7

The Diffusion of the Black Death and Today's Global Epidemics

It was the eminent mathematician Daniel Bernoulli (1766) from Basel who had the idea of describing the development of an epidemic using a differential equation, in particular with the aim of showing how the epidemic could be mitigated by vaccination. Bernoulli calculated the risk of dying from the vaccination against pox in his day as 1 to 200 and set it in relation to the expected prolongation of life of 3 years and 2 months. To Bernoulli's surprise, people resisted vaccination; they preferred the probability of dying earlier to the risk, though low, of dying immediately. Clearly, Bernoulli was not as brilliant as a psychologist as he was as a mathematician: he could not understand that people have no idea of probability, a concept too abstract for a non-scientist.

We face this phenomenon in modern times; most people are sceptical about nuclear power because people died as a result of the Chernobyl catastrophe. According to the World Health Organization (WHO), 25 people died whose names are known; other sources give a few hundred.

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This is the total mortality burden of nuclear power in Europe since 1950. Nevertheless, people prefer fossil energy, though it is known that its emissions kill many thousands per year in Europe through various diseases. But this is an abstract danger, and the victims cannot be traced directly to the exhaust of a specific power plant. Probability is no criterion for the average citizen.

Bernoulli considered the temporal development of an epidemic, but neither he nor later researchers on diseases considered modelling their spatial spread using mathematical methods. It was the American physicist J. V. Noble in 1974 who first got involved with this problem. He considered a plague that terrified people in Europe, the Black Death.

7.1 The Black Death

In December 1347, the first cases of the Black Death were diagnosed in southern France. It can be assumed that the contagion was imported from the Levant, where it had already raged for some time. It died out after a few weeks wherever it raged, only to reach a new climax a few kilometres away. Three years later, it had arrived at Europe's outermost limits and died away (Fig. 7.1). But during these 3 years 20% of Europe's population, that is, 20 million people had died.

While still doing its work in one place, the Black Death moved on to the next. A few kilometres further on, it found people who were still healthy and ready for contagion and there did its work until in the new place as well the susceptible people had died and others were healed and now immune. The spread of the Black Death in Europe right up to the far north was the typical progress of a diffusion front as described by Fisher when talking about the spread of an advantageous gene, that is, a gene with

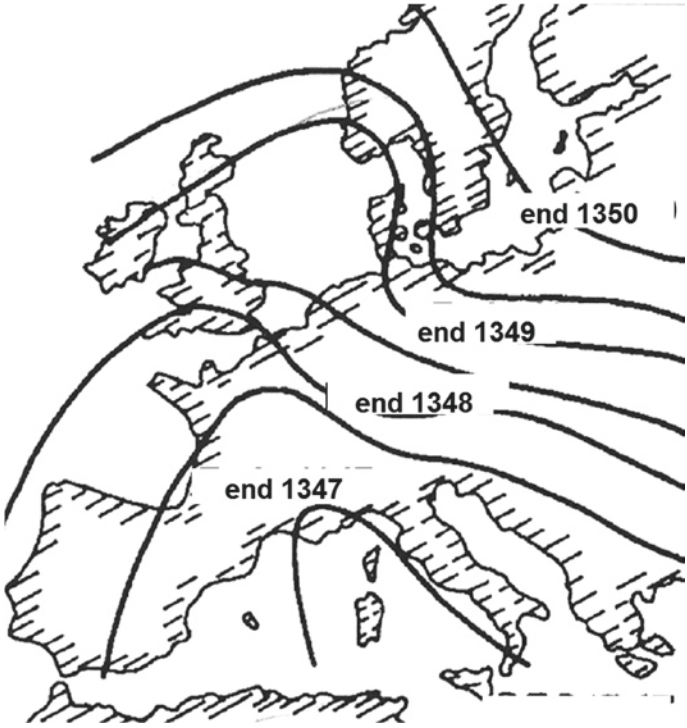

Fig. 7.1 The spread of the first wave of the Black Death in Europe since Roman times from 1347 to 1350. Redrawn from (Langer 1964)

higher advantage (Chap. 4). Noble was the first to describe this spread of an epidemic using equations (Noble 1974). His treatment is based on reaction–diffusion, i.e. Noble stands on the shoulders of Fisher, as yet without simulations at that early time of computer applications.

Noble points out that, because of people's great fear, no more extensive human emigration was tolerated, meaning that no human drift over larger distances took place, unlike the purposefulness that might argue against simple diffusion in the case of the Neolithic farmers migrating

from the Near East to Europe. Therefore, the spread of the Black Death is in fact a classical diffusion process that can be described with equations. It is one step more complicated than the spread of a growing population, as found, for example, in the case of the oak tree, since the spread of the epidemic implies 'finding' and increasing the number of infected people until the point at which these have either died or been healed.

The infection spread across Europe just like Fisher's wave of advance which we have got to know in Chap. 4. Fisher's growth term is now the increase in the percentage of infected people. This means that the interesting factor α is not growth by reproduction as for the oak trees or Neolithic farmers (see Chaps. 4 and 5), but rather the rate of infection of still healthy but susceptible people (that is, people not immune from the start). The number of infected people cannot increase to infinity, however, since the number of people is limited, and not every one of them is susceptible. Hence, after a certain amount of time, every susceptible person has been infected, and the epidemic has reached its climax. Afterwards, the plague declines because all susceptible people have either died or else have been healed and are now immune. Noble put forward a reaction–diffusion equation for the infected population similar to that proposed by Fisher about 20 years earlier for the spread of an advantageous gene. Noble replaced Fisher's growth rate α of the advantageous gene with the growth rate α of the infected population (which he described as the product of the susceptible population and the rate of infection). In addition, Noble had to consider that a proportion of the infected persons would die and that others would recover. Therefore, the epidemic would die out after some time.

All this can be read from Fig. 7.2, a computer simulation with somewhat realistic parameters.

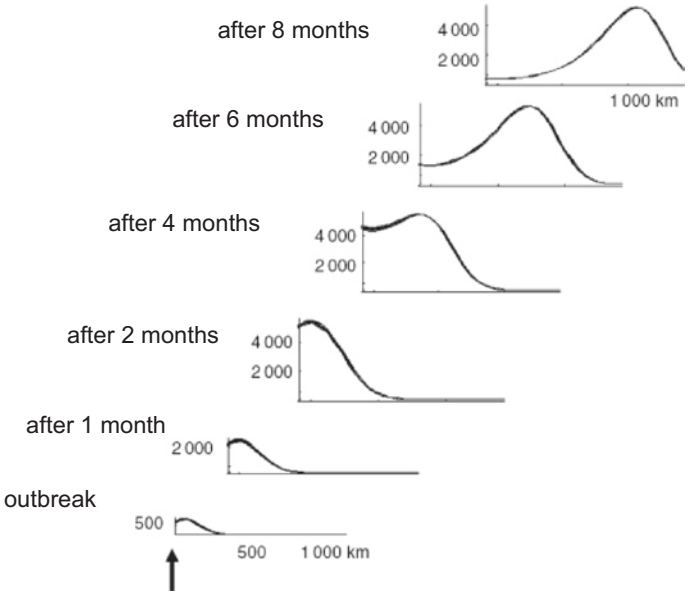

Fig. 7.2 Schematic representation of the number of infected people per 1,000 km^2 at different times and at different distances from the place of outbreak (arrow) of an epidemic. For simplicity's sake, one-dimensional spread is assumed. It is evident that the number of infected people at the place of outbreak first increases (here from 500 infected per 1,000 km^2 to 2,000 after 1 month and 4,000 after 2 months) and then declines again. After 4 months, the climax of the epidemic is already 500 km away from the place of outbreak and 1,000 km away after 8 months. At the place of outbreak, the epidemic is then already extinct

Extensive reports exist about that first wave of the Black Death in medieval times, but Noble still had to make a couple of assumptions regarding the preconditions for a person to fall ill. Noble's assumptions are daring, perhaps unsustainably so, and therefore, we will look at them a little more closely.

The bacillus of the Black Death is transferred from sick persons to healthy persons by fleas, which by preference

live on rats and other rodents; it is sometimes also transferred directly from person to person. The infected persons, if susceptible, fall ill and either die or become immune after having survived the illness. The Black Death has then 'consumed' the region and loses its strength. To make an estimation of the area in which a sick person can infect others, Noble assumed that people move on the average with a speed of 1–2 km per hour and that fleas jump over a distance of 2 m to their next victim, with a chance of infection of 10%. Noble attained a 'cross section' of an infected person for infecting others of about 1 km^2 per year. In plain text, this implies that during the time of her/his illness a person will infect everybody who sojourns in this area and is susceptible.

Noble investigated further whether the spread velocity of the Black Death was compatible with other 'diffusion speeds' of people in Europe in medieval times, which were characterized by their radii of interaction with other people and with the contemporary population densities, assuming that 85 million Europeans were alive in 1347. Now comes one of those drastic simplifications characteristic of and necessary for calculations of historic events because, as emphasized earlier, in the humanities, unlike in the exact sciences, we cannot modify the real parameters, since they were fixed in the past. Noble assumed that people were equally distributed over Europe, thus arriving at an average population density of 20 people per square kilometre. And Noble assumed further, based on historical reports, that rumours and gossip spread in Europe with a speed of 100 miles per year in the manner of a random walk and that the Black Death spread at the same speed, since it also relied on personal contact. With Einstein's formula for movements on the surface of the

earth (Chap. 3) Noble received a value for the diffusivity. He admitted that information on important events such as the outbreak of a war and the death or the coronation of a king or emperor will have spread much faster.

Based on all these assumptions, which Noble, of course, openly declared to be 'educated guesses', i.e. reasonable estimates, Noble calculated the velocity of the wave front of the spread of Black Death, using Fisher's equation for the velocity of the wave of advance $v = 2\sqrt{D \cdot \alpha}$ (Chap. 4). Noble came up with $v = 700$ km per year, in satisfactory agreement with the speed of spread as derived by historians.

Here we recognize an essential difference from the computer simulations on the spread of ragweed (Chap. 4) or of modern languages (Chap. 8). For these investigations, most details are known and to neglect them would be shameful. All the complexity of the real situations is apparent and to deal with them with only simple reaction–diffusion equations would not fulfil the requirements, whereas computer simulations can better deal with the complexities (Prochazka and Vogl 2017). Descriptions of spread problems in the past, on the other hand, can be dealt in this way, since only a few parameters of these processes are known. An educated guess, such as those made by Noble or by Ammerman and Cavalli-Sforza for the demic diffusion in the Neolithic (Chap. 5), is in order but, of course, yields only rather general information.

7.2 The Spread of Epidemics in the Twenty-First Century

7.2.1 Random Walk

In modern times, long-range travel of invading neophytes or infectious diseases cannot be dealt with using Fisher equations with just one wave front. Just as with the spread of invasive plants or animals from various centres (Chap. 4) or of language spread (Chap. 8), methods have to be applied that search in a random way. Figure 7.3 compares temporal snapshots of simulated spread processes of ragweed from Richter (2013a) and of an epidemic from Brockmann (2018) in order to demonstrate that the spread of neophytes and of diseases can follow similar patterns. Both infestations, by neophytes and by bacteria, can propagate randomly from non-connected areas.

7.2.2 Long Distance Spread

As argued by Chapman et al. (2016) (see Chap. 4) for invasive plants (neophytes), repeated introduction to multiple areas and by multiple trade pathways is a feature of many invasions and leads to dispersal from newly emerging nuclei. Brockmann (2018) considers the same mechanism for the propagation of infectious diseases. Through air transport of people, the spread of epidemics over long distances appears even more probable than the spread of invasive plants, which in principle could be drastically mitigated. The possibility of transmitting diseases to distant locations can yield new seeds far away that can eventually become the nuclei of new waves of propagation.

This implies that the distance from an initial outbreak location can no longer be used as a measure for estimating

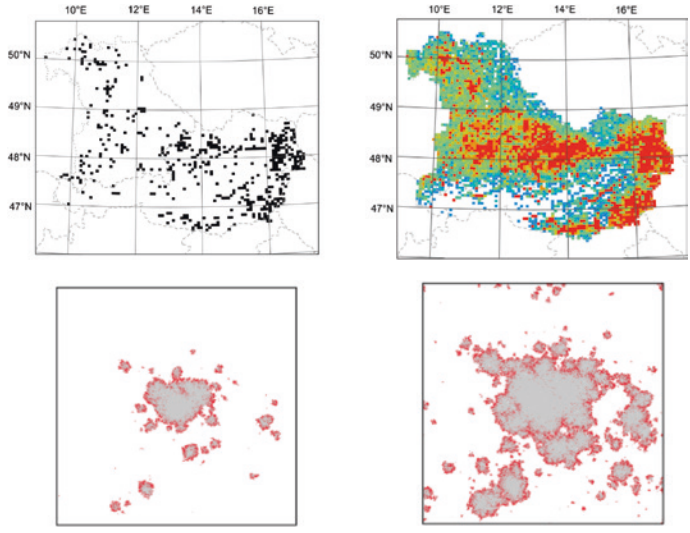

Fig. 7.3 Random spread of infestation. Top row: model of ragweed infestation of Austria and Bavaria in 2005 (left) and as predicted for 2050 (right). In this model, a once infested cell will never recover. Transmission occurs not only to nearest-neighbour sites but also to farther sites, though with a lower probability controlled by a distribution similar to a Gaussian (Richter et al. 2013a, Richter 2015, see also Chap. 4). Bottom row: model of the spread of a disease. An infected site (red) can transmit an infection to a susceptible (white) neighbouring site. Left, earlier point in time, right, later time. Please note that in this model, infected sites recover and become immune (grey). Also here, in addition to nearest-neighbour transmission, with a small probability, a transmission to a distant site can occur (Brockmann 2018)

or computing the time that it takes for an epidemic to arrive at a certain location. From Fig. 7.4, it can be seen that the dispersion of SARS, the severe acute respiratory syndrome, an epidemic caused by a virus, did not progress by means of a geographical wave. It sprang up in China and infected victims in the USA and in Europe earlier than in geographically much closer countries such as Korea or India. To treat traffic and consequently infections

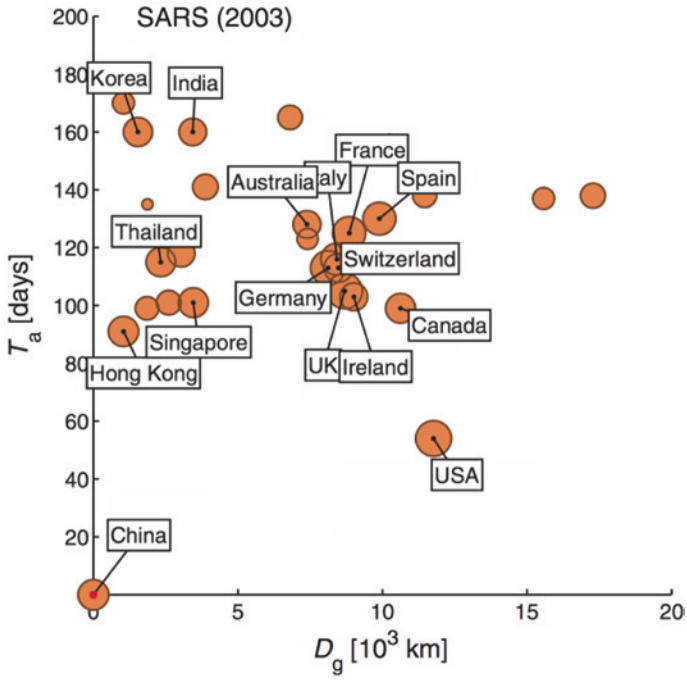

Fig. 7.4 Arrival time T_a of the SARS epidemic in 2003 as a function of distance D_g from the initial outbreak location in China. Note that the epidemic arrived earlier in USA and Europe than in Korea or India. (Brockmann 2018)

spreading across many locations worldwide, a new idea is needed: Brockmann (2018) replaces the diffusion fluxes by the probabilities that susceptible, infected and recovered persons move from one location to another one and back.

To treat such complex questions relating to motion from and to many nodes, Brockmann and Helbing (2013), Brockmann (2018) recently developed the idea of replacing the traditional geographic distance with the notion of an effective distance derived from the structure of the global air-transportation network. If two locations in the air-transportation network exchange a large number of passengers, they should be effectively close because

a larger number of passengers imply that the probability of an infectious disease being transmitted between them is comparatively higher than if these two locations were linked only by a small number of travelling passengers. This leads the authors to define the shortest path between each pair of airports. Figure 7.5 depicts the shortest path tree

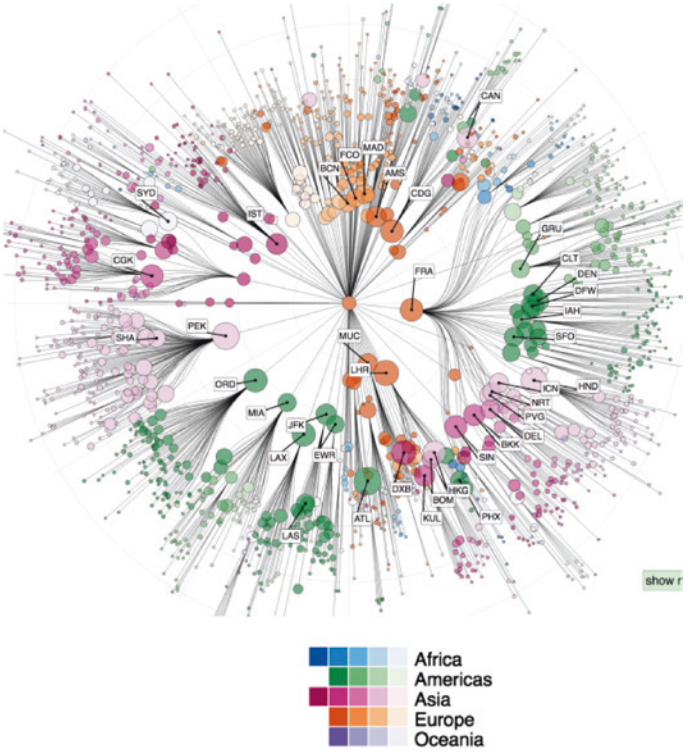

Fig. 7.5 Shortest path trees and effective distance from the perspective of Tegel airport (TXL) in Berlin. TXL is the central node. Radial distance in the tree quantifies the effective distance to the reference node TXL. Large European airports such as Frankfurt (FRA), Munich (MUC) and London Heathrow (LHR) are effectively close to TXL. However, some airports that are geographically distant, such as Chicago (ORD) and Beijing (PEK), are actually effectively closer than smaller European airports. (Brockmann 2018)

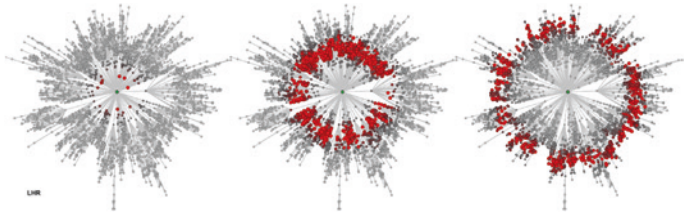

Fig. 7.6 Temporal snapshots of computer-simulated hypothetical scenarios corresponding to an epidemic initially starting at LHR (London). The networks depict the shortest effective distance to the corresponding seed airports. (Brockmann 2018)

for the Berlin airport Tegel, TXL. You can see that large European airports are effectively close to TXL, as you might expect. However, large Asian and American airports such as Beijing (PEK) and New York (JFK) are also comparatively close to TXL. Because the shortest path tree also represents the most probable routes for spreading, this method can be used to identify airports that are particularly important in terms of distributing an infectious disease throughout the network.

Figure 7.6 employs the effective distance and shortest path tree representation from the perspective of the outbreak location of an epidemic. Similar to a reaction obeying simple reaction–diffusion dynamics, which spreads as a wave of advance with constant speed over distance, infection processes spread at a constant speed over *effective* distance (Brockmann 2018). One can predict the arrival time of an epidemic wave front, knowing the speed of the effective wave of advance and the effective distance. For example, if the spreading commences shortly after an epidemic outbreak and the initial spreading speed is determined, arrival times can be forecast.

7.3 What Has Changed with Globalization?

Just as I was writing these lines, I read an item in the newspaper about a tragic case: a 4-year-old girl in hospital in Italy was possibly bitten by a mosquito (anopheles), which had presumably escaped from a trunk belonging to patients just arrived from the Tropics. The girl died because the doctors could not imagine that her disease was malaria and so did not give her the correct treatment. Transport over large distance is a modern phenomenon, whereas in medieval times—with the exception of the crusades or long journeys by merchants such as Marco Polo—people moved by random walk in a very limited area.

Globalization has completely revolutionized the spread dynamics of diseases: whereas 700 years ago it took a ship arriving from the Levant to import the Black Death, which then needed several years to spread over Europe, today the outbreak of a global disease may be a matter of days. On the other hand, the mitigation of the Black Death was impossible, the numerous 'plague walls' all over Europe proving useless, whereas computer simulations such as those of Brockmann may be expected to lead towards models that might accurately predict contagion patterns and thus prevent the invasion.

Brockmann and Helbing suggest that it seems promising to extend the effective distance method to other contagion phenomena, such as human-mediated bioinvasion or the spread of rumours or violence promoted by the Internet and mobile devices, a subject of evermore importance in an increasingly connected society. That human transport of seeds appears indeed as the trigger of the invasion and of the future geographical progress of invasive species is already considered by Chapman et al. (Chap. 4). The contagion leading

to violence and the concomitant spread of violence are by now known to everyone as a globally connected plague.

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