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Climate variability and infectious diseases nexus: Evidence from Sweden

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ABSTRACT

Many studies on the link between climate variability and infectious diseases are based on biophysical experiments, do not account for socio-economic factors and with little focus on developed countries. This study examines the effect of climate variability and socio-economic variables on infectious diseases using data from all 21 Swedish counties. Employing static and dynamic modelling frameworks, we observe that temperature has a linear negative effect on the number of patients. The relationship between winter temperature and the number of patients is non-linear and "U" shaped in the static model. Conversely, a positive effect of precipitation on the number of patients is found, with modest heterogeneity in the effect of climate variables on the number of patients across disease classifications observed. The effect of education and number of health personnel explain the number of patients in a similar direction (negative), while population density and immigration drive up reported cases. Income explains this phenomenon non-linearly. In the dynamic setting, we found significant persistence in the number of infectious and parasitic-diseased patients, with temperature and income observed as the only significant drivers.

1. Introduction

Climate change has become a topical issue globally, as the physical and biological systems on all continents are already being affected by recent changes in climatic conditions (Asante & Amuakwa-Mensah, 2014). Climate change, including climate variability, has multiple influences on human health and these are expected to be either direct or indirect (Costello et al., 2009; IPCC, 2014, 2007). The impacts of climate change on human health include intensity of transmission of vector-borne, tick-borne and rodent-borne diseases, food- and water-borne diseases, and changes in the prevalence of diseases associated with air pollutants and aeroallergen. Climate change could alter or disrupt natural systems, making it possible for diseases to spread or emerge in areas where they had been limited or had not existed, or for diseases to disappear by making areas less hospitable to the vector or the pathogen (National Research Council, 2001). The direct and immediate effects such

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as deaths due to heat waves and floods which are mostly dramatic provoke immediate policy responses. However, long-term effects act through changes in natural ecosystems and in most cases impact on disease vectors, waterborne pathogens, and contaminants (National Research Council, 2001).

Until recently, the climate-health nexus did not feature prominently in the climate change discourse. In the past, discussions on climate change focused on the effects of the phenomenon on the global economic outlook and eco-systems sustainability (McMichael, Neira, Bertollini, Campbell-Lendrum, & Hales, 2009). Increasingly, scientists have become interested in the potential effects of global climate change on health (Campbell-Lendrum, Corvalán, & Prüss–Ustün, 2003; Carson, Hajat, Armstrong, & Wilkinson, 2006; Costello et al., 2009; IPCC, 2014; McMichael, Woodruff, & Hales, 2006; Nerlander, 2009; Woodward, Lindsay, & Singh, 2011; Wu, Lu, Zhou, Chen, & Xu, 2016). According to McMichael et al. (2006), climate change already has and will continue to have a negative impact on the health of human populations. Evidence already exists that climate change affects the rates of malnutrition, diarrhoeal diseases, malaria and deaths as a result of changing precipitation and high temperatures (McMichael & Woodruff, 2005). This is because there is ample evidence that links most of the world's emerging and re-emerging infectious diseases to climatic variations. Climate change according to Costello et al. (2009) was responsible for 5.5 million disability adjusted life years (DALYs) lost in 2000. These initial assessments and figures of the disease burden attributable to climate change were conservative and relate only to deaths caused by cardiovascular diseases, diarrhoea diseases, malaria, accidental injuries during coastal and inland floods, landslides and malnutrition. Not all of the effects of climate change will be harmful to human health but the damages are projected to outweigh the benefits (Confalonieri et al., 2007). A warmer climate is expected to bring benefits to some populations, including reduced mortality and morbidity in winter and increase local food production, particularly in northern high latitudes. Against this background, the negative effects of climate change on health are likely to be greater and are more strongly supported by evidence than are the possible benefits.

Developed countries are also not immune to the health impact of climate change. As presented in Table 1, climate-dependent infectious diseases are likely to impact on most developed countries (Panic & Ford, 2013). For example, waterborne and food-borne diseases which are caused by environmental or climatic factors are likely to affect almost all developed countries. Also, Northern European countries (particularly Sweden) are expected to be affected by tick-borne diseases which are predominantly caused by increased daily precipitation, humidity, changing patterns of seasonal precipitation, increased average temperatures and extreme heat.

Although the impact of climate change on health is anticipated, few studies have really used data to empirically estimate the effect on health outcomes, specifically infectious diseases. Most of the few studies which exist are based on biophysical experiments and do not control for socioeconomic covariates. In Sweden for example, Lindgren (1998), Lindgren, Tälleklint, and Polfeldt (2000) and Lindgren and Gustafson (2001) examined the link between climate change and infectious diseases. These studies ignored socioeconomic factors in their analysis and also focused on only one infectious disease (i.e. tick-borne encephalitis). We therefore contribute to the literature by analyzing the effect of climate variability and socioeconomic factors on infectious disease patients in Sweden. Our study utilizes panel data from in-patient care diagnoses records on infectious and parasitic diseases, climate indicators (e.g. temperature and precipitation) and socio-economic variables for twenty one counties in Sweden. The study employed both static and dynamic analysis, and also accounts for county and year fixed effects. We considered a pooled estimation where all the infectious and parasitic diseases are lumped together, and also a disaggregated estimation where dominant infectious and parasitic diseases (such as intestinal infectious diseases and other

Table 1
Climate-Dependent infectious diseases and sample countries likely to experience health hazards linked to changes in disease exposure.

Disease Type	Disease	Environmental factors impacting disease dynamics	Countries likely to be affected
Mosquito-borne diseases	Malaria	Increased average temperatures, precipitation	Australia, New Zealand, Chile, Southern Europe
	West Nile Virus	Increased average temperatures, drought	USA, Southern Europe, Canada, Australia, New Zealand, Chile
	Dengue, Chikungunya fever, Yellow fever	Increased average temperatures	New Zealand, Mediterranean region (coastal areas in Spain, Portugal and France), Chile
Tick-borne diseases	Lyme borreliosis, tick-borne encephalitis,	Increased daily precipitation, humidity, changed patterns of seasonal precipitation, Increased average temperatures, extreme heat	Northern Europe, Canada, USA
Waterborne diseases	Sewage and sanitation: Vibrio vulnificus and Vibrio cholera, E.Coli, Campylobacter, Salmonella, Cryptosporidium, Giardia, Yersinia, Legionella	Increased rainfall and storm frequency, flooding, landslides, increased average temperatures, extreme heat episodes	All countries
Food borne diseases	Salmonellosis, campylobacteriosis	Extreme rainfall, flooding, increased average temperatures, increased frequency of extreme heat, changed seasonal patterns	All countries

Source: Adapted from Panic and Ford (2013) with modifications.

bacterial diseases) are separated from less dominant ones. We use different temperature values (i.e. summer, winter and average) to examine the seasonal effect of temperature on the number of infectious disease patients. Although we recognized that infectious and parasitic diseases are dynamic in nature, we also consider a static model in the analysis because the dataset for this study is based on annual records and as such we assume that the spread of the disease would reach its steady-state within a year.

The remainder of the paper is organized as follows. Section two discusses how climate change and socio-economic factors affect health while section three presents an analysis of the theoretical and empirical methods, and data issues. Section four discusses the empirical results and the final section, five concludes the study.

2. How climate change and socio-economic factors affect health

Generally, health outcomes can be affected by climate, socio-economic and ecological factors. In this section we discuss how climate change affects health while paying attention to the potential effect of socio-economic factors (including migration dynamics) on infectious diseases. The likely effects and outcomes of climate change on human health is summarized by Confalonieri et al. (2007, pp. 391–431) and they conclude that climate change has both positive and negative effects on health outcomes, with the negative effects most likely to outweigh the positives. Woodward et al. (2011) observe that the risk of climate change to health results mainly from the effects of the phenomenon on local food production, severity and frequency of storms and floods, threats to water supplies and the direct effect of heat on people. Confalonieri et al. (2007) also classify human exposure to the effects of climate change into two (i.e. direct and indirect). People are affected directly through changing weather patterns and indirectly through food and water quality and quantity, agriculture, among others. Exposure to any of these conditions can cause morbidity and even death. Most literature on the implications of climate change suggests that climate change may affect human health through three pathways: directly, indirectly and through social and economic disruptions (Asante and Amuakwa-Mensah, 2014; Confalonieri et al., 2007; IPCC, 2014, 2007; Panic & Ford, 2013; Wu et al., 2016).[1]

2.1. Health effects due to direct and indirect exposure to changes in climatic variables

Changes in climatic conditions are expected to affect the distribution of morbidity and mortality through the physical effects of exposure to high or low temperature (Campbell-Lendrum et al., 2003). Human beings are able to cope well with mid-range[2] temperatures and are only stressed by temperatures that are 'uncommonly' high or low (Woodward et al., 2011). Significant increase or reduction in temperature adversely affects body temperature and metabolism processes within the body. The early effect of high temperature is usually reduced physical and mental work capacity. Further and sustained exposure leads to dehydration, exhaustion and heat stroke (Kovats & Kristie, 2006). These have direct effects on productivity (IPCC, 2007; Nerlander, 2009).

Heat waves are expected to have tremendous effect on human health. According to Robine et al. (2008), the heat wave in Europe in 2003 caused about 70,000 deaths principally from cardiovascular diseases. Other studies in California by Knowlton et al. (2009) found similar results. Another direct impact of climate is cold waves which usually affects people who spend a lot of time outdoors (e.g. the homeless). In the polar and temperate regions, cold waves can still increase mortality when electricity and heating systems malfunction (Confalonieri et al., 2007, pp. 391–431). Cold related mortality has declined in most European countries since 1950 (Carson, Peterson, & Higgins, 2003). Many attribute the reduction in winter time mortality to decline in cold days and nights. Carson et al. (2006) however reports that the reduction in cold temperature accounts for a small proportion of the reduction in winter time mortality. Schwartz (2005) also found socio-demographic characteristics and medical conditions can increase the risk of death associated with extreme temperatures. He indicated that while patients with diabetes had a higher risk of dying on hot days, women had higher risk of dying on cold days. Studies by D'Ippoliti et al. (2010) confirmed the results of an earlier study by Schwartz (2005) that the effect of heat waves was highest among people with respiratory diseases and women aged between 75 and 84 years.

Indirectly, climate change affects human health through air, food and water quality and quantity, agriculture and the ecology of vectors (IPCC, 2007). Malnutrition and food insecurity are also affected indirectly by climate change as high temperatures and erratic rainfall reduce crop yields (Costello et al., 2009). Contact between food and pest species, especially flies, rodents and cockroaches, is also temperature-sensitive. Fly activity is largely driven by temperature rather than by biotic factors (Goulson, Derwent, Hanley, Dunn, & Abolins, 2005). Malnutrition, according to the IPCC (2014; 2007) increases the risk of morbidity and mortality from infectious diseases. Aziz et al. (1990) confirmed this in his study on Bangladesh. In Bangladesh, drought and lack of food were linked to an increasing possibility of dying from a diarrhoeal disease.

Changes in rainfall patterns affect surface water flow. Reduction in rainfall leads to reduced river flows and increased water temperature leading to declining water quality because the dilution of contaminants in the water is reduced. Less oxygen is

[1] Intergovernmental Panel on Climate Change.

[2] Parsons (2002) assess that normal temperature for humans is about 98.6 °F. (37.0 °C.). Individual differences in metabolism, hormone levels, physical activity, and even the time of day can, however, cause it to be as much as 1 °F. (0.6 °C.) higher or lower in healthy individuals. For elderly people, it is also normal for core body temperature to be lower.

therefore dissolved in the water and microbiological activity is enhanced (Confalonieri et al., 2007). This notwithstanding, several studies document the linkage between microbial load in water as a result of extreme rainfall events and runoff and cases of human disease is not very clear (Schwartz, Woods, Porte, Seeley, & Baskin, 2000). Work by Senhorst and Zwolsman (2005) in the Netherlands associated low quality of water during 2003 to low river flows during the dry summer. The marked seasonal outbreaks of cholera in the Amazon and sub-Saharan Africa are often associated with reductions in rainfall, floods and faecal contamination of water supplies (Confalonieri et al., 2007). In the United States, Curriero, Patz, Rose, and Lele (2001) found an association between extreme rainfall events and monthly reports of outbreak of water-borne diseases. Common forms of food contamination such as salmonellosis have been found to be associated with high temperatures (IPCC, 2007). Readers can refer to Wu et al. (2016) for a detailed review of the impact of climate change on infectious diseases.

2.2. Socio-economic factors and infectious diseases

Infectious diseases can also spread through human travel patterns. Thus migration is one of the means by which diseases spread, either because migrants bring new pathogens with them to their destinations or because the migrants themselves constitute susceptible populations and lack immunity to endemic diseases in their areas of settlement (National Research Council, 2001). This situation is true for both forced migration (such as those based on political, religious and natural disasters) and for voluntary migration of people seeking new social or economic opportunities. Also, modern transportation such as jet transportation is an avenue through which pathogens and vectors can be spread rapidly from one area to another within a continent or from one continent to another. An example of such situation is that of influenza, where it appears that new strains initially spread from Southeast Asia to other areas of the world (National Research Council, 2001). Furthermore, individuals who are infected with infectious diseases and who may be asymptomatic can infect fellow passengers and susceptible people at their destinations.

Transportation has been found to be the easiest means by which non-respiratory infectious diseases may be introduced into new areas (National Research Council, 2001). For instance, gonorrhea initially was found in Asia and then spread to the United States (Knapp, Faley, Ekeberg, & Dubois, 1997). The recent Ebola outbreak in West Africa in the years 2014 and 2015 is an example, where the disease spread rapidly to other countries and continents through travels. Also, the means of transportation themselves can contribute to the spread of vectors to new areas. For example, the concept of "airport malaria" which is associated with the outbreaks of malaria among populations surrounding airports in temperate non-endemic areas such as the United States, England, and Northern Europe (National Research Council, 2001). This concept emerged from the clustering of cases around international airports, where an experiment confirmed that anopheline mosquitos could survive a long-distance flight in the wheel wells of jet aircraft, demonstrating the potential for air transportation to facilitate the spread of disease vectors (National Research Council, 2001; Guillet et al., 1998). An example of such occurrence is also the case where one of the Asian vectors of dengue, the mosquito *Aedes albopictus*, was transported to Houston in wet tires through container shipment (Moore & Mitchell, 1997).

Population density is another important factor to be considered since population concentration may facilitate the spread of infectious diseases if there are persons in the population who are infected. In most cases, population density has often been linked to increasing ease with which airborne infections, waterborne diseases, and sexually transmitted infections are spread among the populace (National Research Council, 2001). Other social and demographic factors which may encourage the spread of infectious disease include but not limited to poverty level, household design and architecture, and water development projects.

2.3. Healthcare in Sweden

Healthcare in Sweden is built on a government-controlled decentralized welfare system. With a population of approximately 9.4 million, Sweden ranks amongst the highest number of health care and care workers per capita who deliver care mostly in primary care facilities or in the home (Anell, Glenngard, & Merkur, 2012). The medical system is largely public with the majority of services provided in primary care and outpatient clinic facilities. Private firms provide 20% of public hospital care and 30% of public primary care. With few exceptions, health care workers do not work extra hours to supplement their income maintaining the 50 h work-week prescribed for health care workers (Anell et al., 2012). The rate of employees with long working hours is low in Sweden, 91% of all employees *do not* work long hours (Eurostat LFS 2006).

Non-communicable diseases like cancers, cardiovascular diseases and type 2 diabetes have received significant attention in the recent past because of their high cost to care and significant impact on health globally (Lim, 2013; Lozano et al., 2012). In Sweden alone, between 2007 and 2013, the prevalence of diabetes increased from 5.8% to 6.8% whilst incidence remained constant at 4.4 per 1000 population (Andersson, Ahlbom, & Carlsson, 2015). Despite the high cost to care of non-communicable diseases such as diabetes, great strides have been made in reducing morbidity and mortality, and in increasing the life expectancy in prevalent cases (Lennartsson & Heimerson, 2012).

Of increasing concern, however, infectious diseases are estimated to account for approximately 10% of the total burden of disease in the European Union. In Sweden, this burden has also increased over the last decade (Quaglio, Demotes-Mainard, & Loddenkemper, 2012; van Lier, Havelaar, & Nanda, 2007). According to Baker-Austin et al. (2016), 2014 was the warmest year on record since record keeping started in 1860. In 2014, the number of domestically acquired *Vibrio* infections in Sweden and Finland more than doubled. *Vibrio* species grow preferentially in low-salinity warm water (above 15°), and recreational

exposure to water during heat waves seems to have been responsible for these cases (Baker-Austin et al., 2016). The *Vibrio* species are responsible for outbreaks of oral-fecal diarrheas.

Similar observations have been made about the increasing trend in Shiga toxin-producing *Escherichia coli* over the last decade; with incidence increasing not only in Sweden but in also in Ireland, the Netherlands and Denmark (ECDC, 2015). Other infectious diseases such as Campylobacter enteritis, *Chlamydia* and Tularemia have all seen a rise in incidence that has made their surveillance of utmost importance (Al, 2015; ECDC, 2015; Harvala et al., 2016; Holmberg, 2012).

The changing landscape of emerging and re-emerging infectious diseases has made it necessary to determine contributing factors to the observed epidemiological shifts that have precipitated the rise in the number of cases. To the best of our knowledge, there has been no research done on the national burden of infectious disease in Sweden over the last few years.

3. Methodology and data

3.1. Theoretical framework

The theoretical framework for this study follows the work of Graff Zivin and Neidell (2013) where we relate climate variability to health. Based on Grossman's (1972) postulation which characterizes health as an investment good, Graff Zivin and Neidell (2013) extended how health can influence productivity through the extensive margin (that is, a process where illness reduces labour supply hence affecting productivity) to an intensive margin. The intensive margin is when productivity is affected assuming a fixed labour supply. Through the intensive margin the theoretical model is able to capture more precise health effects. Graff Zivin and Neidell (2013) modelled the representative individual's health production function as a function of ambient pollution levels, mitigation activities to pollution exposure in the form of avoidance behaviour and medical care that reduces the negative health consequences from pollution exposure. Based on this, we redefine the health production function to examine the role of climate variability on health. Thus, our health production function depends on climate variability (CV), mitigation of the harmful effect of climate change by avoidance behaviour (A) and medical care (M). This is expressed as:

$$H = f(CV, A, M) \tag{1}$$

Both avoidance behaviour (A) and consumption of medical care (M) reduces the health burden from climate variability. There is however distinction in timing and cost associated with avoidance behaviour and medical care consumption. Following Graff Zivin and Neidell (2013), we rewrite Equation (1) in order to better examine how environmental variables affect health. Thus, we create a distinction between individual's health (H) and illness incidence (ϕ). Therefore, the health production function is given as:

$$H = f[M(\phi), \phi(CV, A)] \tag{2}$$

From Equation (2), climate variability and avoidance behaviour jointly determine the incidence of illness attributed to climate variability. Also, medical expenditure in turn depends on these illness incidences. Moreover, health of the individual depends on medical expenditure and incidence of illness. Medical expenditure is assumed to reduce severity of illness. In our analysis we impose the normal concavity assumption on the health production function and its subparts shown in Equation (2). The utility function of a representative individual is assumed to be a function of health (H), consumption goods (X) and leisure (L). That is:

$$U = u(H, X, L) \tag{3}$$

Also, the individual allocates his/her wage and non-wage income on consumption goods, mitigation activities through avoidance behaviour and medical expenditure. Thus, the budget constraint is given by:

$$I + w(H)[T - L] = P_X X + P_A A + P_M M \tag{4}$$

where I is non-wage income, $w(H)$ is wage income which is dependent on H, T is time, L is leisure, and P_X, P_A and P_M are prices of X, A and M, respectively. The individual's utility problem is to maximize the utility function in Equation (3) subject to the budget constraint presented in Equation (4). Solving the first conditions from the maximization problem (see appendix) together with the budget constraint gives us the optimal avoidance and medical treatment which are functions of climate variability (CV), the function that translate climate change into illness incidence (ϕ) and the costs of avoidance behaviour (P_A), medical cares (P_M) and all other consumption goods (P_X). Thus, the optimal avoidance behaviour and medical treatment is expressed as:

$$M = g(CV, \phi, P_M, P_A, P_X) \tag{5}$$

$$A = h(CV, \phi, P_M, P_A, P_X) \tag{6}$$

From Equations (5) and (6), the optimal avoidance behaviour and medical treatment depends on climate variability. As a result, we can derive an expression for the relationship between climate variability and health by finding the total derivative of Equation (2). That is:

$$\frac{dH}{dCV} = \underbrace{\left(\frac{\partial H}{\partial M}\frac{\partial M}{\partial \phi} + \frac{\partial H}{\partial \phi}\right)}_{\frac{dH}{d\phi}} \bullet \underbrace{\left(\frac{\partial \phi}{\partial CV} + \frac{\partial \phi}{\partial A}\frac{\partial A}{\partial CV}\right)}_{\frac{d\phi}{dCV}} \tag{7}$$

From Equation (7) it is obvious that the effect of climate variability on health has two parts, which are the relationship between climate variability and illness (that is, $d\phi/dCV$), and the degree to which illness is translated into health status (that is, $dH/d\phi$). The second expression of Equation (7) describes the net effect of climate variability on illness incidence based on individuals' exposure level. The expression has two components: the first term $(\partial\phi/\partial CV)$ and the second term $((\partial\phi/\partial A)(\partial A/\partial CV))$. The first term $(\partial\phi/\partial CV)$ represents the pure biological effect of climate variability whereas the second term $((\partial\phi/\partial A)(\partial A/\partial CV))$ shows the role of avoidance behaviour in averting illness incidence by putting in place mitigation measures against the harmful effect of climate variability. From the net effect of climate variability (that is, $d\phi/dCV$), there is the possibility of observing no change in illness despite the existence of biological effect if the avoidance behaviour is very productive in mitigating the harmful effect of climate variability. However, if the avoidance behaviour is impossible or ineffective, then the biological effect and the reduced form effects (that is, $d\phi/dCV$) will be identical (Graff Zivin & Neidell, 2013).

Similarly, the first expression in Equation (7) has two components: the first term $((\partial H/\partial M)(\partial M/\partial\phi))$ and second term $(\partial H/\partial\phi)$. The term $((\partial H/\partial M)(\partial M/\partial\phi))$ shows the degree to which medical treatment, which is a post-exposure intervention, reduces the negative effects of climate variability on health. Also, the term $(\partial H/\partial\phi)$ represents how health responds to illness, which reflects the degree to which climate-induced illness incidence are not treated, either due to the illness being untreatable or individuals do not seek treatment.

3.2. Empirical model and variable description

In estimating an empirical model to examine the effect of climate variability on health, we modify the optimal medical treatment function in Equation (5) by aggregating the number of individuals who seek medical treatment at the county level. The focus here is to investigate the effect of climate elements in explaining infectious and parasitic diseases. Thus, we consider how climate elements together with socio-economic variables explain the incidence of infectious and parasitic diseases in Sweden. This study relies on panel annual data from 21 counties in Sweden from 1998 to 2013. Our empirical model from Equation (5) is given as:

$$M = g(CV, D) \tag{8}$$

where M is the number of individuals who seek medical treatment due to incidence of infectious and parasitic diseases, CV is climate variables (that is, temperature and precipitation) and D is a vector of socio-economic and control variables which include income, education, number of healthcare personnel, population density and immigration.[3] Inclusion of socio-economic factors helps to fulfill external validity of the results. For the dependent variable we consider the number of patients per 100,000 inhabitants. Infectious and parasitic diseases in this study relate to all diseases classified as infectious and parasitic by the National Board of Health and Welfare in Sweden.[4] All data on health variables are based on in-patient care diagnoses. We express the dependent variable as non-linear in climate variables (that is, temperature and precipitation) and income (that is, the dependent variable is a quadratic function of climate variables and income). The introduction of climate variables and income in quadratic form is to test whether the number of infectious and parasitic disease patients is non-linear in climate variables and income. We use income (i.e. GDP per capita) as a proxy for the capacity of the county to detect infectious diseases.

We estimate Equation (8) under two different assumptions. First, we assume a static model where current number of infectious disease patients is not dependent on previous number of infectious disease patients. The static model is considered because the data used in this study an annual data and most infectious and parasitic disease spread within a period of days, weeks or months. We assume that the spread of the disease is likely to reach its steady-state within a year and as such the static model is worth considering. Therefore the static model is expressed as:

[3] Immigration is based on the definition by the Swedish Statistics board. We do acknowledge the limitation of this variable in our study. For instance, considering immigrants as potentially bringing new pathogens when immigrants are from developed countries, is less relevant than considering Swedish residents coming back from poor countries as potentially bringing these new pathogens.

[4] See http://www.socialstyrelsen.se/statistics/statisticaldatabase/inpatientcarediagnoses or the appendix of Amuakwa-Mensah, Marbuah, and Mubanga (2016) for the list of the diseases.

$$M_{it} = \beta_0 + \beta_1 Temp_{it} + \beta_2 Temp^2{}_{it} + \beta_3 Precip_{it} + \beta_4 Precip^2_{it} + \delta' D_{it} + \eta_i + \gamma_t + \varepsilon_{it} \quad (9)$$

Each variable in Equation (9) is a panel data set for county i in time period t. The term $\delta' D_{it}$ in Equation (9) is the product of the vector of socio-economic and control variables and their corresponding parameter, η_i is the county fixed effect variable and γ_t captures year fixed effect. In estimating Equation (9), we utilize panel fully modified ordinary least squares (FMOLS) estimation technique and accounted for both county and year-fixed effects to capture any county and year specific effect on the dependent variable. FMOLS developed by Phillips and Hansen (1990) is a semi-parametric model that is robust to endogeneity and serial correlation problems. Also, it provides consistent and efficient estimates even in the absence of cointegration relation. Further, it is robust to both stationary and non-stationary series in a single cointegration (see Phillips, 1995). Given that our panel data have relatively long time period, using FMOLS within a panel setting helps us to circumvent problems of serial correlation and non-stationarity. In order to estimate the model using FMOLS, the variables are first modified and then the system estimates directly to eliminate the existing nuisance parameters. The structure of the FMOLS has a correction term for endogeneity and serial correlation. By accounting for county fixed effects we take into consideration average differences across counties in any observable or unobservable predictors, such as differences in climate conditions, economic activities, etc. The fixed effect coefficients soak up all the cross-group action and as such provide consistent estimates.

In our second scenario, we assume the possibility of infectious and parasitic diseases to portray persistence despite the annual nature of the data for the study. That is, current number of infectious disease patients is likely to be affected by previous trends. In such a case, estimates using OLS technique are biased due to the endogenous lag of the dependent variable which is a covariate in the model. In order to address the problem of endogeneity we rely on the one-step system generalised method of moment (GMM) to estimate Equation (10).

$$M_{it} = \beta_0 + \kappa M_{it-1} + \beta_1 Temp_{it} + \beta_2 Temp^2{}_{it} + \beta_3 Precip_{it} + b_4 Precip^2_{it} + \boldsymbol{\delta' D_{it}} + \eta_i + \gamma_t + \varepsilon_{it} \quad (10)$$

The one-step system GMM method unlike the Arellano and Bond (1991) estimation technique addresses both the problem of individual fixed effects in addition to the problem of endogenous variable arising from the use of lag dependent variable as a regressor. Thus, the Arellano and Bover (1995)/Blundell and Bond (1998) technique or the system GMM augments Arellano-Bond by making additional assumption that the first differences of the instrumental variables are uncorrelated with the fixed effects. In order to estimate the dynamic model, the instrument used is mostly a transformation of the lagged endogenous (or predetermined) variables. To estimate consistent estimators for the instruments that are the lagged dependent variable with further lags of the same variable, the assumption of no serial correlation in the error term is very relevant.

In testing this assumption of no serial autocorrelation, the null hypothesis of no autocorrelation is rejected for the first lag but accepted for the higher lags. In our study we use a significance level of 5% for the test of no serial correlation. We further carry out the Sargan test to examine the over-identification restriction. This test has a chi square distribution and the null hypothesis is that over-identifying restrictions are valid. The acceptance of the null hypothesis implies that the population moment conditions are correct, thus the over-identifying restrictions are valid. We treat the lag of number of infectious disease patients (i.e. the dependent variable) as endogenous and the instruments we use for it are the same variables lagged enough periods to avoid higher order autocorrelation in the residuals. The other explanatory variables in Equation (10) are treated as exogenous in the estimation. The system automatically uses different forms of the exogenous variables as instruments in addition to the lags of the dependent variable in generating the consistent and unbiased estimates.

For the static and dynamic models, we estimate the models using three different temperature variables. Specifically, we consider mean annualized winter, summer and average temperatures. However, for the precipitation variable, we consider only annualized average precipitation. The variables considered in our analysis are in line with the argument that transmission of infectious diseases is determined by many factors including social, economic and ecological conditions, access to health care, and intrinsic human immunity (Jones et al., 2008; Semenza & Menne, 2009). With the exception of temperature and precipitation, we transform all the variables by taking the natural logarithm. Because of the relatively long time period of our panel data, we proceeded our estimation by carrying out a panel unit root test using the Phillips-Perron and Augmented Dickey-Fuller (ADF) unit root test. With the exception of the number of health personnel and immigration, all the variables are stationary in levels. We present the variable description and summary statistics in Table 2. From the summary statistics, we observe much variations in the climate variables (that is, temperature and precipitation) across counties over the years. For example, the average annual mean temperature deviation from the normal temperature for winter is about 1.6 °C with a standard deviation of about 2.1 °C. Also, the average annual mean precipitation deviation from the normal is about 12.9 mm with a standard deviation of about 14.2 mm.

Fig. 1 shows the distribution of patients across various infectious and parasitic disease types. Majority of patients reported of other bacterial diseases (i.e. A30-A49)[5] which comprises of Listeriosis, Diphtheria, Whooping cough, Scarlet fever,

[5] See http://www.socialstyrelsen.se/statistics/statisticaldatabase/inpatientcarediagnoses for complete list of diseases in each classification.

Table 2
Variable description, data sources and descriptive statistics.

Variables	Description	Source	N	Mean	Std	Min	Max
Patients	natural log of the number of patients per 100,000 inhabitants	NBHW	357	6.034	0.137	5.653	6.437
Temperature winter	annual winter mean temperature deviation from the normal (°C)	SMHI	357	1.595	2.062	−3.300	6.300
Temperature winter squared	Temperature winter squared	SMHI	357	6.785	6.950	0	39.69
Temperature summer	annual summer mean temperature deviation from the normal (°C)	SMHI	357	0.764	0.953	−1.500	3
Temperature summer squared	Temperature summer squared	SMHI	357	1.489	1.859	0	9
Temperature average	annual average temperature deviation from the normal (°C)	SMHI	357	1.109	0.740	−1.600	2.500
Temperature average squared	Temperature average squared	SMHI	357	1.776	1.414	0.01000	6.250
Precipitation	annual average precipitation deviation from the normal (mm)	SMHI	357	10.89	14.28	−21.70	56.80
Precipitation squared	Precipitation squared		357	322.07	447.81	0	3226.2
Income	natural log of GDP per capita	SCB	357	5.658	0.208	5.193	6.347
Income squared	Income squared	SCB	357	32.06	2.364	26.97	40.29
Education	natural log of the number of the population with post-secondary education three years or more	SCB	357	10.27	0.927	8.117	12.97
Health personnel	natural log of the number of health personnel	NBHW	336	7.777	0.0979	7.553	8.048
Population density	natural log of population density	SCB	357	3.192	1.131	0.916	5.820
Immigration	natural log of the number of immigrants	SCB	357	7.638	1.055	4.860	10.44

Note: where NBHW, SMHI, SCB and RUS are National Board of Health and Welfare (http://www.socialstyrelsen.se/statistics/statisticaldatabase/inpatientcarediagnoses), Swedish Meteorological and Hydrological Institute (http://www.smhi.se/klimatdata/framtidens-klimat/ladda-ner-scenariodata?area=swe&sc=rcp85&var=n&seas=ar&sp=en), Statistics Sweden (http://scb.se/en_/) and National emission database (http://projektwebbar.lansstyrelsen.se/rus/Sv/statistik-och-data/nationell-emissionsdatabas/Pages/default.aspx), respectively.

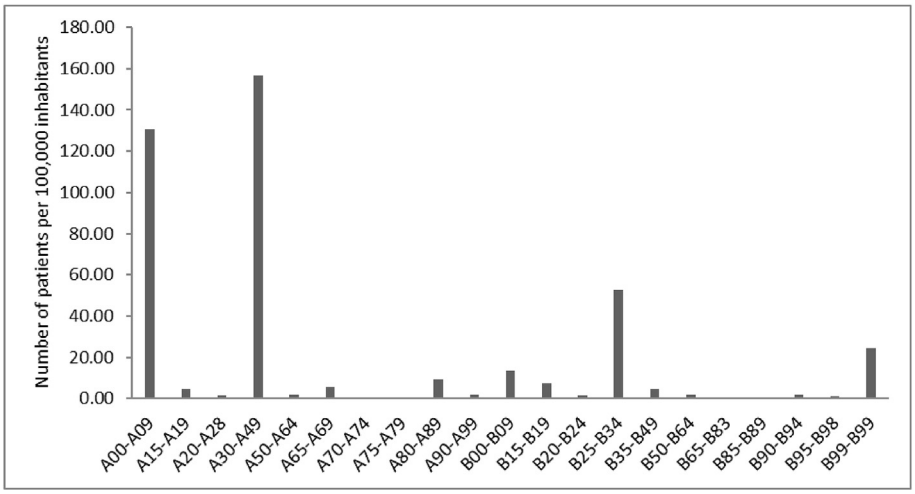

Fig. 1. Distribution of patients across various infectious and parasitic disease (1998–2014).
Note: A00-A09 Intestinal infectious diseases; A15-A19 Tuberculosis; A20-A28 Certain zoonotic bacterial diseases; A30-A49 Other bacterial diseases; A50-A64 Infections with a predominantly sexual mode of transmission; A65-A69 Other spirochaetal diseases; A70-A74 Other diseases caused by chlamydiae; A75-A79 Rickettsioses; A80-A89 Viral infections of the central nervous system; A90-A99; Arthropod-borne viral fevers and viral haemorrhagic fevers; B00-B09 Viral infections characterized by skin and mucous membrane lesions; B15-B19 Viral hepatitis; B20-B24 Human immunodeficiency virus [HIV] disease; B25-B34 Other viral diseases; B35-B49 Mycoses; B50-B64 Protozoal diseases; B65-B83 Helminthiases; B85-B89 Pediculosis, acariasis and other infestations; B90-B94 Sequelae of infectious and parasitic diseases; B95-B98 Bacterial, viral and other infectious agents; B99-B99 Other infectious diseases.
Source: National Board of Health and Welfare database, Sweden. Accessed on 28/04/2016. http://www.socialstyrelsen.se/statistics/statisticaldatabase/inpatientcarediagnoses

Meningococcal infection, etc. The next dominant category is patients reporting for intestinal infectious diseases (i.e. A00-A09), followed by viral (i.e. B25-B34) and other infectious diseases (i.e. B99-B99).

Fig. 2 shows the distribution of infectious and parasitic diseases patients across counties averaged over the year 1998–2014. The average number of patients reporting for infectious and parasitic diseases between the years 1998 and 2014 in Sweden is about 421 patients per 100,000 inhabitants. The county with the highest number of infectious and parasitic disease patients during the period under consideration is Västerbotten (with about 506 patients per 100,000 inhabitants), followed by Gotland (with about 502 patients per 100,000 inhabitants) and Dalarna (with about 475 patients per 100,000 inhabitants). The counties with the least number of infectious and parasitic disease patients are Kronoberg (with about 344 patients per 100,000 inhabitants), followed by Uppsala (with about 362 patients per 100,000 inhabitants).

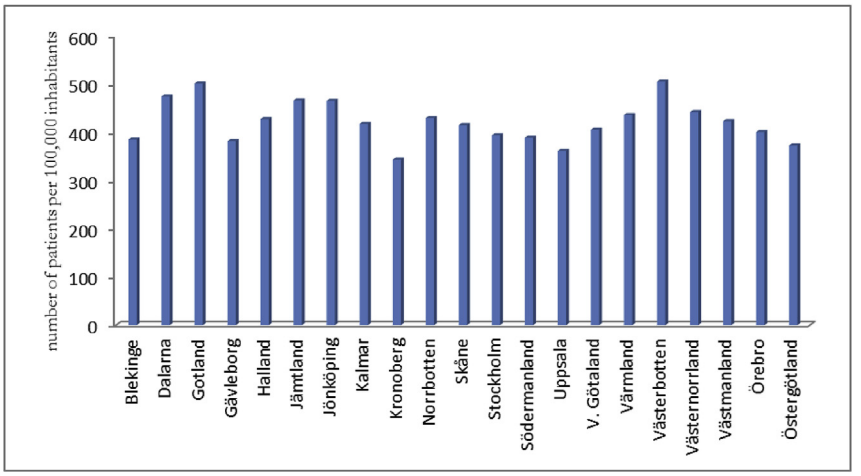

Fig. 2. Distribution of infectious and parasitic disease patients across counties (1998–2014).
Source: National Board of Health and Welfare database, Sweden. Accessed on 28/04/2016.

4. Empirical results and discussions

4.1. Static analysis

The static analysis of factors affecting the number of infectious and parasitic disease patients is shown in Table 3. The models (1) to (3) show results based on aggregated reported cases of infectious and parasitic diseases and the other models (that is, (4)–(9)) show results of disaggregated reported cases. Models (4)–(6) represent results of the two main dominant class of diseases reported, while (7)–(9) are the results of the categories of less cases of diseases reported. In each case we have shown results when winter, summer and average temperature values are used in the model. Our results show that the effect of climate variables on the number of infectious and parasitic disease patients depends on whether winter, summer and average temperature values are used. Using winter temperature values in the models, the findings show that temperature is the only climate variable which explain the number of infectious and parasitic disease patients based on models (1) and (4). However, both temperature and precipitation explain the number of patients in model (7). In all these cases the relationship between winter temperature and the number of patients portray a "U" shape relation, indicating that an increase in winter temperature above the normal average reduces the number of infectious and parasitic disease patients initially but eventually increases in the long run. In other words, winter temperature has non-linear impact on the number of infectious disease patients.

The rate of decrease of the number of infectious and parasitic disease patients in the short run is higher than the rate of increase in the long run. In other words, the coefficient of the linear term of temperature is higher than that of the non-linear term using the winter temperature values.

Using summer temperature values, we find the effect of temperature on the number of patients to be monotonically decreasing especially in models (2) and (8). In the case of the aggregated reported cases (that is, model (2)), summer temperature has no significant effect on the number of patients based on the linear term, however the quadratic term for temperature is significant and negative. From model (8), the decreasing effect of summer temperature on the number of patient is observed for both the linear and non-linear terms. Precipitation has no significant effect on the number of patients based on the linear term, however, the effect is significant for the non-linear term but this effect is negligible when summer temperature values are used (see model 2). With regard to the disaggregated reported cases, the effect of precipitation on the number of patients follows a "U" shape for the dominant reported categories (i.e. A00-A09 & A30-A49) as shown in model (5). On the contrary, the effect of precipitation on the number of patients is monotonically increasing for the least reported category of diseases (see model 8). Using average temperature values in our models, we observed a linear negative relationship between temperature and number of infectious and parasitic disease patients. On the contrary, precipitation has positive effect on the number of patients. These findings are confirm by Martens, Jetten, Rotmans, and Niessen (1995), National Research Council, (2001) and Panic and Ford (2013). According to these studies, the suitability of vector habitats is determined by precipitation levels and an increase in precipitation has a higher possibility to create conducive environment for vectors, which will in turn increase infectious and parasitic disease patients.

Further insights from our results suggest that infectious diseases like vector-borne and tick-borne diseases are mostly affected by temperature. This affects the survival and reproduction rate of the vector and the ticks, which in turn affect the habitat suitability, distribution, intensity and the pattern of their activities like biting rate. Whereas some of the vectors develop and reproduce during lower temperature, others develop and reproduce in higher temperature. As suggested by

Table 3
Static analysis of the factors affecting infectious and parasitic disease patients.

Variables	All			A00-A09 & A30-A49			Others		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Winter	Summer	Average	Winter	Summer	Average	Winter	Summer	Average
Climate variables									
Temperature	−0.0309***	−0.00362	−0.0698***	−0.0211***	−0.00298	−0.0530***	−0.0515***	−0.0214***	−0.102***
	(0.00201)	(0.00431)	(0.00610)	(0.00263)	(0.00561)	(0.00801)	(0.00250)	(0.00549)	(0.00779)
Temperature squared	0.00267***	−0.00885***	0.000906	0.00125**	−0.00283	0.00285	0.00616***	−0.0186***	−0.00332
	(0.000400)	(0.00174)	(0.00251)	(0.000524)	(0.00226)	(0.00329)	(0.000497)	(0.00222)	(0.00320)
Precipitation	0.000178	−0.000124	0.000259**	−0.000274	−0.000427**	−0.000230	0.00110***	0.000453***	0.00126***
	(0.000131)	(0.000134)	(0.000130)	(0.000171)	(0.000174)	(0.000171)	(0.000162)	(0.000171)	(0.000166)
Precipitation squared	2.34e-06	8.84e-06**	5.30e-06	5.66e-06	8.78e-06*	8.20e-06*	−3.45e-06	1.16e-05**	−1.71e-07
	(3.70e-06)	(3.81e-06)	(3.67e-06)	(4.85e-06)	(4.96e-06)	(4.81e-06)	(4.60e-06)	(4.86e-06)	(4.68e-06)
Socioeconomic variables									
Income	0.731***	0.966***	1.013***	0.390	0.513	0.567	1.924***	2.474***	2.420***
	(0.268)	(0.270)	(0.267)	(0.351)	(0.352)	(0.350)	(0.333)	(0.344)	(0.341)
Income squared	−0.0879***	−0.113***	−0.115***	−0.0453	−0.0594*	−0.0627**	−0.212***	−0.268***	−0.257***
	(0.0234)	(0.0236)	(0.0233)	(0.0307)	(0.0308)	(0.0306)	(0.0291)	(0.0301)	(0.0297)
Education	−0.363***	−0.362***	−0.385***	−0.370***	−0.366***	−0.382***	−0.382***	−0.373***	−0.423***
	(0.0444)	(0.0448)	(0.0445)	(0.0582)	(0.0583)	(0.0584)	(0.0552)	(0.0571)	(0.0568)
Health personnel	−0.404***	−0.441***	−0.467***	−0.698***	−0.713***	−0.737***	0.152***	0.0670	0.0393
	(0.0402)	(0.0404)	(0.0400)	(0.0526)	(0.0526)	(0.0526)	(0.0499)	(0.0515)	(0.0511)
Population density	0.418***	0.447***	0.437***	−0.0659	−0.0445	−0.0510	1.479***	1.534***	1.505***
	(0.0360)	(0.0362)	(0.0357)	(0.0472)	(0.0472)	(0.0468)	(0.0447)	(0.0462)	(0.0455)
Immigration	0.128***	0.126***	0.131***	0.150***	0.149***	0.153***	0.0860***	0.0807***	0.0907***
	(0.00724)	(0.00726)	(0.00718)	(0.00948)	(0.00945)	(0.00943)	(0.00900)	(0.00925)	(0.00918)
Constant	8.692***	8.241***	8.528***	13.68***	13.36***	13.55***	−4.778***	−5.865***	−5.016***
	(0.866)	(0.874)	(0.859)	(1.134)	(1.138)	(1.128)	(1.076)	(1.114)	(1.098)
Observations	335	335	335	335	335	335	335	335	335
County FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Year FE	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Adjusted R-squared	0.685	0.675	0.684	0.676	0.672	0.676	0.560	0.546	0.556
Long Run SE	0.0147	0.0148	0.0146	0.0193	0.0192	0.0192	0.0183	0.0188	0.0186
Bandwidth(neweywest)	69.03	69.05	68.95	69.03	69.05	68.95	69.03	69.05	68.95

Standard errors in parentheses *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Values for the covariates are beta-type coefficients.

Semenza and Menne (2009) and Lindgren et al. (2000), since the late 1950s all cases of encephalitis admitted in the Stockholm county in Sweden have been serologically tested for tick-borne diseases. These have been attributed to milder and shorter winters, which results in longer tick-activity seasons. On the other hand, higher temperature also prevents the development and activities of some disease vectors which reduces the incidence of infectious diseases.

From Table 3, the results show that socio-economic factors such as income, education, number of health personnel, population density and immigration have significant effects on the number of infectious and parasitic disease patients. Generally, income and the number of patients portray a non-linear relationship, that is, an inverted "U" shaped relationship. This means that as the income per capita in Swedish counties increase, there is more coverage to track individuals with infectious and parasitic diseases hence the number of infectious patients increases. However, beyond a certain income threshold the number of patients decreases as income increases. This result is intuitive since during any epidemic outbreak, investment or higher income in the region implies more income will be channeled into the health sector and this will help to trace or access individuals with the disease so the number of recorded cases will definitely increase. Also, the increase in the number of infectious and parasitic disease cases may be probably due to investment in scientific research to uncover various infectious and parasitic diseases. As the investment or income increases, complemented with necessary treatment, the number of cases starts decreasing after a certain income threshold. Thus, increase in income per capita may imply more resources for public health services which significantly affects the spread of diseases, since the very purpose of such services is to stem the spread of the disease. Through the activities of public health such as vaccination which is a specific intervention aimed at preventing the occurrence of diseases in individuals, the incidence of infectious diseases would be reduced among the population. Also, an increase in income per capita in the county goes a long way to help in the development of anti-microbial agents which has the tendency of altering the pattern of infectious and parasitic diseases.

Our results further show a negative effect of education on the number of infectious and parasitic disease patients. This effect is robust in all the models. This result suggests that as the number of the population with post-secondary education of three years or more increases, the number of infectious and parasitic disease patients will reduce. The result conforms to our expectation since the more educated is presumed to have more information about the causes and prevention of diseases, and as such counties with more educated people are likely to reduce the number of patients. The elasticity of the number of patients to education is between −0.362 and −0.423 from our models. Similarly, the number of health personnel have negative effect on the number of patients for models (1) to (6). This means that as the number of health personnel increases, it

reduces the patients to personnel ratio hence health care workers are likely to relatively spend more time with patients to explain the causes and prevention of the disease. As patients are informed about the causes and prevention of the diseases, it makes early disease detection and treatment possible, and thus decreases the risk of transmission. The elasticity of the number of patients to the number of health personnel ranges between −0.404 and −0.737. Contrary to our expectation, the number of health personnel has a positive effect on the number of patients in model (7).

As expected, our results show a positive effect of population density on the number of infectious and parasitic disease patients, with exception to models (4)–(6). The positive effect suggests that higher concentration of population may facilitate the spread of infectious diseases if there are persons in the population who are infected. Our elasticity estimations show that estimates from models (7)–(9) are relatively elastic, indicating that the number of patients in these classifications are more responsive to population density. Diseases in these classification include, for example airborne infections, waterborne diseases, and sexually transmitted infections, which are likely to spread among the populace when they are concentrated (National Research Council, 2001). In a similar vein, our results show a positive effect of immigration on the number of infectious and parasitic disease patients. This implies that the spread of infectious disease agents is greatly affected by human travel patterns and the inflow of migrants. Thus, the inflow of immigrants into Sweden is one of the means by which diseases spread, either because migrants bring new pathogens with them to their destinations or because the migrants themselves constitute susceptible populations and lack immunity to endemic diseases in their areas of settlement. The positive effect of migration on infectious diseases brings to mind the concept of “airport malaria” which arose from numerous reports of limited malaria outbreaks among populations surrounding airports in temperate non-endemic areas such as the United States, England, and Northern Europe (National Research Council, 2001). The effect of immigration on the number of patient is robust and the elasticity ranges from 0.086-0.15.

4.2. Dynamic analysis

Here, we present results for the dynamic analysis. We report the results for only aggregated estimation since the results for disaggregated analysis are qualitatively the same as the one shown in Table 4. As said earlier we made use of one-step system GMM in our estimations and tested the over-identification and no serial correlation restrictions. Using a significance level of 5%, the population moment condition which shows the validity of the instruments used, are correct for all models shown in Table 4, since the null hypothesis for the Sargan's test are not rejected. The serial correlation test shows that all the results for the variant system GMM models fulfil the no serial correlation assumption as autocorrelation is significant at the first order but insignificant for the second order autocorrelation. These guarantee the consistency of the estimates and the validity of the instruments used.

Our results in Table 4 show that infectious and parasitic diseases portray persistence. This means that current number of infectious and parasitic disease patients is likely to be affected by previous patients. This situation is very common in cases where the disease is not totally eradicated from patients or potential patients are not identified and vaccinated. Comparing models (1) and (2), the coefficient of the lag of the number of patients in winter is higher than that of summer. Meaning that the number of infectious and parasitic disease patients are likely to exhibit more persistence in winter relative to summer seasons. In relation to climate variables, temperature is the only variable which affects the number of patients, with the exception of model (2) where climate variables do not explain the number of patients. Unlike the static analysis, temperature has a linear effect on the number of patients. And the effect of temperature on the number of patients is negative similar to the static analysis. From models (1) and (3), a unit increase in temperature above the normal winter and average temperatures will lead to a decrease in the number of patients by about 4 and 7, respectively.[6]

Income is the only socioeconomic factor which explain the number of infectious and parasitic disease patients within a dynamic framework, when we account for both county and year fixed effects in our analysis. The relationship between income and the number of patients follow an inverted “U” shape, similar to the static analysis. The results show a high responsiveness of the number of patients to income at lower income levels, where the number of patients is rising, than higher income levels, where the number of patients is falling. In absolute terms, the effect of income on the number of patients is relatively higher when summer temperature values are used (that is, model (2)) than when winter temperature values are used (that is, model (1)).

5. Conclusions

We have so far examined the effect of climate variability and socioeconomic factors on the incidence of infectious diseases using panel data for the period 1998–2013 for all 21 Swedish counties. Both static and dynamic analysis were considered. We considered the number of infectious and parasitic disease of patients as the outcome variable in this paper. Following the theoretical model by Graff Zivin and Neidell (2013), we observe that medical treatment is a function of four components: climate variability, a function that translate climate variability into illness incidence and the costs of avoidance behaviour, medical care and all other consumption goods. Also, the effect of climate variability on health can be decomposed into the

[6] Since the dependent variable (that is, number of patients) is a natural logarithm transformation and the temperature values are not, the coefficients of temperature is multiplied by 100 in order to ascertain the unit change in the number of patients resulting from a unit change in temperature.

Table 4
Dynamic analysis of the factors affecting infectious and parasitic disease patients.

Variables	(1)	(2)	(3)
	Winter	Summer	Average
Patients (−1)	0.427***	0.331***	0.325***
	(0.114)	(0.109)	(0.106)
Climate variables			
Temperature	−0.0416***	−0.0106	−0.0749**
	(0.0116)	(0.0287)	(0.0350)
Temperature squared	−0.00151	0.00316	0.00762
	(0.00213)	(0.0114)	(0.0134)
Precipitation	0.000499	0.000193	0.000354
	(0.000693)	(0.000726)	(0.000689)
Precipitation squared	−4.34e-06	−4.55e-07	6.73e-06
	(2.12e-05)	(2.27e-05)	(2.16e-05)
Socioeconomic variables			
Income	6.642*	7.504*	7.471**
	(3.777)	(3.847)	(3.765)
Income squared	−0.581*	−0.676**	−0.677**
	(0.333)	(0.340)	(0.332)
Education	−0.372	−0.558	−0.505
	(0.493)	(0.505)	(0.493)
Health personnel	0.598	0.412	0.401
	(0.379)	(0.385)	(0.377)
Population density	0.164	0.471	0.506
	(0.419)	(0.421)	(0.410)
Immigration	−0.0565	−0.0272	−0.0299
	(0.0423)	(0.0425)	(0.0419)
Constant	−12.20	−13.13	−13.15
	(10.97)	(11.03)	(10.88)
Observations	315	315	315
Number of counties	21	21	21
Wald chi2	450.1	421.1	442.4
Sargan's test	15.45	12.31	13.58
1st order autocor.	−2.45**	−2.68***	−2.62***
2nd order autocor	0.107	0.76	0.59
County FE	Yes	yes	Yes
Year FE	Yes	yes	Yes

Standard errors in parentheses *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Values for the covariates are beta-type coefficients.

relationship between climate variability and illness, and the degree to which illness is translated into health status. The relationship between climate variability and illness is described as the net effect of climate variability on illness incidence based on individuals' exposure level.

Based on the theoretical model we empirically estimated the relationship between the number of infectious and parasitic disease patients and climatic variability together with socioeconomic factors. We observed from a static analysis that temperature generally has a linear negative effect on the number of patients. However, the relationship between winter temperature and the number of patients is non-linear and “U” shaped. Contrary to temperature, we found precipitation positively influence the reported number of patients with the disease. There is slight heterogeneity in the effect of climate variables on the number of patients across disease classification. Whereas education and the number of health personnel have negative effect on the number of patients, population density and immigration show the opposite effect on the number of patients. We found a non-linear relationship between income and the number of patients, which is inverted “U” shape. The dynamic analysis showed that the number of infectious and parasitic disease patients exhibit persistence, and temperature and income are the only dominant variables that explain the number of patients. Whereas temperature was found to have a linear negative impact on the number of patients, income showed an inverted “U” shape relationship with the outcome variable.

In summary, our study suggest that the number of infectious and parasitic disease patients in Sweden may be influenced by climatic and socio-economic variables. This suggests that investment into public health services in the long run will have negative impact on the number of reported cases of the these diseases. There should therefore be adaptation and mitigation strategies to address the impact on climate variability on health. These strategies may include but not limited to enhanced public awareness through public health education and prevention, vaccination programmes, disease surveillance, investment into protective technologies, weather forecasting and early warning systems, emergency management and disaster preparedness, among others. Furthermore, inclusion of climate sensitive infectious and parasitic diseases on the list of notifiable diseases should be paramount. In addition, population density and growth can be checked. Migration policies are also very critical in addressing infectious diseases in Sweden and should engage the attention of the Swedish Migration Board and public health authorities. Relevant routine screening for potential introduction of targeted infectious diseases at the various points of entry into Sweden should be considered and implemented with regard to appropriate legal frameworks (local,

regional and international) that ensures respect for individual rights and freedoms while safeguarding the general interest of the larger population.

To the best of our knowledge, our study is the first to model the effect of climate change on infectious diseases in Sweden using such unique data. The comprehensive nature of the Swedish registers has allowed us to access a wealth of possible confounders, infectious disease records and climate pattern trends. Due to the wealth of information in these databases, we are confident that our results are generalizable to the other regions that have experienced similar climate change patterns.

Certain limitations however need to be taken into consideration. Although the Swedish registers provide a wealth of information, our study used aggregate data and not individual level data. These data are collected for administrative and not research purposes and thus some important information may have been omitted. Although Sweden has strict rules about access to medication, the registers cannot account for individuals who self-medicate, or allow self-limiting disease conditions to run their course or start treatment abroad. This would misclassify those with disease to undiseased. We also cannot exclude the possibility that some of the increase in disease incidence could be attributed to increased travel amongst local Swedish people to tropical environments. However, by using data over several years, the observed increase cannot be accounted for only by travel abroad.

In addition, we make too strong an assumption on the use of the static model. That infectious diseases do not spread among individuals is not very plausible. This limitation was primarily driven by the annual data at our disposal. Even though the dynamic model sought to complement the static analysis, a more detailed inventory of higher frequency data on infectious diseases and its drivers would have allowed a much deeper investigation which could have provided more insight. Another issue of concern is the large cross-unit (that is, N) and small time unit (T) assumption for the system GMM technique used for the dynamic model. There is the tendency that a small N may limit the number of instruments used for estimation and this may have consequences on the properties of the estimator. It has however been proven that if there exist some persistence in the variable then system GMM estimator yields results with the lowest bias even if the cross-unit (N) is relatively small (Soto, 2009). In the case of the FMOLS, it performs very well when the time period is very long since it corrects the dependent variable using the long-run covariance matrices.

In summary, our sensitivity analysis alleviated some of the concerns introduced by using aggregate data and possible misclassification bias by those who do not visit health facilities. The concerns raised discourage a conclusion of causal inference from our findings.

Appendix

$$\underset{X,A,L,M}{Max}\ \ell = u(H,X,L) + \lambda[I + w(H)[T-L] - P_X X - P_A A - P_M M] \tag{1a}$$

The first order conditions by finding the partial derivatives of Equation (1a) with respect to X, A, L and M are as follows:

$$\ell_X = \frac{\partial \ell}{\partial X} = \frac{\partial U}{\partial X} - \lambda P_X = 0 \tag{2a}$$

$$\ell_L = \frac{\partial \ell}{\partial L} = \frac{\partial U}{\partial L} - \lambda w = 0 \tag{3a}$$

$$\ell_A = \frac{\partial \ell}{\partial A} = \frac{\partial U}{\partial H}\left(\frac{\partial H}{\partial M}\frac{\partial M}{\partial \phi}\frac{\partial \phi}{\partial A} + \frac{\partial H}{\partial \phi}\frac{\partial \phi}{\partial A}\right) - \lambda\left(P_A + \frac{\partial w}{\partial H}\left(\frac{\partial H}{\partial M}\frac{\partial M}{\partial \phi}\frac{\partial \phi}{\partial A} + \frac{\partial H}{\partial \phi}\frac{\partial \phi}{\partial A}\right)(T-L)\right) = 0 \tag{4a}$$

$$\ell_M = \frac{\partial \ell}{\partial M} = \frac{\partial U}{\partial H}\frac{\partial H}{\partial M} - \lambda\left(P_M + \frac{\partial w}{\partial H}\frac{\partial H}{\partial M}(T-L)\right) = 0 \tag{5a}$$

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