



Soil Borne Human Diseases

S. Jeffery and W.H. van der Putten



EUR 24893 EN - 2011

The mission of the JRC-IES is to provide scientific-technical support to the European Union's policies for the protection and sustainable development of the European and global environment.

European Commission
Joint Research Centre
Institute for Environment and Sustainability

Contact information

Address: Land Management & Natural Hazards Unit
Institute for Environment & Sustainability (IES) - T.P 280
Via E. Fermi 2749,
I-21027 Ispra (VA)
ITALY
E-mail: simon.jeffery@jrc.ec.europa.eu
Tel.: +39 0332 783682
Fax: +39 0332 786394

<http://ies.jrc.ec.europa.eu/>
<http://www.jrc.ec.europa.eu/>

Legal Notice

Neither the European Commission nor any person acting on behalf of the Commission is responsible for the use which might be made of this publication.

***Europe Direct is a service to help you find answers
to your questions about the European Union***

**Freephone number (*):
00 800 6 7 8 9 10 11**

(* Certain mobile telephone operators do not allow access to 00 800 numbers or these calls may be billed.

A great deal of additional information on the European Union is available on the Internet. It can be accessed through the Europa server <http://europa.eu/>

JRC 65787

EUR 24893 EN

ISBN 978-92-79-20796-9 (print)
ISBN 978-92-79-20797-6 (pdf)

ISSN 1018-5593 (print)
ISSN 1831-9424 (online)

doi:10.2788/36703

Luxembourg: Publications Office of the European Union

© European Union, 2011

Reproduction is authorised provided the source is acknowledged

Printed in Italy

Soil Borne Diseases of Humans

S. Jeffery¹ and W.H. van der Putten²

¹Institute of Environment and Sustainability, Joint Research Centre, European Commission, Ispra, Italy

²Department of Terrestrial Ecology, Netherlands Institute of Ecology (NIOO-KNAW), P.O. Box 50, 6700 AB Wageningen, The Netherlands
And Laboratory of Nematology, Wageningen University and Research Centre, P.O. Box 8123, 6700 ES Wageningen, The Netherlands

Table of Contents

Abstract	6
1. Introduction	6
1.1 Aims of this report.....	7
1.2 Estimates of number of infections and mortalities.....	7
1.3 Soil borne human diseases	8
1.3.1 Euedaphic versus soil transmitted pathogens and parasites	8
1.4 Other non-infectious soil associated diseases	10
1.4.1 Silicosis	10
1.4.2 Geophagia (pica associations).....	10
2. Discussion of Soil Borne Diseases.....	11
2.1 Viruses.....	11
2.1.1 Enteroviruses	11
2.1.2 Hantavirus	12
2.1.3 Other viruses.....	13
2.2 Bacteria.....	13
2.2.1 Actinomycosis	14
2.2.2 Anthrax	14
2.2.3 Botulism	15
2.2.4 Campylobacteriosis	16
2.2.5 Escherichia coli	17
2.2.6 Gas Gangrene	18
2.2.7 Leptospirosis	19
2.2.8 Listeriosis	20
2.2.9 Lyme Disease	21
2.2.10 Pseudomonas aeruginosa.....	21
2.2.11 Q fever	22
2.2.12 Salmonellosis.....	23
2.2.13 Shigellosis	24
2.2.14 Tetanus	25
2.2.15 Tularemia.....	26
2.2.16 Yersiniosis	27
2.2 Protozoa.....	28
2.2.1 Amoebiasis	28
2.2.2 Balantidiasis	29
2.2.3 Cryptosporidiosis.....	29
2.2.4 Cyclosporiasis	29
2.2.5 Giardiasis.....	30
2.2.6 Isoporiasis.....	31
2.2.7 Toxoplasmosis.....	31
2.3 Fungi.....	32
2.3.1 Aspergillosis.....	33
2.3.2 Blastomycosis.....	33
2.3.3 Coccidioidomycosis	34
2.3.4 Histoplasmosis.....	34
2.3.5 Sporotrichosis.....	35
2.3.6 Mucormycosis	35
2.3.7 Mycetoma.....	35
2.4 Helminths	36

2.4.1 Ascariasis.....	36
2.4.2 Echinococcosis	37
2.4.3 Hookworm.....	38
2.4.4 Strongyloidiasis	38
2.4.5 Trichuriasis	39
2.4.6 Trichinellosis	39
2.4.7 Other Helminth infections	39
2.5 Prions.....	40
3. Mapping of soil borne diseases	40
4. Discussion	42
4.1 Possible Factors affecting SBDs	42
4.1.1 Land use, change and management.....	42
4.1.2 Use of pesticides and herbicides	43
4.1.3 Use of antibiotics.....	43
4.1.4 Climate change.....	44
5. Recommendations	45
6. Conclusions	46
8. Acknowledgements	47
9. References	47

Abstract

Soils are home to a remarkable array of biodiversity with some estimates stating that 25% of the Earth's species find their home in the soil. Of these organisms, the vast majority are not of any threat to human health, but rather function to provide numerous ecosystem services which emerge through the multitude of complex interactions between organisms within the soil and the soil itself. These ecosystem services range from those which are vital for maintaining life on Earth, such as the formation of soil, the cycling of nutrients with the result of maintaining soil fertility, and the filtering of water, as well as provision of useful compounds such as antibiotics, the majority of which have been isolated from soil organisms.

However, soils also contain microorganisms which are capable of causing diseases in humans. They act either as opportunistic pathogens which take advantage of susceptible individuals, such as those who are immuno-compromised; or as obligate pathogens which must infect humans in order to complete their life-cycles. These organisms may be capable of surviving within the soil for extended periods of time before infecting humans who come into contact with contaminated soil.

This report provides an overview of the various soil borne diseases which can affect humans, including a discussion of the literature where available for each disease, and an analysis of the evidence for why each disease may be considered to be soil borne. Information from the World Health Organisation (WHO) and the European Centre for disease prevention and control (ECDC) on infection and mortality rates within the EU27 is also presented. However, limitations with the data sets prevent accurate qualitative analysis such as which diseases have the highest recovery rates in which member state, numbers of non-lethal disease cases, etc. A discussion of the factors which may affect the incidence of such diseases; including land management practices or land use change, climate change, and the use of antibiotics in livestock, is presented. Finally, areas of future research which are needed are highlighted to aid further investigation of this important and yet understudied area.

1. Introduction

Before progressing any further with this report it is vital to state that while the diseases discussed in the following pages are caused by organisms which inhabit the soil, these organisms form by far and away the minority of soil organisms in the vast majority of situations, both in terms of abundance and species richness. Soils are arguably the most complex systems on the planet, when comparing the scale of heterogeneity in both time and space with other ecosystems. They are home to a remarkable array of biodiversity with some estimates stating that 25% of the Earth's species find their home in the soil. Of these organisms, the vast majority are not of any threat to human health, but rather function to provide numerous ecosystem services which emerge through the multitude of complex interactions between organisms within the soil and the soil itself. These ecosystem services range from those which are vital for maintaining life on Earth, such as the formation of soil, the cycling of nutrient with the result of maintaining soil fertility, and filtering of water (MEA 2005), as well as provision of useful compounds such as antibiotics, the majority of which have been isolated from soil organisms (Turbé et al. 2010). The fact that many antibiotics have been isolated from soil organisms is due to the competitive nature of life within the soil. Soils, particularly at the micro-scale at which most soil organisms exist, consist of finite or even sparse resources in the form of water, food and space. It is because of this competitive existence, with organisms striving to utilise resources often while fighting off the advances of organisms with which they are competing, that some organisms engage in a form of chemical warfare by excreting compounds that can kill or interfere with the growth of other nearby microorganisms which come into contact with these compounds. Humans have isolated and utilise many of these compounds as antibiotics which aid in the fight against human disease. However, these adaptations in some soil organisms which may help survival in the soil can sometime be turned against humans if

these organisms infect susceptible individuals, and so can become the causative agents of soil borne diseases in humans.

This risk of exposure to infectious organisms from the soil has been known for centuries. For example, Hippocrates, in 4th century BC, stated that it is necessary to beware of "...the soil too, whether bare and dry or wooded and watered, hollow and hot or high and cold". It is clear that infection from soil is by no means a new phenomenon although our understanding of mechanisms has obviously increased greatly since the time of Hippocrates. Owing to vaccines and antibiotics we have been slowly winning the battle against disease over the last few decades, but with the rise of antibiotic resistance in some pathogens it is clear that prevention is better than the cure. So, which diseases are soil borne? Is there anything that we can do with regard to land management practices or other precautions that we can take which will help control the incidence of soil borne diseases? And are there any human activities that enhance the risk of soil borne diseases?

1.1 Aims of this report

This report aims to be a first step towards bringing information together from various sources into one easily accessible source of information on soil borne diseases, covering all groups of pathogenic organisms of humans that are associated with the soil.

The first step is to identify which diseases are soil borne. Each of these diseases will then be discussed in turn, providing a brief overview of the disease, its cause, progression in humans and general prognosis. Research will be pulled together to discuss how each organism is associated with the soil, where available, which will then be critically analysed to identify areas of further research. Data available publically from the World Health Organisation (WHO) and European Centre for Disease Prevention and Control (ECDC) on infection rates and mortalities will be presented where available and discussed.

Finally, a literature review of anthropogenic activities which may affect soil borne pathogenic organisms and the incidence of diseases associated with such organisms will be performed, and recommendations made for the next steps towards achieving a full understanding of soil borne pathogenic organisms, their role in the environment, their impact on human health within Europe, and any steps that may be possible to limit or reduce the number of soil borne diseases in humans. For example, the misuse of soil by humans which can lead to soil degradation processes might also enhance soil borne disease incidence, for example through increasing wind erosion and so the amount of airborne spores from the soil. This report will attempt to identify whether or not such factors may play a role in the incidence of human disease from the soil

1.2 Estimates of number of infections and mortalities

All data quoted in this report for infection rates were obtained from The European Centre for Disease Prevention and Control (ECDC) who compile data obtained through The European Surveillance System (TESSy), whereby member states are required to track and report data concerning rates of infection of communicable diseases as described in Decision No2119/98/EC. Of those communicable diseases which are tracked, 18 have been identified as soil borne diseases within the confines of this report, and the data which is available (from 2006-2009) are discussed in this report.

Data on mortality rates are logged with the WHO that collates data from death certificates for approximately 100 member states of the WHO. Data were extracted from this database for mortalities attributed to the soil borne diseases identified in this report for each of the 27 EU member states where available on the ICD10 codes. The ICD10th revision was adopted by the World Health Assembly in 1990. It came into effect as from 1993 with the number of countries submitting their underlying causes of death data using ICD10th revision to WHO increasing from 4 countries in 1995 to 75 in 2003 and to over 100 in 2007. This means that data is not always available for the same timeframe in each country concerning each disease.

1.3 Soil borne human diseases

So, what are soil borne human diseases in the context of this report? Many infectious diseases may be considered to be soil borne to some extent. However, many of those would likely require quite contrived situations which, while theoretically possible, are seemingly unlikely. An example of such a disease is measles. This is a highly infectious viral disease. While it can theoretically be transmitted through soil, the virus is known to survive only very poorly in the air and on surfaces (approximately a 30 – 70% reduction in the number of viable virus particles in the first minute of leaving the body depending on the relative humidity of the air). Therefore, the risk of transmission through air, and particularly from other surfaces such as the soil, is relatively low to minimal, particularly if the infected individual is no longer present.

To include all of the diseases which may be transmitted through the soil in cases where more or less immediate exposure after contamination is required for infection to occur would mean the inclusion of the majority of transmissible diseases. This carries the risk of obscuring those diseases which truly are soil borne, i.e. those diseases which make their home in soil but which are capable of causing disease in humans, or those diseases that can survive for a sufficient time in the soil that the original infected human can be long departed before the organism infects another human host. With such diseases it can be hypothesised that environmental factors such as land management practices, may have an influence on the incidence of these diseases and as such it may be possible to find land management practices which positively impact on human health.

Therefore, soil borne human diseases, within the context of this report are defined as being:

“A human diseases resulting from any pathogen or parasite, transmission of which can occur from the soil, even in the absence of other infectious individuals”

1.3.1 Euedaphic versus soil transmitted pathogens and parasites

Table 1 (below) lists all of the human diseases so far identified which fall under the definition of being soil borne diseases. The pathogens responsible for causing such diseases can be divided into two groups: Euedaphic pathogenic organisms (EPOs), being potential pathogens which are true soil organisms, i.e. their usual habitat is the soil. This list includes most of the bacterial pathogens and all of the fungal pathogens. The other group consists of soil transmitted pathogens (STPs). These are organisms which, while they may be able to survive in soil for extended periods of time, are not true soil organisms, but rather are obligate pathogens who must infect a host in order to complete their life cycles. It should be noted that as with many classifications and groupings made, strict categorisation has its limitations, as rather a continuum is likely to exist with some overlap of organisms between the two groupings (for example, strongyloidiasis and shigellosis). This is an initial attempt at defining these organisms which may have to be revised as future works is conducted (Table 1).

Table 1: Soil borne infectious diseases (bold) and their causative agents (*italics*) split into two groups, “Euedaphic pathogenic organisms (EPOs)” and “Soil Transmitted Pathogens (STPs)”, depending on the closeness of their relationship with soil.

Euedaphic pathogenic organisms	Soil Transmitted Pathogens
Actinomycetoma: (e.g. <i>Actinomyces israelii</i>)	Poliovirus
Anthrax: <i>Bacillus anthracis</i>	Hantavirus
Botulism: <i>Clostridium botulinum</i>	Q Fever: <i>Coxiella burnetii</i>
Campylobacteriosis: e.g. <i>Campylobacter jejuni</i>	Lyme disease: <i>Borrelia</i> sp.
Leptospirosis: e.g. <i>Leptospira interrogans</i>	Ascariasis: <i>Ascaris lumbricoides</i>
Listeriosis: <i>Listeria monocytogenes</i>	Hookworm: e.g. <i>Ancylostoma duodenale</i>
Tetanus: <i>Clostridium tetani</i>	Enterobiasis (Pinworm)
Tularemia: <i>Francisella tularensis</i>	Strongyloidiasis: e.g. <i>Strongyloides stercoralis</i>
Gas Gangrene: <i>Clostridium perferingens</i>	Trichuriasis (Whipworm): <i>Trichuris trichiura</i>
Yersiniosis: <i>Yersinia enterocolitica</i>	Echinococcosis: e.g. <i>Echinococcus multicularis</i>
Aspergillosis: <i>Aspergillus</i> sp.	Trichinellosis: <i>Trichinella spiralis</i>
Blastomycosis: e.g. <i>Blastomyces dermatitidis</i>	Amoebiasis: <i>Entamoeba histolytica</i>
Coccidioidomycosis: e.g. <i>Coccidiodes immitis</i>	Balantidiasis <i>Balantidium coli</i>
Histoplasmosis: <i>Histoplasma capsulatum</i>	Cryptosporidiosis: e.g. <i>Cryptosporidium parvum</i>
Sporotrichosis: <i>Sporothrix schenckii</i>	Cyclosporiasis: <i>Cyclospora cayetanensis</i>
Mucormycosis: e.g. <i>Rhizopus</i> sp.	Giardiasis: <i>Giardia lambila</i>
Mycetoma: e.g. <i>Nocardia</i> sp.	Isosporiasis: <i>Isospora belli</i>
Strongyloidiasis: e.g. <i>Strongyloides stercoralis</i>	Toxoplasmosis: <i>Toxoplasma gondii</i>
	Shigellosis: e.g. <i>Shigella dysenteriae</i>
	<i>Pseudomonas aeruginosa</i>
	<i>Eschericia coli</i>
	Salmonellosis: e.g. <i>Salmonella enterica</i>

Once this division is made it can be used to highlight areas of further research and analysis. For example, when analysing infection rates it is highly probable that infection with EPOs have occurred from the soil, but with STPs it is much less certain; salmonellosis can be contracted as a result of poor hygiene by an infected individual who is preparing food.

Another utility in this distinction is that it is probable that EPOs provide ecosystem services within the soil, such as breaking down organic matter, or affecting soil erodibility as in the case of some fungi for example. While it is possible that their role is minimal if the pathogens are only present at low abundances (as would be expected to be the case for STPs but may not necessarily be the case for EPOs which cause diseases opportunistically), in areas which have low species richness the effects of some species on their local environment can be disproportionate to their abundance.

For example, many of the potential diseases causing fungi are hyphal (or dimorphic) and these fungal types have been shown to play an important role in stabilising the soil surface. In deserts environments with low species richness, the removal of the potential disease causing fungus from the soil may have

an impact on the soil surface stability and hence increased effects of wind erosion and decreased water holding capacity etc.

Conversely, it is likely that STPs will provide fewer ecosystem services when in the soil as it is not necessarily their “usual” habitat. However, while STPs may not provide ecosystem services while in the soil environment, they may well provide ecosystem functions. Soil foodwebs are generally very complicated, with several trophic levels and a high level of interconnectedness. While in the soil environment, STPs may well provide a food source for other organisms and so their presence may have other effects, for example by maybe temporarily allowing an increase in the level of diversity through being a novel food source. However, owing to the relatively low levels of abundance of these species in the soil any effects are likely to be highly localised.

While we acknowledge that these differences are currently hypothetical this distinction may also help guide policy in the future as well as highlighting the need for research in this area. For example, if it can be shown the land or agricultural practices may reduce the abundance of STPs within the soil, or the length of time that they remain viable infectious organisms, then implementation of such practices may have positive impacts on human health. However, due to the fact the EPOs may provide ecosystem services, reducing their abundance further in the soil may have negative effects and enhance soil degradation processes.

1.4 Other non-infectious soil associated diseases

Other diseases which can be obtained from the soil or are associated with the soil include silicosis and geophagia which is associated with pica (defined as being a persistent ingestion of eating non-food materials) neither of which is caused by an infectious agent.

1.4.1 Silicosis

Silicosis is generally considered an occupational lung disease as it is most commonly contracted as a result of work or occupational activity. It is caused by the inhalation of silica dust and is most common in miners and quarry workers but can also affect agricultural workers.

Symptoms of silicosis include shortness of breath, a cough, inflammation of the lungs, pain while breathing and possibly a fever. It is an irreversible condition which has no cure and which continues to worsen even after exposure has ended owing to the body’s inability to remove the silica particles from the lungs.

Silicosis is not contagious as there is no biological component and as such is not discussed in detail in this report. However, land management practices that reduce wind erosion, which has a possibility of lifting silica particles from the soil into the air where they may be inhaled, are likely to have twin benefits of reducing soil erosion and concurrently reducing the incidence of silicosis.

1.4.2 Geophagia (pica associations)

There is a history of geophagia (eating soil) in various areas throughout the world, from a range of cultures, and has been documented in many different species including mammals, birds and reptiles (Diamond 1999).

Apart from its obvious role as a potential source of inoculation, geophagia has been recorded as a form of pica. However, pica is not a communicable disease and is not transmitted through the soil.

Therefore it does not fall within the scope of this report and so is discussed no further here.

2. Discussion of Soil Borne Diseases

The following section provides a discussion of the various soil borne diseases, split up into polygenetic taxonomic groups for ease of reference. Infectious organisms capable of causing disease come from five major phylogenetic groups being viruses, bacteria, protozoa, fungi and helminths (nematodes). The final group, helminths, is the only group for which a specific focal point currently exists within the WHO for soil transmitted pathogens or parasites, that of “soil transmitted helminths”; currently no focal point exists for soil borne infections caused by any of the other four main groups of infectious organisms.

Graphical representations of infection rates by country are included using data available from TESSy for those diseases for which data is available within the EU27. Where data were not available for a member state for a given disease, this has been mentioned in the figure caption. Several of the diseases identified in this report are not tracked by TESSy, including all of the fungal diseases discussed, and as such no graph is provided showing infection rates for these diseases.

2.1 Viruses

Viruses are highly host specific and are incapable of multiplying outside of host cells. Therefore, human viruses almost certainly have no functional role within the soil system. This means that they can all be considered to be soil transmitted pathogens, as defined by this report (Section 1.4.1), as opposed to euedaphic pathogens.

Some viruses are capable of surviving within the soil system for extended periods of time and to be able to adsorb to soil particles and so resist elution (e.g. poliovirus). Others adsorb less strongly to the soil and so can become mobilised after rainfall and either washed deeper into the soil profile or to different areas depending on the soil hydrology (e.g. other enteroviruses) (Landry et al. 1979). Factors which most affect the survival of human pathogenic viruses in the soil are pH level, moisture content, temperature, exposure to sunlight and the presence of soil organic matter (WHO 1979).

2.1.1 Enteroviruses

Human enteric viruses (also known as enteroviruses) are a genus of picornaviruses that include polioviruses, coxsackieviruses and echoviruses. They generally infect the gastrointestinal tract of humans but may also spread and infect tissues in other areas of the body, particularly the nervous system. They have been found to be able to survive for extended periods in some soil environments with survival times of up to 170 days reported for virus particles in loamy and sandy loamy soils (WHO 1979). Enteric viruses have been recovered at considerable distances from their source owing to their generally relatively poor binding to soil particles and soil organic matter meaning that they are easily eluted and carried in rainwater. The exception to this is poliovirus which seems to adsorb relatively strongly to soil particles (Landry et al. 1979).

Polioviruses are a specific group of enteroviruses. They have been shown to be able to survive within the soil environment for between 80 days (Duboise et al 1976) and 96 days (WHO 1979). While these viruses seem to adsorb readily to both biological and non-biological surfaces within the soil system, it has been demonstrated that this adsorption does not inactivate the virus particles (Carlson et al. 1968) and so they remain infectious.

No infections were reported by the ECDC for the years 2006-2008 although 1 infection was reported in Poland in 2009. The WHO has 6 deaths registered as being attributed to acute poliomyelitis under the ICD10 codes in the EU 27 between 1994 and 2008, although of these only Bulgaria has reported any deaths attributed to acute poliomyelitis since 1996 (1 in 2006 and 2 in 2008).

2.1.2 Hantavirus

Hantaviruses are generally considered to be zoonotic, being carried by wild rodents. Infection in humans generally occurs either from rodent bites or through coming into contact with infected rodent excreta which may be in the soil.

Hantavirus infection in humans can cause either “hantavirus pulmonary syndrome” (HPS) or “hemorrhagic fever with renal syndrome” (HFRS) (Jonsson et al. 2010). The first outbreak of HFRS occurred during the Korean War and so HFRS was initially called Korean hemorrhagic fever. HFRS is the less fatal form of hantavirus infection with mortality rates in the region of 12% as compared to a 60% mortality rate with HPS (Jonsson et al. 2010).

Symptoms are initially flu-like and occur two to three weeks after exposure to the virus. As the disease progresses symptoms usually develop into coughing and difficulty breathing which may be accompanied by feeling dizzy, headaches and stomach pain and diarrhea and/ or vomiting (Dugdale et al. 2011).

While HPS and HFRS can be fatal in humans, and may even become life threatening within only a few days of early symptoms, the earlier the infection is treated the better as the chances for recovery. The virus spreads among the rodent population either through direct contact between infected and non-infected individuals, or through non infected individuals coming into contact with infected rodent excreta in the soil. Humans generally become infected through inhalation of aerosolised viral particles (Wesley et al. 2010). This may occur through land management practices such as tillage which disturbs the soil and may eject virus particles from the soil into the air, although further research is required to investigate how much effect this mechanism and others have on the incidence of the disease.

In 2006, 15 cases of hantavirus infection were reported in Estonia, the only country for which data were reported in that year. Seven cases were reported in Estonia in 2007, with Germany and The Netherlands reporting 0 cases that year and no other countries within the EU27 providing data. Finland reported the highest rate of infection for the two years that data is available (2008 & 2009). In 2008 a total of 4479 cases of hantavirus infection were reported within the EU27, with all countries with the exception of Cyprus, the Czech Republic, Denmark, France and Portugal providing data to the ECDC. In 2009 a total of 2438 cases of hantavirus infection were reported, with the Czech republic providing data in 2009, but no data being provided by Cyprus, Denmark, France or Portugal.

The WHO reported 27 deaths within the EU27 between 1996 and 2008 as attributed to “haemorrhagic fever with renal syndrome” which included epidemic Korean, Russian, Hantan virus disease and Hantavirus disease with renal manifestations under the ICD10 codes. Information concerning mean annual infection rates per country can be seen in Fig 1.

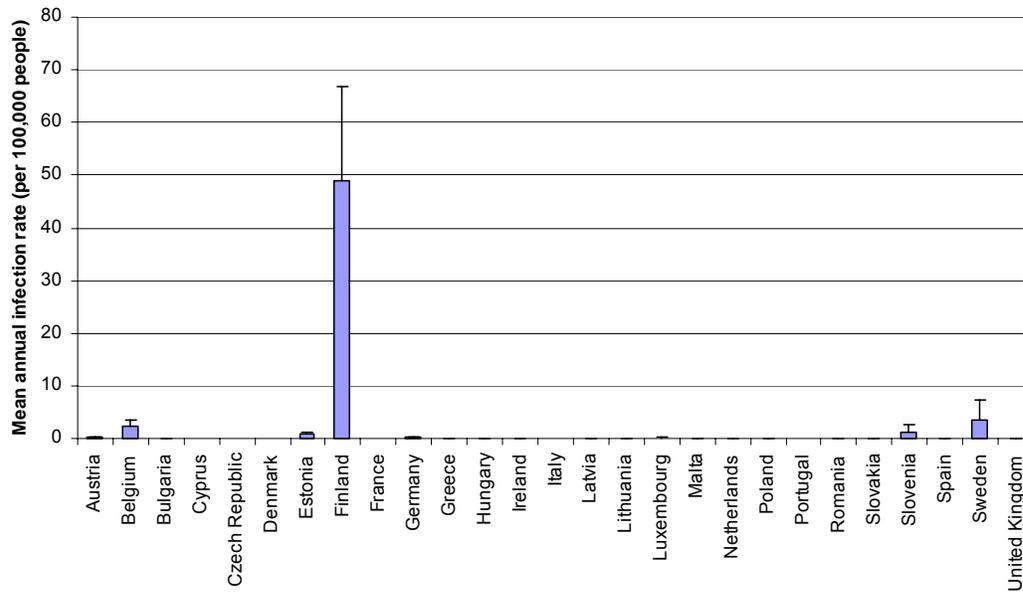


Fig 1: Mean annual infection rates of Hantavirus infection in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error of between year data. Data not available for Cyprus, Denmark, France, Italy or Portugal.

2.1.3 Other viruses

Many other human viruses have either been shown or are thought to only survive relatively poorly in the soil system although studies on many viruses are still currently lacking. However, the rubella virus, mumps virus, rhinovirus and parainfluenza virus among others, have all been found to survive in the external environment, including in the soil, for only a matter of hours and occasionally for a day or two (Walther and Ewald 2004). For this reason these diseases are not considered within the scope of this report and so are not discussed further.

2.2 Bacteria

The bacteria are a remarkably diverse group of organisms. They form one of the three domains of life, along with Archea and Eukaryota, used in modern taxonomy. Bacteria exist at relatively high levels of abundance in all ecosystems on the planet that have so far been studied and they play a vital role in global functioning through the ecosystem services and functions that they provide. However, there are several groups of bacteria which are pathogenic in humans. These include obligate pathogens, and opportunistic pathogens which can be divided into different groups. For example, enteric bacteria are rod shaped, gram negative bacteria which occur most commonly in the intestines of humans and other animals where they may cause disease, mainly diarrhea, in some instances. They include bacteria from the genera *Campylobacter*, *E. coli* and *Shigella* sp. which are discussed in more detail below. Worldwide, enteric bacteria are estimated to causes between 4 and 6 million deaths each year and are the second most common cause of infant mortality globally. Other groups of bacteria, such as the actinomycetes, are found more commonly in the external environment but there are some species which may be capable of causing disease in humans. A full discussion of the main soil borne pathogenic groups follows:

2.2.1 Actinomycosis

Actinomycosis is caused by infectious actinomycete species, often *Actinomyces israelii*, a soil dwelling species that is found in decaying organic matter (Roque 2010). Actinomycetes are generally soil inhabiting saprophytes although some species are capable of causing diseases in plants, animals or humans. They are generally anaerobic or facultative anaerobic organisms, with some genera such as *Frankia* forming symbiotic relationships with the roots of non-leguminous plants including trees. Here they play an important role in maintaining soil fertility due to their ability to fix nitrogen.

As well as inhabiting the soil, many species of actinomycete can also be found colonising the gut, mouth and vagina of humans although the majority of these do not cause diseases.

Actinomycosis usually presents as a chronic infection that commonly affects the face and neck and can include the formation of abscesses in the lungs, intestine or mouth caused by the infection of tissues, normally in conjunction with other bacterial species. The disease is only very rarely fatal with only 4 mortalities recorded in the WHO mortalities database under the ICD 10 codes (2 in Denmark in 1996 and 2 in Hungary in 2002; WHO 2011). Data on actinomycosis infection rates is not collected by the ESDC and so no information is currently readily available on actinomycosis infection rates within the EU27.

2.2.2 Anthrax

Anthrax is a zoonotic disease caused by bacteria of the species *Bacillus anthracis*, which are gram positive, rod shaped, aerobic, spore forming bacteria. The disease is a problem in many countries world-wide where it can affect livestock and wildlife. Infections in humans are more usually secondary, i.e. passed on to humans by infected animals (Blackburn et al. 2007). Animals generally get anthrax from grazing on soils which contain spores of the bacterium *B. anthracis*. Humans then may become infected through touching infected animals or animal products where the bacterial spores may enter into wounds if present, or be inhaled as is the case for “wool sorter’s diseases”; an often fatal infection resulting from the handling of infected wool (Van Ness 1971). Anecdotal evidence exists that soil management practices such as tillage may increase the risk of human infection of anthrax due to inhalation of spores, although there is a paucity of empirical evidence in this regard and further research is required to assess the effects of land management practices on the incidence of this disease in humans.

When in spore form, these bacteria are highly resistant to desiccation and other environmental stresses and can remain in this inactive form in soil for many years, only becoming active once environmental conditions become favourable again.

The ECDC has between 3 and 14 infections recorded annually attributed to anthrax within the EU27 between 2006 and 2009 with the highest mean infection rate being in Bulgaria. The WHO mortality database has 5 deaths registered as attributed to anthrax septicaemia under the ICD10 codes within the EU27, between 2001 and 2004, 4 in Romania, and 1 in the United Kingdom. No deaths were attributed to anthrax under the ICD 10 codes in Bulgaria despite having the highest infection rates. Whether this is the result of the disease being cured more readily there, or due to deaths being attributed to other factors cannot be known from the data which is currently available. Information concerning mean annual infection rates per country can be seen in Fig 2.

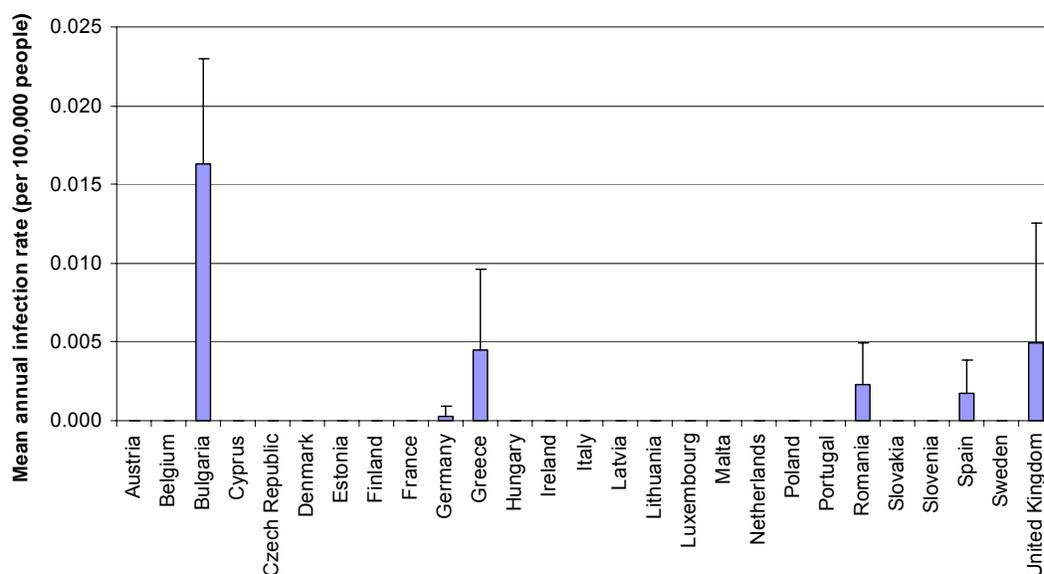


Fig 2: Mean annual infection rates of anthrax in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error.

2.2.3 Botulism

The bacterium *Clostridium botulinum* is the causative agent of botulism. *C. botulinum* is not a well defined species of bacterium but rather refers to distinct groups of bacteria that produce a total of seven distinct toxins (called A to G), all of which have similar pharmacological actions (Smith 1979). However, of these groups, only types A, B, E and more rarely F are capable of causing botulism in humans. Types C, D, E and G cause illness in other mammals, birds and fish (WHO 2002).

C. botulinum are spore forming, anaerobic bacteria whose principal habitat is the soil, although their distribution can be highly regional. While the bacteria are soil borne, infection generally occurs through eating contaminated food, although it can also be transmitted directly into wound infections directly from the soil.

Symptoms usually present within 12 – 36 hours and are caused by toxins produced by the bacteria rather than by the organisms themselves. However, thorough cooking of contaminated foods (heating to >85°C for five minutes or boiling for a few minutes) is sufficient to destroy the toxins as well as the bacteria (WHO 2002). Early symptoms usually include fatigue, weakness and vertigo, followed by a dry mouth and difficulty swallowing and speaking, and blurred vision. The disease can progress to weakness in the neck and arms after which respiratory muscles and muscles of the lower body become affected and this paralysis may make breathing difficult (WHO 2002).

Antitoxin administration as soon as possible after diagnosis is necessary to ensure the best chance of recovery, although severe cases of the disease may also require supportive treatment such as mechanical ventilation for periods of weeks or even months. Administration of antibiotics is also necessary in the case of wound infection.

The ECDC reported between approximately 100 and 130 infections per year attributed to botulism within the EU27 with 41 deaths having occurred between 1999 and 2008 as registered under the ICD10 codes in the WHO mortality database. Information concerning mean annual infection rates per country can be seen in Fig 3.

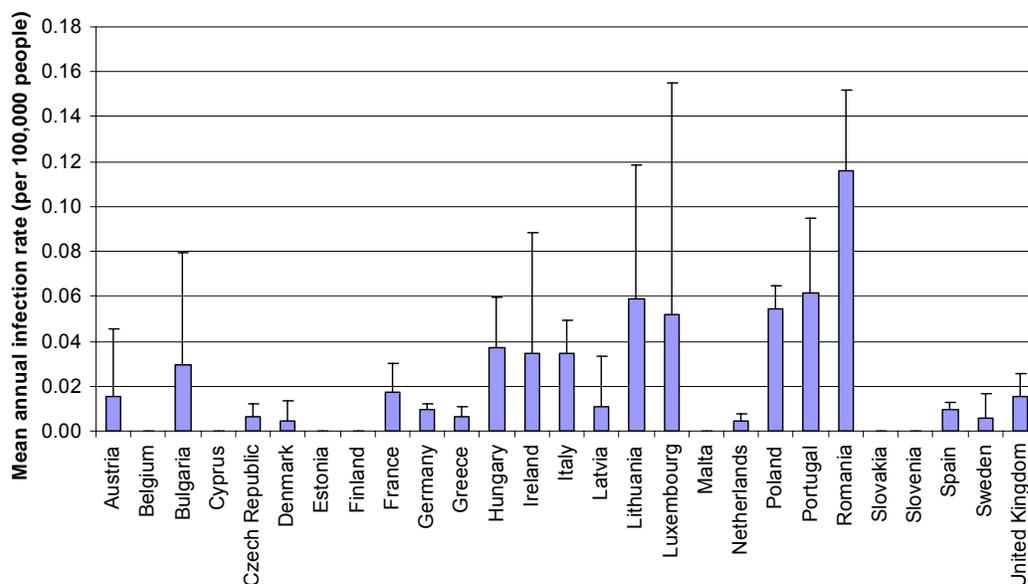


Fig 3: Mean annual infection rates of botulism in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error.

2.2.4 Campylobacteriosis

Bacteria from the genus *Campylobacter*, most commonly the species *Campylobacter jejuni*, are the causative agents of campylobacteriosis. It is an enteric bacterium and is one of the most common forms of bacterial infections in humans. Symptoms of campylobacteriosis usually present within 2-5 days with only a relatively small number of organisms (1,000-10,000 bacteria) need to cause infection (Javid and Ahmen 2009). Symptoms include: fever, which can reach 40°C; headaches; and diarrhea, which is classified as inflammatory diarrhea which may be bloody (also known as dysentery). *C. fetus* may also cause bacteraemia (i.e. infection of the blood), usually as an opportunistic disease in immuno-compromised hosts, where it may be associated with systemic illness, meningitis, vascular infections, abscesses and/or nonspecific abdominal pain (Javid and Ahmen 2009).

The main route of transmission of *Campylobacter* sp. are the ingestion of contaminated food or water (particularly undercooked poultry or unpasteurised milk), the faecal-oral route, or person to person contact. However, *C. jejuni* has been demonstrated to survive in the soil for at least 25 days, with indications that it can survive considerably longer (Ross and Donnison 2006) meaning that the soil is also a possible route of transmission, particularly owing to the relatively small infectious dose need to cause disease.

Between 175,000 and 200,000 infections of campylobacteriosis were reported within the EU27 between the years of 2006 and 2009, being the highest rate of infection of all of the soil borne diseases identified by this report which are tracked by the ECDC. However, despite this relatively high infection rate, only 46 mortalities were attributed to the diseases as logged on the WHO mortalities database under the ICD10 codes for the EU27 between 1998 and 2008. Information concerning mean annual infection rates per country can be seen in Fig 4.

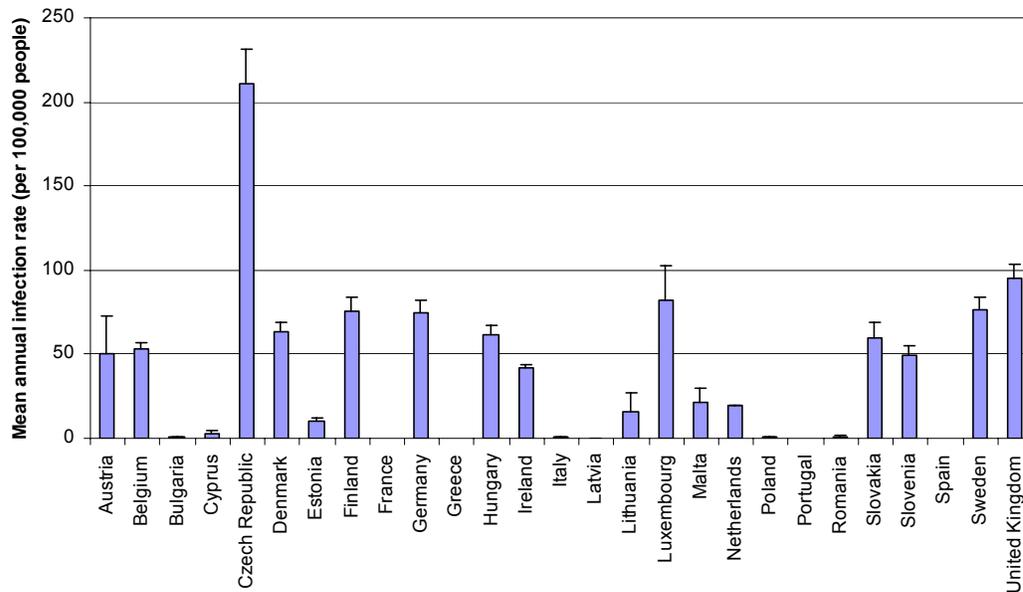


Fig 4: Mean annual infection rates of campylobacteriosis in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for France, Greece, Portugal or Spain.

2.2.5 *Escherichia coli*

Many strains of the bacterium *Escherichia coli* are harmless and in fact are prevalent within the human gut and do not cause diseases. However, some strains produce toxins which can cause disease in humans which in some cases can be severe depending on the host's health before infection and the strain of *E. coli*, and the associated toxin, which is causing infection.

The most common form of pathogenic *E. coli* is Enterotoxigenic *E. coli* (ETEC), which is the most common cause of bacterial diarrhea in children in the developing world as well as among travellers to developing countries (WHO 2009). ETEC accounts for several hundred million cases of diarrhea and tens of thousands of deaths globally each year (WHO 2009).

Other serotypes of pathogenic *E. coli* include Enteropathogenic *E. coli* (EPEC) which causes diarrhea but also contains virulence factors which are similar to *Shigella* (discussed in more detail below; Section 2.2.12) and Verotoxigenic *E. coli* (VTEC) which causes bloody diarrhea ("Vero" indicates that this serotype produces the "Shig" toxin; See Section 2.2.12 for more information).

The final serotype discussed in this report, known as Enterohaemorrhagic *E. coli* (EHEC), came to prominence most recently due to the strain 0104:H4 which caused the outbreak in Europe in 2011. This is a particularly virulent strain of the bacterium which was identified in Germany and caused the third largest outbreak of *E. coli* with about 2200 infected patients, as well as one of the most lethal with at least 22 dead (as of 6/6/2011; Nettleman 2011). The infectious dose of only 10 to 100 bacteria needed to cause disease, as opposed to over a million bacteria needed for most other pathogenic strains of *E. coli* demonstrates the relatively high virulence of this serotype (Greig et al. 2010). This new strain produces shiga toxin (and so is also VTEC) and is very similar to the 0157:H7 strain about which more is known. However, this strain also has the ability to attach to cells within the gastrointestinal tract in much the same way as Enteroaggregative (EAEC) strains (Nettleman 2011). It is this combination of factors which are likely responsible for the relatively high levels of virulence and mortality. While firm data are not currently included in this report, the strain clearly warrants monitoring and any updated versions of this report should include data where available.

Although the main routes of transmission of *E. coli* are poor hygiene or sanitation leading to contamination of food or water, *E. coli* can also survive in the soil for sufficient periods of time to lead to infection even when the infectious individual that was the source of the contamination has long left

the area. The main factor affecting the length of time that *E. coli* can survive in soil appears to be soil moisture content, with cells surviving for 14 days in dry soils and longer in wet soils (Chandler and Carven 1980). However, Avery et al. (2004) demonstrated that the pathogenic strain O157 could survive on surface vegetation for up to 6 weeks, or in the underlying soil for 8 weeks. The survival of *E. coli* in compost heaps was also affected by moisture content but conversely the bacteria were capable of surviving higher temperatures at lower moisture contents, although survivability was generally measured in minutes as opposed to days at temperatures of 54-67°C (Gong et al. 2005). Only data on infection rates of VTEC are logged through TESSy and so are available through the ECDC. Approximately 3000 cases of VTEC were reported within the EU27 each year between 2006 and 2009. The WHO logs mortalities due to enterohemorrhagic *E. coli* (7 deaths between 1997 and 2006), enteroinvasive *E. coli* (10 deaths between 1999 and 2008), enteropathogenic *E. coli* (10 deaths between 1994 and 2006) and enterotoxigenic *E. coli* (1 death in 2002). Information concerning mean annual infection rates per country can be seen in Fig 5.

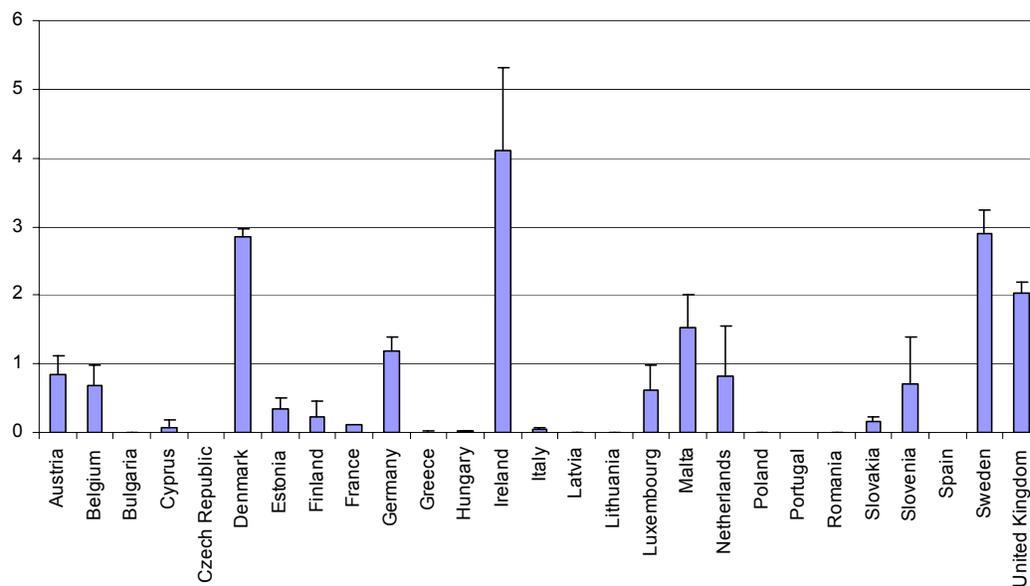


Fig 5: Mean annual infection rates of *E. coli* (VTEC type only) in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for Czech Republic, Portugal or Spain. Note that data regarding the 0104:H4 strain which caused the outbreak in Europe in 2011 discussed by Nettleman (2011) are not included in this figure.

2.2.6 Gas Gangrene

The causative agent of gas gangrene is, in 80-90% of cases, the bacterium *Clostridium perfringens* (previously known as *Clostridium welchii*). The other 10-20% of cases are caused by the bacteria *C. novio*, *C. septicum*, *C. histolyticum*, *C. bifermentans* and *C. fallax* (Revis 2008). If untreated, gas gangrene is always fatal, although with treatment this mortality rate drops to 20-30% (60% if the chest or abdomen are infected; Revis 2008).

C. perfringens, like other *Clostridia* species such as *Clostridium botulinum*, are bacilli which are gram positive, anaerobic and spore forming. They are highly prevalent in soils (DeSpain Smith and Gardner 1949), as well as the intestinal tract of humans and animals (Bryan 1969). It has been reported that *C. perfringens* is more widely spread than any other pathogenic bacterium (Matches et al. 1974). However, no data are currently available in the literature as to the effects of land management practices on the abundance of this bacterium, nor which in which soil types or under which environmental conditions it is most prevalent.

More than 50% of cases occur as the result of trauma which introduces the bacteria into the body through wounds (Revis 2008). However, infection only occurs if the organisms are inoculated into a tissue where the oxygen tension is below 30%, such as in some deep cuts. Incubation is usually between 12-24 hours, although this can be as short as 1 hour in some instances, or may take several weeks (Revis 2008). Initial symptoms are usually the increasing of pain after surgery or a trauma, which is out of proportion to what would be expected from the surgery or wound, and which may have a sudden onset. Discolouration of the skin around the wound usually occurs, which can be purplish black and may also have the occurrence of blister like structures called bullae. The ECDC does not keep records of gas gangrene infection rates. The WHO reported 566 deaths due to gas gangrene under the ICD10 codes within the EU27 between the years 1994 and 2008.

2.2.7 Leptospirosis

Leptospirosis, also known as Weil's diseases in its more serious form, is caused by a species of aerobic spirochete bacteria from the genus *Leptospira*. Bacteria of this genus are found in aquatic ecosystems as well as the soil, although they are generally found in higher abundances in the soil adjacent to water than the water itself (Henry and Johnson 1978).

The genus *Leptospira* is most often divided into two complexes, the parasitic complex and the biflexia (non-parasitic) complex. The parasitic complex includes 13 named and 4 unnamed species (Agent 2005), of which *L. interrogans* is the most well known, and has over 200 known pathogenic serologic variants (Agent 2005).

Transmission of leptospirosis generally occurs through contact with fresh water, soil, or possibly vegetation which has been contaminated by the urine of infected animals. The bacteria are capable of surviving for at least 42 days in acidic soil (pH ~5.5; Hellstrom and Marshall 1978) and up to 74 days in more neutral soils (Zaitsev et al. 1989). This demonstrates that while pathogenic species require an infected individual to transfer them to the soil environment through their urine, they are then capable of surviving a considerable time after the infected individual has left the area until a susceptible host comes along and so fall under the classification of STPs. The fact that the bacteria can survive for extended periods in water means that they can be transported relatively large distances in times of floods, or through being transported via overland flow and in waterways. Increased risk of contracting the disease is associated with farmers, vets, loggers, sewer workers and those that partake in water sports including fresh water swimming, kayaking and canoeing.

Pathogenic *Leptospira* sp. can enter the body through ingestion of contaminated food or water, through broken skin or through mucous membranes. The bacteria then enter the blood and can migrate around the body, particularly to the kidneys, and within 7 to 10 days the bacteria can be passed in urine.

Symptoms of the diseases can be very diverse and generally have two phases. The first phase usually involves flu-like symptoms including strong headache. The disease then passes through a brief moment in which the patient may be asymptomatic before the second phase begins which can include meningitis, renal failure and liver damage.

The WHO registered 357 deaths within the EU27 between 1997 and 2008 under the ICD10 codes as attributed to leptospirosis (including Weil's diseases and unspecified forms of leptospirosis). The ECDC registered between 541 and 844 cases of infection of leptospirosis between 2006 and 2009 in the EU27. Information concerning mean annual infection rates per country can be seen in Fig 6.

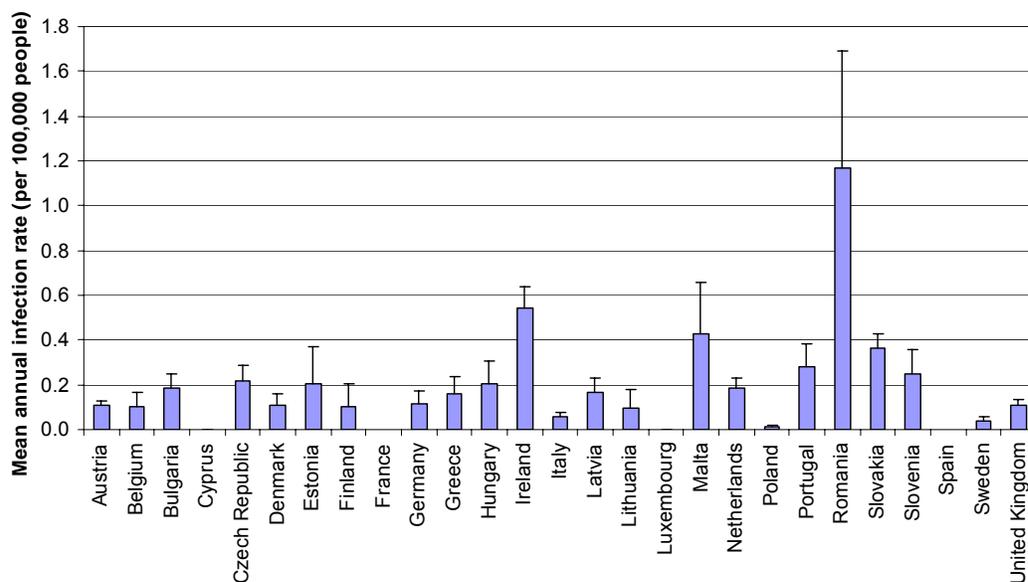


Fig 6: Mean annual infection rates of leptospirosis in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for France or Spain.

2.2.8 Listeriosis

The causative agent of listeriosis is the gram-positive bacterium *Listeria monocytogenes*. Infection usually occurs in humans after eating food contaminated with the bacterium. It mainly affects infants, the elderly and immuno-compromised patients (Hof 1996). While it is generally considered to be a food borne disease, the bacterium has been demonstrated to survive for extended periods in soil (Welshimer 1960) as well as being associated with sheep and cattle (Weinstein 2011). The main factor affecting the survival of *Listeria monocytogenes* in soil was found to be soil moisture content with survival averaging up to approximately 67 days in soils where the moisture content was not controlled as compared to up to 295 days in soils which were protected from evaporation (Welshimer 1960).

Symptoms of listeriosis usually include fever, vomiting and myalgia which last for 7-10 days.

However, in some instances the infection may spread to the nervous system where it can cause meningitis, or the blood, both of which are much more serious conditions. Overall the mortality rate of listeriosis is 20-30% with intravenous antibiotics being necessary as treatment.

The ECDC had approximately 1,500 cases of infection registered annually between 2006 and 2009 in the EU27. The WHO reports 579 deaths in the EU27 reported under the ICD10 codes between the years 1996 and 2008. Information concerning mean annual infection rates per country can be seen in Fig 7.

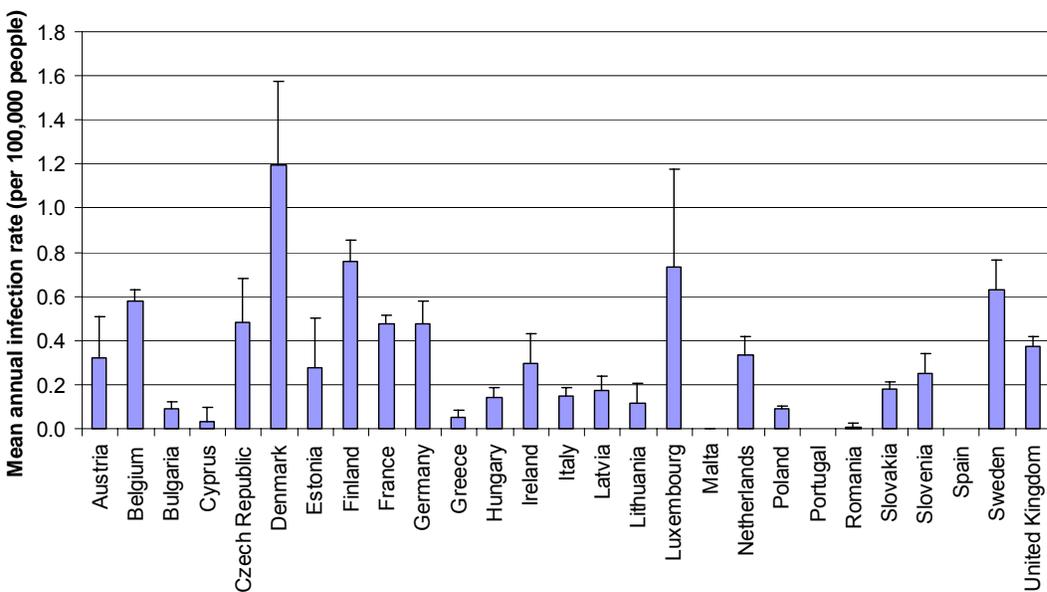


Fig 7: Mean annual infection rates of listeriosis in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for Portugal or Spain.

2.2.9 Lyme Disease

Lyme disease is classified as a zoonosis, as it is transmitted to humans from a natural reservoir among rodents by ticks that feed on both sets of host.

Lyme disease, or Lyme borreliosis as it is sometimes known, is an infectious disease which is caused by at least three species of spirochete bacteria belonging to the genus *Borrelia*. In the United States the causative agent of this disease is usually *Borrelia burgdorferi* is the main cause of Lyme disease, whereas most European cases are caused by either *Borrelia afzelii* or *Borrelia garinii*. Lyme diseases presents due to a combination of infection with *Borrelia* bacteria combined with the body's immune response to the infection (Meyerhoff 2011).

Transmission of the bacteria occurs through being bitten by an infected tick from the genus *Ixodes*. However, transmission is quite rare, with only about 1% of recognized tick bites resulting in Lyme disease. This is thought to be because an infected tick must be attached for at least a day for transmission to occur.

Although ticks responsible for spreading Lyme disease are parasitic and hence rely on the presence of hosts for feeding, Guerra et al. (2002) demonstrated that the presence and abundance of the ticks was variable even when the host population was adequate and that the presence of ticks is positively associated with deciduous, dry to mesic forests and sandy or sandy loam textured soils.

The ECDC does not track infections of Lyme disease. The WHO has 97 deaths attributed to Lyme disease (including "meningitis due to Lyme disease" and "other neurologic disorders in Lyme disease") as having occurred in the EU 27 between 1995 and 2008.

2.2.10 *Pseudomonas aeruginosa*

Pseudomonas aeruginosa is a gram negative rod shaped bacterium which is widely distributed in soil and water around the world, as well as being found on plants and humans (Qarah and Cunha 2009). It is an opportunistic pathogen which rarely causes disease in healthy individuals but can cause a wide range of diseases in immuno-compromised individuals including respiratory tract infection, bacteraemia (infection of the blood), endocarditis (infection of the heart), urinary tract, gastrointestinal infection, as well as infecting bones, joints and the central nervous system. Therefore, it can be considered to be an EPO.

Symptoms usually include inflammation of the infected area and sepsis, and if critical organs become infected can be fatal (Balcht and Smith 1994). One characteristic of *Pseudomonas aeruginosa* which is of particular concern is its low antibiotic susceptibility (Hirulkar and Soni 2011).

The ECDC does not track infections of *Pseudomonas aeruginosa* and the WHO does not track deaths attributed to it in its ICD10 codes beyond those "...as the cause of diseases classified to other chapters".

2.2.11 Q fever

The causative agent of Q fever is the bacterium *Coxiella burnetii* which is a small, gram negative obligate intracellular parasite meaning that it has to infect the cells of a host in order to complete its life cycle. Therefore, it is classified as an STP.

The disease was originally described in Brisbane, Australia where the pathogen was discovered in 1937. It is considered to be a zoonotic disease with a reservoir in cattle, goats and sheep. The bacterium is found in all parts of the world with the exception of New Zealand (Greenslade et al. 2003) and Antarctica (Jones et al. 2011).

Coxiella burnetii is very hardy and resistant to heat, desiccation and many common disinfectants, and so is able to persist in the environment, including soil, for up to 150 days (Jones et al. 2011). Infection occurs from the inhalation of the bacteria in spore form, usually from farmyard dust. The presence of vegetation and increased soil moisture appear to reduce the transmission of *Coxiella* by reducing the amount of dust available for dispersion of the bacteria (van der Hoek et al. 2011). The bacterium can also be contracted through contact with milk, urine or bodily fluids of infected animals as well as through ticks. The bacterium is highly virulent in humans with an infective dose of just one organism being necessary to cause disease in some instances (Tigertt et al 1961).

Symptoms of acute Q fever usually appear 2-3 weeks after infection, although as many as half of the people infected with *C. burnetii* do not show symptoms. If symptoms do present they can vary greatly from person to person and can include high fever, severe headache, nausea, vomiting, diarrhea, abdominal and/or chest pain, myalgia, chills and/or sweats and a non-productive cough.

Most individuals who develop Q fever recover with the antibiotic doxycycline usually being very effective as treatment. However, complications may occur including pneumonia, inflammation of the liver or heart or central nervous system complications. Q fever can also occur in a chronic form which occurs in <5% of infected patients, particularly in pregnant women, immuno-compromised patients and people with pre-existing heart valve defects, and usually presents as endocarditis.

The ECDC reports that approximately 600 cases of Q fever infection occurred annually within the EU27 in 2006 and 2007 and then there was a large increase to 1667 cases in 2008 and 1962 in 2009 with the majority of cases being reported in The Netherlands which saw a more than 100 fold increase in infection rate across those 4 years. The WHO reported 11 deaths in the EU27 reported under the ICD10 codes between the years 1999 and 2007. Information concerning mean annual infection rates per country can be seen in Fig 8.

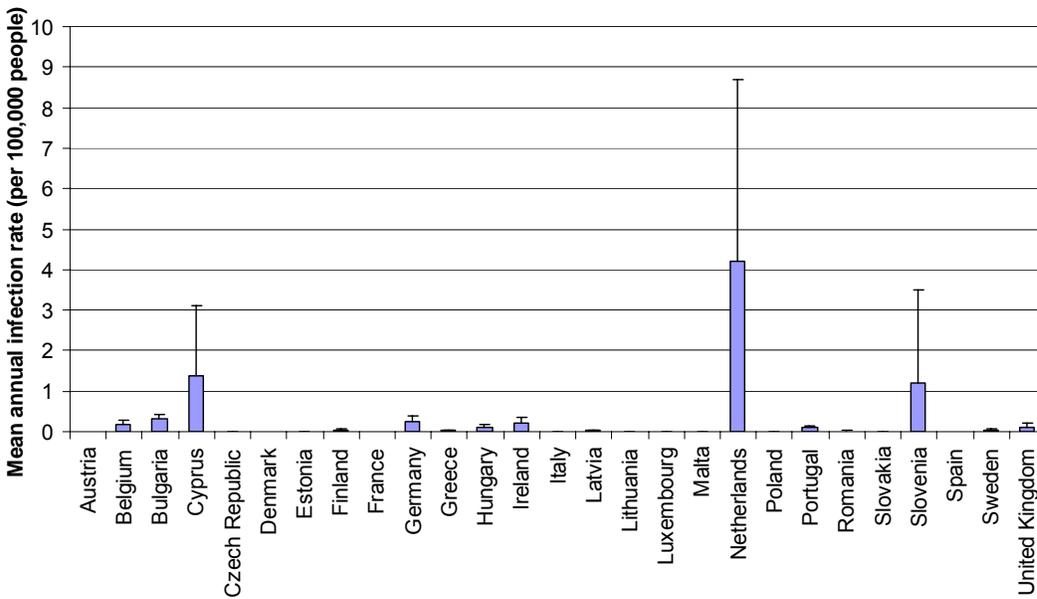


Fig 8: Mean annual infection rates of Q fever in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for Austria, Denmark, France, or Spain.

2.2.12 Salmonellosis

Salmonellosis, sometimes called salmonella, is caused by a group of gram-negative motile bacteria from the genus *Salmonella*. The genus includes many species of bacteria and each species may contain many serotypes. For example, the species *S. enterica* has over 2,500 different serotypes described, some of which are host restricted and others of which may have a very broad host range (Grassi and Finlay 2008).

Salmonella, along with *E. coli* and *Campylobacter*, are the most common forms of food borne illness. Although humans generally contract salmonellosis through eating contaminated food, one of the main routes in which pathogenic *Salmonella* bacteria come into contact with vegetables is from the soil (Islam et al. 2004).

Salmonella sp. along with other human enteric pathogens such as *E. coli* and *Campylobacter*, are often introduced into the soil with liquid manure on agricultural land (Bech et al. 2010). Once introduced into the soil, pathogenic species of *Salmonella* have been demonstrated to be able to persist for up to 231 days and to be capable of contaminating vegetables grown in such soils (Islam et al. 2004). Salmonellosis presents as one of three distinct syndromes, typhoid (enteric) fever, non-typhoidal enterocolitis, or non-typhoidal focal disease (Klotchko and Wallace 2011), all of which are caused by different serotypes of salmonella bacteria.

Typhoid fever can present with nonspecific features such as diarrhea, vomiting or respiratory symptoms. Approximately 10-15% of patients develop severe diseases which presents with persistent and high fever which generally is accompanied by relative bradycardia (slower than normal heart rate) as well as rose spots on the back, arms and legs in 25% of cases. The tongue is usually coated, the abdomen tender and the liver and spleen may become inflamed.

Non-typhoidal enterocolitis usually presents with loose bloodless stools. In rare instances large volume diarrhea may also be present which typically resolves in 3-7 days. Fever, headache, abdominal cramps and myalgia are also common with the fever usually resolving itself within 48 hours.

Non-typhoidal focal disease occurs due to bacteraemia if salmonella bacteria enter the blood. It may affect almost any organ with organs which have pre-existing structural anomalies are generally most vulnerable and prone to being affected (Klotchko and Wallace 2011).

A decline in the incidence of salmonella infection between 2006, where approximately 162,000 cases of infection were recorded, down to approximately 110,000 cases in 2009 within the EU27 (ECDC 2011). 960 deaths were recorded by the WHO under the ICD 10 codes as attributed to salmonella infection (all types) between the years 1994 and 2008. Information concerning mean annual infection rates per country can be seen in Fig 9.

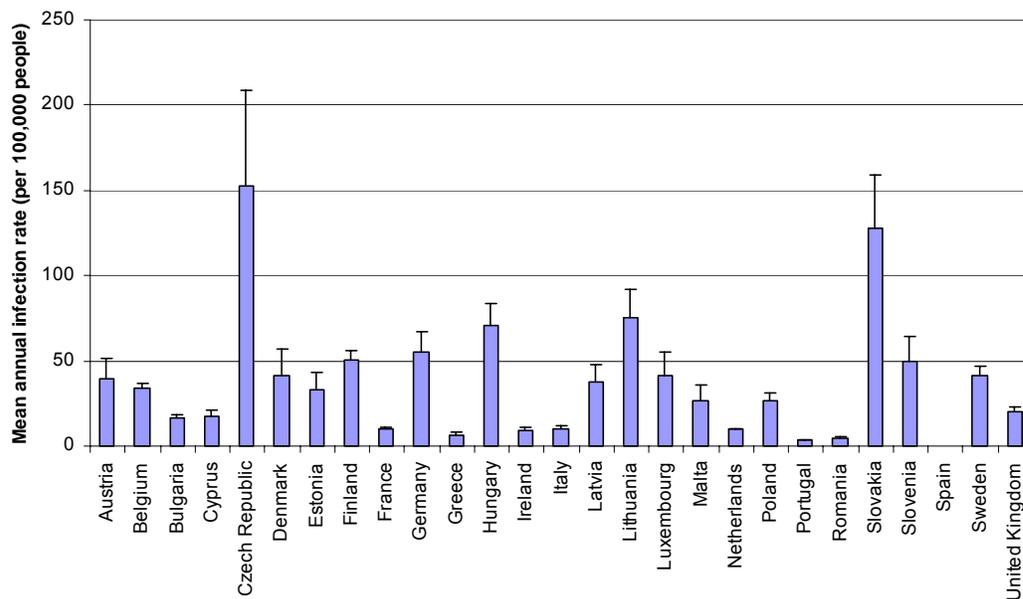


Fig 9: Mean annual infection rates of salmonellosis in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for Spain.

2.2.13 Shigellosis

Shigellosis is caused by one of four different species of bacteria from the genus *Shigella*: *S. flexneri* (also known as “Type B”); *S. sonnei* (also known as “Type D”); *S. boydii*; and *S. dysenteriae*. These bacteria are capable of causing an acute intestinal disease which may present with symptoms ranging from relatively mild abdominal pains, up to stomach cramps, diarrhea, fever, vomiting and blood in stools. In its more extreme presentations shigellosis is often referred to as dysentery (which is an umbrella term for similar symptoms caused by a range of viruses, bacteria or protozoa), and can be fatal if left untreated

Transmission of *shigella* bacteria can occur through eating infected food, contaminated soil or water, or human to human transmission can occur through blood, saliva, sexual contact, the faecal-oral route or from the mother to fetus. Shigellae bacteria have been found to survive for up to approximately 40 days in the soil, with the strain of bacteria having a greater influence on their viability than soil type and temperature (Leonardopoulos et al. 1980). This means that soil may remain as a source of infection even after an infectious individual may have long left the area.

Shigella species are responsible for approximately 165 million cases of severe dysentery globally each year. The majority of these cases occur in developing countries in children under 5 years of age; more than one million people are estimated to die from *Shigella* infection each year. Within the EU27 approximately 6,000 – 8,000 cases of *Shigella* infection were reported annually between 2006 and 2009 according to data available from the ECDC. However, only 85 deaths have been attributed to *Shigella* within the EU27 since 2000 (WHO 2011). These relatively low infection and mortality rates when compared to the global average are likely due to the improved sanitation generally found within the EU27 when compared to the developing world, and the ready access to medical facilities to counter

dehydration (the main risk associated with *Shigella* infection due to the diarrhea it causes), as well as the ready access to antibiotics which are responsible for the greatly reduced mortality rates seen within the EU27 despite *S. dysenteriae* Type 1 having formed resistance to several antimicrobial drugs including ampicillin, chloramphenicol, streptomycin, tetracycline and sulfadiazine (Olarie et al. 1976). Information concerning mean annual infection rates per country can be seen in Fig 10.

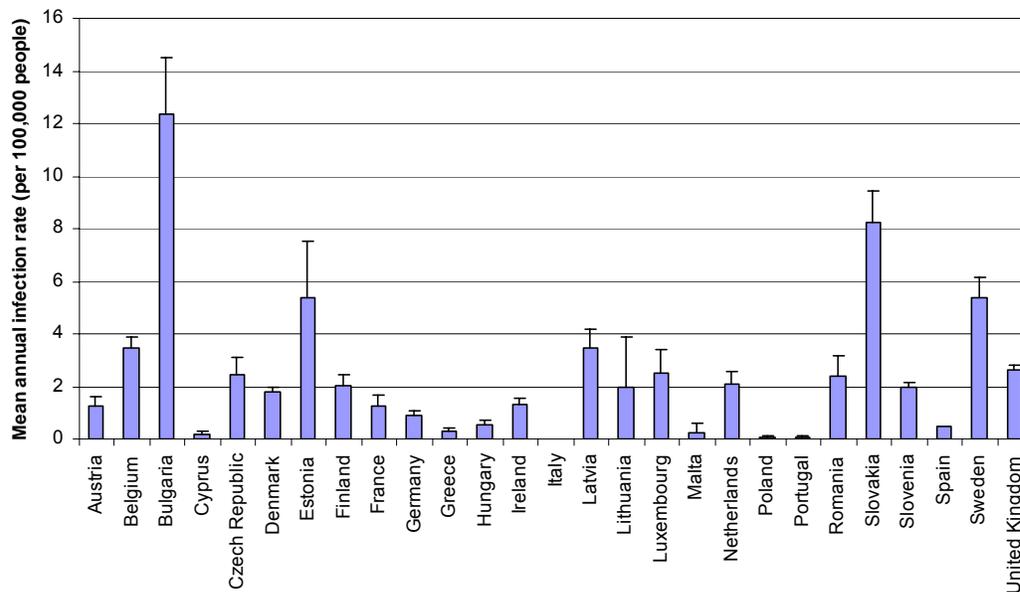


Fig 10: Mean annual infection rates of shigellosis in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for Italy.

2.2.14 Tetanus

Clostridium tetani is the causative agent of the diseases tetanus. *C. tetani* bacteria, as all other species of *Clostridium*, are spore forming, gram positive and obligate anaerobes.

The *C. tetani* bacterium, which has a global distribution, is generally found in the soil as well as in animal waste. The association that many people have with tetanus and rusty metals is somewhat misconstrued. The bacterium does not have any propensity to grow on rusty metal, but rather owing to its highly resilient spores is capable of surviving on rusty metal. The rusty metal itself sometimes provides a means of infection through piercing the skin of individuals who cut themselves on the metal or stand on rusty nails etc.

The tetanus disease is caused by a toxin which is produced by the bacterium and affects the central nervous system. The incubation period can vary from a few days to a few weeks. The first signs of tetanus infection are usually general tiredness or weakness, followed by muscle spasms. The spasms can be particularly prevalent in the jaw muscles at the onset of the disease, causing the jaw to become tightly closed due to these muscle spasms and leading to the alternative name for the diseases “lockjaw”.

Diagnosis of tetanus is based on the presentation of symptoms as there are currently no blood tests which are capable of detecting tetanus. Furthermore, *C. tetani* bacteria are only recovered from the wound in approximately 30% of cases, as well as occasionally being isolated from wounds of individuals who do not have symptoms of tetanus which is testament to how widespread this species of bacteria is.

Tetanus is a preventable condition owing to the availability of an effective vaccine. Most people within the developed world are offered vaccination against tetanus as a part of each countries standard vaccination schedule. Infection with tetanus as registered with the ECDC has seen a continuous decline within the EU27 from 163 infections in 2006, down to 78 infections in 2009. However, 412

deaths are registered in the WHO mortality database as attributed to “other tetanus” under the ICD 10 codes between the years 1996 and 2008. Information concerning mean annual infection rates per country can be seen in Fig 11.

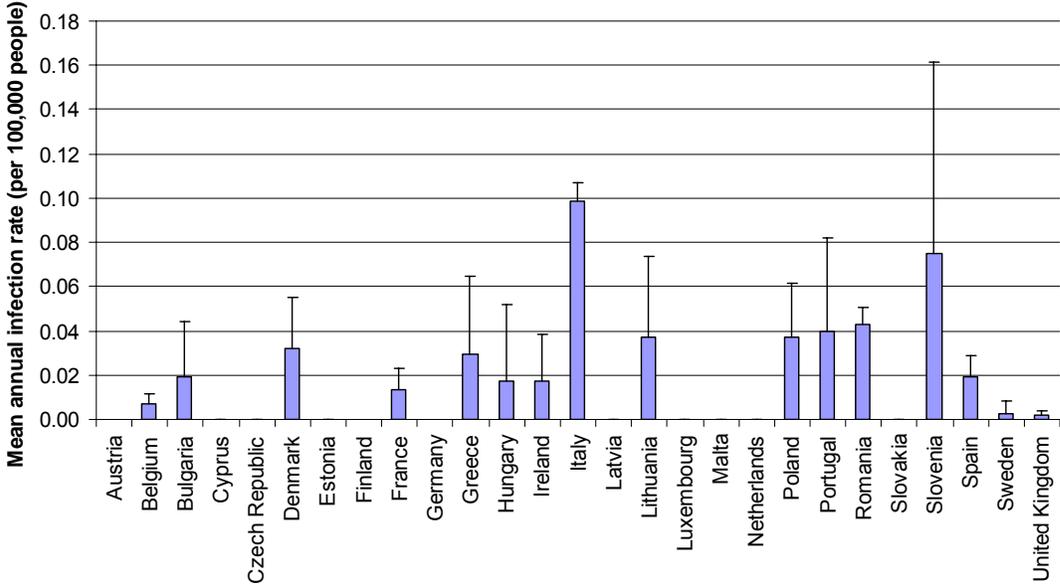


Fig 11: Mean annual infection rates of tetanus in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for Austria, Finland or Germany.

2.2.15 Tularemia

Tularemia is caused by the bacterium *Francisella tularensis* which is a non-motile gram-negative coccobacillus of which several serotypes exist, each with varying degrees of virulence. The bacterium is a facultative intracellular pathogen which causes tularemia in humans and animals. The disease is something known as rabbit fever, or deer fly fever owing to the fact that the primary vectors are deer flies or ticks, as well as other arthropods. As well as bites from arthropods, the disease can be contracted through direct contact between a break in the skin with an infected animal or its dead body (most often rabbits, hence the name rabbit fever; Schaffner 2007). The disease may also be contracted through inhalation or ingestion of infected contaminated dust or soil (Reintjes et al. 2002). Inoculation or inhalation of as few as 10 cells of *F. tularensis* bacteria can be sufficient to cause disease in humans (Abd et al. 2002).

The bacterium is known to survive for weeks at low temperatures in water, soil and animal carcasses, and for more than a year in mud in some instances (Parker et al 1951). Evidence suggests that the main reservoir of the bacterium in the environment may be the cells of protozoa, particularly the protozoa *Acanthamoeba castellanii* which is ubiquitous in the environment (Abd et al. 2002).

The incubation period after infection is usually 3 to 5 days after exposure and the illness usually starts very suddenly and may continue for several weeks. Symptoms include chills, fever, headache, joint stiffness and muscle pain and red spots on the skin which grow to become ulcers. Tularemia is fatal in approximately 5% of untreated cases, but less than 1% of treated cases (Dugdale et al. 2011)

Between approximately 550 (2006) and 1200 cases (2007) were been reported within the EU 27 between 2006 and 2009. The disease is generally cured with antibiotics, although a high relapse rate has been observed with tetracycline and chloramphenicol (Dugdale et al. 2011). Only 5 deaths were recorded by the WHO under the ICD 10 codes as attributed to tularaemia between the years 1995 and 2003 within the EU27. Information concerning mean annual infection rates per country can be seen in Fig 12.

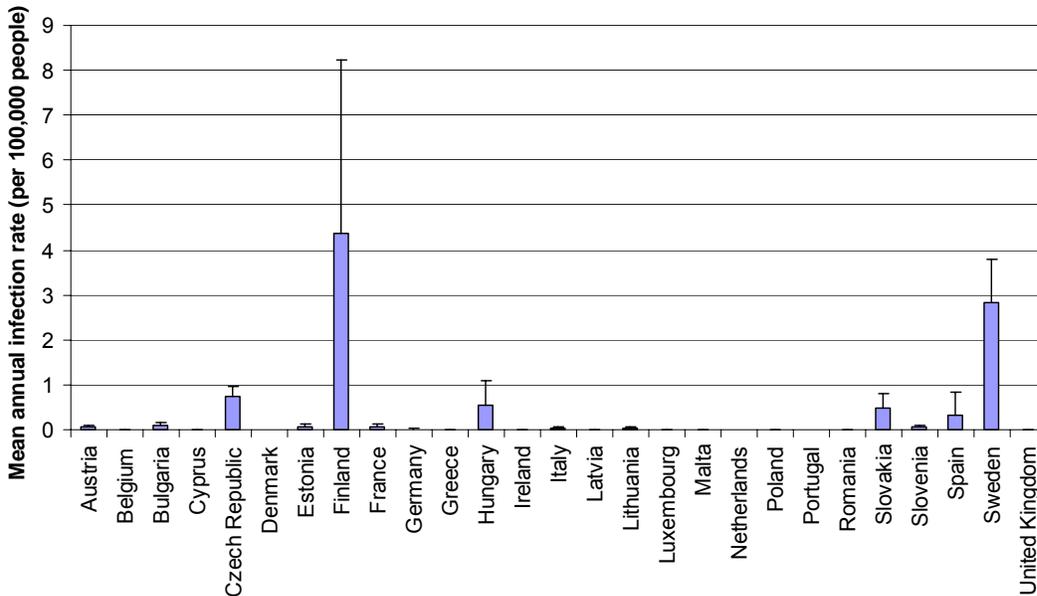


Fig 12: Mean annual infection rates of tularemia in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for Denmark, The Netherlands or Portugal.

2.2.16 Yersiniosis

Yersinia enterocolitica is the causative agent of yersiniosis. It is a species of gram negative coccobacillus bacterium which is part of the family Enterobacteriaceae, along with *Salmonella* and *E. coli*. It is a zoonotic disease which can be transmitted from many different animal including pigs, cattle and deer. *Yersinia enterocolitica* consists of a relatively diverse group of bacteria which can be divided into either 18 or 54 different serotypes depending on the criteria used (Weagant and Feng 2001).

The survival of *Yersinia enterocolitica* in soil is strongly affected by environmental conditions, particularly soil moisture, as the bacterium does not cope well with desiccation. For example, it has been demonstrated that air drying soil over 10 days reduced the number of viable cells to only 0.1% of the original population (Chao et al. 1988), although different strains of the bacterium have different survival rates (Tahiro et al 1991).

Symptoms usually develop between 4 and 7 days after infection which usually occurs through ingestion of the bacteria, usually from undercooked food, particularly pork products. Symptoms of yersiniosis include fever, diarrhea (which may be bloody) and abdominal pain. In some instances joint pain, particularly in the knees, wrists or ankles may also occur, although this is relatively rare. A skin rash may also occur on the legs and torso, which is also rare but slightly more common in women than men.

The disease usually resolves itself within 1 to 3 weeks, although in some cases treatment through the use of antibiotics is required, particularly in susceptible individuals such as those who are immunocompromised. In very young infants (i.e. <3 months or so in age) the bacteria is sometimes capable of infecting the blood (bacteraemia) and as such young infants who contract the diseases may require hospital treatment.

Between approximately 7,600 and 9,000 cases of yersiniosis were reported within the EU27 annually between 2006 and 2009 according to the ECDC. The WHO registered 340 deaths in the EU27 between 1994 – 2008, under the ICD10 codes, as being attributed to enteritis caused by *Yersinia enterocolitica*. Information concerning mean annual infection rates per country can be seen in Fig 13.

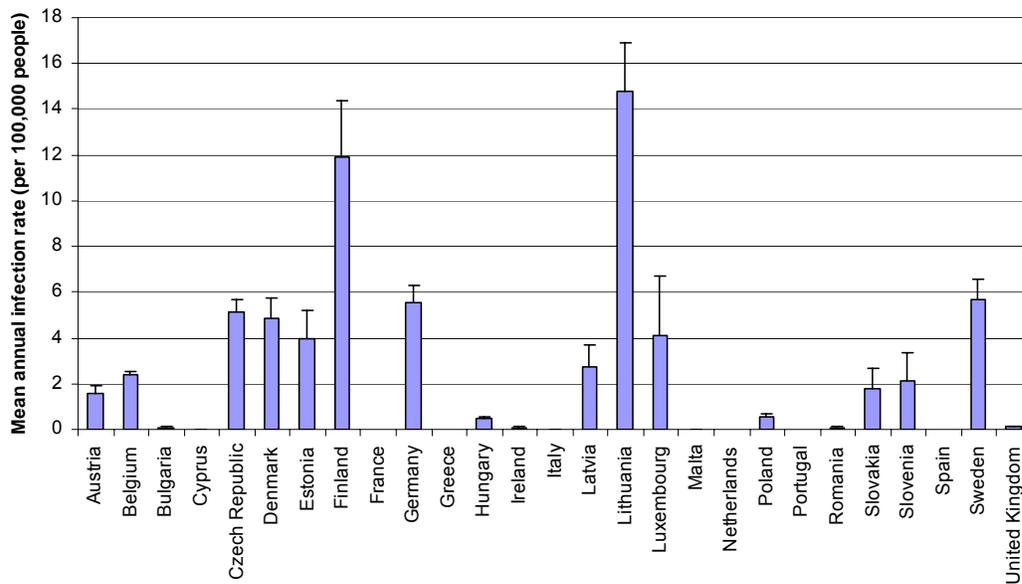


Fig 13: Mean annual infection rates of yersiniosis in the EU27, by member state, per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for France, Greece, The Netherlands or Spain.

2.2 Protozoa

Protozoa are a highly diverse group of single celled eukaryotic organisms such as amoebae. They are distributed globally and are found in high abundances in soils where they feed on bacteria, fungi and organic matter within the soil and in turn provide a food source for other soil invertebrates. Some species are pathogenic in humans, however, the most famous of which being *Plasmodium* sp., the causative agents of malaria. However, unlike the *Plasmodium* sp. which has mosquitoes as a vector, several groups can be soil borne, and these are discussed in more detail below.

2.2.1 Amoebiasis

The causative agent of amoebiasis is the protozoan *Entamoeba histolytica* which is found worldwide and is an anaerobic parasitic protozoan. The highest incidences of amoebiasis are in the developing world, particularly in areas where there is inadequate sanitation and hygiene. Amoebiasis is second only to malaria with regard to deaths caused by protozoa globally (Lacasse and Cleveland 2009) although deaths from amoebiasis are relatively rare within the EU27.

Infection with *E. histolytica* can be asymptomatic or may cause diarrhea and abdominal pain.

However, dysentery can occur in some cases as well as invasive extra intestinal disease, which most commonly involves the liver but may also affect other organs (Lacasse and Cleveland 2009).

Infection generally occurs through the faecal-oral route which may involve the organism being in the soil for some time, although other routes of transmission have also been reported (Lacasse and Cleveland 2009). *E. histolytica* is capable of forming cysts and in this form can survive for weeks or months in soil (Beaver and Deschamps 1949), and so infect hosts who later consume food or water contaminated with such soil.

The ECDC does not track infection rates of amoebiasis. The WHO registered 20 deaths as being attributed to amoebiasis within the EU27 under the ICD10 codes between 2000 and 2007, including amoebic liver and lung abscesses and amoebic non-dysenteric colitis.

2.2.2 Balantidiasis

Balantidiasis is caused by the ciliated protozoan *Balantidium coli*. It is found worldwide but is most common in Latin America, Southeast Asia, The Philippines and Papua New Guinea. It is thought that pigs are the most likely reservoir of this protozoan (Chijide 2008). However, under favourable conditions with regard to temperature and humidity *Balantidium coli* can survive for weeks to months in soil in cyst form (Goldsmith 1997).

Infection with *B. coli* is often asymptomatic. However, some individuals may present with diarrhea, which may be watery or bloody and which may lead to severe fluid loss, as well as with nausea and vomiting. Abdominal pain may be present, along with loss of appetite, headache and fever.

The ECDC does not track infection rates of balantidiasis. The WHO has no registered deaths as being attributed to balantidiasis within the EU27 under the ICD10 codes. However, it should be noted that the WHO does have deaths registered to non-specific diseases such as “Diarrhoea and gastroenteritis of presumed infectious origin” of which over 11,000 deaths have occurred within the EU27 between 1994 and 2008 and which may include balantidiasis. This means that it is not possible to say with confidence that no deaths have occurred due to balantidiasis within the EU27 in recent years. There is insufficient clarity in the data to be able to draw a conclusion in this regard.

2.2.3 Cryptosporidiosis

Cryptosporidiosis is caused by spore-forming protozoa from the genus *Cryptosporidium* such as *C. parvum* and *C. muris*. As with the other protozoan diseases it is usually spread through the faecal-oral route and so is most prevalent in areas of poor sanitation where it mostly affects children (Cabada and White 2011).

Infection with *Cryptosporidia* is often asymptomatic. For those infected individuals that do present with symptoms diarrhea is the most common which is often watery and incapacitating. Nausea, low grade fever and abdominal cramps are also common symptoms. Symptoms usually last for 5-10 days, although they may persist for up to 4 weeks (Cabada and White 2011).

In cyst form, *Cryptosporidia* is capable of surviving in soil for weeks or months. Like other protozoa, while in cyst form *Cryptosporidia* are non-motile. However, they have been found to leach down through soil due to rainfall (Mawdsley et al. 1996) and as such may possibly migrate from the initial area of contamination, particularly if they enter a water source such as a river.

The ECDC does not track infection rates of cryptosporidiosis. The WHO registered 10 deaths as being attributed to Cryptosporidiosis within the EU27 under the ICD10 codes between 1999 and 2006, 80% of which occurred within the United Kingdom between 2002 and 2006.

2.2.4 Cyclosporiasis

Cyclosporiasis is caused by the coccidian protozoan *Cyclospora cayetanensis* which only infects humans (Shoff and Behrman 2010). As with the other protozoan diseases it is usually spread through the faecal-oral route and so is most prevalent in areas of poor sanitation. The protozoan is capable of surviving for extended periods in soil and as such contact with contaminated soil has been suggested to be an important mode of transmission (Chacin-Bonilla 2007; 2010).

Infection with *Cyclospora cayetanensis* usually presents with diarrhea which may be explosive and accompanied with abdominal cramps, fatigue, malaise and weight loss and which may be interspersed with periods of remission. The diarrhea may persist for weeks to months if left untreated (Shoff and Bahrman 2010).

The ECDC does not track infection rates of cyclosporiasis. The WHO has no registered deaths as being attributed to cyclosporiasis within the EU27 under the ICD10 codes. However, as above for balantidiasis, it should be noted that the WHO does have deaths registered to non-specific diseases such as “Diarrhoea and gastroenteritis of presumed infectious origin” of which over 11,000 deaths have occurred within the EU27 between 1994 and 2008 and which may include cyclosporiasis and as

such it is not possible to say that no deaths have occurred due to cyclosporiasis within the EU27 in recent years. There is insufficient clarity in the data to be able to draw a conclusion in this regard.

2.2.5 Giardiasis

The causative agent of giardiasis is the flagellate protozoan *Giardia lamblia* which is the most common protozoan intestinal parasite worldwide. It is a zoonotic disease which has been isolated from the stools of various animals including cats, dogs, sheep, cattle and various rodents.

Giardia lamblia is found throughout the world. It does not strongly bind to soil particles and so in the event of rainfall leading to overland flow, the protozoan is easily washed away and so can be transported for long distances (Dai and Boll 2006). Cysts of *G. lamblia* are capable of surviving for up to 3 months in water at 4°C. While data on survivability in soil is not currently available it seems likely that it will survive for a similar amount of time in soil in its resistant spore form, although this may well be dramatically reduced in dry soils (Sattar 1999).

Giardiasis usually presents with a broad spectrum of symptoms with some people experiencing an abrupt onset of explosive watery diarrhea with abdominal cramps, vomiting, fever, malaise and foul flatus which last for 3-4 days before transition into the more common symptoms of giardiasis.

However, most people experience a slower onset of symptoms. These include stools which become malodorous, mushy and greasy which may alternate with watery diarrhea and constipation. Upper and mid abdominal cramps may occur, as well as sulphurous belching, substantial burning and acid indigestion as well as loss of appetite, fatigue and malaise.

Between approximately 14,400 and 18,100 cases of giardiasis were logged at the ECDC within the EU27 between 2006 and 2009. The WHO registered 3 deaths as being attributed to giardiasis within the EU27 under the ICD10 codes between 2004 and 2006, all of which occurred within the United Kingdom. Information concerning mean annual infection rates per country can be seen in Fig 14.

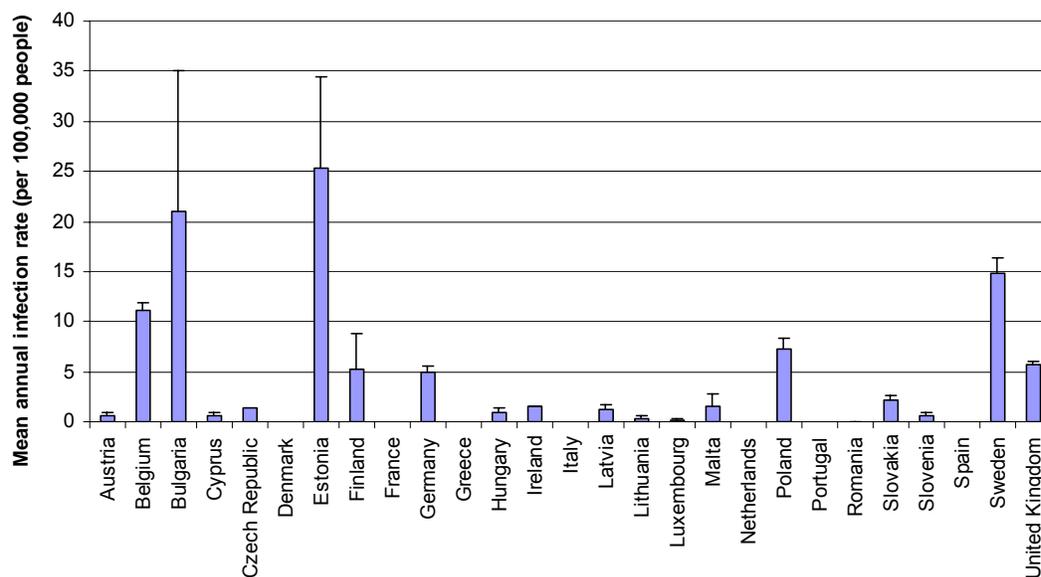


Fig 14: Mean annual infection rates of giardiasis in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for Denmark, France, Italy, Portugal or Spain.

2.2.6 Isoporiasis

Isoporiasis is a relatively uncommon illness caused by the coccidian protozoan *Isospora belli*. Humans are the only known hosts for the protozoan which is distributed globally, but more common in the tropics and subtropics. The protozoan is an obligate intracellular parasite meaning that it must enter a human cell in order to complete its lifecycle. However, the organism also has an external stage whereby oocysts need to enter the external environment, usually water contaminated with faeces, but also possibly soil, in order for the oocysts to mature (Tolan et al. 2011).

As with other infectious protozoa, the main route of infection is the faecal-oral route. No data is currently available on the survivability of *I. belli* in soil, but as with other protozoa it is likely to be soil moisture dependent, with moist soil favouring longer survival times.

The disease usually presents with profuse, watery, offensive smelling diarrhea with abdominal cramps, loss of appetite and low grade fever. Headache and myalgia may also present, although more rarely. The incubation period of the disease ranges from 3 – 14 days and symptoms usually persist for 2-3 weeks (Tolan et al. 2011)

The ECDC does not track infection rates of isoporiasis and the WHO has no deaths registered as being attributed to isoporiasis within the EU27 under the ICD10 codes.

2.2.7 Toxoplasmosis

Toxoplasmosis is caused by the protozoan *Toxoplasma gondii* which is an obligate intracellular parasite. Humans are only intermediate hosts of the protozoan, along with other mammals, with cats being the primary hosts.

T. gondii is very common and it is estimated that approximately one third of the world's population of humans have been exposed to it (Montoya and Liesenfeld 2004). However, the disease is usually asymptomatic with symptoms only usually presenting in immuno-compromised individuals and in new born infants who may acquire the disease *in utero* from their mothers.

T. gondii has been shown to be detectable in soils where infected cats defecate and so can be highly localised (Afonso et al 2008). When in the soil, the oocysts of *T. gondii* have been found to be able to survive for at least 3 months (Lindsay et al. 2002) meaning that soils can remain infectious for long periods of time after the originally infected individual has left.

Symptoms, when present, include flu-like symptoms with prominent lymphadenopathy (swollen lymph nodes). Ocular infection may occur and is usually painful and may lead to impaired vision and the presence of floaters.

In immuno-compromised patients the central nervous system may become infected, leading to seizures, headaches and altered mental states. The lungs may also become infected leading to a non productive cough, chest discomfort and dyspnoea.

Congenital toxoplasmosis can cause jaundice, developmental delay, visual defects including blindness, and cerebral calcification (Becker et al. 2010).

Toxoplasmosis is relatively rare within the EU27 with only approximately 20 – 30 cases reported annually to the ECDC between 2006 and 2009. However, the WHO has 159 deaths reported as attributed under the ICD 10 codes to various forms of toxoplasmosis including toxoplasma meningoencephalitis, toxoplasma hepatitis and “toxoplasmosis with other organ involvement” between 1996 and 2008 within the EU27. Information concerning mean annual infection rates per country can be seen in Fig 15.

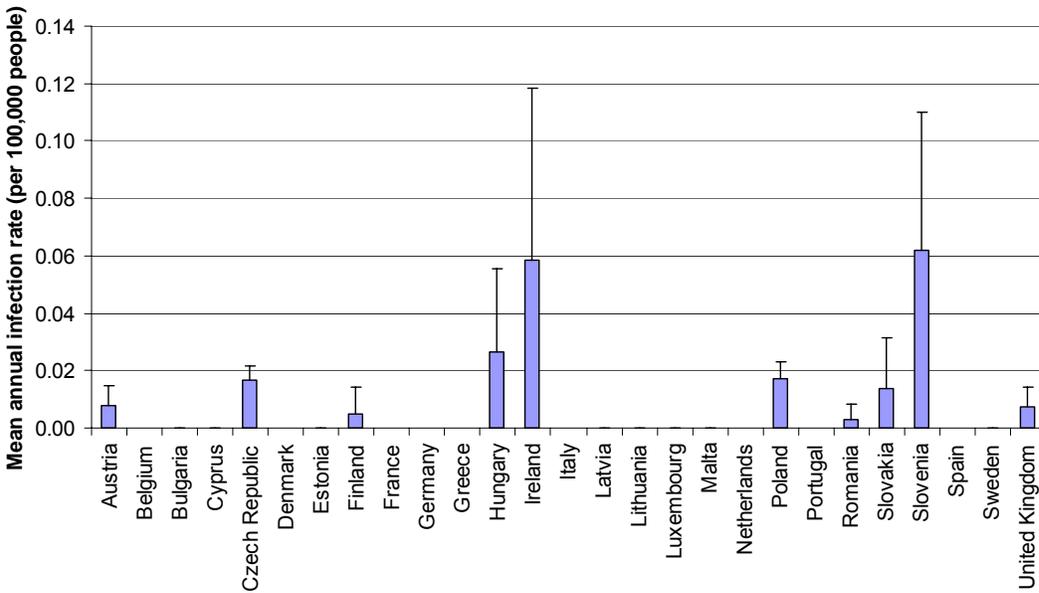


Fig 15: Mean annual infection rates of toxoplasmosis in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for Denmark, France, Germany, Greece, Italy, the Netherlands, Portugal or Spain.

2.3 Fungi

The fungi, like humans, are from the domain “Eukaryota”. This means that fungal cells are more similar to human cells than they are to bacterial cells. This means that complications arise with regard to treatment of fungal diseases as the two types of cell (human and fungus) are relatively similar when compared to bacterial cells. Therefore, that the chemicals which can be used to combat fungal infection are relatively limited when compared to the antibiotics used to fight bacterial infection, as they need to be able to attack the invading fungal cells without doing too much damage to the bodies own cells.

A little over 400 species of fungi are known to cause diseases in animals and far fewer cause diseases in humans. Generally they are caused by opportunistic pathogenic strains of fungi which pose the greatest risk to immuno-compromised patients. A rise in the incidence of these diseases had been reported (Low and Rotstein 2011), with one possible explanation for this rise being the increase in the number of patients that are immuno-compromised as a result of chemotherapy, taking anti-rejection drugs post transplant, people with AIDS and other long term health conditions such as diabetes (Low and Rotstein 2011). Many fungal diseases which occur in humans are superficial, affecting the skin, nails or hair, such as ringworms (generally caused by *Microsporum canis*) and “athlete’s foot” (caused by *Trichophyton mentagrophytes* and *Trichophyton rubrum*; Pelayo Ulacia and Dafhnis 1980), with only relatively few fungal diseases which are potentially fatal in humans.

The majority of human pathogenic fungi are soil inhabiting saprotrophs (i.e. they feed on dead organic matter within the soil). However, when coming into the contact with immuno-compromised patients or other susceptible individuals, such as in an open wound or through the inhalation of spores, some fungi can become very aggressive forms of infection. Furthermore, owing to the eukaryotic nature of fungi, meaning that their cells are more similar to human cells as previously discussed, many antibiotics are ineffective and different antifungal medication is required.

2.3.1 Aspergillosis

The name aspergillosis relates to an infection, growth or allergic reaction caused by a fungus of the genus *Aspergillus*. *Aspergillus* fungi are relatively common in soils and leaf litter all around the world including forest, wetland, cultivated soils and particularly in desert soils (Klich 2002). In the soil *Aspergillus* sp. function as saprophytes, and as with most other soil fungi play an important role in the breakdown of organic matter.

Despite being very wide spread globally, meaning most people are frequently exposed to the fungus, infections only occur very rarely in people with normal immune systems (Zieve et al 2010).

Aspergillosis can occur in three different forms in humans. Pulmonary aspergillosis – allergic bronchopulmonary type, which is an allergic reaction to the fungus which may develop in people who already have lung problems such as cystic fibrosis or asthma; Pulmonary aspergillosis – invasive type, which is a serious infection with pneumonia which may spread to other parts of the body. This type occurs almost exclusively in immuno-compromised patients such as those with AIDS, those undergoing chemotherapy or on medication after organ transplants; and finally Aspergilloma, which is a growth of fungus that develops in an area where previous lung damage has occurred through lung disease such as tuberculosis or a lung abscess (Zieve et al. 2010).

The ECDC does not track infection rates of aspergillosis within the EU27. The WHO logged 2,357 deaths under the ICD10 codes as attributed to aspergillosis (all forms) between the years of 1994 and 2008 within the EU27.

2.3.2 Blastomycosis

Blastomycosis refers to fungal infections caused by the dimorphic fungus *Blastomyces dermatitidis*, which is endemic to portions of North America (Kayser et al. 2005) or the fungus *B. brasiliensis* (also known as *Paracoccidioides brasiliensis*) which is endemic to some areas of South America (Kayser et al. 2005). Dimorphic refers to the ability of these fungi in two undertake growth forms, either hyphal growth like a mould, or single cellular growth like a yeast.

Infection can be caused by either spores or mycelium from the soil and may occur in any part of the body, although primary infection tends to be pulmonary due to transmission from the soil via inhalation. Less commonly infection may occur through the skin after trauma should infectious cells enter the wound (Steele and Shetty 2011). Symptoms normally include low grade fevers, chest pain, a mild but persistent productive cough and haemoptysis (i.e. coughing up of blood) (Feng et al. 2007). Secondary infections may also be detected as lesions in the skin which may remain localised or, in some instances, may spread throughout the body leading to extensive ulceration (Steele and Shetty 2011). It has been reported that males are more susceptible to infection with the ratio being as high as 10 infected males for every 1 infected female (Morris et al. 2004), a fact which is generally attributed to males being more likely to be associated with activities with an increased risk of infection such as hunting, camping and logging (Fang et al. 2007). In South America, blastomycosis is most commonly observed among farmers. There is no evidence of transmission of the diseases from either infected humans or infected animals to other humans, even if they are susceptible to the diseases, i.e. through being immuno-compromised (Kayser et al 2005). There is currently no effective treatment for blastomycosis.

The ECDC does not track infection rates of blastomycosis within the EU27. The WHO has 9 deaths registered in its mortality database under the ICD 10 codes attributed to blastomycosis in various forms (e.g. acute pulmonary blastomycosis, disseminated blastomycosis) between 2001 and 2008. However, it is not possible to say whether these individuals were also infected from within the EU27 or whether they contracted the disease from other areas where the fungus is more common or endemic.

2.3.3 Coccidioidomycosis

The causative agents of coccidioidomycosis (also known as valley fever, among other names) are the fungi *Coccidioides immitis* and *Coccidioides posadasii* (Oppenheimer et al. 2010). Both species of fungus are morphologically identical but are distinct both genetically and epidemiologically. *C. immitis* is endemic to the San Joaquin valley region of California, whereas *C. posadasii* is endemic to desert areas of South and Central America, Northern Mexico and the south west of the United States of America (Oppenheimer et al. 2010). Cases of coccidioidomycosis have been reported in Europe, but these have been associated with patients who have travelled to the United States of America and other places where the fungus is endemic (Asgari et al. 2001).

The number of cases of coccidioidomycosis have been reported to increase markedly in the later summer and early autumn (i.e. the dry season) where soil disturbances due to wind erosion, or other anthropogenic factors, such as agricultural practices, are thought to make the fungus airborne and thereby increases the risk of its inhalation (Oppenheimer et al. 2010).

Infection usually presents as an acute but benign and respiratory disease which is generally self-limiting. Furthermore, once the infected individual recovers, they are likely to be permanently immune from further infection by the same fungus.

The ECDC does not track infection rates of coccidioidomycosis within the EU27. The WHO has 4 deaths registered in its mortality database under the ICD 10 codes attributed to coccidioidomycosis. However, as with the case for blastomycosis, it is not possible to say whether these individuals were also infected from within the EU27 or whether they contracted the disease from other areas where the fungus is more common or endemic.

2.3.4 Histoplasmosis

Histoplasmosis is caused by the fungus *Histoplasma capsulatum*. It is also known as Darling's diseases, Ohio valley diseases or Cave disease. The disease primarily affects the lungs after infection due to the inhalation of spores disturbed from the soil or from bird or bat droppings. The fungus is endemic to the Ohio, Missouri and Mississippi river valleys in the United States as well as in caves in southern and East Africa and other river valleys of North and Central America, eastern and southern Europe, eastern Asia and Australia (Fayyaz and Lessnau 2010).

Histoplasma capsulatum is a dimorphic fungus which grows in mycelial form while in the soil at ambient temperatures but which grows in a single celled yeast form at body temperature in mammals (Fayyaz and Lessnau 2010). While the most infectious soil occurs in areas inhabited by birds and bats, birds cannot transmit the diseases, while bats can. Bird excrement, however, enriches the soil and provides favourable conditions for the fungus to grow, i.e. acidic damp soil which is high in organic matter. In contrast, bats can become infected with the fungus and can transmit it through their guano. Owing to the fact that *Histoplasma capsulatum* can survive well and even thrive within the soil, contaminated areas can remain infectious for years (Fayyaz and Lessnau 2010).

Approximately 90% of people infected with *Histoplasma capsulatum* remain asymptomatic with histoplasmosis usually only developing in immuno-compromised individuals. When symptoms occur they can take different forms; either acute or chronic pulmonary histoplasmosis where symptoms include fever, chills, cough (including possibly coughing up of blood), and chest pain which is usually associated with inhalation (Dugdale et al. 2008); or disseminated histoplasmosis where the fungus spreads beyond the lungs via the blood and may affect multiple organs, and may include headache, neck stiffness, mouth sores and skin lesions (Dugdale et al. 2008). Disseminated histoplasmosis is usually fatal if left untreated (Kauffman 2007).

Data on infection rates is not kept by the ECDC. The WHO has 9 deaths logged under their ICD10 codes as attributed to histoplasmosis between the years of 2000 and 2007 in the EU27.

2.3.5 Sporotrichosis

Sporotrichosis is a chronic fungal skin infection which is caused by the fungus *Sporothrix schenckii*. The fungus is wide spread in the soil with a global distribution (Lima Barros et al. 2011) and may also be found on plant materials such as hay. Infection usually occurs via infection through broken skin while handling plant materials and as such is often associated with farmers, horticulturalist and gardeners. Disseminated sporotrichosis (i.e. sporotrichosis which has spread through the infected individual's body) may also occur in immuno-compromised people who inhaled dust containing spores.

Symptoms usually include a red lump which develops at the site of infection and which eventually turns into an ulcer. As the fungus sometimes grows through the lymphatic channels in the body small ulcers may appear in lines on the skin passing up a leg or arm from the initial site of infection. Treatment is required to allow such sores or ulcers to heal which takes the form of antifungal medicine such as itraconazole or fluconazole. Disseminated sporotrichosis may lead to lung and breathing problems as well as infection of bone or the central nervous system.

The ECDC does not track infection rates of sporotrichosis. The WHO has only 1 death registered as attributed to sporotrichosis under the ICD 10 codes as occurring within the EU27 which occurred in Poland in 2002 (Lymphocutaneous sporotrichosis) demonstrating that the disease is only very rarely fatal.

2.3.6 Mucormycosis

Mucormycosis is the name given to a range of diseases which are caused by fungi of the order Mucorales, which is the largest order of zygomycete fungi, and which are sometimes referred to as pin moulds. Mucoralean fungi are generally saprotrophic and grow on organic matter such as dead plant or animal material within soil. However, some species of Mucorales are parasites or pathogens of plants or animals, including humans. Fungi from the genera *Rhizopus* are the most common causative agents of these diseases although fungi from the genera *Mucor*, *Absidi* and *Cunninghamella* are also frequently implicated in causing diseases (Crum-Cianflone 2008).

Infection usually occurs in the lungs, sinuses or brain, although may also occur in the gastrointestinal tract and other organs, and is most common in immuno-compromised patients such as those with undergoing cancer treatment, or suffering from AIDS.

Mucormycosis is very rare but has a high mortality rate (50-85%) which can be even higher in individuals with gastrointestinal or pulmonary infection (Crum-Cianflone 2008). It is necessary for infected tissues to be removed for survival combined with medical therapy including antifungal medication. Infections have been found to increase in areas after tornados which can lift spores from the soil into the air where they can then infect wounds of individuals struck by debris as a result of the tornado (Williams 2011).

The ECDC does not track infection rates of mucormycosis. Owing to the rarity of infection only 11 mortalities are registered with the WHO between 1998 and 2006 under the ICD10 codes within the EU27, despite its high mortality rate once individuals are infected.

2.3.7 Mycetoma

Mycetoma refers to two diseases, caused by either actinomycetes or fungi, which result in chronic subcutaneous infection. Mycetoma which is caused by actinomycetes is called actinomycetoma and that caused by true fungi is called eumycetoma. More than 20 species of fungi and actinomycetes can cause mycetoma, with actinomycetes causing almost three times as many cases of the disease as true fungi (Ania et al. 2008). Mycetoma may result from the infection of one of a number of different soil dwelling actinomycetes or fungi including organisms of the genera *Nocardia*, *Streptomyces*, *Madurella* and *Pseudoallescheria*.

The disease is most common in Africa, but is also found in Central and South America, India, and the Middle and Far East. It is most common between latitudes of 15°S and 30°N.

The disease usually presents with subcutaneous swelling which is painless, and usually occurs at the site where a previous penetrating injury has occurred which has inoculated the actinomycete or fungus into the wound. Development of the disease takes place over several years and can eventually lead to massive swelling and possible skin rupture.

The ECDC does not track infection rates of mycetoma. The WHO registered 78 deaths as being attributed to mycetoma within the EU27 under the ICD10 codes between 1997 and 2007 (including nocardial mycetoma).

2.4 Helminths

The name helminth refers to a group of parasitic worms which consist of a number of phyla, many of which are unrelated genetically, but bear superficial similarities. The four main phyla are Annelida, Platyhelminths, Nematoda, and Acanthocephala. The majority of helminths which infect humans are from the two phyla Platyhelminth and Nematoda.

Helminths are internal parasites, meaning that unlike external parasites such as fleas and lice, they live inside the host, generally inside the digestive tract where they can receive nutrients from food consumed by the host as it passes through the gut. This nutrient acquisition occurs in competition with nutrient uptake by the host's gut and so can lead to malnutrition as well as diseases in the host.

Not all helminths remain confined to the intestinal tract. In some instances larvae may penetrate the mucosa of the small intestine and invade the blood stream, and so migrate to other parts of the body. Some species may also enter the host through the skin, most commonly on the feet and ankles, and from there can be transported around the body. While the majority of helminth infections occur in less developed countries, particularly in areas of poor sanitation, and infection rates in Europe have been in decline and are minimal compared to decades or centuries ago, they are often soil transmitted and so fall well within the scope of this report. For this reason, a more in depth discussion is given for each group below.

2.4.1 Ascariasis

The causative agent of ascariasis is *Ascaris lumbricoides* which is the largest intestinal nematode (nematodes are also known as roundworms) and is the most common form of human helminth infection worldwide. Along with hookworms and whipworms, *Ascaris* make up a group known as soil-transmitted helminths.

Ascariasis is most common in tropical and sub-tropical areas and is generally associated with poor sanitation. *A. lumbricoides* are capable of reaching more than 30 cm in length, which is more than two orders of magnitude greater than most nematodes which are found in the soil. This disparity in size is due to the relatively rich conditions with regard to nutrient availability, as well as lack of predation which occurs in the human intestine when compared to the soil environment.

The life cycle of *Ascaris* involves eggs from an infected individual being excreted along with faeces which may contaminate the soil if a person defecates outside, or if the faeces of an infected individual are used as a fertilizer. Ascariasis is then spread when a non-infected individual ingests eggs from the soil, either through fingers which have come into contact with contaminated soil being put into the mouth, or by eating fruit or vegetables which have come into contact with the infected soil which have not been adequately washed, cooked or peeled.

Ascariasis may present with no symptoms in some individuals. However, other individuals may develop a compromised nutritional status, abdominal pains, low grade fever, and in the case of more heavy infection, intestinal blockage or rectal prolapse. In some instances, larvae may penetrate the wall of the intestine and enter the blood stream where they can migrate to other parts of the body, most commonly the lungs, where they cause a form of the diseases known as ascaris pneumonitis. In the lung, the nematodes can cause inflammation and haemorrhaging and can increase the propensity for the individual to contract bacterial lung infections.

The ECDC does not track ascariasis infection within the EU27. The WHO has 8 deaths registered under the ICD 10 codes as having occurred within the EU between 1994 and 2006.

2.4.2 Echinococcosis

Echinococcosis is caused by parasitic plathyhelminthes from the genus *Echinococcus*. Different species of this genus are associated with different forms of the disease. The life cycle of these parasitic worms involve several different hosts, each of which is associated with a different stage in the life cycle of the worm and transmission between which may involve the soil.

Carnivores are the definitive hosts of the worm, with foxes being the predominant definitive hosts of the species *E. multicularis* within Europe (rodents generally act as intermediate hosts). Infection with larvae of this species causes alveolar echinococcosis which resembles a slow growing malignant tumour. It originates in the liver and may spread to other organs through metastases, and is lethal without treatment (Vuitton 2010).

Human infection occurs through contact with egg-containing faeces, soil or plants and occurs through hand to mouth transfer (Eckert and Deplazes 2004). The eggs have been found to be easily killed as high temperatures but highly resistant to cold, as well as all known antiseptics (Scott et al. 2011) and soil moisture has been found to play a key role in the survival times of eggs within soil (Hansen et al. 2003).

The disease presents with abdominal pain in the upper right quadrant in approximately 30% of cases which may last for several years before lesions develop. Jaundice develops over time and is present in 20% of cases with inflation of the liver occurring in 16% of cases (Vuitton 2010). Other symptoms may include bone pain, skin tumours, shortness of breath, fever and possible gallstone like symptoms. The ECDC registered between 966 cases (2007) and 789 cases (2009), although data is unavailable for infection rates in Denmark and Italy. The WHO registered 597 mortalities as attributed to echinococcosis of various forms (e.g. *Echinococcus granulosus* infection of lung or liver or echinococcosis, unspecified, of liver) under the ICD10 codes in the EU 27 between 1997 and 2008. Information concerning mean annual infection rates per country can be seen in Fig 16.

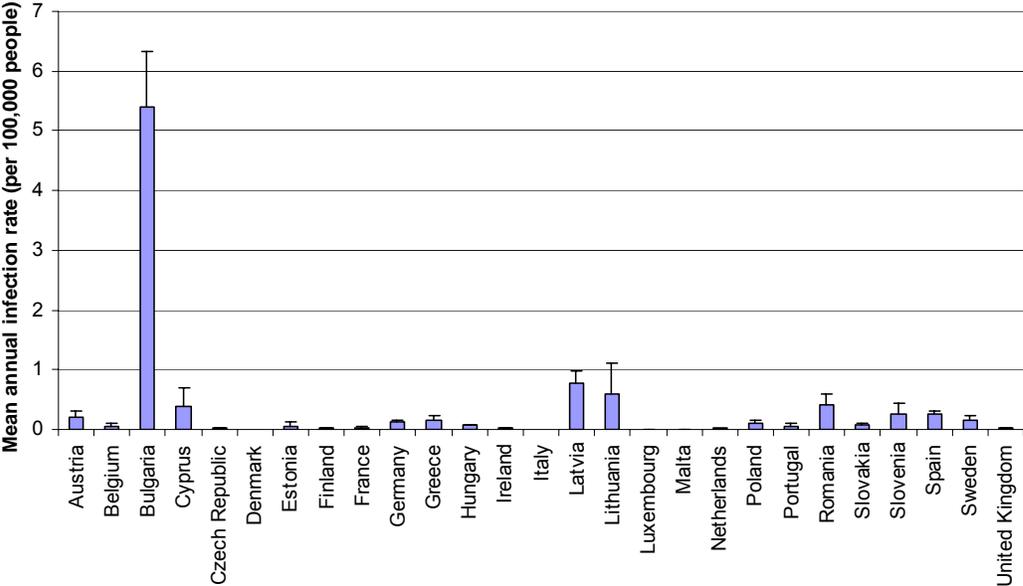


Fig 16: Mean annual infection rates of echinococcosis in the EU27 by member state per 100,000 people for 2006 - 2009. Bars show standard error. Data not available for Denmark, Italy or The Netherlands.

2.4.3 Hookworm

Two species of parasitic nematodes which infect humans are known as hookworms. These are *Ancylostoma duodenale* and *Necator americanus*. Both species are found throughout the tropics and sub tropics, with overlapping distributions, although *A. duodenale* is most common in North Africa, India and the Middle East, and formerly in southern Europe. *N. americanus* on the other hand, is most common in the Americas, Sub-Saharan Africa, Indonesia, China and Southeast Asia. Both species of hookworm combined are estimated to infect more than 740 million people globally (Tam et al. 2011). Hookworms are considerably smaller than *Ascaris lumbricoides* which is also a species of nematode (Section 2.4.1). This means that the risk of intestinal obstruction which may occur with *A. lumbricoides* is much less with hookworms. The most significant risk of hookworm infection is anaemia due to loss of iron from the gut, the uptake of which is competitive between the hookworms and their host.

Humans are the only known reservoir for hookworms. Infection occurs through contact with soil which has been contaminated with faeces. Larvae are able to penetrate the skin, usually through the feet which may cause a stinging or burning sensation. The larvae then migrate to the gut of their host. Infection may also occur through ingestion of eggs or larvae, through the hand to mouth route after touching infected soil or water.

Infection rates can be as high as 80% in lesser developed countries in the most tropics, but is only 10-20% in drier climates, probably owing to the relatively poor survivability of the eggs and larvae in dry soils. Particle size distribution of soils and organic matter content have also been shown to play an important role in the survival of hookworm larvae in soils (Mabaso et al. 2004) possibly due to their impact on soil water retention.

The ECDC does not track infection by hookworms within the EU27, although cases of infection are likely to be minimal if any. The WHO has 1 death registered as attributed to hookworm disease under the ICD 10 codes which occurred in France in 2003. However, it seems highly likely that the disease itself was contracted outside of France.

2.4.4 Strongyloidiasis

The causative agents of strongyloidiasis are two species of parasitic nematode from the genus *Strongyloides* (also known as threadworms), *S. stercoralis* which is the most common of the two which cause infection in humans, and *S. fulluborni* which is mainly found in Papua New Guinea and some areas of Africa. Many hosts infected with *Strongyloides* remain asymptomatic although the disease may cause life threatening infections in immuno-compromised hosts with a 60-85% mortality rate (Asdomongkol et al. 2006).

Infection with *Strongyloides* may be as high as 40% in some areas where soil is moist and sanitation is poor.

Strongyloides can survive for extended periods in the soil owing to the fact that it has two types of life cycles. One of these is a free-living life cycle, meaning the organisms can survive for generations and so persist indefinitely in soil. However, infection may be transmitted to humans when a person comes into contact with soil containing *Strongyloides* in their larval “filariform” stage. These larvae can enter a host through the skin, where they enter the circulatory system via the lymphatic system and are carried to the lungs. Once in the lungs they migrate upwards to the throat where they are then swallowed and enter the digestive tract. The infected host may be asymptomatic for this entire time, with the exception of hives at the point of entry which may be mistaken for insect bites. Once in the lower tract of the digestive system, *Strongyloides* may be excreted in faeces. The time taken from the initial penetration of the skin, through migration to the lungs and back down into the intestine to be passed out in faeces once more is in the range of 17-28 days. *Strongyloides* are the only helminth to secrete larvae and not eggs in faeces (Chandrasekar et al. 2011).

The ECDC does not track infection by hookworms within the EU27, although cases of infection, if any, are likely to be minimal. The WHO has 7 deaths registered as attributed to strongyloidiasis within the EU27 between 1998 and 2006.

2.4.5 Trichuriasis

The causative agent of trichuriasis is *Trichuris trichiura*, also known as whipworm which is a very common intestinal parasite with approximately one quarter of the world's population being through to carry the nematode. The name "whipworm" is due to their shape as they have a wider posterior end with a longer, thinner anterior end which means that they can look like a whip with a handle.

Infection occurs through ingestion of eggs from soil in areas of poor sanitation. Eggs which are deposited onto the soil become embryonated after 10 – 14 days in the soil which is the infective stage of their life cycle (Donkor and Lundberg 2011). Once ingested, the larvae develop within the small intestine before passing into the cecum where they penetrate the mucosa before going on to complete their development into adult worms in the large intestine. Adult worms bury their thin, threadlike anterior parts into the intestinal mucosa and feed on tissue secretions (not including blood).

Approximately three months is needed for eggs to develop into mature worms which may live for between 1 and 5 years. Adult females can lay eggs for up to 5 years and may shed up to 20,000 eggs per day which can survive for up to 14 days in soil (Acha and Szyfres 2003).

T. trichiura is often found in association with other helminth infections which inhabit similar conditions such as *A. lumbricoides*.

Infections usually present asymptotically apart from in individuals infected with numerous worms. Symptoms, where present, include vague abdominal discomfort, stunted growth, nocturnal loose stools, possible dysentery, and in some case rectal prolapse.

Whipworm infection is estimated to affect nearly 25% of the world's population, with infection being more common in less developed countries. However, 2.2 million individuals are thought to be infected in the rural South East area of the United States (Donkor and Lundberg 2011).

The ECDC does not register data on infection rates of trichuriasis within the EU27. The WHO has 5 deaths registered as attributed to trichuriasis in the EU27, all of which occurred in Germany in 1998.

2.4.6 Trichinellosis

Trichinellosis is also known as trichinosis, the causative agents of which are nematodes from the genus *Trichinella*, most commonly *T. spiralis*. Trichinellosis is a zoonotic illness which is spread through pigs via various pork products if not cooked sufficiently. *Trichinella* are not usually transmitted through the soil; rather infection is spread through encysted worms in infected muscle tissue is eaten by carnivores, mainly pigs, rats or humans. However, a theoretical mechanism in which encysted worms are excreted into the soil in faeces and then ingested by individuals who may come into contact with the soil may be possible, in a similar way to the other helminths discussed in this report.

However, data on the possibility of this mechanism or the survivability of *Trichinella* within the soil is not currently available in the literature.

The disease is of some clinical importance within the EU27. Between 688 and 787 cases of trichinellosis are registered with the ECDC as occurring within the EU 27 between 2006 and 2009 (except for Denmark for which no data is available). The majority of cases within the EU 27 were in Romania (75% of cases in 2008) or Bulgaria (54% of cases in 2009). The WHO has 14 deaths registered as attributed to trichinellosis in the EU27 between 1996 and 2005.

2.4.7 Other Helminth infections

Schistosomiasis, caused by platyhelminthes of the genus *Schistosoma*, and enterobiasis caused by *Enterobius vermicularis* (also known as pinworms) are not discussed within this report. This is because while there is a theoretical possibility of them being transmitted by the soil the means of infection is overwhelmingly aquatic in the case of Schistosomiasis or from person to person contact in the case of enterobiasis that they do not fall within the scope of this report.

2.5 Prions

Prion diseases are caused by specific, mis-folded proteins which can function as infective agents. They propagate by transmitting the mis-folded state and thereby converting properly folded proteins to the diseases associated prion form. Of the five main prion diseases which have been identified to occur in humans: Creutzfeldt-Jakob Disease (CJD); Variant Creutzfeldt-Jakob Disease (vCJD); Kuru; Fatal familial insomnia; Gerstmann-Straussler-Scheinker Syndrome, it seems most probable that only CJD and vCJD are possible to be transmitted through the soil. This is because Fatal familial insomnia; Gerstmann-Straussler-Scheinker Syndrome both seem to require at least a genetic predisposition to the disease if the disease is not hereditary, and Kuru is associated with cannibalism and so is not currently associated with transmission from the soil. For this reason on CJD and vCJD are discussed in this report although it should be noted that the soil as a route of transmission is currently hypothetical and further research is needed to confirm or reject this hypothesis.

Prions have been shown to be spread orally, and to be able to cause disease in individuals who ingest prions, in humans in the case of Kuru, and from the environment in the case of deer (Balchandran et al. 2010). As to whether humans can contract a prion disease straight from the environment (e.g. through the ingestion of prions from contaminated soil or dust) is not yet known. However, prions have been shown to survive for several years in the soil (Brown and Gajdusek 1991), and that binding of prions to montmorillonite, a common soil mineral, can increase the oral transmissibility of prion disease (Johnson et al. 2007). However, as the infectious dose of prions necessary to cause disease via oral transmission is unclear further research is necessary to elucidate whether this means of horizontal transmission of prion disease does, or may occur in humans.

Some preliminary evidence suggests that control of the longevity of prions within soil may be possible, although difficult at large scales. Nachitaylo et al. (2010) demonstrated that some soil enzymes are capable of increasing the rate of degradation of prions within soil, but that proteolytic enzymes which are capable of degrading prions in the soil are inhibited by aprotinin and leupeptin, two organic compounds which are often produced and released into the environment by the soil microbiota. If a method of reducing the production of these compounds within the soil could be found it is possible that the longevity of prions within the soil may be reduced.

3. Mapping of soil borne diseases

Several efforts have been made to produce maps of diseases incidence to aid spatial analysis and so help identify underlying mechanisms of various diseases. The first attempt at mapping diseases at a global scale appears to have been made by Finke in 1792, although this manuscript appears to have been lost or destroyed (Barrett 2000). Further attempts were made throughout the 19th century and through the 20th Century (Abrahams 2006) and include both national (Howe 1963) and international (May 1952) attempts at the production of maps showing the incidence of diseases.

In order to fully assess the possible impacts of factors such as climate, land management practices, the impact of livestock etc, a high resolution database logging the incidence of infection of a soil borne disease, as identified in this report, would be highly advantageous for allowing the testing of hypotheses, for example by comparing the incidence of soil borne disease such as aspergillosis, the spread of which may be exacerbated by tillage of the soil causing spores to be released into the air. By comparing areas where conventional tillage is prevalent with other areas where min or conservation tillage are prevalent the testing of the hypothesis that conventional tillage leads to increased incidence of aspergillosis would be testable. Clearly this would need a high resolution database, pulling together information on infections as identified by hospitals on a local level, ideally from several if not all

member states of the EU27 in order for the interactions with different land management practices under different climatic conditions and at different latitudes to be tested.

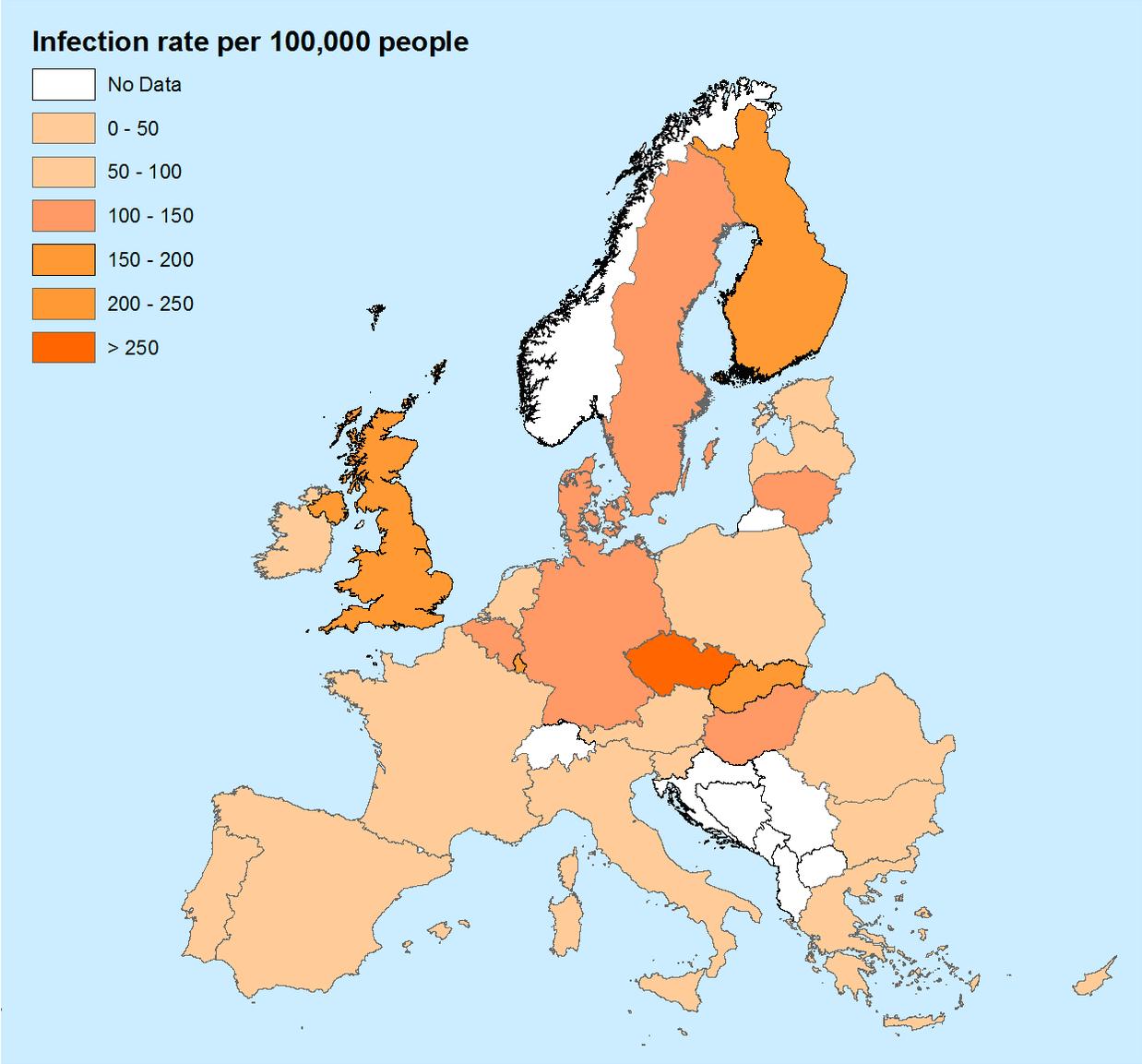


Fig 17. Total number of infections of soil borne human diseases for which data is available through TESSy for the EU27, summed over the years 2006-2009. It should be noted that not all member states have provided data on infection rates for each of the soil borne diseases discussed in this report. This greatly limits analysis as data sets are incomplete meaning that inevitably some countries are shown with underreported infection rates.

In an ideal situation it would be possible to track the incidence of each of the soil borne human diseases discussed in this report at, at least the country level, annually, although a higher resolution of spatial data would be preferable. This would facilitate the monitoring of changes in disease incidence to possibly allow an investigation into the mechanisms behind such changes in disease incidence.

4. Discussion

The fact that not all countries implemented the 10th revision of the ICD codes at the same time means that information concerning date rates is not the same for all periods of time, for each country within the EU for which data are available. For this reason, the span of time for which mortalities are reported for each disease are always given along with the time span over which they occurred, which varies from disease to disease. Amalgamation of the different ICD coded WHO databases, if possible, would overcome this weakness and allow more data to be included in analyses as well as historical data to allow the tracking of changes in incidence of soil borne diseases over longer time frames than the 10-12 years which is currently possible.

Comparison between the TESSy database on infection rates by country and the WHO mortality database can not currently be made with a sufficiently high level of confidence to allow the undertaking of statistical analyses, such as calculating the mortality rate as a function of the number of infections for each soil borne disease for which data is available. This is because differences exist in the methods by which data is collected. For example, as the WHO mortality database uses data obtained from death certificates only one cause of death is usually given. This means other underlying illness may have not been reported. Furthermore, those mortalities which have been attributed to a soil borne disease may have been contracted outside of the EU27 and the initial infection not logged. Therefore, it cannot be said with confidence that of the X number of individuals reported as having been infected with a soil borne diseases, as registered in TESSy, Y number of them died as a result of the disease as registered in the WHO mortality database. While this is not currently possible with the data sets available and used in the construction of this report, this is clearly an aim which would be worthwhile achieving to allow for the identification of diseases hot spots where mortality rates are higher which can then help guide resource allocation or prompt further research as to why survival rates after infection vary.

The resolution of the information provided within the WHO mortality database is not always sufficiently high to allow effective analysis to be undertaken. For example, ICD A09 is listed as “Diarrhoea and gastroenteritis of presumed infectious origin”. As will become clear in the following discussion of symptoms attributed to each soil borne disease, there are many which may lead to death through this mechanism. This means that any analysis of diseases which may include this as a symptom may under-represent the mortality rate owing to the fact that the underlying disease responsible for these deaths cannot be identified.

With data concerning infection rates, one further weakness in the data with regard to analyses which can be undertaken for this report is that the source of the infection is unknown. While all of the diseases discussed within this report are capable of being caught from or transmitted through the soil, other means of infection are also possible in many cases, such contaminated food or water or possibly even person to person transmission.

Another problem with the data which is currently available is the lack of spatial resolution. There is currently no easy way to access data on infection rates across Europe at anything other than the country scale. This means that it is not possible to model as to whether various types of soil borne disease and more associated with agricultural regions, or other predominant land types/uses.

4.1 Possible Factors affecting SBDs

4.1.1 Land use, change and management

Several of the diseases outlined and discussed in Section 2 are capable of transmission from the soil by become airborne, for example in dust (e.g. Q Fever, aspergillosis, tularemia, sporotrichosis). These diseases are therefore likely to be those most directly affected by land management practices and land use change. For example, any activity which is associated with increased wind erosion, on which there is abundant information in the primary research literature, seem likely to increase the incidence of these diseases in the surrounding area. Such activities could include land use change, for example by

ploughing grassland to make arable land, ploughing or tilling soil that is too dry, etc. These conditions may also increasingly occur under climate change, as that can lead to enhanced incidence of drought periods, besides periods with heavy rainfall. These conditions can be enhanced by more intense soil use e.g. when converting most primary production into biofuels, which reduces soil organic matter status and therefore erosion susceptibility. Reduced soil tillage could be part of a solution, however, no-till systems are usually associated with increased soil moisture and increased temperatures as well as increased crop residue, which might enhance the incidence of crop diseases, especially in short rotation cropping systems. The effects that these conditions will have on the abundance of the soil borne diseases identified by this report are not always clear, although the majority of the organisms discussed survive for longer periods of time in moist soil compared to dry soil.

4.1.2 Use of pesticides and herbicides

Some anecdotal evidence suggests that herbicides, particularly glyphosate, can reduce the abundance of neutral or beneficial soil organisms and so remove the biocontrol mechanisms which may help repress diseases causing organisms within the soil through factors such as competition for resources (Smith 2011). For example a 500% increase in *Fusarium* sp., a fungus, was noted in the roots of Roundup Ready soybean (Smith 2011). However, it should be noted that the information presented in this report is only anecdotal and no studies on this currently appear in the primary literature.

Toxins produced by *Fusarium* sp. are suspected of being able to cause disease in humans (European Commission 1999). For example, the soil-borne plant pathogen *Fusarium culmorum* can cause marram grass (*Ammophila arenaria*) planter's disease, particularly when the plant material had been stored under conditions favourable for fungal multiplication. The disease is not deadly, but can cause severe inconvenience to the planters (Snijders et al 1996). There may be many more such examples with non-lethal consequences, but severe inconvenience with associated socio-economic costs, and is an area requiring further research to investigate.

In the case of Roundup Ready soybean, research is necessary to investigate firstly, whether glyphosate does lead to increases in diseases causing organisms in crop roots, and secondly whether toxins produced by these organisms, if they do exist at increased abundance, can be transferred to humans in sufficient concentrations to cause disease. The consequences of Genetically Modified Organisms (GMOs) especially in relation to modified crops may also need to be evaluated from the perspective of their influence on soil borne human diseases.

4.1.3 Use of antibiotics

Antibiotics are often used in livestock, both prophylactically and to cure diseases. They have been shown repeatedly to be able to survive passing through the animals and for 30-90% of the antibiotic to be excreted via urine or faeces as intact bioactive substances (USCDC 2011). Once released into the environment, the fate of the antibiotic varies depending on the type of antibiotic, its chemical structure etc. but has been shown to be capable of leading to increased resistance to antibiotics within soil microbial communities (Heuer et al. 2010).

A full review is available on the movement of veterinary antibiotics in soils which can be highly variable depending on their molecular structure and atomic composition (Tolls 2001). Individual studies have investigated ways to restrict the mobility and control the fate of veterinary antibiotics in the environment. Heuer et al (2010) found that agroforestry and grass buffer systems have the potential to mitigate the spread of veterinary antibiotics from agricultural systems, and that root exudates, by enhancing microbial growth and potential degradation of the antibiotics through provision of an additional carbon source, may reduce the longevity of antibiotics introduced into the environment. However, as shifts in microbial community characteristics may occur depending on which species are sensitive to antibiotics and so are suppressed; predictions of the likely effects of antibiotics on the soil community are problematic. However, the fact that antibiotics can affect soil microbial communities and enzyme activities (Liu et al 2009) shows that further to increasing the possibility of antibiotic resistance developing and spreading, effects on soil fertility may also occur.

From the viewpoint of soil borne diseases of humans, when such veterinary antibiotics enter the environment they can be expected to place an evolutionary pressure on the communities with which they come into contact. In the soil, these communities are generally microbial and it can be expected that the evolutionary pressure placed via antibiotics on soil microbial communities is towards an increase in resistance to that antibiotic.

Once antibiotic resistance has developed within a community, horizontal gene transfer which occurs frequently between bacterial species in the environment means that the resistance can spread and while it may not initially develop in an organism which is capable of causing disease in humans, it may be passed to such organisms (Allen et al 2010). This has been shown to have occurred with regard to tetracycline resistance which was found to develop in an aquaculture environment but which was then passed on to a human pathogenic bacteria (Rhodes et al. 2000). It seems likely therefore that the same has happened, or will happen within the soil environment leading to antibiotic resistance in other human pathogenic bacteria. Steps should be urgently taken to investigate how common such occurrences are and which steps can be taken to minimise the development of antibiotic resistance through such mechanisms.

4.1.4 Climate change

Prediction of the effects of climate change on the distribution of diseases causing organisms is problematic owing to uncertainties regarding the effects of climate change on climatic conditions at the local scale. However, it is notable that many soil borne diseases have relatively limited distributions across the world, with the majority of cases being restricted to the tropics or sub-tropics in some instances (e.g. Isoporiasis, Ascariasis, hookworms), or highly localised as for the fungus *Coccidioides immitis* which is endemic to the San Joaquin valley region of California. Changes in weather patterns will lead to changes in their associated ecoregions and in instances where these changes favour soil borne pathogens or parasites of humans these diseases are likely to spread.

Further to this, changes in weather patterns may affect diseases incidence in areas where diseases causing organisms are endemic. For example, increased rainfall leads to higher soil moisture levels which have been shown to enhance the longevity of STPs (Welshimer 1960; Chandler and Carven 1980; Chao et al 1988). Conversely, drier soils can lead to increased wind erosion increasing the chances of inhaling soil particles from the air which be contaminated with disease causing organisms as well as of inhaling spores of infectious organisms.

One large scale effect of climate change for which some evidence already exists is the apparent “migration towards the poles” of organisms where by the species range of several groups of organisms have been found to be spreading towards the poles. This has raised concern with the possible spread of malaria to previously malaria free areas owing to the migration towards the poles of malaria carrying mosquito species (e.g. Martens et al. 1999). The majority of soil transmitted helminth diseases occur in the tropics and sub-tropics and this range may well increase as a result of global warming. A further example is mycetoma, a fungal disease which is currently most common between latitudes of 15°S and 30°N; this may change with climate change.

Investigations as to which mechanisms are responsible for the limited species range of some soil borne diseases causing organism, which are likely to be a combination of climatic effects and soil physical, chemical and biological properties, may aid in predicting the likely effects of climate change on the species range of diseases causing organisms. Current information available in the literature suggests that changes in weather patterns which lead to moister soils may mean SBDs can survive for longer in new places, whereas changes in weather patterns which lead to drier soils may mean more incidences of SBDs which can be inhaled.

5. Recommendations

This report is only a first step towards a thorough investigation of this relatively understudied area and only contains a relatively limited analysis of the data that is available on this matter. The logical next step following this report would be to put together an expert working group covering aspects such as epidemiology, medicine, statistics, soil biology, database management and GIS, and economics. This cross-disciplinary working group would allow the production of a comprehensive reference report to the European Commission containing analysis of factors such as the socio-economic impacts of soil borne diseases within the EU27. Furthermore a cross member-state analysis of diseases incidence versus mortality would allow the identification of member states with the highest survival rates for soil borne diseases and may help provide insights into treatment efficacy etc.

Such a group would allow the identification of questions which may already be answerable with the current data sets and to present such answers in the reference report. For example, an economical analysis of the cost of soil borne diseases to the European economy may be able to be undertaken with the information available on incidence of diseases within the EU27, accounting for both working days missed and the cost of treating infections. This exercise would also help quantify the socioeconomic effects of soil borne diseases. A sensitivity analysis of the data currently available would also provide a measure of the confidence at which any result obtained from the current data can be accepted.

Data currently available with the ECDC are incomplete with not all member states providing figures on infection rates for all diseases for each year. This greatly limits the analyses that can be done and so inhibits investigation in to such factors as whether factors such as climate, latitude, land use etc. currently have any effect on disease incidence and whether such relationships are changing over time, in response to land use or climate change for example.

Inclusion of colleagues from the ECDC and WHO in the working group would allow monitoring and discussion of data acquisition techniques and possible synergising of information available in data bases. This will increase the confidence with which comparisons of infection rates versus mortality rates for each disease can be made to give an indirect measure of survival rates. Analysis of differences between survival rates in member states may allow increased understanding of the most efficient ways to treat soil borne diseases.

An expert working group would allow the identification of soil borne diseases which have the potential to cause a similar impact with regard to health and socio-economic factors as the EHEC strain 0104:H4 which caused the outbreak in Europe in 2011. These should be considered from the viewpoint of current changes in land/soil use and climate change and steps identified in a range of scenarios to help contain any such outbreak and to minimise the socio-economic and health impacts of such outbreaks.

Georeferenced information at a high spatial resolution, ideally individual hospitals where diagnosis occurs, would allow the mapping of areas at increased risk of various soil borne diseases. Furthermore, it would then be possible to combine this information with that available from Corine Land Cover to investigate which land use types are most closely correlated with disease incidence as well as investigating the effects of land use change over time.

While this is not currently possible with the data sets available and used in the construction of this report, this is clearly an aim which would be worthwhile achieving to allow for the identification of diseases hot spots where infection rates are higher, as well as identification of areas where human survival rates after infection may be lower. This can then help guide resource allocation or prompt further research as to why survival rates after infection vary.

Discussion of the utility of sub-groupings of soil borne diseases further to the initial step made in this report could be undertaken from an inter-disciplinary point of view. For example, with the current groupings of ESPs and STP will allow some further investigation of the data available allowing for the fact that ESPs can be said with confidence to have been obtained from the soil whereas STPs cannot. This is important information for interpreting the data and any analyses. However, it is possible that from a socio-economic viewpoint a different division of the diseases may be useful, for example into occupational versus non-occupational. A multi-disciplinary team is necessary to have the required expertise to allow assessment of soil borne diseases from a range of viewpoints.

Finally the working group would be able to identify and priorities areas of our understanding soil borne diseases which are currently lacking and require further (experimental) investigation and so would aid in resource funding allocation. For example, investigations as to which mechanisms are responsible for the limited species range of some soil borne diseases causing organism, which are likely to be a combination of climatic effects and soil physical, chemical and biological properties, may aid in predicting the likely effects of climate change on the species range of diseases causing organisms.

6. Conclusions

In conclusion, this report is now able to answer the questions posed in Section 1.1.

1. Which diseases are, or can be soil borne?

A range of diseases have been identified in Table 1 showing that soil borne diseases can be caused by a wide range of organisms coming from disparate taxonomic groups which vary greatly in their virulence and prognoses. Soil borne pathogens and parasites can be split into two groups; those that reside in the soil as true soil organisms which can potentially cause diseases in humans, the euedaphic pathogenic organisms (EPOs); and soil transmitted pathogens (STPs) which are capable of surviving for extended periods in soil and so can cause infection even when there are no other infectious humans in the vicinity.

2. Is there anything that we can do with regard to land management practices or other precautions that we can take which will help control the incidence of soil borne diseases?

Some soil borne diseases have been shown to have higher incidence in people of some occupations, such as farmers, as well as to be affected by climatic, environmental and anthropogenic factors. Steps should be taken to identify which factors have the largest effect on disease incidence for each disease and so allow for measures to be taken or policy to be written to minimise the destructive effects and socioeconomic costs of soil borne diseases.

3. Are there any human activities that enhance the risk of soil borne diseases?

Evidence suggests that human activities which increase soil degradation processes can enhance soil borne disease incidence. For example, soil degradation or poor land management leading to increased wind erosion can lead to concurrent increases in some soil borne diseases owing to the increased abundance of fungal and bacterial spores in the air. Steps taken to reduce soil degradation processes and are likely to have concurrent benefits in some areas with a reduction in incidence of soil borne diseases. Other factors, like intensified carbon extraction from production systems for biofuel production, can be predicted to influence soil borne disease incidence, for example because lowered soil organic matter may enhance soil susceptibility to drying out and eroding, thereby spreading soil borne diseases. This needs further study.

Finally, a key recommendation of this report is the formation of an expert working group on soil borne diseases. This would allow working towards identifying practices in land management practices which minimise the impacts of soil borne diseases, both in terms of human health and socio-economics factors. A concerted effort by an inter-disciplinary team would allow the identification of future risks of outbreaks or organisms such as EHEC strain 0104:H4 which caused the outbreak in Europe in 2011 and which proactive steps can be taken to minimise the chances of an outbreak and to ensure the impacts are minimal should one occur. Furthermore, the construction of an integrated database made available to the expert working group and pooling information on mortalities from across WHO ICD codes, combined with information on soil borne disease infection rates available from the ECDC would allow monitoring of treatment efficacy in different member states, highlight areas where diseases are of particular risk and how these risks are changing over time.

8. Acknowledgements

Data concerning mortalities used in this report are publically available from the WHO. However, we would like to acknowledge the helpful advice and information that we received regarding data availability from Dr. Michal Krzyzanowski, Dr Andrey Egorov, and Dr. Antonio Montresor.

We would also like to acknowledge the TESSy team of the ECDC for helping with data acquisition and interpretation, particularly Dr. Sergio Brusin for his help and advice.

The work carried out for this study contributes to EU-funded research Ecofinders (ENV.2010.2.1.4-4).

Finally we would like to acknowledge and thank Prof. Diana H. Wall for initiating and encouraging interest in this issue, and Dr. Luca Montanarella for making this report possible.

9. References

- Abd H., Johansson T., Golovliov I., Sandström G. & Forsman M. (2003) Survival and Growth of *Francisella tularensis* in *Acanthamoeba castellanii*. *Appl. Environ. Microbiol.*, 69, 600-606
- Abrahams P.W. (2006) Soil, geography and human disease: a critical review of the importance of medical cartography. *Progress in Physical Geography*, 30, 490-512
- Acha P.N. & Szyfres B. (2003) Trichuriasis of Animal Origin. . In: *Zoonoses and Communicable Diseases Common to Man and Animals*, pp. 302-304. Pan American Health Organisation, Washington D.C.
- Afonso E., Lemoine M., Poulle M.L., Ravat M.C., Romand S., Thuillez P., Villena I., Aubert D., Rabilloud M., riche B. & Gilot-Fromont E. (2008) Spatial distribution of soil contaminated by *Toxoplasma gondii* in relation to cat defecation behaviour in an urban area. *International Journal for Parasitology*, 38, 1017-1023
- Agent E. (2005) Leptospirosis
- Allen H.K., Donato J., Wang H.H., Cloud-Hansen K.A., Davies J. & Handelsman J. (2010) Call of the wild: antibiotic resistance genes in natural environments. *Nat Rev Micro*, 8, 251-259
- Ania B.J., Ansenjo M. & Kiel R.J. (2008) Mycetoma. URL <http://emedicine.medscape.com/article/211459-overview>
- Anonymous (2009) Hookworm Infections (*Ancylostoma duodenale* and *Necator americanus*). *Red Book*, 2009, 375-376
- Asdamongkol N., Pornsuriyasak P. & Sungkanuparph S. (2006) Risk factors for strongyloidiasis hyperinfection and clinical outcomes. *Southeast Asian Journal of Tropical Medicine and Public Health*, 37, 875-884
- Asgari N, Hahné S, Heiskanen-Kosma T, Siikamäki H & M M. (2001) Cases of coccidioidomycosis in Europe due to exposure in California, United States. *Eurosurveillance*, 5
- Avery L.M., Hill P., Killham K. & Jones D.L. (2004) *Escherichia coli* O157 survival following the surface and sub-surface application of human pathogen contaminated organic waste to soil. *Soil Biology and Biochemistry*, 36, 2101-2103
- Balachandran A., Harrington N.P., Algire J., Soutrine A., Spraker T.R., Jeffrey M., González L. & O'Rourke K.I. (2010) Experimental oral transmission of chronic wasting disease to red deer (*Cervus elaphus elaphus*): early detection and late stage distribution of protease-resistant prion protein. *The Canadian Veterinary Journal*, 51, 169-178
- Balcht A.L. & Smith R.P. (1994) *Pseudomonas aeruginosa: infections and treatment*. Marcel

- Dekker, New York.
- Baloda S.B., Christensen L. & Trajcevska S. (2001) Persistence of a *Salmonella enterica* Serovar Typhimurium DT12 Clone in a Piggery and in Agricultural Soil Amended with *Salmonella*-Contaminated Slurry. *Appl. Environ. Microbiol.*, 67, 2859-2862
- Bech T.B., Johnsen K., Dalsgaard A., Laegdsmand M., Jacobsen O.H. & Jacobsen C.S. (2010) Transport and Distribution of *Salmonella enterica* Serovar Typhimurium in Loamy and Sandy Soil Monoliths with Applied Liquid Manure. *Appl. Environ. Microbiol.*, 76, 710-714
- Becker J.U., Singh D. & Sinert R.H. (2010) Toxoplasmosis in Emergency Medicine Medication. URL <http://emedicine.medscape.com/article/787505-overview>
- Blackburn J.K., McNyset K.M., Curtis A. & Hugh-Jones M.E. (2007) Modeling the Geographic Distribution of *Bacillus anthracis*, the Causative Agent of Anthrax Disease, for the Contiguous United States using Predictive Ecologic Niche Modeling. *The American Journal of Tropical Medicine and Hygiene*, 77, 1103-1110
- Brown P. & Gajdusek D.C. (1991) Survival of scrapie virus after three years' interment. *Lancet*, 337, 296-270
- Bryan F.L. (1969) What the sanitarium should know about *Clostridium perfringens* foodborne illness. *Journal of Milk and Food Technology*, 32, 381-389
- Bystrom J. (2007) Anthrax - what you need to know. URL [http://www1.agric.gov.ab.ca/\\$department/deptdocs.nsf/all/agdex11466/\\$file/663-48.pdf?OpenElement](http://www1.agric.gov.ab.ca/$department/deptdocs.nsf/all/agdex11466/$file/663-48.pdf?OpenElement)
- Cabada M.M. & White J., A.C (2011) Cryptosporidiosis clinical presentation. URL <http://emedicine.medscape.com/article/215490-clinical>
- Carlson G.F.J., Woodard F.E., D.F W. & O.J. S. (1968) Virus inactivation on clay particles in natural waters. *Journal of the Water Pollution Control Federation*, 40, R89-R106
- Chacín-Bonilla L. (2008) Transmission of *Cyclospora cayetanensis* infection: a review focusing on soil-borne cyclosporiasis. *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 102, 215-216
- Chacín-Bonilla L. (2010) Epidemiology of *Cyclospora cayetanensis*: A review focusing in endemic areas. *Acta Tropica*, 115, 181-193
- Chandler D.S. & Craven J.A. (1980) Relationship of soil moisture to survival of *Escherichia coli* and *Salmonella typhimurium* in soils. *Crop and Pasture Science*, 31, 547-555
- Chao W.L., Ding R.J. & Chen R.S. (1988) Survival of *Yersinia enterocolitica* in the environment. *Canadian Journal of Microbiology*, 34, 753-756
- Commission E. (1999) Opinion on Fusarium Toxins. In: (ed. Directorate-General HaCP). European Commission
- Crum-Cianflone N.F. (2008) Mucromycosis treatment & management. URL <http://emedicine.medscape.com/article/222551-treatment#a1128>
- Dai X. & Boll J. (2006) Settling velocity of *Cryptosporidium parvum* and *Giardia lamblia*. *Water Research*, 40, 1321-1325
- de Jong J.G. (1965) The survival of measles virus in air, in relation to the epidemiology of measles. *Archives of Virology*, 16, 97-102
- de Lima Barros M.B., Schubach A.O., de Vasconcellos Carvalhaes de Oliveira R., Martins E.B., Teixeira J.L. & Wanke B. (2011) Treatment of Cutaneous Sporotrichosis With Itraconazole - study of 645 Patients. *Clinical Infectious Diseases*, 52, e200-e206
- DeSpain Smith L. & Gardner M.V. (1949) The occurrence of vegetative cells of *Clostridium perfringens* in soil. *Journal of Bacteriology*, 58, 407-408
- Diamond J.M. (1999) Evolutionary biology: Dirty eating for healthy living. *Nature*, 400, 120-121
- Donkor K.A.P. & Lundberg S. (2011) Trichuris Trichiura. URL <http://emedicine.medscape.com/article/788570-overview>
- Duboise S.M., Moore B.E. & Sagik B.P. (1976) Poliovirus survival and movement in a sandy

- forest soil. *Applied and Environmental Microbiology*, 31, 536-543
- Dugdale III D.C. & Vyas J.M. (2011) Tularemia. URL
<http://www.nlm.nih.gov/medlineplus/ency/article/000856.htm>
- Dugdale III D.C., Vyas J.M. & Zieve D.Z. (2008) Histoplasmosis. URL
<http://www.nlm.nih.gov/medlineplus/ency/article/001082.htm>
- Dugdale III D.C., Vyas J.M. & Zieve D.Z. (2011) Hantavirus
- Eckert J. & Deplazes P. (2004) Biological, Epidemiological, and Clinical Aspects of Echinococcosis, a Zoonosis of Increasing Concern. *Clin. Microbiol. Rev.*, 17, 107-135
- Fang W., Washington L. & Kumar N. (2007) Imaging Manifestations of Blastomycosis: A Pulmonary Infection with Potential Dissemination. *RadioGraphics*, 27, 641-655
- Fayyaz J. & Lessnau K.D. (2010) Histoplasmosis clinical presentation. URL
<http://emedicine.medscape.com/article/299054-clinical>
- Gessel P.D., Hansen N.C., Goyal S.M., Johnston L.J. & Webb J. (2004) Persistence of zoonotic pathogens in surface soil treated with different rates of liquid pig manure. *Applied Soil Ecology*, 25, 237-243
- Goldsmith R.S. (1997) Infectious diseases: protozoal & helminthic. In: *Current Medical Diagnosis and Treatment* (ed. al. MSe). Appleton & Lange, Stamford
- Gong C.M., Koichi I., Shunji I. & Takashi S. (2005) Survival of pathogenic bacteria in compost with special reference to *Escherichia coli*. *Journal of Environmental Sciences (China)*, 17, 770-774
- Grassl G.A. & Finlay B.B. (2008) Pathogenesis of enteric Salmonella infections. *Current Opinion in Gastroenterology*, 24, 22-26
- Greig J.D., Todd E.C.D., Bartleson C. & Michaels B. (2010) Infective doses and pathogen carriage. In: *2010 Food Safety Education Conference*, Atlanta, Georgia, USA
- Guerra M., Walker W., Jones C., Paskewitz S., Cortinas M.R., Stancil A., Beck L., Bobo M. & Kitron U. (2002) Predicting the Risk of Lyme Disease: Habitat Suitability for *Ixodes scapularis* in the North Central United States. *Emerging Infectious Diseases*, 8, 289-297
- Guo X., Chen J., Brackett R. & LR. B. (2002) Survival of Salmonella on tomatoes stored at high relative humidity, in soil, and on tomatoes in contact with soil. *Journal of Food Protection*, 65, 274-279
- Guzman J. (2010) Soil pathogen fate and transport: Biopores facilitating *Escherichia coli* transport. In: *Graduate College of Oklahoma State University*, p. 124. Oklahoma State University, Oklahoma
- Hansen F., Tackmann K., Jeltsch F., Wissel C. & Thulke H. (2003) Controlling *Echinococcus multilocularis* - ecological implications of field trials. *Preventive Veterinary Medicine*, 60, 91-105
- Hauser P.M., Burdet F.X. & M C.O.H.K.L.T.P.S.D.P. (2010) Comparative genomics suggests that the fungal pathogen *Pneumocystis* is an obligate parasite scavenging amino acids from its host's lungs. *PLoS ONE*, 5, e15152
- Hellstrom J.S. & Marshall R.B. (1978) Survival of *Leptospira interrogans* serovar pomona in an acidic soil under simulated New Zealand field conditions. *Research in Veterinary Science*, 25, 29-33
- Henry R.A. & Johnson R.C. (1978) Distribution of the genus *Leptospira* in soil and water. *Appl. Environ. Microbiol.*, 35, 492-499
- Heuer H., Binh C.T.T., Kopmann C., Zimmerling U., Krogerrecklenfort E. & Smalla K. (2010) Effects of veterinary medicines introduced via manure into soil microbial communities. *Interdisciplinary Studies on Environmental Chemistry - Biological Responses to Contaminants*, 9-13
- Hirulkar N.B. & Soni B. (2011) Incidence of Antibiotic-Resistant *Pseudomonas aeruginosa* Isolated from Drinking Water. *International Journal of Pharmaceutical & Biological Archives*, 2, 724-733

- Islam M., Morgan J., Doyle M.P., Phatak S.C., Millner P. & Jiang X. (2004) Persistence of *Salmonella enterica* serovar typhimurium on lettuce and parsley and in soils on which they were grown in fields treated with contaminated manure composts or irrigation water. *Foodborne pathogens and disease*, 1, 27-35
- Javid M.H. & Ahmed S.H. (2009) Campylobacter infections clinical presentation. URL <http://emedicine.medscape.com/article/213720-clinical>
- Johnson C.J., Pedersen J.A., Chappell R.J., McKenzie D. & Aiken J.M. (2007) Oral Transmissibility of Prion Disease Is Enhanced by Binding to Soil Particles. *PLoS Pathog*, 3, e93
- Johnson C.J., Pedersen J.A., Chappell R.J., McKenzie D. & Aiken J.M. (2007) Oral Transmissibility of Prion Disease Is Enhanced by Binding to Soil Particles. *PLoS Pathog*, 3, e93
- Johnson C.J., Phillips K.E., Schramm P.T., McKenzie D., Aiken J.M. & Pedersen J.A. (2006) Prions Adhere to Soil Minerals and Remain Infectious. *PLoS Pathog*, 2, e32
- Jones R.M., Hertwig S., Pitman J., Vipond R., Asp n A., B llske G.r., McCaughey C., McKenna J.P., van Rotterdam B.J., de Bruin A., Ruuls R., Buijs R., Roest H.-J. & Sawyer J. (2011) Interlaboratory Comparison of Real-Time Polymerase Chain Reaction Methods to Detect *Coxiella burnetii*, The Causative Agent of Q Fever. *Journal of Veterinary Diagnostic Investigation*, 23, 108-111
- Jonsson C.B., Figueiredo L.T.M. & Vapalahti O. (2010) A Global Perspective on Hantavirus Ecology, Epidemiology, and Disease. *Clin. Microbiol. Rev.*, 23, 412-441
- Karkowska-Kuleta J., Rapala-Kozik M. & Kozik A. (2009) Fungi pathogenic to humans: molecular bases of virulence of *Candida albicans*, *Cryptococcus neoformans* and *Aspergillus fumigatus*. *Acta Biochimica Polonica*, 56, 211-224
- Kauffman C.A. (2007) Histoplasmosis: a clinical and laboratory update. *Clin. Microbiol. Rev.*, 20, 115-132
- Kayser F.H., Bienz K.A., Eckert J. & Zinkernagel R.M. (2005) *Medical Microbiology*. Thieme, Stuttgart, Germany.
- Klich M.A. (2002) Biogeography of *Aspergillus* species in soil and litter. *Mycologia*, 94, 21-27
- Klotchko A. & Wallace M.R. (2011) Salmonellosis clinical presentation. URL <http://emedicine.medscape.com/article/228174-clinical#a0218>
- Landry E.F., Vaughn J.M., McHarrell T.Z. & Beckwith C.A. (1979) Adsorption of enteroviruses to soil cores and their subsequent elution by artificial rainwater. *Appl. Environ. Microbiol.*, 38, 680-687
- Leonardopoulos J., Papakonstantinou A., Kourti H. & Papavassiliou J. (1980) Survival of shigellae in soil. *Zentralbl Bakteriol Mikrobiol Hyg B.*, 171, 459-465
- Lindsay D.S., Blagburn B.L. & Dubey J.P. (2002) Survival of nonsporulated *Toxoplasma gondii* oocysts under refrigerator conditions. *Veterinary Parasitology*, 103, 309-313
- Liu F., Ying G.G., Tao R., Zhao J.L., Yang J.F. & Zhao L.F. (2009) Effects of six selected antibiotics on plant growth and soil microbial and enzymatic activities. *Environmental Pollution*, 157, 1636-1642
- Low C.Y. & Rotstein C. (2011) Emerging fungal infections in immunocompromised patients. *F1000 Med. Reports*, 3
- Mabaso M.L., Appleton C.C., Hughes J.C. & Gouws E. (2004) Hookworm (*Necator americanus*) transmission in inland areas of sandy soils in KwaZulu-Natal, South Africa. *Tropical Medicine & International Health*, 9, 471-476
- Martens, P., Kovates, R.S., Nijhof, S., de Vries, P., Livermore, M.T.J., Bradley, D.J., Cox, J. & McMichael, A.J. (1999) Climate change and future populations at risk of malaria. *Global Environmental Change*, 9, 1, S89-S107
- Matches J.R., Liston J. & Curran D. (1974) *Clostridium perfringens* in the environment. *Applied Microbiology*, 28, 655-660
- Mawdsley J.L., Brooks A.E. & Merry R.J. (1996) Movement of the protozoan pathogen

- Cryptosporidium parvum* through three contrasting soil types. *Biological Fertility of Soils*, 21, 30-36
- Meyerhoff J.O., Edlow J.A., Elston D.M., Madappa T., Mowad C.M., Schwartz R.A., Thomas F.P., Vinson R.P. & Wu S. (2011) Lyme Disease.
- Morris S.K. & Nguyen C.K. (2004) Blastomycosis. *University of Toronto Medical Journal*, 81, 172-175
- Nechitaylo T.Y., Timmis K.N., Byzov B.A., Kurakov A.V., Belogolova E., Jones D.L., Ferrer M. & Golyshin P.N. (2007) Fate of prions in soil: Degradation of recombinant prion in aqueous extracts from soil and casts of two earthworm species. *Soil Biology and Biochemistry*, 42, 1168-1171
- Nettleman M.D. (2011) Escherichia coli 0157:H7 (E. coli 0157:H7) Overview. URL http://www.emedicinehealth.com/e_coli_escherichia_coli_0157h7_e_coli_0157h7/page11_em.htm
- Nielsen K., Heitman J. & Jay C.D. (2007) Sex and Virulence of Human Pathogenic Fungi. In: *Advances in Genetics*, pp. 143-173. Academic Press
- Olarte J., Filloy L. & Galindo E. (1976) Resistance of *Shigella dysenteriae* type 1 to ampicillin and other antimicrobial agents: strains isolated during a dysentery outbreak in a hospital in Mexico City. *Journal of Infectious Diseases*, 133, 572-575
- Oppenheimer A.P., Arsur E.L. & Hospenthal D.R. (2010) Coccidioidomycosis. URL <http://emedicine.medscape.com/article/215978-overview>
- Pachepsky Y., Yu O., Karns J., Shelton D., Guber A. & van Kessel J. (2008) Strain-dependent variations in attachment of *E. coli* to soil particles of different sizes. *International Agrophysics*, 22, 61-66
- Parker R.R., Steinhaus G.M., Kohls G.M. & Jellison W.L. (1951) Contamination of natural waters and mud with *Pasteurella tularensis* *Pasteurella tularensis* and tularemia in beavers and muskrats in the north western United States. *National Institutes of Health Bulletin*, 193, 1-61
- Pelayo Ulacia S. & Dafhnis D. (1980) Isolation of the causative agent and histopathology of athlete's foot (preliminary report). *Revista Cubana de Medicina Tropical*, 32, 227-232
- Pennardt A. (2009) Giardiasis in Emergency Medicine Medication. URL <http://emedicine.medscape.com/article/782818-overview>
- Platz S. (1980) Experimental studies on survival of *Salmonella typhimurium* in different soil types. *Zentralblatt fur Bakteriologie Mikrobiologie und Hygiene I Abt. Originale B* 171, 256-268
- Poore G.V. & Lond F.R.C.P. (1899) Earth in relation to the preservation and destruction of contagia. *The British Medical Journal*, 457-460
- Ralf Reintjes, Isuf Dedushaj, Ardiana Gjini, Tine Rikke Jorgensen, Benvon Cotter, Alfons Liefertucht, Fortunato D'Ancona, David T. Dennis, Michael A. Kosoy, Gjyle Mulliqi-Osmani, Roland Grunow, Ariana Kalaveshi, Luljeta Gashi & Humolli I. (2002) Tularemia Outbreak Investigation in Kosovo: Case Control and Environmental Studies. *Emerging Infectious Diseases*, 8, 69-73
- Revis D.R.J. (2008) Clostridial gas gangrene clinical presentation
- Rhodes G., Huys G., Swings J., McGann P., Hiney M., Smith P. & Pickup R.W. (2000) Distribution of Oxytetracycline Resistance Plasmids between Aeromonads in Hospital and Aquaculture Environments: Implication of Tn1721 in Dissemination of the Tetracycline Resistance Determinant Tet A. *Appl. Environ. Microbiol.*, 66, 3883-3890
- Rigou P., Rezaei H., Grosclaude J., Staunton S. & Quiquampoix H. (2006) Fate of prions in soil: adsorption and extraction by electroelution of recombinant ovine prion protein from montmorillonite and natural soils. *Environmental Science & Technology*, 40, 1497-1503
- Roque M.R., Roque B.L. & Foster C.S. (2010) Actinomycosis in Ophthalmology URL <http://emedicine.medscape.com/article/1203061-overview>

- Ross C.M. & Donnison A.M. (2006) Campylobacter jejuni inactivation in New Zealand soils. *Journal of Applied Microbiology*, 101, 1188-1197
- Sattar S.A. (1999) *Giardia cyst and Cryptosporidium oocyst survival in watersheds and factors affecting inactivation*. AWWA Research Foundation, USA.
- Schaffner W. (2007) Tularemia and other Francisella infections. In: *Cecil Medicine* (eds. Goldman L & Ausiello D). Saunders Elsevier, Philadelphia
- Scott P.C., Godard V. & Benabderrahmane C. (2011) Modeling risk areas for Echinococcus multilocularis egg survival in an urban park in France. In: *Anais XV Simpósio Brasileiro de Sensoriamento Remoto*, pp. 5989-5996. INPE, Curitiba, Brazil
- Semenov A.V., van Overbeek L. & van Bruggen A.H.C. (2009) Percolation and survival of E. coli O157:H7 and Salmonella enterica serovar Typhimurium in soil amended with contaminated dairy manure or slurry. *Appl. Environ. Microbiol.*, AEM.01791-08
- Sharma M., Ingram D.T., Patel J.R., Millner P.D., Wang X., Hull A.E. & Donnenberg M.S. (2009) A novel approach to investigate the uptake and internalization of Escherichia coli O157:H7 in spinach cultivated in soil and hydroponic medium. *Journal of Food Protection*, 72, 1513-1520
- Smith J.M. (2011) Monsanto's Roundup Triggers Over 40 Plant Diseases and Endangers Human and Animal Health. URL http://www.foodconsumer.org/newsite/Non-food/Environment/roundup_0118110818.html
- Smith L.D. (1975) Inhibition of Clostridium botulinum by Strains of Clostridium perfringens Isolated from Soil. *Appl. Environ. Microbiol.*, 30, 319-323
- Smith L.D.S. (1979) *Clostridium botulinum*: characteristics and occurrence. *Clinical Infectious Diseases*, 1, 637-641
- Snijders, C.H., Samson, R.A., Hoekstra, E.S., Ouellet, T., Miller, J.D., de Rooj-van der Goes, P.C., Baar, A.J., Dubois, A.E., Kauffman, H.F. (1996) Analysis of Fusarium causing dermal toxicosis in marram grass planters. *Mycopathologia*, 135, 119-128
- Steele R.W. & Shetty A. (2011) Pediatric Blastomycosis Clinical Presentation
- Tam A.B., Hexdall A. & Weiss E.L. (2011) Hookworm in Emergency Medicine. URL <http://emedicine.medscape.com/article/788488-overview>
- Tashiro K., Kubokura Y., Kato Y., Kaneko Y. & Ogawa M. (1991) Survival of Yersinia enterocolitica in soil and water. *The Journal of Veterinary Medical Science*, 53, 23-27
- Tigertt W.D., Benenson A.S. & Gochenour W.S. (1961) Airborne Q fever. *Microbiol. Mol. Biol. Rev.*, 25, 285-293
- Tolan R.W., Minnaganti V.R. & Fennelly G. (2011) Isoporiasis. URL <http://emedicine.medscape.com/article/219776-overview>
- Tolls J. (2001) Sorption of veterinary pharmaceuticals in soils: A review. *Environmental Science & Technology*, 35, 3397-3406
- Turbé A., Toni A.D., Lavelle P., Benito P., Lavelle P., Ruiz N., van der Putten W.H., Labouze E. & Mudgal S. (2010) Soil Biodiversity: functions, threats and tools for policy makers. In. European Commission, Brussels
- van der Hoek W., Hunink J., Vellema P. & Droogers P. (2011) Q fever in The Netherlands: the role of local environmental conditions. *International Journal of Environmental Health Research*, 11, 1-11
- Van Ness G.B. (1971) The ecology of Anthrax. *Science*, 172, 1303-1307
- Volkova V.V., Bailey R.H. & Wills R.W. (2009) Salmonella in broiler litter and properties of soil at farm location. *PLoS ONE*, 4, e6403
- Walther B.A. & Ewald P.W. (2004) Pathogen survival in the external environment and the evolution of virulence. *Biological Reviews*, 79, 849-869
- Weagant S.D. & Feng P. (2001) *Yersinia enterocolitica*. In: *Bacteriological Analytical Manual*. U.S. Food and Drug Administration, Washington DC, USA
- Weinstein K.B. (2011) Listeria monocytogenes treatment & management. URL <http://emedicine.medscape.com/article/220684-treatment>

- Welshimer H.J. (1960) Survival of *Listeria monocytogenes* in soil. *Journal of Bacteriology*, 80, 316-320
- Wesley C.L., Allen L.J. & Langlais M. (2010) Models for the spread and persistence of hantavirus infection in rodents with direct and indirect transmission. *Mathematical Biosciences and Engineering*, 7, 195-211
- WHO (1979) Human viruses in water, wastewater and soil. In: *WHO Technical Report Series* 639. World Health Organisation, Geneva
- WHO (2002) Botulism; Factsheet N°270. In. WHO Media Centre, Geneva
- Zaitsev S.V., Chernukha I.G., Evdokimova O.A. & Belov A.S. (1989) Survival rate of *Leptospira pomona* in the soil at a natural leptospirosis focus. *Zh Mikrobiol Epidemiol Immunobiol.*, 2, 64-68
- Zieve D.Z., Eltz D.R. & Dugdale III D.C. (2010) Aspergillosis. URL <http://www.nlm.nih.gov/medlineplus/ency/article/001326.htm>

European Commission

EUR 24893 EN – Joint Research Centre – Institute for Environment and Sustainability

Title: Soil Borne human Diseases

Author(s): S. Jeffery and W. H. van der Putten

Luxembourg: Publications Office of the European Union

2011 – 49 pp. – 21 x 29.5 cm

EUR – Scientific and Technical Research series – ISSN 1018-5593 (print), ISSN 1831-9424 (online)

ISBN 978-92-79-20796-9 (print)

ISBN 978-92-79-20797-6 (pdf)

doi:2788/36703

Abstract

Soils are home to a remarkable array of biodiversity with some estimates stating that 25% of the Earth's species find their home in the soil. Of these organisms, the vast majority are not of any threat to human health, but rather function to provide numerous ecosystem services which emerge through the multitude of complex interactions between organisms within the soil and the soil itself. These ecosystem services range from those which are vital for maintaining life on Earth, such as the formation of soil, the cycling of nutrients with the result of maintaining soil fertility, and the filtering of water, as well as provision of useful compounds such as antibiotics, the majority of which have been isolated from soil organisms.

However, soils also contain microorganisms which are capable of causing diseases in humans. They act either as opportunistic pathogens which take advantage of susceptible individuals, such as those who are immuno-compromised; or as obligate pathogens which must infect humans in order to complete their life-cycles. These organisms may be capable of surviving within the soil for extended periods of time before infecting humans who come into contact with contaminated soil.

This report provides an overview of the various soil borne diseases which can affect humans, including a discussion of the literature where available for each disease, and an analysis of the evidence for why each disease may be considered to be soil borne. Information from the World Health Organisation (WHO) and the European Centre for disease prevention and control (ECDC) on infection and mortality rates within the EU27 is also presented. However, limitations with the data sets prevent accurate qualitative analysis such as which diseases have the highest recovery rates in which member state etc. A discussion of the factors which may affect the incidence of such diseases; including land management practices or land use change, climate change, and the use of antibiotics in livestock, is presented. Finally, areas of future research which are needed are highlighted to aid further investigation of this important and yet understudied area.

How to obtain EU publications

Our priced publications are available from EU Bookshop (<http://bookshop.europa.eu>), where you can place an order with the sales agent of your choice.

The Publications Office has a worldwide network of sales agents. You can obtain their contact details by sending a fax to (352) 29 29-42758.

The mission of the JRC is to provide customer-driven scientific and technical support for the conception, development, implementation and monitoring of EU policies. As a service of the European Commission, the JRC functions as a reference centre of science and technology for the Union. Close to the policy-making process, it serves the common interest of the Member States, while being independent of special interests, whether private or national.

LB-NA-24893-EN-C



ISBN 978-92-79-20796-9



9 789279 207969