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Common Ragweed Invasion in Sweden: Impacts of the Lag Phase on Human Health

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ABSTRACT

Common ragweed (*Ambrosia artemisiifolia*) is a neophyte from North America that has spread rapidly throughout Europe. Because the pollen of common ragweed is highly allergic, many countries have adopted containment and mitigation measures. The neophyte has only recently established flowering populations in Sweden. We use this well documented case to study the early impacts of an invasive species on human health. Our identification strategy relies on spatial and temporal variation in common ragweed populations and health data for 1998-2011, applying panel-data estimation techniques. To ensure the robustness of our findings, we test and correct for heteroskedasticity and autocorrelation. Our estimates show that an invasive species still in its lag phase can yet have adverse impacts on human health.

Keywords

Common ragweed, lag phase, health effects, panel estimation

This paper is work in progress. The latest version is available from here.

1. Introduction

Common ragweed is a neophyte (invasive species) from North America with a wide ecological tolerance and adaptation capacity that has allowed its rapid spread throughout Europe. The plant has harmful impacts on many sectors, particularly agriculture and human health. In addition to increasing weed control costs for farmers, the pollen of common ragweed is known to cause severe allergic reactions. The allergic reactions associated with an exposure to common ragweed pollen are of two kinds: skin reactions (atopic dermatitis) and illnesses of the respiratory system (allergic rhinitis, hayfever and asthma). Most of the published contributions on the health impacts of common ragweed focus on countries with well-established populations of common ragweed. In contrast, little is know about the impacts of common ragweed in countries where ragweed invasion is still in its lag phase.[1] Our study aims at filling this gap and we find evidence that an invasive species still in its lag phase can have adverse impacts on human health. We chose Sweden as our case study because the ragweed population there is newly established, with the first flowering plant having been documented in the early 1990s (Dahl et al., 1999). To identify the impact of common ragweed pollen, we combine Swedish botanical and health data for the period 1998 to 2011. We use a panel-data estimation approach, correcting for heteroskedasticity and autocorrelation. We find small but significant deleterious effects of ragweed presence on human health and find our estimates to be robust to a battery of robustness checks.

The rest of the paper is organized as follows. We start with a review of the health literature on allergic reactions caused by common ragweed pollen. We then provide an overview of the historical and current spread of common ragweed in Sweden. Our empirical approach, data sources and econometric strategy are presented in the methodology section. We then discuss our findings and describe the tests implemented to check the robustness of our findings. We conclude with a summary of our major findings and a presentation of leads for further research.

2. Health literature on allergic reactions

Common ragweed pollen is highly allergic and a wide range of illnesses has been documented in the health literature (Anzivino-Viricel et al., 2011). The pollen of common ragweed causes asthma more often than any other single pollen allergen (Corsico et al., 2000). A pan-European study of common ragweed sensitization[2] has found that on average 10 percent of the European population is sensitive to common ragweed (Burbach et al., 2009). The incidence of allergic reactions is very heterogeneous across Europe (Bullock et al., 2010). Sensitization rates are highest in countries with well-established common ragweed populations, while these rates are substantially lower in countries with incidental populations, which are still in the lag phase. These countries include Sweden and are

[1] Plant population growth is often modeled with four sequential phases: lag phase (adaptation phase), log phase (exponential phase), stationary phase (zero growth phase), and death phase (decline phase). During the lag phase, a population adapts itself to the habitat's conditions and the population size is small.

[2] Sensitization is the production of a specific type of allergic antibody by the immune system resulting in a positive allergy test. It is possible for a person to make a specific allergic antibody to pollen, without experiencing allergic reactions when exposed to this pollen.

found mainly in Northern Europe. Although the incidence of allergic reactions depends on the genetic diversity of the human population as shown by Warner (1999) for instance, we are assuming in our analysis that genetic heterogeneity is not large enough at the county level to generate bias in our estimates. We discuss this issue in more detail and propose strategies to better account for genetic heterogeneity in the discussion section.

A higher concentration of ragweed pollen in the air is linked to an increase in the number of consultations for allergic rhinitis (Breton et al., 2006). Bullock et al. (2010) identified a pollen concentration of 5-20 grains per m^{-3} as the threshold for allergic symptoms. Other studies have shown that people are already affected by 1-2 grains per m^{-3}. Pollen traps in Sweden routinely record air concentration rates of common ragweed pollen at the lower end of this scale (Sikoparija et al., 2013). Patients that are allergic to common ragweed are found more often in urban areas (Shea et al., 2008; D'Amato and Cecchi, 2008), where higher temperatures result in a pollen season that starts earlier and causes the release of more pollen (Ziska, 2008). In addition, sensitization rates are increased by lower air quality and higher urbanization intensity (Ziska et al., 2003). This indicates the possibility of temporal heterogeneity which we address in our identification strategy.

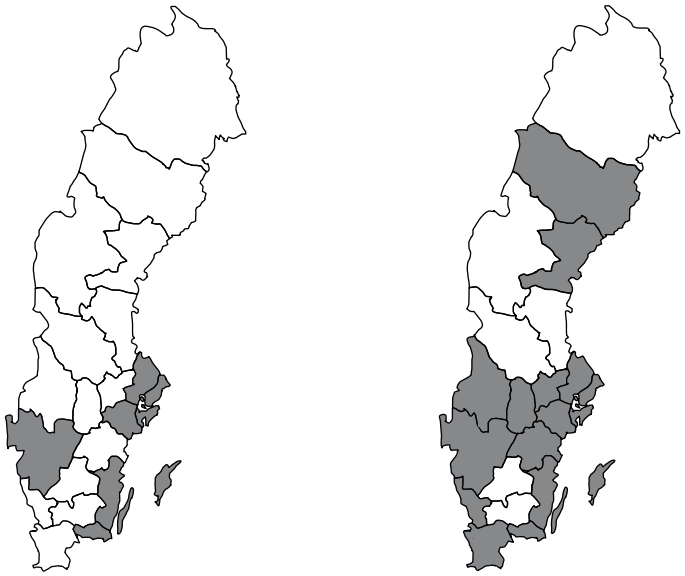
3. Common ragweed in Sweden

The spread of common ragweed in Sweden is well documented. The first plants were identified several decades ago and records of flowering populations can be found dating back to the 1990s (Dahl et al., 1999). Since that time, Swedish pollen traps have registered several periods of high air concentrations of common ragweed pollen. Because we are interest in the impact of local common ragweed populations on human health, the incidence of these events is an insufficient measure of common ragweed presence in the county. Indeed, common ragweed pollen can be transported through the air over several hundred kilometers, which would render our identification strategy invalid (Sikoparija et al., 2013). Hence, we rely on data from the Swedish Species Observation System, which collects reports on the Swedish flora and fauna since the 1980s.[3] We compare the spread of common ragweed in Sweden for 1998 with that of 2011 in **Figure 1**. While only 7 Swedish counties reported populations in 1998, the incidence increased to 15 by 2011. Common ragweed has visibly spread along the coast and inland in that period.

Studies have shown that the natural spreading rate of common ragweed is about 6-10 km per year under favorable climate conditions. Also important for the spread of common ragweed are anthropogenic mechanisms which include agricultural harvesting machineries, road networks or seeds that are dispersed through the trade of birdseed.[4] Adaptation to the cold Nordic climate is crucial for the success of common ragweed in Sweden. In recent years, summers in Sweden have lasted longer with higher temperatures in the crucial growth periods, giving momentum to the ragweed invasion.

[3] For the 1980s, confirmed findings of common ragweed are rare, but since 1995 the number of reported populations has increased substantially.

[4] Major European producers of birdseed are located in areas with successfully established common ragweed populations. Cross-border and cross-region trade of birdseed is large in volume and several countries have imposed trade remedies to close this spreading gateway for common ragweed (Vitalos and Karrer, 2008).



(1) In 1998 **(2)** In 2011

Figure 1: Spatial distribution of common ragweed populations in Sweden.

4. Methodology

In this section we discuss the identification strategy used to investigate the impact of common ragweed populations on human health in Sweden. We also introduce our dataset and describe tests and corrections for heteroskedasticity and autocorrelation that we apply to ensure the validity of our identification strategy.

4.1 Empirical approach

The hypothesis that we test empirically is that *an invasive species, which is still in its lag phase, can yet have adverse impacts on human health.* Indeed, many studies have investigated the impact of common ragweed on human health, but most of these studies focused on the individual, using experimental research designs. Based on this literature, we identified several sources of spatial and temporal heterogeneity that our identification strategy has to deal with. We decided to exploit the variation in botanical and health data with a reduced form panel-data estimation approach. For each health category (illness), we estimate three estimation equations, one for each health indicator. The different health categories and indicators are discussed in the data section below. The estimation equation is given by:

$$y_{it} = \alpha_i + \beta_t + \gamma P_{it} + \epsilon_{it} \tag{1}$$

where $i = 1, \ldots, 21$ denotes the county and $t = 1998, \ldots, 2011$ is the year. We introduce county fixed effects α_i to account for spatial variation. Among others, sources of such time-invariant heterogeneity are sensitivity to common ragweed, genetic diversity and urbanization. To control for temporal heterogeneity, we use time fixed effects β_t. We decided to control for temporal changes with fixed effects because linear or quadratic trends are not able to capture the temporal heterogeneity accurately. For instance, a simple trend can not account for climate and economic shocks, while our country-level time fixed effects can account for a large portion of this variation. We include a total of

21 time-invariant and 14 time-variant fixed effects. The coefficient of interest is γ which measures the impact of common ragweed. The common ragweed variable is a dummy that takes the value 1 if common ragweed is present in a county, otherwise it is 0. Our assumptions regarding the structure of the error term are discussed in the section where we present our estimation strategy.

4.2 Data

The Swedish government collects data on diagnoses in in-patient care (in the hospital) from the National Patient Register since 1998. The Health and Welfare Statistical Database provides data on length of stay (1), admissions (2) and treated patients (3), according to the International Classification of Diseases (ICD-10) distributed over sex, age, home county and principal diagnosis.[5] We use aggregated data over sex and age because we are interest in the aggregated response to common ragweed presence. To control for scale effects, we divide the data on length of stay, admissions and treated patients by the number of inhabitants in each county, creating three new health indicators that are scaled per 100,000 inhabitants. Our analysis covers ICD-10 codes that were identified in the literature to be caused by common ragweed allergens. The categories are listed in **Table 1**.

Table 1: Description and ICD-10 codes for health categories.

Description	ICD-10 code
vasomotor and allergic rhinitis	J30
chronic rhinitis, nasopharyngitis and pharyngitis	J31
asthma	J45
status asthmaticus	J46
allergic contact dermatitis	L23

Note. The Swedish Health and Welfare Statistical Database provides data at the 2-digit disaggregation level.

Data for the spatial and temporal distribution of common ragweed populations in Sweden are taken from the Swedish Species Observation System. The system maintains more than 30 million records on the flora and fauna in Sweden since the 1980s. These data are mainly collected by amateurs and volunteers, but are also provided by professional conservationists. Currently, more than 16,000 active members contribute to the database. They provide GIS position data on common ragweed populations, which we match with the Swedish counties. If a plant was sighted in one of the counties, our dummy variable, which is supposed to capture the effect of common ragweed on human health, takes the value 1 in this country, otherwise the dummy is zero. The descriptive statistic for health and botanic data is presented in **Table 2**.

[5] The codes in brackets are used in later stages of this paper to identify the different health indicators. More details on the definition of health indicators can be obtained from the Swedish Health and Welfare Statistical Database.

Table 2: Descriptive statistic of model variables.

Variables/Indicators		Mean	Std. Dev.	Min.	Max.
J30	(1)	1.05	2.15	0	16.93
	(2)	0.73	1.16	0	8.64
	(3)	0.71	1.12	0	8.64
J31	(1)	0.95	1.87	0	14.24
	(2)	0.46	0.83	0	8.72
	(3)	0.44	0.82	0	8.72
J45	(1)	193.84	105.73	48.65	669.66
	(2)	68.48	27.73	26.12	194.03
	(3)	56.36	20.84	22.08	154.02
J46	(1)	12.74	10.54	0	75.35
	(2)	3.42	2.33	0	17.22
	(3)	2.93	1.8	0	11.96
L23	(1)	1.65	4.52	0	34.98
	(2)	0.29	0.57	0	4.64
	(3)	0.27	0.51	0	4.64
Common ragweed		0.54	0.5	0	1

Note. For each health category and indicator, we have 294 observations.

4.3 Estimation strategy

The estimation of panel-data data models poses several econometric issues that our identification strategy has to address. A first potential issue is heteroskedasticity. It is likely that the variance component is heteroskedastic, which would render hypothesis testing invalid.[6] This is because the ordinary least squares (OLS) estimate of the variance are biased if heteroskedasticity is present in the data. To examine the possibility of heteroskedasticity, we use a Likelihood-ratio (LR) test.[7] A high χ^2-value would attest that the variance component is heteroskedastic, while a low χ^2-value would indicate no issue with heteroskedasticity. We report LR-test results in **Table 3**. The large χ^2-values show that we have to correct for heteroskedasticity in all specifications.

A further possible issue is that of autocorrelation in the idiosyncratic error term. Autocorrelation violates the OLS assumption that the error terms are uncorrelated, resulting in standard errors that tend to be underestimated. To test for autocorrelation, we use the procedure outlined in Wooldridge (2002), which is robust to conditional heteroskedasticity. The procedure uses the residuals from the regression in **Equation 1** in first-differences. If the null of no autocorrelation holds, the residuals should exert a correlation of -0.5.[8]

[6] Our health data have county-specific variation of scale, which is an indicator for panel specific heteroskedasticity.

[7] The test compares the fit of two models, where the homoskedastic model is assumed to be nested in the heteroskedastic model.

[8] A Wald test of this hypothesis was developed by Drukker (2003). We use the user-written Stata program *xtserial*, which allows to inspect autocorrelation in panel-data models.

Table 3 presents the F-test results. We find that except for J30, AR(1) autocorrelation is an issue that we have to deal with. This is indicated by the large F-statistics.[9]

Table 3: Test results for heteroskedasticity and AR(1) autocorrelation.

Health		LR-test		AR(1)-test	
Category	Indicator	χ^2-statistic	probability	F-statistic	probability
J30	(1)	440.35	0.000	0.922	0.348
	(2)	377.13	0.000	0.141	0.711
	(3)	353.07	0.000	0.030	0.865
J31	(1)	446.57	0.000	3.243	0.087
	(2)	506.57	0.000	87.022	0.000
	(3)	418.51	0.000	83.054	0.000
J45	(1)	371.93	0.000	15.771	0.001
	(2)	108.56	0.000	45.056	0.000
	(3)	97.82	0.000	17.427	0.001
J46	(1)	156.41	0.000	3.580	0.073
	(2)	160.90	0.000	13.559	0.002
	(3)	149.32	0.000	20.746	0.002
L23	(1)	756.74	0.000	9.829	0.005
	(2)	312.12	0.000	6.879	0.016
	(3)	313.89	0.000	4.484	0.046

Note. We set the threshold for heteroskedasticity and AR(1) autocorrelation at a probability of 10 percent.

To address heteroskedasticity and autocorrelation, we fit our model with a feasible generalized least squares (FGLS) estimator.[10] The first-order asymptotic properties of FGLS are equal to GLS, which makes the estimator asymptotically more efficient than OLS. A further issue is the correlation of error terms. To deal with this issue, we assume that the variance of panels differs and the error terms are correlated. This gives us the following heteroskedasticity and autocorrelation (HAC) consistent covariance structure:

$$\Omega_{\text{HAC}} = \begin{pmatrix} \sigma^2_{1,1}\mathrm{P}_{1,1} & \sigma_{1,2}\mathrm{P}_{1,2} & \cdots & \sigma_{1,N}\mathrm{P}_{1,N} \\ & \sigma^2_{2,2}\mathrm{P}_{2,2} & \cdots & \sigma_{2,N}\mathrm{P}_{2,N} \\ & & \ddots & \vdots \\ & & & \sigma^2_{N,N}\mathrm{P}_{N,N} \end{pmatrix} \tag{2}$$

[9] We also test for higher levels of autocorrelation by correlating the dependent variables with their lags. Our results show that AR(1)autocorrelation is present in all specifications but no higher levels of correlation are detected. Details can be obtained from the authors.

[10] The properties of FGLS are discussed in Greene (2012). FGLS is similar to generalized least squares (GLS), except that it uses an estimated variance-covariance matrix instead of the true matrix which is not known directly. An advantage of GLS is that it allows us to fit regression models when the variances of the observations are heteroskedastic and/or when there is a certain degree of correlation between the observations. In these cases OLS is statistically inefficient and can give misleading inferences. A detailed exposition of the finite sample properties of FGLS can be found in Wooldridge (2002).

where we replaced the individual identity matrices P_{ij} along the diagonal of Ω with a more general structure:

$$P_{ij} = \begin{pmatrix} 1 & \rho_j & \rho_j^2 & \cdots & \rho_j^{t-1} \\ \rho_i & 1 & \rho_j & \cdots & \rho_j^{t-2} \\ \rho_i^2 & \rho_i & 1 & \cdots & \rho_j^{t-3} \\ \vdots & \ddots & \ddots & \ddots & \vdots \\ \rho_i^{t-1} & \cdots & \cdots & \cdots & 1 \end{pmatrix} \tag{3}$$

This is necessary as the Wald test indicated the presence of AR(1) autocorrelation. We assume that the correlation parameter is unique for each panel, which allows the use of more information on the autocorrelation structure, providing a more reliable estimate of γ.

To check the goodness of fit, we calculate a R-squared value which is the square of the correlation between the model's predicted values and the actual values. This measure has the advantage of referring to the original scale of measurement, with a correlation that ranges from 0 to 1.

5. Results

We summarize our results in **Table 4**. The estimates confirm our hypothesis that an invasive species that is still in its lag phase can yet have adverse impacts on human health. We find statistically significant and positive effects of common ragweed on the length of stay, admissions, and number of patients for almost all health categories. The impact of common ragweed on the length of stay is strongest for asthma patients (J45) and least pronounced for patients with vasomotor and allergic rhinitis (J30). Across the board, the number of admissions to the hospital increased due to the presence of common ragweed in a county. Similar results are found for the number of patients treated in the hospital. Overall, the goodness of fit is not very large, considering that we utilized county and time fixed effects. This indicates a problem with time-variant heterogeneity that is specific to each county. We need to address this problem more carefully to ensure the robustness of our findings.

Table 4: Summary of estimation results.

Health	(1)		(2)		(3)	
Category	Estimate	R-sqd.	Estimate	R-sqd.	Estimate	R-sqd.
J30	0.265***	0.013	0.376***	0.413	0.328***	0.428
	(0.049)		(0.062)		(0.059)	
J31	0.237***	0.148	0.163***	0.386	0.143***	0.373
	(0.069)		(0.048)		(0.029)	
J45	10.325***	0.296	1.218***	0.193	−0.570	0.697
	(3.467)		(0.449)		(0.485)	
J46	2.417***	0.328	0.540***	0.060	0.366***	0.340
	(0.380)		(0.122)		(0.103)	
L23	0.040	0.305	0.051*	0.023	0.042*	0.289
	(0.144)		(0.030)		(0.022)	

Note. Note. Heteroskedasticity and panel-specific AR(1) autocorrelation corrected standard errors in parentheses, * indicates significance at $p < 0.1$, ** at $p < 0.05$, and *** at $p < 0.01$.

6. Sensitivity analysis

A strategy to control for time-variant heterogeneity that is specific to each county would be to use a full set of time-variant county fixed effects. This is not possible because a full set of fixed effects would use up all degrees of freedom (df). Hence, we approximate the full set of fixed effects with time-variant fixed effects for climate regions δ_{qt} with $q = \{1, 2, 3\}$ and for coastal counties θ_{rt}. The estimation equation for our sensitivity analysis is given by:

$$y_{it} = \alpha_i + \beta_t + \gamma P_{it} + \sum_{q=1}^{3} \delta_{qt} + \theta_{rt} + \epsilon_{it} \tag{4}$$

where all indexes have the same meaning as in our baseline specification. The climate regions are set according to the definition used by the Swedish Meteorological and Hydrological Institute. We distinguish the three Swedish climate regions with dummies,[11] which we interacted with the time variable. Such a dummy for q would take the value one at time t, otherwise it is zero. We include time-variant dummies for coastal counties to account for unobserved heterogeneity that is specific to coastal counties. We run the usual statistical tests to identify econometric problems that could render our identification strategy invalid. The LR-test results indicate heteroskedasticity across the board, while the Wald-test results show a problem with AR(1) autocorrelation for J31-J46 and L23.[12]

Our sensitivity analysis supports the findings from our baseline specification (see **Table 5**). We find that the presence of a common ragweed population in a county has positive effects on the length of stay, admissions, and number of patients in Swedish hos-

[11] The Southern climate region (1) is encircled by the Baltic Sea, Norway and the counties Dalarna and Gävleborg, while the central region (2) comprises Dalarna, Gävleborg, Jämtland and Västernorrland. Norrbotten and Västerbotten are included in the Northern climate region (3).

[12] Detailed statistical test results can be obtained from the authors.

pitals. Patients that suffer from asthma (J45) and status asthmaticus (J46) stay longer in the hospital in counties with common ragweed populations, while admissions to the hospital are higher in counties with common ragweed populations for chronic rhinitis, nasopharyngitis and pharyngitis (J31), status asthmaticus (J46) and allergic contact dermatitis (L23). Similar results are found for the number of patients. The overall goodness of fit that we achieved with the sensitivity analysis has increased dramatically, indicating that our time-variant fixed effects for climate regions and the coastal counties capture a large portion of the unobserved variation in data. The results provide further validation of our hypothesis. We find that common ragweed, which is an invasive species that is still in its lag phase, has yet adverse impacts of human health in Sweden.

Table 5: Sensitivity analysis: Summary of estimation results.

Health	(1)		(2)		(3)	
Category	Estimate	R-sqd.	Estimate	R-sqd.	Estimate	R-sqd.
J30	0.021	0.270	0.150	0.482	0.273**	0.492
	(0.193)		(0.108)		(0.120)	
J31	0.030	0.238	0.132***	0.375	0.214***	0.374
	(0.072)		(0.043)		(0.066)	
J45	17.887***	0.806	−0.532	0.709	0.394	0.732
	(4.515)		(0.965)		(0.768)	
J46	2.187***	0.474	0.665***	0.505	0.369***	0.478
	(0.596)		(0.141)		(0.112)	
L23	0.082	0.338	0.093**	0.326	0.085*	0.356
	(0.151)		(0.043)		(0.046)	

Note. Heteroskedasticity and panel-specific AR(1) autocorrelation corrected standard errors in parentheses, * indicates significance at $p < 0.1$, ** at $p < 0.05$, and *** at $p < 0.01$.

7. Conclusions and further research

The scope of this paper is to explore the impacts that an invasive species, which is still in its lag phase, can have on human health. We answer this question with Swedish botanical and health data for the period 1998 to 2011, exploiting spatial and temporal variation with a panel-data estimation approach. To ensure the robustness of our findings, we test and correct for heteroskedasticity and AR(1) autocorrelation. Our baseline estimation results confirm our hypothesis that an invasive species, which is still in its lag phase, can yet have adverse effects on human health. The sensitivity analysis provides similar results, strengthening the found causality with estimates that account for much of the unobserved heterogeneity.

The paper provides several leads that hold good potentials for future research. Firstly, the time span of the study is relative short. A longer period is desired to get a better understanding of the long term trends. Secondly, the spatial resolution of our analysis can be improved. Drug sales data at the pharmacy level would give the opportunity to

investigate the relationship more carefully and get an estimate of the economic cost of an invasive species.

References

Anzivino-Viricel, L., Fontaine, D. and Sonko, A. (2011). 2e rapport en région sur l'Ambroisie en région Rhône Alpes: aAnalyse des données médico- economiques, environnementales et enquête auprés des collectivités territoriales. Tech. rep., Groupe de travail régional sur lambroisie, Observatoire Régional de la Santé Rhône-Alpes.

Breton, M., Garneau, M., Fortier, I., Guay, F. and Louis, J. (2006). Relationship between climate, pollen concentrations of *Ambrosia* and medical consultations for allergic rhinitis in Montreal, 1994-2002. *Science of the Total Environment* 370: 39–50.

Bullock, J., Chapman, D., Schafer, S., Roy, D., Girarfello, M., Haynes, T., Beal, S., Wheeler, B., Dickie, I., Phanf, Z., Tinch, R., Čivić, K., Delbaere, B., Jones-Walters, L., Hilbert, A., Schrauwen, A., Prank, M., Sofiev, M., Niemelä, S., Räisänen, P., Lees, B., Skinner, M., Finch, S. and Brough, C. (2010). Assessing and controlling the spread and the effects of common ragweed in Europe. Final report ENV.B2/ETU/2010/0037, Natural Environment Research Council, UK.

Burbach, G. J., Heinzerling, L. M., Röhnelt, C., Bergmann, K. C., Behrendt, H. and Zuberbier, T. (2009). Ragweed sensitization in Europe - GA(2)LEN study suggests increasing prevalence. *Allergy* 64: 664–665.

Corsico, R., Falagiani, P., Ariano, R., Berra, D., Biale, C., Bonifazi, F., Campi, P., Feliziani, V., Frenguelli, G., Galimberti, M., Gallesio, M., Liccardi, G., Loreti, A., Marcer, G., Marcucci, F., Meriggi, A., Minelli, M., Nardelli, R., Nardi, G., Negrini, C., Papa, G., Piu, G., Pozzan, M., DAmbrosio, F. and Riva, G. (2000). An epidemiological survey on the allergological importance of some emerging pollens in Italy. *Journal of Investigational Allergology and Clinical Immunology* 10: 155–161.

Dahl, A., Strandhede, S.-O. and Wihl, J. (1999). Ragweed - An allergy risk in Sweden? *Aerobiologia* 15: 293–297.

D'Amato, G. and Cecchi, L. (2008). Effects of climate change on environmental factors in respiratory allergic diseases. *Clinical and Experimental Allergy* 38: 1264–1274.

Drukker, D. M. (2003). Testing for serial correlation in linear panel-data models. *Stata Journal* 3: 168–177.

Greene, W. H. (2012). *Econometric Analysis*. New York, NY: Macmillan, 7th ed.

Shea, K. M., Truckner, R. T., Weber, R. W. and Peden, D. B. (2008). Climate change and allergic disease. *The Journal of Allergy and Clinical Immunology* 122: 443–453.

Sikoparija, B., Skjoth, C., Alm Kuebler, K., Dahl, A., Sommer, J., Grewiling, L., Radisic, P. and Smith, M. (2013). A mechanism for long distance transport of Ambrosia pollen from the Pannonian Plain. *Agricultural and Forest Meteorology* 180: 112 – 117.

Vitalos, M. and Karrer, G. (2008). Distribution of *Ambrosia artemisiifolia* L. - Is birdseed a relevant vector? *Journal of Plant Diseases and Protection* 21: 345 – 347.

Warner, J. (1999). Worldwide variations in the prevalence of atopic symptoms: What does it all mean? *Thorax* 54: 46–51.

Wooldridge, J. (2002). *Econometric Analysis of Cross Section and Panel Data*. Cambridge, MA: MIT Press.

Ziska, L. H. (2008). Rising atmospheric carbon dioxide and plant biology: The overlooked paradigm. *DNA and Cell Biology* 27: 165–172.

Ziska, L. H., Gebhard, D. E., Frenz, D. A., Faulkner, S., Singer, B. D. and Straka, J. G. (2003). Cities as harbingers of climate change: Common ragweed, urbanization, and public health. *Journal of Allergy and Clinical Immunology* 111: 290–295.