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Climate variability and infectious diseases nexus: evidence from Sweden

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Abstract

In this paper, we present evidence based on a theoretical model developed that links the impact of climate variability on health. Using Swedish data on infectious diseases, we empirically estimate the causal relationship between climate variability and health outcomes. Generally, we find that the number of infectious disease patients and admissions are significantly driven by indicators of climate variability and socio-economic variables such as income and number of immigrants. Specifically, the effect of temperature variation on the health outcomes is ambiguous and sensitive to the choice of winter, summer or average temperature. Precipitation is relevant in explaining the number of infectious disease patients and admissions only when summer temperature considered in the model. Further, we find that an increase in carbon emissions directly causes the number patients and admissions in the summer. The relationship between infectious disease proxies (i.e. patients and admissions) and income per capita follows an inverted-U shape.

Keywords: Climate change; Infectious diseases; Migration; Sweden

1.0 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 and Amuakwa-Mensah, 2015). Climate change, including climate variability, has multiple influences on human health and these are expected to be either direct or indirect. The impacts of climate change on human health include intensity of transmission of vector -, tick-, 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 (NRC, 2001). The direct and immediate effects such 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 (NRC, 2001).

Until recently, the climate change-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, *et al.*, 2009). Increasingly, scientists have become interested in the potential effects of global climate change on health. 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, 2005). This is because there is ample evidence that links most of the world's killer diseases to climatic variations (Campbell- Lendrum *et al*, 2003). Climate change according to Costello (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. Studies show that small increases in the risk for climate-sensitive disease conditions such as diarrhoea diseases and malnutrition can result in very large increases in the total disease burden (Haines et.al, 2006). Not all of the effects of climate change will be harmful to human health but the damages are projected to outweigh the benefits. A warmer climate is expected to bring benefits to some populations, including reduced mortality and morbidity in winter and greater 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-dependant infectious diseases is likely to impact on most developed countries (Panic and Ford, 2013). For example, water-borne and food-borne diseases which are caused by environmental or climate 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, changed patterns of seasonal precipitation, increased average temperatures and extreme heat.

Table 1: Climate-Dependant 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	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: Panic and Ford (2013)

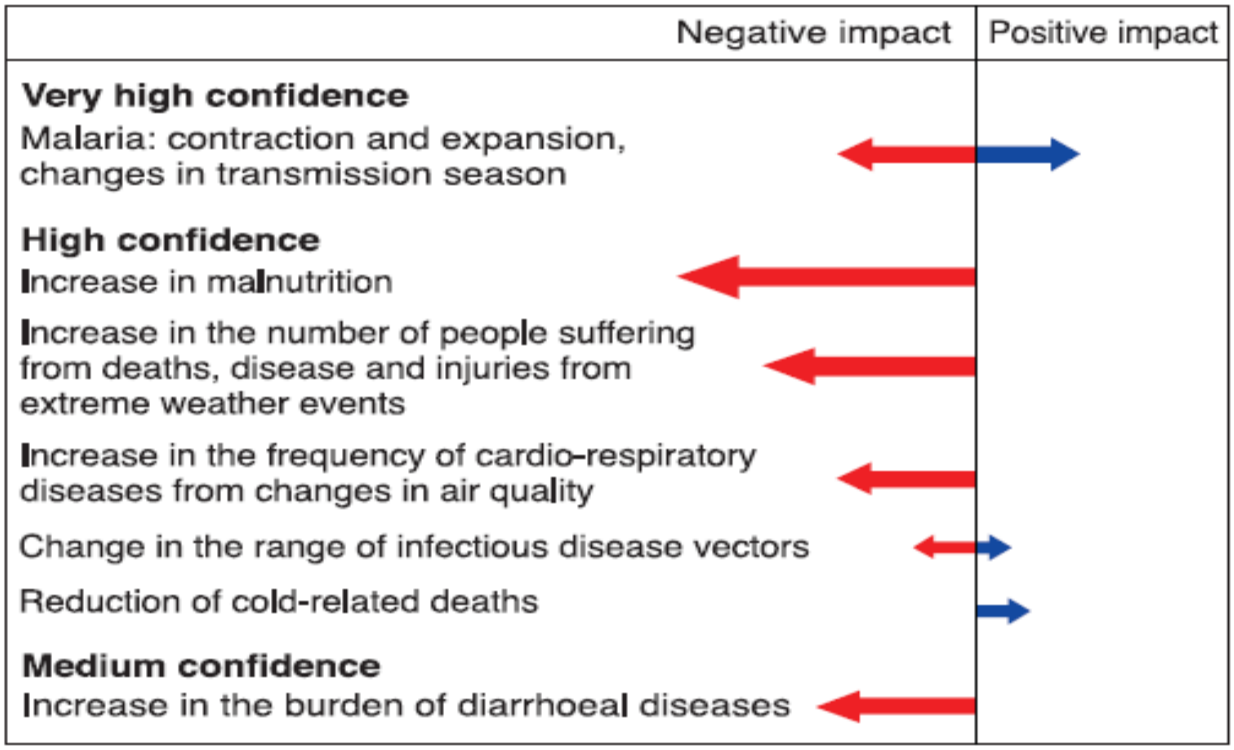
Although the impact of climate change on health is anticipated, few studies have really used data to empirically estimate the effect of climate change on health outcomes, specifically infectious diseases. This study attempts to theoretically and empirically investigate how climate change affects health. We empirically estimate the effect of climate change on infectious diseases using data from Sweden. Moreover, we examine how the number of immigrants affects the number of infectious disease patients and admissions in Sweden. Although it is expected that migration activities have effect on infectious diseases, we are not aware of any study in Sweden that has estimated this effect. This study therefore seeks to fill this gap.

The remainder of the paper is organised as follows. Section two discusses how climate change and socio-economic factors affect health while section three presents an analysis of the conceptual and theoretical linkage between climate change and health outcomes. The fourth section explains the method and data used for the empirical investigation with section five focused on discussion of the empirical results. Section six 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 as summarized by Confalnieri *et al.* (2007) are presented in Figure 1. The figure shows that climate change has both positive and negative effects on health outcomes, with the negative effects most likely to outweigh the positive effects.

Figure 1: Direction and Magnitude of Change of Selected Health Impacts of Climate Change



Source: Confalnieri *et al.* (2007)

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. Confalnieri *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 (IPCC, 2007).

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). Several studies have concluded on the impact of atmospheric temperature on the health status of a given population. Human beings are able to cope well with mid-range temperatures and are only stressed by temperatures that are 'uncommonly' high or low (Woodward, 2011). Significant increase or reduction in temperature adversely affects body temperature and metabolism processes within the body. The early effect of high temperature usually is reduced physical and mental work capacity, further and sustained exposure leads to dehydration, exhaustion and heat stroke (Kovats, 2006). These have direct effects on productivity (IPCC, 2007 and 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 affect people who spend a lot of time outdoors such as the homeless. In the polar and temperate regions, cold waves can still increase mortality when electricity and heating systems malfunction (Confalnieri *et al.*, 2007). Cold related mortality has declined in most European countries since 1950 (Carson *et al.*, 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. Schwarts (2005) also found in his study that 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 Schwarts (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 *et al.*, 2005). Malnutrition according to the Intergovernmental Panel on Climate Change (IPCC) (2007) increases the risk of morbidity and mortality from infectious diseases. Azziz *et al.* (1990) confirmed this in his study in 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 therefore dissolved in the water and microbiological activity is enhanced (Confalnieri *et al.* 2007 and Bates *et al.*, 2008). 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 (Aramin *et al.*, 2000 and Schwartz *et al.*, 2000). Work by Senhorst and Zwolsman (2005) in the Netherlands associated the low quality of water during the 2003 period to low river flows during the dry summer of 2003. The marked seasonal outbreaks of cholera in the Amazon and sub-Saharan Africa are often associated with reductions in rainfall, floods and the faecal contamination of water supplies (Gerolomo and Pema, 1999 and Confalnieri *et al.*, 2007). In the United States, Curriro *et al.*, (2003) 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).

2.2 How socio-economic factors affect 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 (NRC, 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 (NRC, 2001). Furthermore, individuals who are infected with infectious diseases and who may be asymptomatic can infect fellow passengers and susceptible people at their destinations.

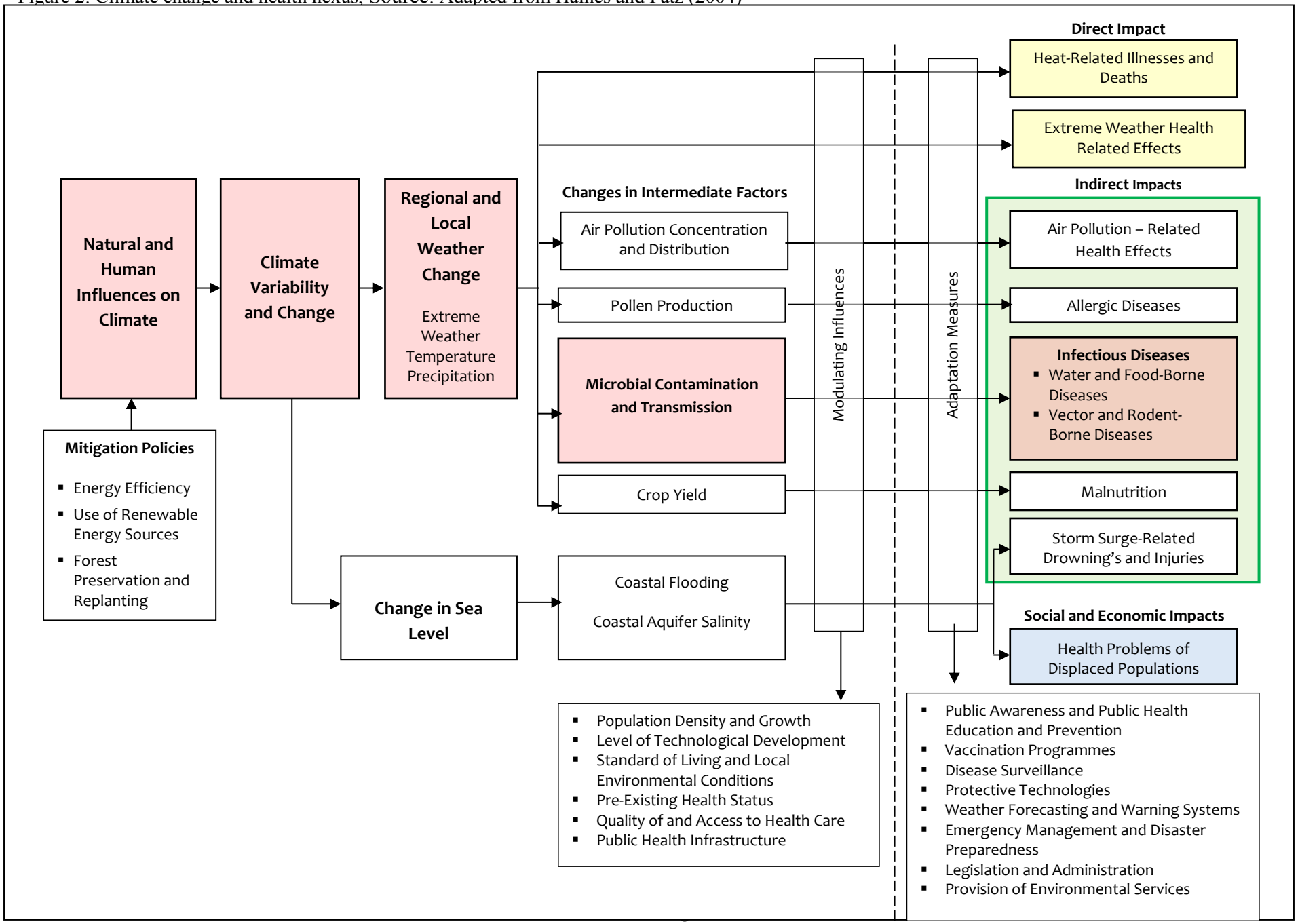
Transportation has been found to be the easiest means by which non-respiratory infectious diseases may be introduced into new areas (NRC, 2001). For instance, gonorrhea initially was found in Asia and then spread to the United States (Knapp *et al.*, 1997). The recent ebola outbreak in West Africa in 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 (NRC, 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 (NRC, 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 and Mitchell, 1997).

Population density is also an important factor to be considered as 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 been often linked with increasing ease to which airborne infections, waterborne diseases, and sexually transmitted infections are spread among the populace (NRC, 2001). Other social and demographic patterns which may encourage the spread of infectious disease include but not limited to poverty level, household design and architecture, and water development projects.

3.0 Conceptual Framework: Climate Change and Health Nexus

One of most comprehensive frameworks that explain the links between climate change or climate variability to health outcomes is provided by Haines and Patz (2004). In the original form, the framework links natural phenomena and human influence to climate change or climate variability. The changes in regional and local climatic conditions manifest as extreme weather conditions, changes in precipitation and rise in oceanic and atmospheric temperature (global warming) occur. These stressors will act directly or indirectly to determine health outcomes (IPCC, 2007). Directly, an increase or decrease in atmospheric temperature causes heat or cold waves leading to heat stroke and other diseases. In addition, climate change will elevate sea levels due to factors such as the melting of arctic ice sheets and rising sea surface temperature. These trigger storm surges and floods that will put coastal settlements especially at risk. Indirectly, through "ecological disturbance" high temperature and changes in patterns of precipitation (stressors) will alter the global pattern of infectious diseases. Global warming will create a suitable environment for disease vectors and pathogens to thrive and enhance the frequency of human contact in most parts of the world. Climate change is also expected to induce tertiary feedbacks through conflicts and displaced population who are likely to increase the pressure on social amenities such as public health services in host communities and/or increase cross infections as well as the outbreak of new diseases. These events could lead to morbidity and/or mortality.

Figure 2: Climate change and health nexus, Source: Adapted from Haines and Patz (2004)



The tertiary effects are broadly captured as social and economic disruptions by IPCC (2007). From the framework (see Figure 2), health outcomes of climate change will be conditioned by the interplay of modulating and adaptation measures. Modulating factors are exogenous of climate change. Adaptive measures, however, are implemented in response to or in anticipation of climate change (IPCC TAR, 2001).

We develop a formal theoretical model following the work of Zivin and Neidell (2013) to relate climate change to health. Based on Grossman's (1972) postulation which characterizes health as an investment good, 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. 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 change on health. Thus, our health production function depends on climate variability (CV), carbon dioxide (CO_2) emissions, mitigation of the harmful effect of climate change by avoidance behaviour (A) and medical care (M). This is expressed as;

$$H = f\left(CV, CO_2, A, M\right) \tag{1}$$

Both avoidance behaviour (A) and consumption of medical care (M) reduces the health burden from climate variability and carbon dioxide emissions. There is however distinction in timing and cost associated with avoidance behaviour medical care consumption. The climate variability and carbon dioxide emission variables in equation (1) constitute climate change (CC). As a result, equation (1) turns equation (1a) below;

$$H = f\left(CC, A, M\right) \tag{1a}$$

Following Zivin and Neidell (2013), we rewrite equation (1a) in order to better examine how environment 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;

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

From equation (2), climate change and avoidance behaviour jointly determine the incidence of illness attributed to climate change. 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 as;

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

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). Thus, the lagrangian expression for this maximization problem is given as;

$$\underset{X,A,L,M}{Max}\ \ell = u(H, X, L) + \lambda\left[I + w(H)[T - L] - P_X X - P_A A - P_M M\right] \tag{5}$$

The first order conditions by finding the partial derivatives of equation (5) with respect to X, A, L and M are as follows:

$$\ell_X = \frac{\partial \ell}{\partial X} = \frac{\partial U}{\partial X} - \lambda P_X = 0 \tag{6}$$

$$\ell_L = \frac{\partial \ell}{\partial L} = \frac{\partial U}{\partial L} - \lambda w = 0 \tag{7}$$

$$\ell_A = \frac{\partial \ell}{\partial A} = \frac{\partial U}{\partial H}\left(\frac{\partial H}{\partial M}\frac{\partial M}{\partial \phi}\frac{\partial \phi}{\partial A} + \frac{\partial H}{\partial \phi}\frac{\partial \phi}{\partial A}\right) - \lambda\left(P_A + \frac{\partial w}{\partial H}\left(\frac{\partial H}{\partial M}\frac{\partial M}{\partial \phi}\frac{\partial \phi}{\partial A} + \frac{\partial H}{\partial \phi}\frac{\partial \phi}{\partial A}\right)(T - L)\right) = 0 \tag{8}$$

$$\ell_M = \frac{\partial \ell}{\partial M} = \frac{\partial U}{\partial H}\frac{\partial H}{\partial M} - \lambda\left(P_M + \frac{\partial w}{\partial H}\frac{\partial H}{\partial M}(T - L)\right) = 0 \tag{9}$$

The standard trade-off between labour and leisure can be derived from solving equations (6) and (7). Our interest is in equations (8) and (9), which gives us the equilibrium condition for avoidance behaviour and medical treatment. Thus, solving equations (8) and (9) gives the intuitive expression that the ratio of marginal productivity of avoidance behaviour and the marginal productivity of medical care should equal the ratio of their prices. That is,

$$\frac{dH/dA}{dH/dM} = \frac{P_A}{P_M} \tag{10}$$

While the left-hand side of equation (10) is the ratio of marginal productivity of avoidance behaviour and marginal productivity of medical care when health stock increases by a unit, the right-hand side is the price ratio of avoidance behaviour and medical treatment. Solving the system of equations (6 to 9) together with the budget constraint gives us the optimal avoidance and medical treatment which are functions of climate change (CC), 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:

$$M = g\left(CC, \phi, P_M, P_A, P_X\right) \tag{11}$$

$$A = h\left(CC, \phi, P_M, P_A, P_X\right) \tag{12}$$

From equations (11) and (12), the optimal avoidance behaviour and medical treatment depends on climate change. As a result, we can derive an expression for the relationship between climate change and health by finding the total derivative of equation (2). That is;

$$\frac{dH}{dCC} = \underbrace{\left(\frac{\partial H}{\partial M}\frac{\partial M}{\partial \phi} + \frac{\partial H}{\partial \phi}\right)}_{\frac{dH}{d\phi}} \bullet \underbrace{\left(\frac{\partial \phi}{\partial CC} + \frac{\partial \phi}{\partial A}\frac{\partial A}{\partial CC}\right)}_{\frac{d\phi}{dCC}} \tag{13}$$

From equation (13) it is obvious that the effect of climate change on health has two parts, which are the relationship between climate change and illness (that is, $d\phi/d\,CC$), and the degree to which illness is translated into health status (that is, $dH/d\phi$).

The second expression of equation (13) describes the net effect of climate change on illness incidence based on individuals' exposure level. The expression has two components: the first term ($\partial\phi/\partial CC$) and the second term$\left((\partial\phi/\partial A)(\partial A/\partial CC)\right)$. The first term ($\partial\phi/\partial CC$) represents the pure biological effect of climate change whereas the second term $\left((\partial\phi/\partial A)(\partial A/\partial CC)\right)$ shows the role of avoidance behaviour in averting illness incidence by putting in place mitigation measures against the harmful effect of climate change. From the net effect of climate change (that is, $d\phi/d\,CC$), 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 change. However, if the avoidance behaviour is impossible or ineffective, then the biological effect and the reduced form effects (that is, $d\phi/d\,CC$) will be identical (Zivin and Neidell, 2013).

Similarly, the first expression in equation (13) has two components: the first term $\left((\partial H/\partial M)(\partial M/\partial\phi)\right)$ and second term$\left(\partial H/\partial\phi\right)$. The term $\left((\partial H/\partial M)(\partial M/\partial\phi)\right)$ shows the degree to which medical treatment, which is a post-exposure intervention, reduces the negative effects of climate change on health. Also, the term $\left(\partial H/\partial\phi\right)$ 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.

4. Methodology and data

4.1 Empirical model and variable description

In estimating an empirical model to examine the effect of climate change on health, we modify the optimal medical treatment function in equation (11). The focus here is to investigate the effect of climate change in explaining infectious and parasitic diseases. Thus, we consider how climate change together with socio-economic variables explains the incidence of infectious and parasitic diseases in Sweden. This study relies on panel data from 21 counties in Sweden from 2005 to 2012. Our empirical model from equation (11) is given as;

$$M = g\left(CV, CO_2, \boldsymbol{D}\right) \tag{14}$$

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), CO_2 is carbon dioxide emissions and $\boldsymbol{D}$ is a vector of socio-economic and control variables which include income, education, number of healthcare personnel, population density and immigration[1]. For the dependant variable we consider the number of admissions per 100,000 inhabitants and number of patients per 100,000 inhabitants. Whereas the number of patients' variable is used as our main dependent variable of interest, the number of admissions is used as a sensitivity check. 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. The list of such diseases is shown in the appendix. All the data on the health variables is based on in-patient care diagnoses. In our analysis, we express the dependant variable as non-linear in income, that is, the dependant variable is a quadratic function of income. Income (that is, GDP per capita) is a proxy for the capacity of the county to detect infectious diseases.

We estimate equation (14) under three different assumptions. First, we assume carbon dioxide is exogenous and estimate how current numbers of individual who seek medical treatment are driven by climate variability, carbon dioxide emission and socio-economic variables (including immigration). That is:

$$M_{it} = \beta_0 + \sum_{j=0}^{1} \beta_{j+1} Temp_{it-j} + \beta_3 \Pr ecip_{it} + \beta_4 CO_{2it} + \beta_5 \operatorname{Im} migrants_{it} + \boldsymbol{\delta' D_{it}} + \eta_i + \varepsilon_{it} \tag{15}$$

Each variable in equation (15) is a panel data set for county i in time period t. The term $\boldsymbol{\delta' D_{it}}$ in equation (15) is the product of the vector of socio-economic and control variables and their corresponding parameter, and η_i is the county fixed effect variable. In estimating equation (15), we utilize fixed effect (FE) panel estimation technique to account for county-fixed effects capturing any county differences which may have effect on the dependent variable.

[1] 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.

Secondly, we assume carbon dioxide emissions to be endogenous since there is the possibility of carbon dioxide emissions in each county to be dependent on economic activities. As such we also estimate equation (15) again by using fixed effect instrumental variable (FEIV) technique. This is to address the endogeneity problem of carbon dioxide emissions and also account for county-fixed effects. We use energy consumption as an instrument for carbon dioxide emissions. This is based on the premise that energy consumption may have a strong correlation with carbon dioxide emission hence satisfying the relevance assumption. Also, there is a weak or no direct relationship between energy consumption and how individuals seek for medical treatment from infectious diseases. Thus, this provides a justification for fulfilling the excludability assumption.

Since it is difficult to have a valid instrument in reality and there is also the possibility of infectious and parasitic diseases to portray persistence, we consider a dynamic model in our third scenario and assume carbon dioxide emission to be endogenous. We use the lag of carbon dioxide emission as an instrument for itself since the lags of a variable is a valid instrument for itself. In this case, we estimate equation (16) below;

$$M_{it} = \beta_0 + \kappa M_{it-1} + \sum_{j=0}^{1} \beta_{j+1} Temp_{it-j} + \beta_3 \Pr ecip_{it} + \beta_4 CO_{2it} + \beta_5 \operatorname{Im} migrants_{it} + \boldsymbol{\delta' D_{it}} + \eta_i + \varepsilon_{it} \quad (16)$$

where CO_2 emission is endogenous and we use its lag as an instrument, κ is the coefficient of the lag of medical treatment. All the other variables maintain their initial definitions. A two-step system generalised method of moment (GMM) is used to estimate equation (16). In all three scenarios, 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. With regard to the control variables we consider GDP per capita, number of the population with post-secondary education three years or more, population density and number of medical personnel. We also consider the number of immigrants since it is a variable of interest.

The variables considered in our analysis are in line with the argument that transmission of infectious diseases is determined by many factors which includes; social, economic and ecological conditions, access to health care, and intrinsic human immunity (Semenza and Menne, 2009; Jones et al., 2008). With the exception of temperature and precipitation, we transform all the variables by taking the natural logarithm. We present the variable description and summary statistics in Table 2. From the summary statistics, we observe great 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 for winter is about 1.4^0C with a standard deviation of about 2.5^0C. Also, the average annual mean precipitation deviation from the normal is about 12.4mm with a standard deviation of about 12.9mm.

Table 2: Variable description, data sources and descriptive statistics

Variable	Description	Source	Obs	Mean	Std. Dev.	Min	Max
Lnadmission	natural log of the number of admissions per 100,000 inhabitants	NBHW	168	6.213	0.132	5.867	6.616
Lnpatients	natural log of the number of patients per 100,000 inhabitants	NBHW	168	6.068	0.130	5.722	6.437
lnco2	natural log of carbon dioxide emissions per capita in thousand tons	RUS & SCB	168	1.765	0.594	0.788	3.752
Lngdppc	natural log of GDP per capita	SCB	168	5.761	0.141	5.509	6.306
Lngdppcsq	lngdppc squared		168	33.211	1.656	30.353	39.769
Lneducation	natural log of the number of the population with post-secondary education three years or more	SCB	168	3.499	0.903	1.656	5.986
Lnpersonel	natural log of the number of health personnel	NBHW	168	7.409	0.092	7.226	7.642
Lnpopuladen	natural log of population density	SCB	168	3.198	1.141	0.916	5.787
Tempav	annual average temperature deviation from the normal (^{0}C)	SMHI	168	1.052	0.880	-1.600	2.400
Tempwint	annual winter mean temperature deviation from the normal (^{0}C)	SMHI	168	1.407	2.525	-3.300	6.300
Tempsum	annual summer mean temperature deviation from the normal (^{0}C)	SMHI	168	0.858	0.791	-0.700	2.600
Precipiav	annual average precipitation deviation from the normal (mm)	SMHI	168	12.373	12.937	-13.800	45.900
Lnimmigration	natural log of the number of immigrants	SCB	168	7.802	1.022	5.056	10.406
Lnenergyconsum	natural log of energy consumption	SCB	126	9.570	0.754	8.156	11.120

NB: 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.

4.2 Model Estimation

As mentioned earlier, the study makes use of FE, FEIV and two step system GMM estimation techniques. The use of the FE panel estimation technique is to provide more consistent estimator, while the FEIV is to correct the problem of endogeneity in the case of carbon dioxide emissions which is a regressor in our model. Also, the two-step system GMM is used to estimate equation (16). This method unlike the Arellano-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-Bover(1995)/ Blundell-Bond (1998) technique or the system GMM augment that of Arellano-Bond by making additional assumption that the first differences of the instrumental variables are uncorrelated with the fixed effects.

The estimation of the fixed effect model is analysed by considering the basic regression model of the form (Wooldridge, 20l0):

$$y_{it} = x'_{it}\beta + z'_i\alpha + \varepsilon_{it} \qquad (17)$$

From equation (17), the vector x_{it} contains *K* regressors which do not include a constant term. The heterogeneity or individual effect is represented by $z'_i a$ where the vector z_i contains a constant term and a set of individual or group specific variables which may be observed or unobserved, all of which are taken to be constant over time *t*. With regard to the fixed effect model, if z_i in equation (17) is unobserved though correlated with x_{it}, then the least squares estimator of β (vector) will be biased and inconsistent due to an omitted variable. In such a case the model from equation (17) will now be;

$$y_{it} = x'_{it}\beta + \eta_i + \varepsilon_{it} \qquad (17a)$$

where $\eta_i = z'_i\alpha$, which represents all observable effects and specifies an estimable conditional mean. The fixed[2] effect approach takes η_i to be a group-specific constant term in the regression model. By using the fixed effect estimation technique we account for 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 across-group action and as such provide consistent estimates.

In relation to the fixed effect instrumental variable technique, there is the tendency for carbon emissions to be endogenous since carbon dioxide emissions may be affected by economic activities and other unobserved variables which affect the likelihood of individuals to seek medical treatment from infectious disease. Thus, we use energy consumption as an instrument for carbon dioxide emissions. There is a strong correlation between carbon dioxide emissions and energy consumption since energy consumption is dependent on the amount of energy produced which in turn affects the level of emissions. With the relationship between carbon dioxide emissions and energy consumption, the relevance assumption characterising the selection of an instrument is met. Our first-stage estimation shows a significant positive effect of energy consumption on carbon dioxide emission, we however do not show the results of the first-stage estimation because of space. With regard to the excludability restriction, the energy level

[2] It should be noted that the term "fixed" as used here signifies the correlation of η_i and x_{it}, not that η_i is non-stochastic

consumed may have weak or no direct relationship with the likelihood of an individual seeking treatment from infectious disease.

However, in reality it is very difficult to have a valid instrument. As such system GMM becomes handy in addressing the endogeneity problem. Also, given the fact that infectious diseases exhibit persistence, the endogeneity problem attributed to the presence of the lag of the dependent variables (in this case infectious diseases) as a regressor in equation (16) can be addressed by using system GMM. The general model within the system GMM framework which considers individual effects is given by equation (18).

$$y_{it} = \sum_{k=1}^{p} \gamma_k y_{i,t-k} + \boldsymbol{x_{it}}' \boldsymbol{\beta} + \varepsilon_{it} \quad (t = p+1,...,T; i = 1,...,N) \tag{18}$$
$$\varepsilon_{it} = \eta_i + \mu_{it}$$

Where the error term (ε_{it}) comprises of the fixed effect (η_i) and the idiosyncratic stock (μ_{it}), $\boldsymbol{x}_{it}$ is the vector of explanatory variables, $\boldsymbol{\beta}$ represents a vector of the associated estimators and *p* is the maximum lag length in the model. From equation (18), *T* is the number of time periods available to each individual *i*. In order for the model to be identified, there should be a restriction on the serial correlation properties of the error term (μ_{it}) and/or the properties of the explanatory variables $\boldsymbol{x}_{it}$. In the model, the error terms are assumed to be independently distributed across individuals with a mean of zero, however arbitrary form of heteroskedasticity across each *i* and time are possible. Also, the explanatory variable $\boldsymbol{x}_{it}$ may or may not be correlated with the individual effects (η_i). Thus, for each of these cases the effects may be strictly exogenous, predetermined, or endogenous variables with respect to the error term (μ_{it}).

We can write the *T* time periods for a random draw *i* based on equation (18) as;

$$y_i = W_i\theta + \iota_i\eta_i + \mu_i \tag{19}$$

From equation (19), θ is a parameter vector which contains the γ_k's and the β's, and W_i is a data matrix containing the time series of the lagged dependent variable and the *x*'s. Also ι_i represents the *T*x1 vectors of unity. We use dynamic panel data to compute various linear GMM estimators of θ with the general form shown in equation (20):

$$\hat{\theta} = \left[\left(\sum_i W_i *' Z_i \right) A_N \left(\sum_i Z W_i * \right) \right]^{-1} \left(\sum_i W_i *' Z_i \right) A_N \left(\sum_i Z_i' y_i * \right) \tag{20}$$

where

$$A_N = \left(\frac{1}{N} \sum_i Z_i' H_i Z_i \right)^{-1} \tag{21}$$

and $W_i *$ and $y_i *$ denote some transformation of W_i and y_i, Z_i represents a matrix of instrumental variables which may or may not be entirely internal. Also H_i is individual specific weighting matrix.

In order to estimate the dynamic model, the instrument used is mostly a transformation of the lagged endogenous (or predetermined) variables. In situations where there is no instrument that is uncorrelated with the individual effect, the transformation we make should be able to eliminate the component of correlation from the error term. 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 essence, if μ_{it} is serially uncorrelated then $\Delta\mu_{it}$ is correlated with $\Delta\mu_{i,t-1}$, however $\Delta\mu_{it}$ will not be correlated with $\Delta\mu_{i,t-k}$ for $k \geq 2$. 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.

Unbiased estimates of the parameters in equation (18) cannot be obtained using the within group or first difference technique in a static panel data model since the transformed lagged dependent variable will be correlated with the transformed error term. The appropriate technique to estimate consistent and efficient estimates of the parameters is the use of a system GMM technique; this is the technique we use in our estimations. Since the unobserved individual effects (η_i) may be correlated with other explanatory variables, expressing equation (18) in first-order difference helps to remove the correlation between the individual effect and the explanatory variables hence avoiding biases in the estimates. In other words, the first differences of the instrumental variables are uncorrelated with the fixed individual effects. 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. Also, carbon dioxide emission is endogenous and its lag is used as an instrument. The other explanatory variables in equation (16) 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 and that of carbon dioxide emissions in generating the consistent and unbiased estimates.

5. Empirical results and discussion

5.1 Results

The results from the empirical estimations are shown in Tables 3 and 4. Whereas Table 3 is the main result of interest, Table 4 is used as a sensitivity check. All the estimated models satisfy the fitness tests. Also, the system GMM estimations satisfy the over-identification and no serial correlation tests. Thus, 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 Tables 3 and 4, since the null hypothesis for the Sargan's test are not rejected. On the no serial correlation test, it can be seen from both tables 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 not significant for the second order autocorrelation. These guarantee the consistency of the estimators and the validity of the instruments used.

From the FE model in Table 3, the results show generally that the number of infectious disease patients are significantly affected by climate variables, income per capita, the squared of income per capita and immigration with variations based on whether winter, summer or average temperature values are used. In the case where winter temperature is used, the lag of winter temperature, income per capita and immigration positively drive the number of infectious disease patients. However, current period's winter temperature and income per capita squared have negative effect on the number of infectious disease patients (see column 1 of Table 3). When summer temperature is used, however, we find lagged summer temperature and current year's precipitation have a negative effect on the number of infectious disease patients (see column 2 of Table 3). Similarly, using average temperature in the estimation, current year's temperature and its lag have a negative effect on the number of infectious disease patients. The effect of income per capita, income per capita squared and immigration in columns 2 and 3 have the same sign as discussed earlier (see column 1 of Table 3).

The results from the FEIV estimation are similar to the FE case with slight differences in the case where winter temperature is used. From column 4 of Table 3, we find that the number of infected patients is significantly caused by current winter temperature, income per capita and its squared. However, in the case of the FE model we observe these variables in column 4 together with the lag of winter temperature and number of immigrants to significantly cause the number of infectious disease patients. In the case where summer and average temperature values are used in estimating the FEIV model in columns 5 and 6 respectively, we find the factors affecting the number of infectious diseases patients to be the same as the case discussed earlier for the FE case (see columns 2 and 3 of Table 3). The signs of the significant variables which affect the number of infectious disease patients when we estimate our model using FEIV are the same as when we used FE.

From the system GMM results where we include the lag of the number of infectious disease patients as a regressor, our results show that the number of infectious disease patients is significantly affected by lag of the number of infectious disease patients, current winter temperature and its lag, income per capita and its squared, when we use winter temperature in the estimation (see column 7 of Table 3). Here, we find the number of immigrants do not affect the number of infectious disease patients unlike in the FE model. The positive effect of previous period's infectious disease patient on current ones in winter implies that current period's patients have the tendency of increasing infection in the next period, especially during winter. This is because most people are indoors during winter and any spring of infectious disease in a place has the tendency of infecting other persons hence increasing the number of infectious disease patients. For example, an influenza which is most common in Sweden in winter, when it is caught up by one person, it has the greater tendency of infecting other persons within the same household, neighbourhood or environment.

In the case of column 8 of Table 3 where summer temperature is used, we find the number of infectious disease patients to be significantly affected by carbon dioxide emissions, lag of summer temperature, current year's precipitation, income per capita and the number of health personnel. Unlike the previous results, the number of health personnel and carbon dioxide emission are key variables in explaining the number of infectious disease patients. The results suggest that the number of health personnel and carbon dioxide emissions have positive effect

on the number of infectious disease patients. The positive effect of health personnel on the number of infectious disease patients when summer temperature is used (see column 8) implies that as the number of health personnel increases the number of infectious disease patients also increase. This result is quiet surprising. However, it can be explained as, the number of health personnel will enhance the health facility's capacity to attend to more patients at a given time. Also, many more patients are likely to report to health facilities with adequate personnel to attend to their needs, especially during summer. As such there will be more reported cases of diseases. In relation to the positive effect of carbon dioxide emissions on the number of infectious disease patients, it means that during summer an increase in carbon emissions will increase the number of infectious disease patients. Ambient pollution which includes carbon dioxide emissions has the tendency of increasing respiratory tract diseases which are infectious hence higher emissions of such pollutants will increase the number of patients. On the contrary, when average temperature is used (see the system GMM results), we observe the number of infectious disease patients to be significantly affected by current and previous temperature, income per capita and its squared (see column 9 of table 3).

5.2 Discussions

Generally, we observe current winter temperature values to have negative effect on the number of infectious disease patients. This means that a reduction in winter temperature increases the incidence of infectious diseases. Also, we observe the lag of summer temperature, average temperature and its lag to have negative effect on the number of infectious disease patients. Thus, a lower temperature especially in winter may increase the incidence of infectious disease like influenza and others. However, the lag of winter temperature has a positive impact on the number of infectious disease patients. Infectious diseases like vector-borne and tick-borne diseases are mostly affected by temperature. Thus, temperature 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 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 our results, the current projection of climate change of high temperature has the tendency of reducing the number of infectious disease patients in Sweden. The negative effect of precipitation on the number of infectious disease patient is contrary to our expectation.

Table 3: Determinants of the number of infectious disease patients

	Fixed Effect Model			Fixed Effect Instrumental Variable Model			System Generalised Method of Moment		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
VARIABLES	Winter	Summer	Average	Winter	Summer	Average	Winter	Summer	Average
Lnpatients (-1)							0.197**	0.135	0.195
							(0.0935)	(0.0905)	(0.124)
LnCO2	0.0164	0.0406	-0.0133	-0.410	-0.338	-0.408	0.157	0.165*	-0.0863
	(0.0949)	(0.0971)	(0.0975)	(0.324)	(0.321)	(0.329)	(0.107)	(0.0960)	(0.141)
Winter Temp.	-0.00838***			-0.0103**			-0.0054***		
	(0.00277)			(0.00411)			(0.00164)		
Winter Temp.(-1)	0.00650*			0.00690			0.0100***		
	(0.00335)			(0.00427)			(0.00226)		
Summer Temp.		0.00681			0.00601			-0.000944	
		(0.00911)			(0.0123)			(0.00674)	
SummerTemp(-1)		-0.0191**			-0.0260**			-0.0272***	
		(0.00886)			(0.0116)			(0.00661)	
Average Temp.			-0.0228***			-0.0336***			-0.0309***
			(0.00775)			(0.0124)			(0.00483)
Avera. Temp(-1)			-0.0208**			-0.0231**			-0.0185***
			(0.00811)			(0.0117)			(0.00431)
Precipitation	-0.000383	-0.001000*	-0.000557	0.000136	-0.000545	0.0000178	-0.000198	-0.00111***	-0.000382
	(0.000557)	(0.000545)	(0.000546)	(0.000737)	(0.000728)	(0.000703)	(0.000354)	(0.000383)	(0.000350)
Precipitation (-1)	0.000258	0.000110	0.000066	0.000279	-0.000091	0.000029	0.000035	0.000577	0.0000175
	(0.000493)	(0.000520)	(0.000482)	(0.000642)	(0.000655)	(0.000608)	(0.000377)	(0.000440)	(0.000370)
LnGDP per cap.	13.46***	13.98***	14.01***	16.15**	17.29**	16.47**	33.23***	16.30*	18.00**
	(4.570)	(4.809)	(4.586)	(6.861)	(6.971)	(6.779)	(12.51)	(9.871)	(8.861)
LnGDPpercap. sq	-1.143***	-1.194***	-1.209***	-1.354**	-1.458**	-1.399**	-2.836***	-1.393	-1.551**
	(0.394)	(0.414)	(0.396)	(0.586)	(0.595)	(0.580)	(1.089)	(0.861)	(0.766)
LnEducation	0.251	0.322	0.0963	-0.170	-0.0546	-0.289	0.209	-0.0939	-0.104
	(0.223)	(0.222)	(0.231)	(0.538)	(0.505)	(0.568)	(0.142)	(0.139)	(0.149)
LnHealthpersonn.	-0.0331	0.0481	-0.105	-0.0836	0.0616	-0.209	0.479	1.239*	-0.216

	(0.441)	(0.448)	(0.442)	(0.565)	(0.567)	(0.548)	(0.365)	(0.654)	(0.493)
LnPopulationden	-0.00350	0.0786	0.145	-0.0806	-0.00843	0.0840	0.00413	0.0597	-0.00284
	(0.346)	(0.356)	(0.348)	(0.659)	(0.659)	(0.672)	(0.0260)	(0.0443)	(0.0252)
LnImmigration	0.0991*	0.162***	0.217***	0.0311	0.111*	0.164**	-0.0424	0.0697	0.0182
	(0.0577)	(0.0504)	(0.0560)	(0.0734)	(0.0651)	(0.0695)	(0.116)	(0.127)	(0.0693)
Constant	-34.89***	-37.87***	-36.15***	-40.05**	-45.60**	-40.51**	-96.61***	-52.25*	-45.26*
	(12.87)	(13.62)	(12.91)	(19.87)	(20.47)	(19.61)	(37.44)	(30.82)	(26.06)
Observations	167	167	167	125	125	125	147	147	147
R-squared	0.405	0.382	0.405	0.346	0.355	0.375			
No. of counties	21	21	21	21	21	21	21	21	21
Sargan's test							9.80 (32)	13.44 (32)	14.74 (32)
1st order autocorr.							-1.998**	-1.73*	-1.867*
2nd order autocor							-0.958	-1.16	-0.668

Standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1.In the Sargan's test we presented the χ^2 values and the degree of freedom are in parentheses. We presented the z values for the autocorrelation test. For the fixed effect instrumental variable estimations, we assume carbon dioxide emission is endogenous so we use energy consumption as an instrument together with other regressors used in the second step. Also, in the system GMM the lag of carbon dioxide emission is used as an instrument for carbon dioxide emission since lags of a variable is a better instrument for itself.

This is only observed in the situation when summer temperature is used in the model, and may explain the negative relationship between precipitation and the number of infectious disease patients. According to Martens *et al.*, (1995) and NRC (2001), the suitability of vector habitats is determined by minimum precipitation levels. Thus, a reduction in precipitation will create conducive environment for vectors which will in turn increase infection of diseases hence more patients.

The positive effect of income per capita and the negative effect of the squared of the income per capita imply that the relationship between the number of infectious disease patients and income per capita is an inverted-U shape. This means that as the income per capita in Swedish counties increase, there is more coverage to track individuals with infectious diseases hence the number of infectious patients increases. However, beyond a certain income level the number of infectious disease 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 channelled into the health sector and this will help access individuals with the disease so the number of recorded cases will definitely increase. As the investment or income increases, complemented with the necessary treatment, the number of cases starts decreasing. Thus, increase in income per capita may imply higher resources for public health services which significantly affect the distribution of diseases, since the very purpose of such services is to stem the spread of disease in populations. 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 antimicrobial agents which has the tendency of altering the pattern of infectious disease.

In relation to the positive effect of immigration on the number of infectious disease patients, the results imply 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 (NRC, 2001).

5.3 Sensitivity Analysis

In Table 4 we use the number of infectious disease individuals admitted as the dependent variable in the estimation. As argued earlier, we use this to check how sensitive our results are. From the FE results in Table 4, we find factors that significantly affect the number of infectious disease admissions to be the same as that of the number of infectious disease patients in Table 3 if average temperature is considered in the model. However, we observe slight variations in the factors when winter and summer temperature values are used. In the case where winter temperature is alternatively included in the estimated model, we find the number of infectious

disease admissions is mainly driven by current and previous temperature in winter, income per capita and its squared, and education. Unlike the evidence shown in Table 3, where the number of infectious disease patients is significantly explained by immigrants when winter temperature is controlled for, the situation in column 1 of Table 4 is different. Also, from column 2 of Table 4 we find all the factors affecting the number of infectious disease patients in column 2 of Table 3 also affect the number of infectious disease admissions. However, education explains the number of infectious disease admission in the case where summer temperature is used (see column 2 of Table 4).

The direction of the effect of the factors affecting the number of infectious disease admissions is the same as that of the number of infectious disease patients discussed earlier. In the case of education, we find a positive effect on the number of infectious disease admission. This means that as the number of individuals having post-secondary education increases the higher the number of infectious disease admissions. This is however contrary to our expectation. It might be that the more educated would seek for more medical services and opt for admission when he/she is infected by infectious disease in order not to spread it to close relatives and friends. In the case of the FEIV results, we find the number of infectious disease admissions to be affected by the same factors as that of the number of infectious disease patients when winter, summer and average temperature variables are used.

Similar to the case of FE in Table 4, results for the system GMM estimation show minor differences to that shown in Table 3. Whereas previous infectious disease patients affect current ones in the case of Table 3 (see column 7), previous infectious disease admissions do not affect current ones in the case of Table 4 (see column 7), when winter temperature value is used. However, the number of health personnel significantly explains infectious disease admissions but not patients when winter temperature value is considered. In the case where summer temperature is used for the system GMM estimation in Table 4, we find the number of infectious disease admission to be affected by previous admissions, income per capita squared, population density in addition to factors which drive infectious disease patients (see results in Table 3). We observe population density to have positive effect on infectious disease admissions after controlling for summer temperature. This means that infection is facilitated by population concentration because infected individuals have a higher probability of contact with susceptible members of the population. In the literature (see NRC, 2001), population density has been linked with increasing ease of transmission of airborne infections, waterborne diseases, and sexually transmitted infections.

Table 4: Determinants of the number of infectious disease admissions

	Fixed Effect Model			Fixed Effect Instrumental Variable Model			System Generalised Method of Moment		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
VARIABLES	Winter	Summer	Average	Winter	Summer	Average	Winter	Summer	Average
Lnadmission (-1)							0.154	0.111*	0.208**
							(0.128)	(0.0669)	(0.105)
LnCO2	0.0473	0.0748	0.00824	-0.352	-0.262	-0.373	0.157	0.195***	-0.00922
	(0.0994)	(0.101)	(0.102)	(0.334)	(0.330)	(0.341)	(0.114)	(0.0752)	(0.109)
Winter Temp.	-0.00763***			-0.00979**			-0.00521***		
	(0.00290)			(0.00424)			(0.00201)		
Winter Temp.(-1)	0.00841**			0.00844*			0.0113***		
	(0.00350)			(0.00441)			(0.00181)		
Summer Temp.		0.00107			0.000690			-0.0113	
		(0.00950)			(0.0126)			(0.00739)	
SummerTemp(-1)		-0.0235**			-0.0295**			-0.0353***	
		(0.00924)			(0.0119)			(0.00767)	
Average Temp.			-0.0243***			-0.0359***			-0.0335***
			(0.00812)			(0.0129)			(0.00605)
Avera. Temp(-1)			-0.0192**			-0.0202*			-0.0200***
			(0.00850)			(0.0122)			(0.00563)
Precipitation	-0.000546	-0.00121**	-0.000734	0.000115	-0.000590	-0.000022	-0.000434	-0.00151***	-0.000753*
	(0.000583)	(0.000569)	(0.000572)	(0.000761)	(0.000747)	(0.000730)	(0.000426)	(0.000450)	(0.000410)
Precipitation (-1)	0.000107	-0.000099	-0.000057	0.000255	-0.000186	0.0000186	-0.000214	0.000450	0.000025
	(0.000517)	(0.000543)	(0.000506)	(0.000663)	(0.000672)	(0.000631)	(0.000351)	(0.000341)	(0.000327)
LnGDP per cap.	13.25***	14.74***	13.97***	14.13**	16.19**	14.61**	29.42**	17.88*	10.86
	(4.784)	(5.015)	(4.807)	(7.085)	(7.156)	(7.035)	(14.15)	(9.321)	(9.792)
LnGDPpercap. sq	-1.123***	-1.258***	-1.206***	-1.180*	-1.366**	-1.240**	-2.500**	-1.524*	-0.940
	(0.413)	(0.432)	(0.415)	(0.606)	(0.611)	(0.602)	(1.232)	(0.807)	(0.844)
LnEducation	0.416*	0.416*	0.210	0.0469	0.131	-0.136	0.154	-0.0657	-0.160
	(0.234)	(0.231)	(0.242)	(0.556)	(0.519)	(0.589)	(0.119)	(0.105)	(0.160)
LnHealthpersonn.	-0.000776	0.0493	-0.0890	-0.0668	0.0525	-0.194	1.054*	1.254**	0.395

	(0.462)	(0.467)	(0.463)	(0.584)	(0.582)	(0.568)	(0.587)	(0.531)	(0.453)
LnPopulationden	-0.181	-0.0470	-0.00919	-0.333	-0.225	-0.120	0.0220	0.0693**	0.00137
	(0.362)	(0.372)	(0.365)	(0.681)	(0.676)	(0.698)	(0.0303)	(0.0349)	(0.0291)
LnImmigration	0.0875	0.178***	0.220***	0.0199	0.123*	0.157**	0.00796	0.0227	0.112**
	(0.0604)	(0.0525)	(0.0587)	(0.0758)	(0.0668)	(0.0721)	(0.107)	(0.0595)	(0.0546)
Constant	-34.46**	-40.05***	-35.95***	-34.10*	-42.26**	-34.97*	-89.98**	-56.62**	-29.55
	(13.48)	(14.21)	(13.54)	(20.52)	(21.02)	(20.35)	(43.50)	(27.05)	(27.23)
Observations	167	167	167	125	125	125	147	147	147
R-squared	0.433	0.416	0.431	0.3897	0.405	0.411			
No. of counties	21	21	21	21	21	21	21	21	21
Sargan's test							10.54 (32)	14.60 (32)	17.34 (32)
1st order autocorr.							-1.702*	-1.732*	-1.676*
2nd order autocor							-0.944	-1.227	-0.782

Standard errors in parentheses, *** p<0.01, ** p<0.05, * p<0.1.In the Sargan's test we presented the χ^2 values and the degree of freedom are in parentheses. We presented the z values for the autocorrelation test. For the fixed effect instrumental variable estimations, we assume carbon dioxide emission is endogenous so we use energy consumption as an instrument together with other regressors used in the second step. Also, in the system GMM the lag of carbon dioxide emission is used as an instrument for carbon dioxide emission since lags of a variable is a better instrument for itself.

We find much difference in the factors which affect the number of infectious disease patients and admissions when average temperature values are used (see column 9 of Tables 3 and 4). With regard to the number of infectious disease admissions, we find previous admissions, average temperature and its lag, precipitation and the number of immigrants to significantly cause the number of infectious disease admissions. Conversely, the number of infectious disease patients is significantly caused by increases in average temperature and its lag, as well as income per capita and its squared.

6.0 Conclusions

In this study, we examine the effect of climate variability, carbon dioxide emissions, migration and other socio-economic variables on the incidence of infectious diseases using on panel data for the period 2005-2012 for all Swedish 21 counties. We consider the number of patients and admissions from infectious diseases as the outcome variables in this paper. By developing a theoretical model based on Zivin and Neidell (2013) to analyse how climate change affects health, we observe that medical treatment is a function of climate change, the function that translate climate change into illness incidence and the costs of avoidance behaviour, medical care and all other consumption goods. Also, the effect of climate change on health can be decomposed into the relationship between climate change and illness, and the degree to which illness is translated into health status. The relationship between climate change and illness is described as the net effect of climate change on illness incidence based on individuals' exposure level.

In order to empirically estimate our theoretical model, we employ three different estimation techniques (FE, FEIV and system GMM) in order to check for the robustness of our results. From the empirical results, we generally observe the number of infectious disease patients and admissions are caused by temperature, income per capita and the number of immigrants. We find mixed effect of temperature on the number of infectious disease patients and admissions depending on whether winter, summer and average temperature are separately included in the estimated model. The relationship between infectious disease proxies (i.e. patients and admissions) and income per capita portray an inverted-U shape. Further, in most cases, the number of immigrants is found to have a positive effect on the number of infectious disease patients and admissions. When summer temperature value is used, we find precipitation to have negative effect on the number of infectious disease patients and admissions.

In few cases we find that the number of infectious disease admissions is caused by education, number of health personnel and population density. Also, carbon dioxide emissions significantly impact on the number of infectious disease patients and admissions in summer (evidence based on the system GMM). In summary, our results show that infectious diseases in Sweden are significantly caused by climate change and socio-economic variables (such as income, number of immigrants, among others). This suggests that investment into public health services in the long run will have negative impact on the number of infectious disease patients and admissions. There should therefore be adaptation and mitigation strategies to address the impact on climate change on health. Further, inclusion of climate sensitive infectious diseases on the list of notifiable

diseases should be paramount. 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.

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Appendix

A00-B99: Infectious and parasitic diseases from National Board of Health and Welfare in Sweden database (http://www.socialstyrelsen.se/statistics/statisticaldatabase/inpatientcarediagnoses)

A00-A09 Intestinal infectious diseases

A00 Cholera; A01 Typhoid and paratyphoid fevers; A02 Other salmonella infections; A03 Shigellosis; A04 Other bacterial intestinal infections; A05 Other bacterial foodborne intoxications; A06 Amoebiasis; A07 Other protozoal intestinal diseases; A08 Viral and other specified intestinal infections; A09 Diarrhoea and gastroenteritis of presumed infectious originshow/hide subcategories

A15-A19 Tuberculosis

A15 Respiratory tuberculosis, bacteriologically and histologically confirmed; A16 Respiratory tuberculosis, not confirmed bacteriologically or histologically; A17 Tuberculosis of nervous system; A18 Tuberculosis of other organsA19 Miliary tuberculosisshow/hide subcategories

A20-A28 Certain zoonotic bacterial diseases

A20 Plague; A21 Tularaemia; A22 Anthrax; A23 Brucellosis; A24 Glanders and melioidosis; A25 Rat-bite fevers; A26 Erysipeloid; A27 Leptospirosis; A28 Other zoonotic bacterial diseases, not elsewhere classifiedshow/hide subcategories

A30-A49 Other bacterial diseases

A30 Leprosy [Hansen's disease]; A31 Infection due to other mycobacteria; A32 Listeriosis; A34 Obstetrical tetanus; A35 Other tetanus; A36 Diphtheria; A37 Whooping cough; A38 Scarlet fever; A39 Meningococcal infection; A40 Streptococcal septicaemia; A41 Other septicaemia; A42 Actinomycosis; A43 Nocardiosis; A44 Bartonellosis; A46 Erysipelas; A48 Other bacterial diseases, not elsewhere classified; A49 Bacterial infection of unspecified siteshow/hide subcategories

A50-A64 Infections with a predominantly sexual mode of transmission

A50 Congenital syphilis; A51 Early syphilis; A52 Late syphilis; A53 Other and unspecified syphilis; A54 Gonococcal infection; A55 Chlamydial lymphogranuloma (venereum); A56 Other sexually transmitted chlamydial diseases; A57 Chancroid; A59 Trichomoniasis; A60 Anogenital herpesviral [herpes simplex] infection; A63 Other predominantly sexually transmitted diseases, not elsewhere classified; A64 Unspecified sexually transmitted diseaseshow/hide subcategories

A65-A69 Other spirochaetal diseases

A65 Nonvenereal syphilis; A66 Yaws; A67 Pinta [carate]; A68 Relapsing fevers; A69 Other spirochaetal infectionsshow/hide subcategories

A70-A74 Other diseases caused by chlamydiae

A70 Chlamydia psittaci infection; A71 Trachoma; A74 Other diseases caused by chlamydiaeshow/hide subcategories

A75-A79 Rickettsioses

A75 Typhus fever; A77 Spotted fever [tick-borne rickettsioses]; A78 Q fever; A79 Other rickettsiosesshow/hide subcategories

A80-A89 Viral infections of the central nervous system

A80 Acute poliomyelitis; A81 Slow virus infections of central nervous system; A82 Rabies; A83 Mosquito-borne viral encephalitis; A84 Tick-borne viral encephalitis; A85 Other viral encephalitis, not elsewhere classified; A86 Unspecified viral encephalitis; A87 Viral meningitis; A88 Other viral infections of central nervous system, not elsewhere classified; A89 Unspecified viral infection of central nervous systemshow/hide subcategories

A90-A99 Arthropod-borne viral fevers and viral haemorrhagic fevers

A90 Dengue fever [classical dengue]; A91 Dengue haemorrhagic fever; A92 Other mosquito-borne viral fevers; A93 Other arthropod-borne viral fevers, not elsewhere classified; A94 Unspecified arthropod-borne viral fever; A95 Yellow fever; A96 Arenaviral haemorrhagic fever; A98 Other viral haemorrhagic fevers, not elsewhere classified; A99 Unspecified viral haemorrhagic feversshow/hide subcategories

B00-B09 Viral infections characterized by skin and mucous membrane lesions

B00 Herpesviral [herpes simplex] infections; B01 Varicella [chickenpox]; B02 Zoster [herpes zoster]; B03 Smallpox; B04 Monkeypox; B05 Measles; B06 Rubella [German measles]; B07 Viral warts; B08 Other viral infections characterized by skin and mucous membrane lesions, not elsewhere classified; B09 Unspecified viral infection characterized by skin and mucous membrane lesionsshow/hide subcategories

B15-B19 Viral hepatitis

B15 Acute hepatitis A; B16 Acute hepatitis B; B17 Other acute viral hepatitis; B18 Chronic viral hepatitis; B19 Unspecified viral hepatitisshow/hide subcategories

B20-B24 Human immunodeficiency virus [HIV] disease

B20 Human immunodeficiency virus [HIV] disease resulting in infectious and parasitic diseases; B21 Human immunodeficiency virus [HIV] disease resulting in malignant neoplasms; B22 Human immunodeficiency virus [HIV] disease resulting in other specified diseases; B23 Human immunodeficiency virus [HIV] disease resulting in other conditions; B24 Unspecified human immunodeficiency virus [HIV] diseaseshow/hide subcategories

B25-B34 Other viral diseases

B25 Cytomegaloviral disease; B26 Mumps; B27 Infectious mononucleosis; B30 Viral conjunctivitis; B33 Other viral diseases, not elsewhere classified; B34 Viral infection of unspecified siteshow/hide subcategories

B35-B49 Mycoses

B35 Dermatophytosis; B36 Other superficial mycoses; B37 Candidiasis; B38 Coccidioidomycosis; B39 Histoplasmosis; B40 Blastomycosis; B41 Paracoccidioidomycosis; B42 Sporotrichosis; B43 Chromomycosis and phaeomycotic abscess; B44 Aspergillosis; B45 Cryptococcosis; B46 Zygomycosis; B47 Mycetoma; B48 Other mycoses, not elsewhere classified; B49 Unspecified mycosisshow/hide subcategories

B50-B64 Protozoal diseases

B50 Plasmodium falciparum malaria; B51 Plasmodium vivax malaria; B52 Plasmodium malariae malaria; B53 Other parasitologically confirmed malaria; B54 Unspecified malaria; B55 Leishmaniasis; B56 African trypanosomiasis; B57 Chagas' disease; B58 Toxoplasmosis; B59 Pneumocystosis; B60 Other protozoal diseases, not elsewhere classified; B64 Unspecified protozoal diseaseshow/hide subcategories

B65-B83 Helminthiases

B65 Schistosomiasis [bilharziasis]; B66 Other fluke infections; B67 Echinococcosis; B68 Taeniasis; B69 Cysticercosis; B70 Diphyllobothriasis and sparganosis; B71 Other cestode infections; B72 Dracunculiasis; B73 Onchocerciasis; B74 Filariasis; B75 Trichinellosis; B76 Hookworm diseases; B77 Ascariasis; B78 Strongyloidiasis; B79 Trichuriasis; B80 Enterobiasis; B81 Other intestinal helminthiases, not elsewhere classified; B82 Unspecified intestinal parasitism; B83 Other helminthiasesshow/hide subcategories

B85-B89 Pediculosis, acariasis and other infestations

B85 Pediculosis and phthiriasis; B86 Scabies; B87 Myiasis; B88 Other infestations; B89 Unspecified parasitic diseaseshow/hide subcategories

B90-B94 Sequelae of infectious and parasitic diseases

B90 Sequelae of tuberculosis; B91 Sequelae of poliomyelitis; B92 Sequelae of leprosy; B94 Sequelae of other and unspecified infectious and parasitic diseasesshow/hide subcategories

B95-B97 Bacterial, viral and other infectious agents

B95 Streptococcus and staphylococcus as the cause of diseases classified to other chapters; B96 Other bacterial agents as the cause of diseases classified to other chapters; B97 Viral agents as the cause of diseases classified to other chaptersshow/hide subcategories

B99-B99 Other infectious diseases

B99 Other and unspecified infectious diseases