ESSAY

The disease triangle: pathogens, the environment and society

Karen-Beth G. Scholthof

Abstract | The primary means to define any disease is by naming a pathogen or agent that negatively affects the health of the host organism. Another assumed, but often overlooked, determinant of disease is the environment, which includes deleterious physical and social effects on mankind. The disease triangle is a conceptual model that shows the interactions between the environment, the host and an infectious (or abiotic) agent. This model can be used to predict epidemiological outcomes in plant health and public health, both in local and global communities. Here, the Irish potato famine of the mid-nineteenth century is used as an example to show how the disease triangle, originally devised to interpret plant disease outcomes, can be applied to public health. In parallel, malaria is used to discuss the role of the environment in disease transmission and control. In both examples, the disease triangle is used as a tool to discuss parameters that influence socioeconomic outcomes as a result of host–pathogen interactions involving plants and humans.

With a global human population approaching 6.5 billion, 800 million people who are chronically hungry or starving and an estimated 2 billion who lack clean water or electricity, it is imperative that we develop better strategies to control and contain disease. Although the role of the environment has long been considered a determinant (or cause) of diseases in plants and man[1,2], it has been undervalued at times in the history of medicine, particularly after germ theory became dogma[3,4]. This was particularly true in the early to mid-twentieth century when the dominant notion was that conquering disease was a job for science, perhaps best recognized by the commercial development and global distribution of antibiotics and vaccines. Although there has been some moderation of this viewpoint, laboratory research increasingly focuses on host–pathogen interactions while generally failing to consider the role of the environment in disease outcome.

This is unfortunate, as plant, animal and human health outcomes are affected by local and global environmental conditions[5–7]. These conditions might include economic or ecological factors, changes in host populations, trade and travel, technology and changes in microorganism populations. Several human diseases, such as HIV, tuberculosis, transmissible spongiform encephalopathies, plague, cholera, influenza and malaria are clearly influenced by one or more of these environmental conditions. Further environmental factors that influence disease outcome include famines, war, the use of biological weapons, and natural or man-made cataclysms such as hurricanes, deforestation, drought, air and water pollution; each of these factors demands flexible approaches to preventing disease and improving the social matrix[8].

Over the past decade Paul Farmer introduced and elaborated the concept of structural violence, in which an individual's health is influenced by a constellation of events[9–11]. This includes various aspects of the social matrix, such as a lack of education, insufficient access to healthcare, and a scarcity of basic needs, including shelter, food, clothing, electricity, clean water and a safe living environment. Farmer's explication of structural violence reveals that life is fragile, yet with relatively little investment individuals can be provided with the means to improve their living conditions.

Curiously, the impact of crop and plant diseases on human welfare is only rarely considered[7,12]. Yet, agricultural losses due to pathogens can transform local economies and even devastate a primary food resource for a community, thereby impacting negatively on the health of the community. When crops lack genetic resistance to pathogens and effective chemical controls are unavailable, serious threats to the economic and food security of small shareholders and their families can occur.

The disease triangle can be used to integrate these different factors (FIG. 1). This model uses the temporal relationships between the environment, the host and an infectious (or abiotic) agent to develop new ideas to predict and control disease. In this article, I use a historical event (the Irish potato famine) to show the mechanics and uses of the disease triangle and the confluence of events that destroyed the potato crop and the health of the disenfranchised poor, who were wholly dependent on the potato. I also discuss how the disease triangle can be used in the present-day setting, using malaria as a case study, to define parameters in agriculture and public health that can improve socioeconomic outcomes.

The disease triangle

During their lifetime, plants are exposed to changing temperatures, humidity, drought or rainfall, soils and nutrients, weeds, insects, nematodes and microorganisms (bacteria, fungi and viruses). Each of these factors might be beneficial or detrimental to plant health. Farmers and scientists must consider economic and market variables, as well as the complex interactions between a host, the pathogen and the environment that determine the quality and value of the crop. Although market concerns drive trends in agriculture, the environment remains the most crucial influence on plant disease incidence from year to year. The disease triangle is a classic plant pathology concept used to examine the role of the environment in disease processes in the life-history of a plant[13].

The disease triangle concept was formalized in the 1960s by George McNew, a scientist at the Boyce Thompson Institute for Plant Research. McNew suggested that the disease triangle could be used "to study the interrelationship of various factors in an epidemic" (REF. 13) and to understand how epidemics might be predicted, limited or controlled. It was intended as an empirical tool for use until research matured to provide new methods to predict and control diseases. McNew broadly defined the parameters of the disease triangle as "the inherent susceptibility of the host, the inoculum potential of the parasite, and the impact of the environment on parasitism and pathogenesis" (REF. 13). As shown in FIGURE 1, six factors interact to determine the potential for a host to develop economically important levels of disease after exposure to a pathogen under favourable environmental and temporal conditions. These factors are the severity of the physical environment (for example, temperature, humidity or rainfall), the duration of the infection period (time), prevalence of the pathogen, virulence of the pathogen, the age or maturity of the host plant and its inherent susceptibility to disease.

McNew's disease triangle gained immediate acceptance in the plant pathology community, perhaps because it provided a means to visualize and articulate observations that three interlocking participants — host, pathogen and environment — determine disease outcome during the life-history of the host. Nowadays there are modified versions of the disease triangle, including the disease pyramid or tetrahedron, that have 'time' and 'man' as additional vertices[14,15]. It is, however, important to carefully study McNew's original triangle (FIG. 1) and his commentary on the nature, origin and evolution of parasitism[13]. In the triangle he prepared for epiphytotics (epidemics on plants), each component of the disease triangle considers temporal conditions (time) in the analysis of the economically important threshold for host–pathogen interactions. McNew's triangle reiterates the rules of Beijerinck: namely that "everything is everywhere — but the environment selects" (REF. 16). The disease triangle was accepted and established as a common feature in plant pathology texts[14,15]. Today, theoretical and applied plant epidemiology are advanced fields of study, incorporating the effects of local and global climate change in the control and management of plant disease[17–22].

Late blight disease of potato

As a case study, the Irish potato famine provides lessons about the relationship between disease and human and plant populations, extending to the elaboration of germ theory[4,23]. In mid-nineteenth century Ireland, potato late blight disease "deprived one-third of the population of virtually their only means of subsistence for several years" (REF. 24). O'Grada has estimated that "one million people, or almost one-eighth of the entire population perished as the result of the potato failures in the 1840s" (REF. 24).

Knowledge of the origins and use of potatoes had a significant role in understanding late blight disease and the Irish famine. Ranked fourth in global food production after wheat, maize and rice, potatoes have been cultivated as a crop for at least 7,000 years[25,26]. From their centre of origin in South America[27], potatoes arrived in Europe in the late 1500s and in North America in the early 1600s. Potatoes are ordinarily propagated as seed potatoes using either the entire potato tuber or the vegetative meristems (the 'eyes') to generate a new plant.

The centre of diversity of *Phytophthora infestans*, the causal agent of late blight disease of potato, is in the central highlands of Mexico where sexual mating types A1 and A2 are found[26,28–32]. Using mitochondrial DNA haplotype analyses, field surveys and herbarium specimens, a lively debate is underway to determine the centre-of-origin of *P. infestans*[28,30,33–36]. The pathogen, which reproduces clonally, presumably 'piggy-backed' on potato tubers as the crop was established worldwide by global trade and travel[37–39]. *P. infestans* is classified as an oomycete or water mould[40]. Oomycetes, pathogens of plants and animals, are more closely related to brown algae than to filamentous fungi[41]. *P. infestans* is found in the soil, crop residue and infected tubers, where it can over-winter until a new crop is planted. Potato plants are susceptible to late blight infection throughout their lifespan, from tuber to full-grown plants[40,42]. Today, the pathogen is endemic wherever potato is grown, accounting for some losses each year[43].

The Irish potato famine

The first reports of the potato disease in the United States were in 1842–1843 in Massachusetts, New York and Pennsylvania[23,44]. By 1844 the disease was widespread in the northeastern United States and adjacent regions of Canada[44,45]. In Europe, the disease was reported in Belgium at the end of June 1845, extending to southeast England by mid-August, and throughout most of the United Kingdom, including the east coast of Ireland by mid-September. By mid-October 1845, the

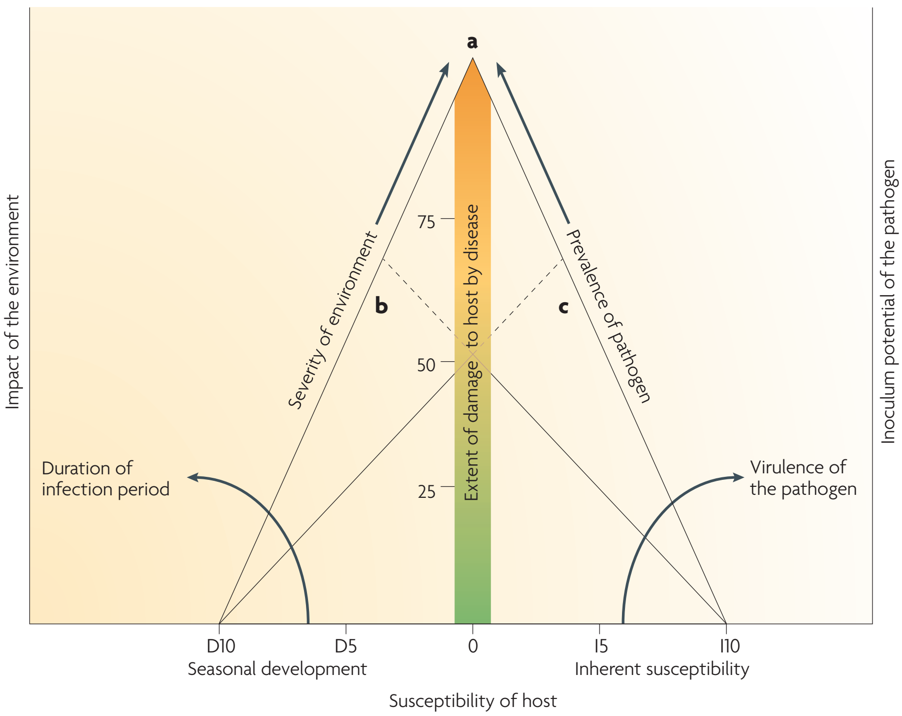

Figure 1 | **The triangle of factors that limit an epidemic.** The full impact of a pathogen might be avoided either by reducing the host susceptibility, inoculum potential of the parasite or environmental conditions favourable to pathogenesis. The peak might be reduced from a maximum point (shown here as **a**, which is the economic effect of the disease in a host) to some lower level (shown here as **b** and **c**). This would restrict the factors in an epidemic, and would reduce disease from a maximum of 100 to some lower value (such as 50, as shown in this figure). All values are assigned empirically and cannot be given precise values at this stage of knowledge. The preferred treatment for a disease shifts progressively clockwise from the left corner to the lower right side of the base as the specialization in pathogenesis increases (from moderate to severe disease). Determinants that affect host outcome include seasonal development (D) and inherent susceptibility (I). It is also important to note that temporal effects are implied for each factor related to McNew's disease triangle. Figure and legend modified with permission from REF. 13 © (1960) Elsevier.

blight had spread throughout Ireland, ushering in the first winter of the famine[1,24,45].

With devastating effect, potato late blight disease added to the ingrained social, political, religious and economic inequalities of the poor in Ireland. In the summer of 1845, as narrated by E. C. Large in *The Advance of the Fungi*[1], the potato plants were luxuriant throughout Ireland, resulting in the formation of a continuous canopy of leaves. By August 16, 1845, plants were at least halfway through the growing season when a correspondent from the Isle of Wight, reported in *The Gardeners' Chronicle* "a blight of unusual character" and wondered "whether this plague be…an entirely new one" (REF. 46). The following week, the alarm was sounded: "a fatal malady has broken out among the potato crop. On all sides we hear of the destruction that has overtaken this valuable product, excepting the north of England...there is hardly a sound sample in Covent Garden Market" (REF. 47). Together, the genetically uniform potato crop (monoculture), the dependence of the poor on a single food source, the presence of *P. infestans*, and permissive environmental conditions, resulted in ecological and social disaster in Ireland[1,4]. These people "whose food was gone were, in fact, beyond the pale of all mercantile system — they had lived upon the produce of their potato-gardens, and had been customers of no shop" (REF. 48). The destitute paupers, at the mercy of their landlords and the politics of the day, found themselves without food, money or other resources; the equivalent structural violence remains all too common today for billions of people in the 'modern' global economy[10,11,49,50].

The disease triangle and monoculture

Assuming that the virulence of the clonal population of the pathogen was unchanged from previous years [37,38], we can narrow the parameters on the disease triangle to look at the social and agricultural environments and try to understand the extent of damage by late blight disease on potato. In Ireland, the virtual reliance of the peasants on a single crop — the potato — set the stage for a food crisis. From first-hand accounts and meteorological reports, it is likely that the outbreak of *P. infestans* on potato in Europe in the mid-1800s was due to climatic conditions that were unusually wet and cool, as described in detail in *The Advance of the Fungi*[1]. Rain, temperature, wind and humidity affect the dispersal, germination, transmission and survival of spores, and the general health or susceptibility of the host plant (FIG. 1). For example, an 1845 report in *The Gardeners' Chronicle*, noted that it was unusually warm in early July, "suddenly succeeding a period of cold ungenial weather" followed by "cold, gloomy, watery days of August, after the forced growth of July" (REF. 47). The cool, cloudy and wet conditions were suitable for the development of potato late blight infections and considerable losses occurred (TABLE 1).

Other factors contributed to losses caused by potato late blight infection. In Ireland, potato was the staple food; in other countries potato losses were just as acute, but the diversity of crops and diet generally warded off the dire effects of famine. By contrast, "Ireland was almost one great potato patch" (REF. 44). In Ireland "nothing so destructive as this new murrain had ever been seen before. It struck down the growing plants like frost in the summer. It spread faster than the cholera amongst men" (REF. 1.)

The potato leaves and stems were destroyed by *P. infestans* infections, and infected tubers quickly rotted, either in the ground or immediately after harvest[1,24,26,51]. As the late blight infection progresses on the potato leaf, spores are produced and spread to adjacent plants, and the developing tubers can be infected when the spores are washed into the soil[52]. Tubers that might have escaped the infection (based on a healthy appearance) were planted the following year, only to become blighted during the season, and yields were further reduced each subsequent year (TABLE 1). So, the environmental conditions, a uniformly susceptible plant host and the presence of the pathogen had devastating repercussions on the plants, people, politics and economy of Ireland.

The use and features of the disease triangle have been used to develop plant disease forecasting, or phytopathometry[53], the science that produces epidemiological models. Forecasting was established first by observation, then developed into a predictive science based on mathematical modelling of environmental conditions using contemporary and historic weather data, including precipitation, humidity, wind and temperature. By establishing daily severity values, these forecasts predict when disease is most likely to occur and when prophylactic measures, such as fungicide treatments of the crop, should be used. Today, LATEBLIGHT, a sophisticated computer modelling system, is used in New York State (USA) and has been adapted for use in Peru to predict the potential for potato late blight outbreaks and to respond with timely and appropriate control measures[17,18].

The disease triangle and social impact

The potato sustained and nourished the poor, who accounted for at least one-third of the population of Ireland in the mid-nineteenth century. As subsistence tenant farmers, they were economically destitute and dependent on the potato crop that they raised on their small plots. The diet consisted of 3.5 to 4.5 kilograms of boiled potatoes per day, which provided essential nutrients and calories when supplemented with buttermilk or salted fish, and field greens in the spring[54]. The productivity of potatoes on small plots is attributed to a doubling of the Irish population, from ~4 million to 8 million, between 1840 and 1845 (REFS 24,26,51).

A near total loss of the potato crop in 1845 and the lack of an alternative source of cheap, nutritious foods led to the 'Great Irish Famine' (REFS 1,24,26,51). The potato was the "arbiter between a bare subsistence and starvation" (REF. 51). The potato epidemic persisted into the 1850s and the conditions of hunger, crowding, lack of housing and poverty — in other words, the conditions of social structural violence[11] — caused more than 1 million deaths and an equal number of Irish nationals emigrated to the United States and to British colonies, especially Canada and Australia (TABLE 1).

The lessons learned from the famine can be applied to contemporary situations. The single crop system of agriculture (monoculture), the lack of understanding about infectious diseases, the lack of chemical control, overwhelming disease pressure and weather conditions made late blight an intractable problem. Factoring in the inherent structural violence[10,11,24] leads to the retrospective and inescapable finding that a disaster to plants and people was inevitable. *Phytophthora* — which translates as the plant destroyer — is an apt name for the late blight pathogen of potato. Even today, late blight is an economically important pathogen of potato and tomato[37,55], providing a constant reminder of a continuing need to identify political crises and to relieve structural violence. Such measures would include using appropriate technologies[7] to ensure healthy, nutritious, inexpensive food, and to prevent or forestall socioeconomic

Table 1 | **Potato cultivation, trade and the Irish population during the great famine***

Year	Potatoes (million acres)	Yield‡ (tons per acre)	% Normal§ (versus 1840)	Price equivalent§	Population‖ (millions)	Emigration¶
1840	2.1	6	100	1	8.2	<30,000
1845	2.1	4	67	1.2–1.5×	8.5	61,000
1846	1.7	1.5	25	3.25×	ND	106,000
1847	0.3	7.2	NA	2–2.25×	ND	200,000
1848	0.8	3.9	63	~2×	ND	178,000
1851	ND	ND	ND	~2×	6.5	250,000

*Data compiled from several sources (REFS 1,24,26,51,54). ‡Yields are not adjusted for seed sown at ~0.8 tons per acre. From 1847–1871 yield average is ~4 tons per acre. Net yields of pre-famine versus famine years are 5.2 versus 3.2 tons per acre. This is a 30% yield decrease. §The average data from the 1840s were used as a base of 100% for yield, or a value of 1 for price equivalents, for the basis of comparisons during the famine years. For 1847, the potato yield per acre was good, but so little acreage was planted that it was not considered a source of food. ‖Figures for populations from 1845 to 1850 are not reliably known. Population census data is provided for 1841 and 1851. ¶Until 1858, more than 1% of the population emigrated per year, accounting for more than 1 million emigrants between the mid-1840s and early 1850s. It was estimated that in the 65 years before the famine, 1.75 million Irish emigrated, resulting in the rough estimate of ~30,000 per year in the 1840s. NA, not applicable; ND, no data or incomplete data.

conditions leading to famine and other dehumanizing events.

Malaria and the disease triangle

The disease triangle should be considered in developing interdisciplinary environmental and economic solutions to diseases[49]. Malaria offers a pertinent public health example to explain specifically how the disease triangle can be used to understand disease development and control. Malaria is caused by four species of the *Plasmodium* parasite that are naturally transmitted to humans during blood meals of female *Anopheles* mosquitoes. Malaria causes 1 million deaths annually and more than 90% of these occur in Africa; there are 300 million infected individuals worldwide[56]. I will discuss two successful strategies that indicate that farmers are eager to be engaged in reducing the seasonal incidence of malaria.

One strategy is to use climatic data to develop early warning systems that predict an increased incidence of malaria[57,58]. This type of forecasting has developed from the climate forecasting that was used to make recommendations for planting maize in Zimbabwe[59]. Both are extensions of the early work by E. C. Large who developed the concepts of phytopathometry to reduce the incidence of potato late blight disease losses on potato[1,53]. In Cane *et al.*[59], a drought index for crops was formulated based on water temperatures in the Pacific Ocean and atmospheric conditions using predictive models and satellite imagery[59]. Predictive climatology is an additional valuable component of integrated pest management (IPM) strategies to reduce disease pressure, pesticide and fertilizer applications, and to improve crop yields and local socioeconomic conditions. The use of climate data to forecast malaria outbreaks is yet another approach of "fighting poverty with maps" (REF. 49). Analyses of ocean surface temperature, precipitation and the annual incidence of malaria were used to define the parameters to predict an outbreak[57,58] and by extension to 'break the triangle' of disease transmission.

A second cogent strategy to improve malaria control was suggested by Levins and co-workers in a rice-growing area of Sri Lanka, using a community-based approach[60,61]. They found that farmers should be considered as an important resource for disease control, including changes in agricultural practices and spray regimens, with a long-term goal of improved individual and public health. These reports are particularly compelling, since Levins is both an ecologist and a farmer, having experience in cropping systems in subtropical Puerto Rico. He brings hands-on experience to IPM strategies to facilitate ecologically orientated malaria control efforts. In a 20-week period, participants significantly increased their understanding of mosquito ecology and disease epidemiology, and environmentally sound preventative measures such as the use of bed nets, elimination of breeding sites, applying oil, salt or fish to stored water and wells, and cleaning up surroundings. Furthermore, 94.1% of the intervention group changed at least one of their agriculture practices to reduce mosquitoes by levelling land and cleaning canals, or draining rice paddies[61]. In addition, educational intervention and an understanding of epidemiology were most effective when women were trained, possibly because they are responsible for household health and management[61]. Although farmers did not decrease herbicide usage, the odds of decreasing or ending their use of pesticides was 5.6 times higher than in the control village[60]. Reducing the use of pesticide is a first step in developing robust IPM programmes[7], including the use of natural predators and reducing pesticide accumulation in fish or other animals in paddy rice fields that might be used as human food sources. Increasing mosquito predators (birds, bats, amphibians and fish) will reduce costs to farmers, and allow for community-based approaches to disease control. As discussed by van der Hoek "agricultural and malaria links are complex" (REF. 62), and as shown by Levins and co-workers[60,61], success in reducing or eliminating malaria must use an integrative agroecosystems approach, that is the whole disease triangle. In particular it is crucial to develop collaborations with agricultural specialists to "focus on the most feasible and acceptable control strategies that increase household incomes, water-use efficiency and food security" (REF. 62) and to facilitate global improvements in human health by looking more carefully at our intimate links with agriculture and plant health[7].

Conclusions

My intent has been to argue that wider dissemination of the disease triangle concept allows us to ask better and more integrative questions about infections and diseases (biotic and abiotic) from three interconnected perspectives — host, pathogen and the environment. The disease triangle might be considered under-articulated, but in fact, this strengthens its use in that it allows for both a complex analysis of relationships and provides the flexibility to prompt new (and easily comprehensible) ideas about the role of the environment in disease. The disease triangle is a holistic, empirical tool that encourages the consideration of habitat, edge-effects[63], climate change, migration and politics[64] as factors that contribute to health (or disease). From the triangle, beginning at any edge, it is possible to evaluate and discern the triggers that manifest disease and affect plant, animal or human health.

Potato late blight and malaria were presented as parallel, and converging, examples of the effects of the environment on host–pathogen interactions to gain insight into developing new strategies for disease control. Disease forecasting and careful monitoring of environmental and climatic conditions provides a powerful tool to predict and control disease outcome and reduce structural violence. It can be ecologically and economically advantageous in reducing chemical (insecticides and drugs) inputs with the potential advantage of decreasing the incidence of chemical resistance in the targeted pathogenic organisms. With increased crowding and disease pressure on the human population, and incidents such as war, disease outbreaks, malnutrition and natural disasters, there is much to be learnt from studying modern and historical responses to plant disease outbreaks. As Irish Statesman Richard Southwell Bourke (1822–1872) noted during the famine, what is needed is "less politics and more ploughing, less argument and more action, less debating and more doing" (REF. 48). The disease triangle provides another strategy to evaluate the role of the environment in disease control and to emphasize the need to bring plant pathologists and agricultural specialists into the global efforts to improve human welfare.

Karen-Beth G. Scholthof is at the Department of Plant Pathology and Microbiology, Texas A&M University, 2132 TAMU, College Station, Texas, 77843 USA.
e-mail: kbgs@tamu.edu

doi:10.1038/nrmicro1596
Published online 27 December 2006

1. Large, E. C. *The Advance of the Fungi* (APS Press, St. Paul, USA, 2003).
2. Reiter, P. From Shakespeare to Defoe: malaria in England in the Little Ice Age. *Emerg. Infect. Dis.* **6**, (2000).
3. Dubos, R. *Mirage of Health: Utopias, Progress, and Biological Change* (Harper & Row, New York, 1959).
4. Kelman, A. & Peterson, P. D. Contributions of plant scientists to the development of the germ theory of disease. *Microbes Infect*. **4**, 257–260 (2002).
5. Patz, J. A., Campbell-Lendrum, D., Holloway, T. & Foley, J. A. Impact of regional climate change on human health. *Nature* **438**, 310–317 (2005).
6. Foley, J. A. *et al.* Global consequences of land use. *Science* **309**, 570–574 (2005).
7. Sanchez, P. A. & Swaminathan, M. S. Hunger in Africa: the link between unhealthy people and unhealthy soil. *Lancet* **365**, 442–444 (2005).
8. Wilson, M. E., Levins, R. & Spielman, A. (eds) *Disease in Evolution: Global Changes and Emergence of Infectious Diseases* (New York Academy of Sciences, 1994).
9. Castro, A. & Farmer, P. Understanding and addressing AIDS-related stigma: from anthropological theory to clinical practice in Haiti. *Am. J. Public Health* **95**, 53–59 (2005).
10. Farmer, P. *Infections and Inequalities* (University of California Press, Berkeley, 1999).
11. Farmer, P. Pathologies of power: rethinking health and human rights. *Am. J. Public Health* **89**, 1486–1496 (1999).
12. Sachs, J. D. & Hotez, P. J. Fighting tropical diseases. *Science* **311**, 1521 (2006).
13. McNew, G. L. The nature, origin, and evolution of parasitism. In *Plant Pathology: An Advanced Treatise* (eds Horsfall, J. G. & Dimond, A. E.) 19–69 (Academic Press, New York, 1960).
14. Agrios, G. N. *Plant Pathology* (Academic Press, New York, 2005).
15. Schumann, G. L. *Plant Diseases: Their Biology and Social Impact* (APS Press, St. Paul, 1993).
16. de Wit, R. & Bouvier, T. 'Everything is everywhere, but, the environment selects'; what did Baas Becking and Beijerinck really say? *Environ. Microbiol.* **8**, 755–758 (2006).
17. Andrade-Piedra, J. L., Hijmans, R., Forbes, G. A., Fry, W. E. & Nelson, R. J. Simulation of potato late blight in the Andes. I: Modification and parameterization of the LATEBLIGHT model. *Phytopathology* **95**, 1191–1199 (2005).
18. Andrade-Piedra, J. L., Hijmans, R., Forbes, G. A., Fry, W. E. & Nelson, R. J. Simulation of potato late blight in the Andes. II: Validation of the LATEBLIGHT model. *Phytopathology* **95**, 1200–1208 (2005).
19. Campbell, C. L. & Madden, L. V. *Introduction to Plant Disease Epidemiology* (John Wiley & Sons, New York, 1990).
20. Garrett, K. A., Dendy, S. P., Frank, E. E., Rouse, M. N. & Travers, S. E. Climate change effects on plant disease: genomes to ecosystems. *Annu. Rev. Phytopath*. **44**, 489–509 (2006).
21. Madden, L. V. Botanical epidemiology: some key advances and its continuing role in disease management. *Eur. J. Plant Pathol*. **115**, 3–23 (2006).
22. Scherm, H., Ngugi, H. K. & Ojiambo, P. S. Trends in theoretical plant epidemiology. *Eur. J. Plant Pathol*. **115**, 61–73 (2006).
23. Peterson, P. D., Campbell, C. L. & Griffith, C. S. James E. Teschemacher and the cause and management of potato blight in the United States. *Plant Dis.* **76**, 754–756 (1992).
24. O'Grada, C. *Black '47 and Beyond: The Great Irish Famine in History, Economy, and Memory* (Princeton University Press, Princeton, 1999).
25. Mullins, E., Milbourne, D., Petti, C., Doyle-Prestwich, B. M. & Meade, C. Potato in the age of biotechnology. *Trends Plant Sci.* **11**, 254–260 (2006).
26. Salaman, R. N. *The History and Social Influence of the Potato* (Cambridge University Press, Cambridge, 1985).
27. Spooner, D. M., McLean, K., Ramsay, G., Waugh, R. & Bryan, G. J. A single domestication for potato based on multilocus amplified fragment length polymorphism genotyping. *Proc. Natl Acad. Sci. USA* **11**, 14694–14699 (2005).
28. Fernández-Pavía, S. P., Grünwald, N. J., Díaz-Valasis, M., Cadena-Hinojosa, M. & Fry, W. E. Soilborne oospores of *Phytophthora infestans* in central Mexico survive winter fallow and infect potato plants in the field. *Plant Dis*. **88**, 29–33 (2004).
29. Grünwald, N. J. *et al.* Potato cultivars from the Mexican National Program: sources and durability of resistance against late blight. *Phytopathology* **92**, 688–693 (2002).
30. Grünwald, N. J. & Flier, W. G. The biology of *Phytophthora infestans* at its center of origin. *Annu. Rev. Phytopath*. **43**, 171–190 (2005).
31. Niederhauser, J. S. The blight, the blighter, and the blighted. *Trans. N. Y. Acad. Sci*. **19**, 55–63 (1956).
32. Niederhauser, J. S. & Mills, W. R. Resistance of *Solanum* species to *Phytophthora infestans* in Mexico. *Phytopathology* **43**, 456–457 (1953).
33. Avila-Adame, C. *et al.* Mitochondrial genome sequences and molecular evolution of the Irish potato famine pathogen, *Phytophthora infestans*. *Curr. Genet*. **49**, 39–46 (2006).
34. Andrivon, D. The origin of *Phytophthora infestans* populations present in Europe in the 1840s: a critical review of historical and scientific evidence. *Plant Pathol*. **45**, 1027–1035 (1996).
35. Ristaino, J. B. Tracking historic migrations of the Irish potato famine pathogen, *Phytophthora infestans*. *Microbes Infect*. **4**, 1369–1377 (2002).
36. Ristaino, J. B., Groves, C. T. & Parra, G. R. PCR amplification of the Irish potato famine pathogen from historic specimens. *Nature* **411**, 695–697 (2001).
37. Fry, W. E. & Goodwin, S. B. Re-emergence of potato and tomato late blight in the United States. *Plant Dis*. **81**, 1349–1357 (1997).
38. Fry, W. E. & Goodwin, S. B. Resurgence of the Irish potato famine fungus. *BioScience* **47**, 363–371 (1997).
39. Goodwin, S. B., Cohen, B. A. & Fry, W. E. Panglobal distribution of a single clonal lineage of the Irish potato famine fungus. *Proc. Natl Acad. Sci. USA* **91**, 11591–11595 (1994).
40. Judelson, H. S. & Blanco, F. A. The spores of *Phytophthora*: weapons of the plant destroyer. *Nature Rev. Micriobiol*. **3**, 47–58 (2005).
41. Kamoun, S. Molecular genetics of pathogenic Oomycetes. *Eukaryotic Cell* **2**, 191–199 (2003).
42. Kamoun, S. & Smart, C. D. Late blight of potato and tomato in the genomics era. *Plant Dis*. **89**, 692–699 (2005).
43. Garelik, G. Taking the bite out of potato blight. *Science* **298**, 1702–1704 (2002).
44. Whetzel, H. H. The relation of plant pathology to human affairs. In *Mayo Foundation Lectures, 1926–1927*. 151–178 (W. B. Saunders Co., Philadelphia, 1928).
45. Bourke, P. M. A. Emergence of potato blight, 1843–1846. *Nature* **203**, 805–808 (1964).
46. Salter, T. B. Home Correspondence. Potato Blight. *The Gardeners' Chronicle* **33**, 561 (1845).
47. Lindley, J. *The Gardeners' Chronicle* **34**, 575 (1845).
48. Anonymous. Ireland. *New Englander* **6**, 457–473 (1848).
49. Center for International Earth Science Information Network. *Where the Poor Are: an Atlas of Poverty* (Columbia University, Palisades USA, 2006).
50. Farmer, P. E., Nizeye, B., Stulac, S. & Keshavjee, S. Structural violence and clinical medicine. *PLoS Med*. **3**, e449 (2006).
51. Solar, P. M. The great famine was no ordinary subsistence crisis. In *Famine: The Irish Experience, 900–1900* (ed. Crawford, E. M.) 112–133 (John Donald Publishers, Inc., Edinburgh, 1989).
52. Schumann, G. L. & D'Arcy, C. J. Late blight of potato and tomato. *The Plant Health Instructor* [online], http://www.apsnet.org/education/LessonsPlantPath/LateBlight/default.htm (2000).
53. Large, E. C. Measuring plant disease. *Annu. Rev. Phytopath*. **4**, 9–26 (1966).
54. Crawford, E. M. Dearth, diet, and disease in Ireland, 1850: a case study of nutritional deficiency. *Med. Hist*. **28**, 151–161 (1984).
55. Goodwin, S. B. *et al.* Genetic change within populations of *Phytophthora infestans* in the United States and Canada during 1994 to 1996: Role of migration and recombination. *Phytopathology* **88**, 939–949 (1998).
56. Greenwood, B. M., Bojang, K., Whitty, C. J. M. & Targett, G. A. T. Malaria. *Lancet* **365**, 1487–1498 (2005).
57. Thomson, M. C. *et al.* Malaria early warnings based on seasonal climate forecasts from multimodel ensembles. *Nature* **577**, 576–579 (2006).
58. Thomson, M. C., Mason, S. J., Phindela, T. & Connor, S. J. Use of rainfall and sea surface temperature monitoring for malaria early warning in Botswana. *Am. J. Trop. Med. Hyg*. **73**, 214–221 (2005).
59. Cane, M. A., Eshel, G. & Buckland, R. W. Forecasting Zimbabwean maize yield using eastern equatorial Pacific sea surface temperature. *Nature* **370**, 204–205 (1994).
60. Yasuoka, J., Levins, R., Mangione, T. W. & Spielman, A. Community-based rice ecosystem management for suppressing vector anophelines in Sri Lanka. *Trans. R. Soc. Trop. Med. Hyg.* **100**, 995–1006 (2006).
61. Yasuoka, J., Mangione, T. W., Spielman, A. & Levins, R. Impact of education on knowledge, agricultural practices, and community actions for mosquito control and mosquito-borne disease prevention in rice ecosystems in Sri Lanka. *Am. J. Trop. Med. Hyg*. **76**, 1034–1042 (2006).
62. van der Hoek, W. How can better farming methods reduce malaria? *Acta Tropica* **89**, 95–97 (2004).
63. Deming, A. H. *The Edges of the Civilized World* (Picador, New York, 1998).
64. Myers, N. & Kent, J. (eds) *The New Atlas of Plant Management* (University of California Press, Berkeley, 2005).

Acknowledgements
I greatly appreciate the helpful comments and encouragement that were generously provided by A. Brandt, C. Rosenberg, H. Scholthof and undergraduate students in my course 'Pathogens, the Environment and Society'. The Aaron Morris & Anne E. Goldberg Memorial Fund helped to support travel and research associated with this project.

Competing interests statement
The author declares no competing financial interests.