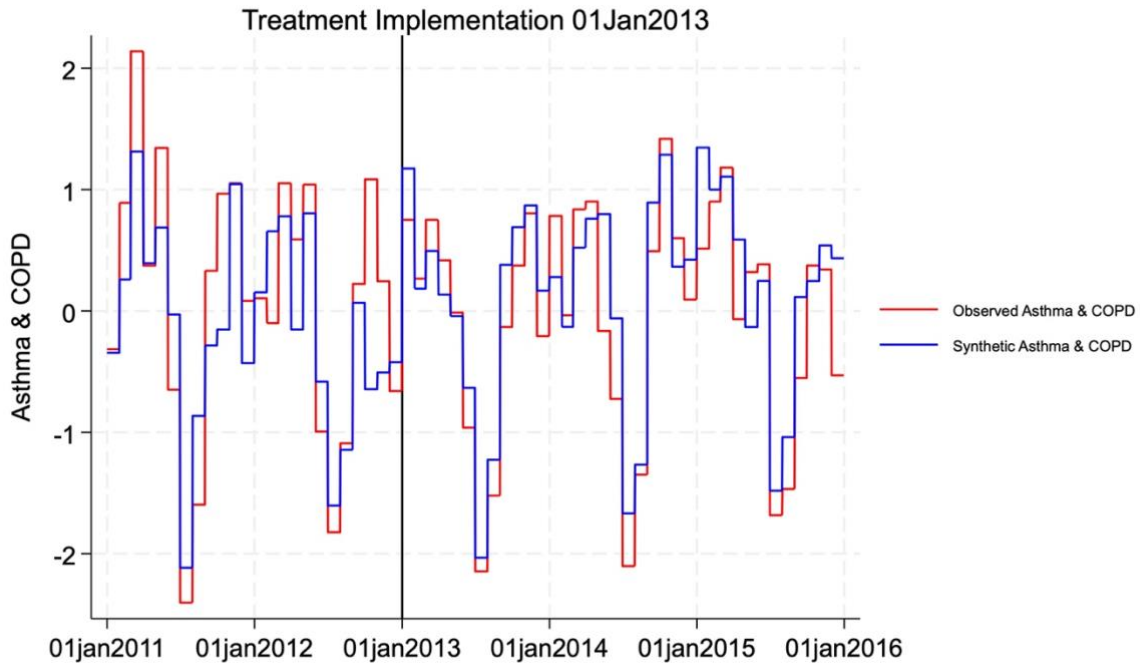
Note: The figure shows the average NO_2 level for asthma & COPD, split into 20 equal parts of the distribution, each constituting 5%.

Appendix 1.2: Synthetic fit of Nitrogen dioxide (NO_2), 2 years prior to treatment



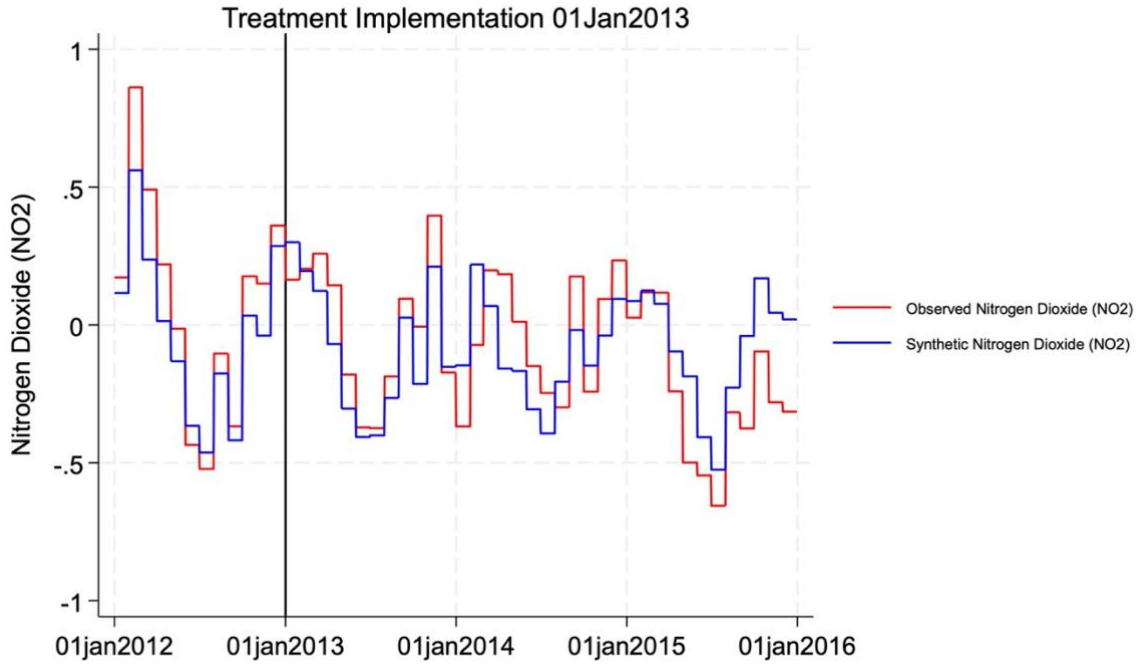
Note: The figure shows the synthetic fit for Nitrogen dioxide (NO_2) after changing the pre-treatment years to 2 years for the in-time validation robustness test.

Appendix 1.3: Synthetic fit of Asthma & COPD, 2 years prior to treatment



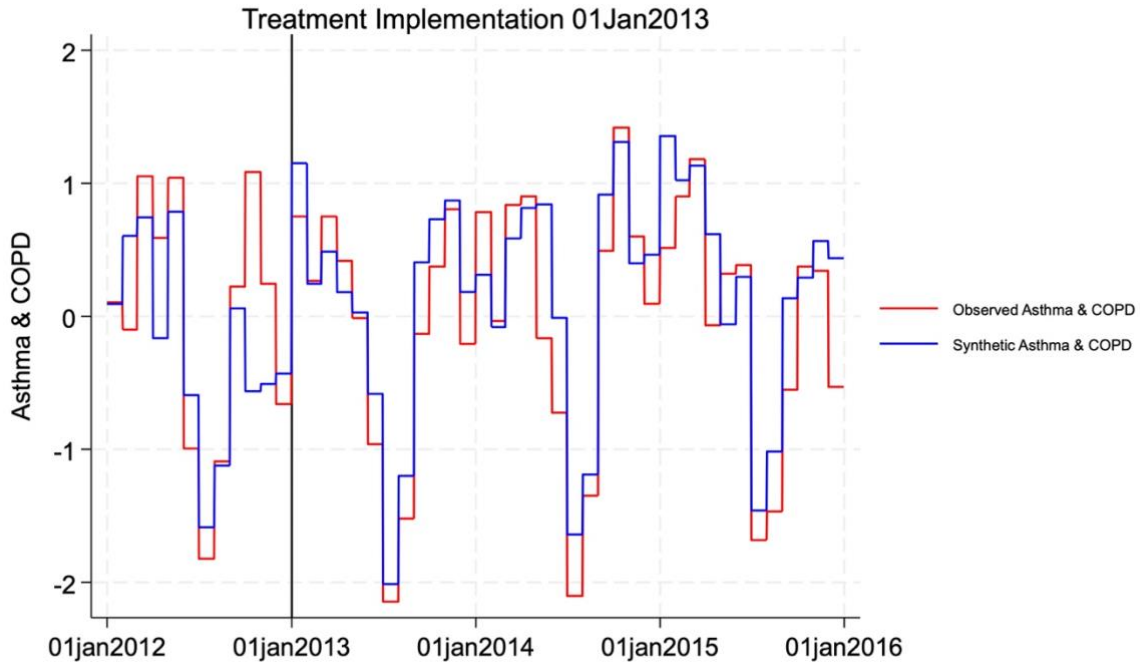
Note: The figure shows synthetic fit for asthma and chronic obstructive pulmonary disease (COPD) after changing the pre-treatment years to a 2-year in-time validation robustness test.

Appendix 1.4: Synthetic fit of Nitrogen dioxide (NO_2), 1 year prior to treatment



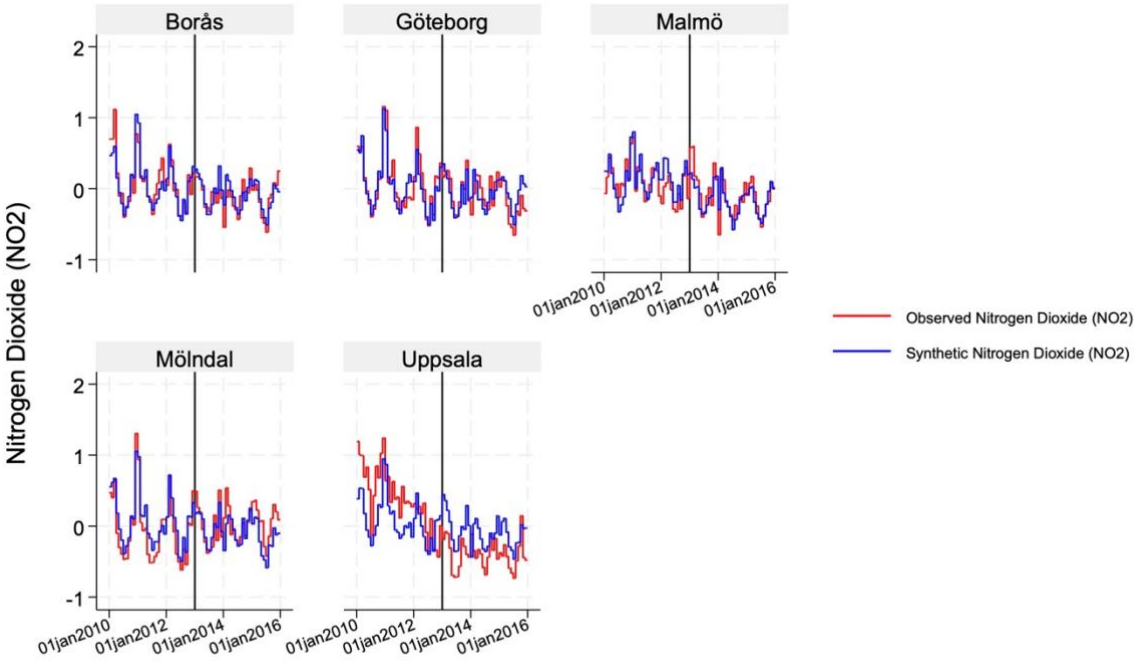
Note: The figure shows the synthetic fit for Nitrogen dioxide (NO_2) after changing the pre-treatment years to a 1-year in-time validation robustness test.

Appendix 1.5: Synthetic fit of Asthma & COPD, 1 year prior to treatment



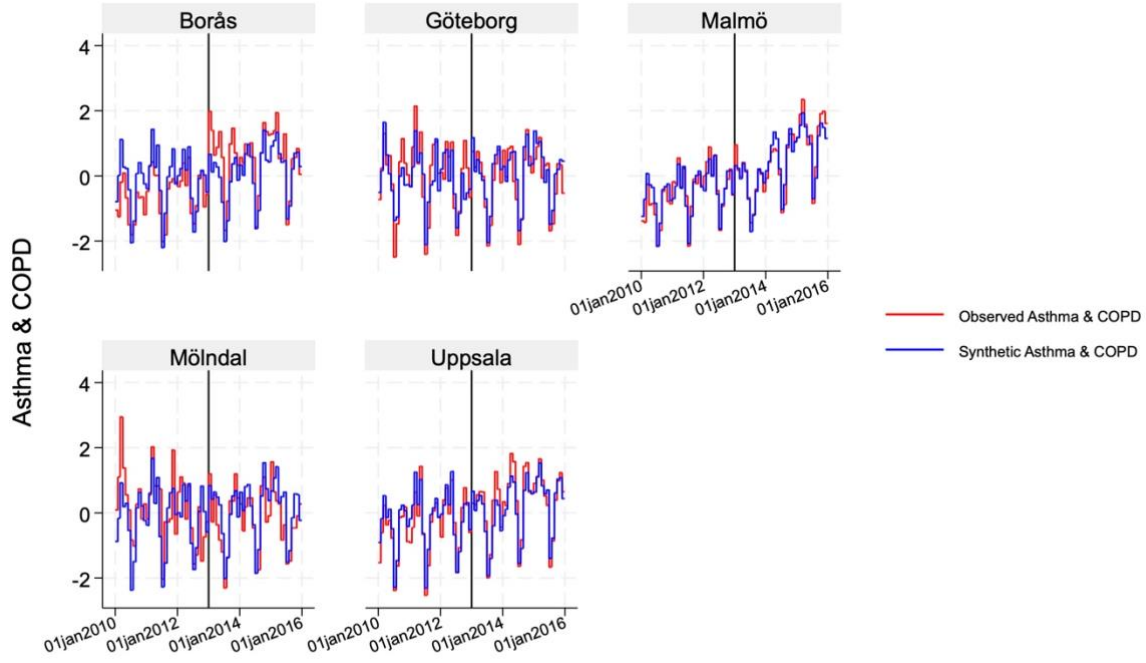
Note: The figure shows synthetic fit for asthma and chronic obstructive pulmonary disease (COPD) after changing the pre-treatment years to a 1-year in-time validation robustness test.

Appendix 1.6: Synthetic fit of Nitrogen dioxide (NO_2), from in-space placebo test



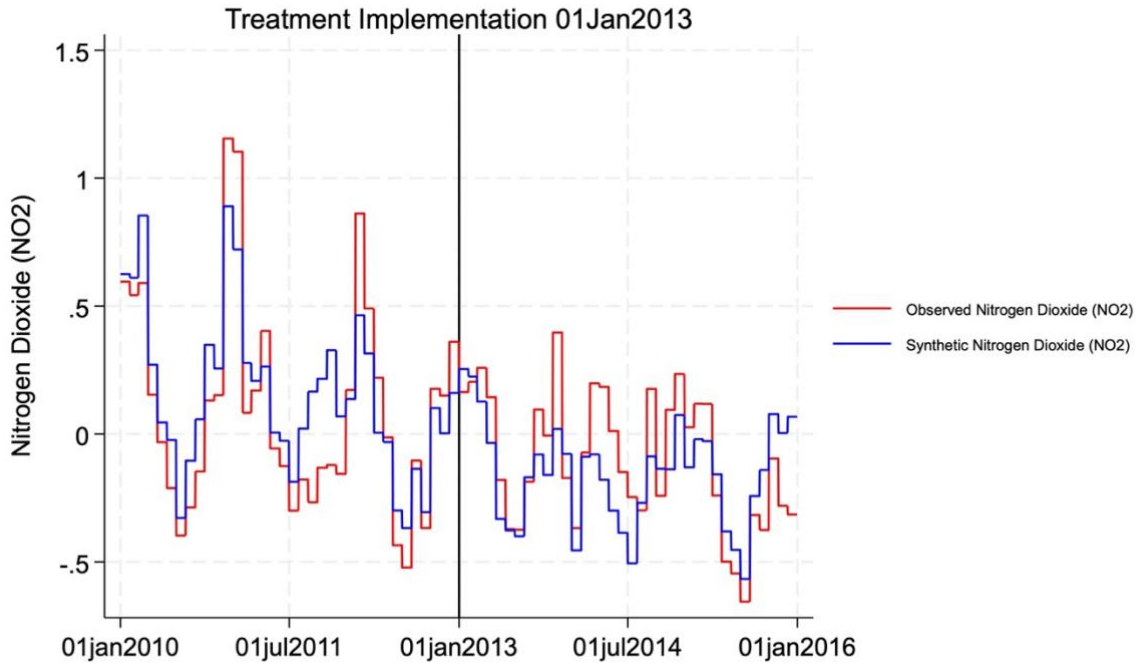
Note: After assigning treatment to them as an in-space placebo test, the synthetic fit for nitrogen dioxide (NO_2) for each municipality in the donor with non-zero weight is shown.

Appendix 1.7: Synthetic fit of Asthma & COPD, from in-space placebo test



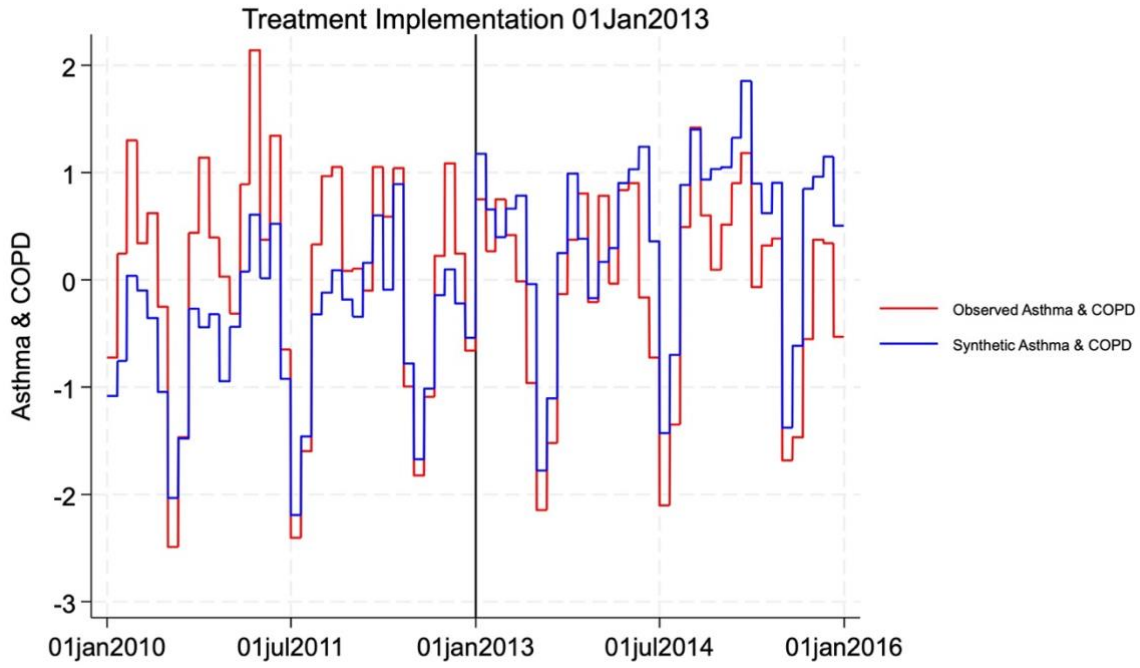
Note: The shows synthetic fit for asthma and chronic obstructive pulmonary disease (COPD) for each municipality in the donor with non-zero weight after assigning treatment to them as an in-space placebo test.

Appendix *1.8: Synthetic fit of Nitrogen dioxide (NO_2), Leaving Mölndal out of the donor pool*



Note: The figure shows the synthetic fit for Nitrogen dioxide (NO_2) after excluding Mölndal from the donor pool in the leave-one-out placebo test.

Appendix 1.9: Synthetic fit of Asthma & COPD, Leaving Mölndal out of the donor pool



Note: The figure shows synthetic fit for asthma and chronic obstructive pulmonary disease (COPD) after excluding Mölndal from the donor pool in the leave-one-out placebo test.