The Nature and Classification of Diseases. Alan Macfarlane

There is a tangled web of relationships between the various actors in the disease drama. There is firstly the disease entity. These are a variety of organisms. The major distinction is between the smallest of these, viruses, the next size up, protozoa, and the largest, the bacteria. Each of these have different properties. The viruses, for instance, are so small that they can float in the air and can hence be transmitted in the form of droplets, through the respiratory tract. Each of them can survive for a different length of time outside the host. It has been suggested that the longer they can survive, the higher the death rate they cause - perhaps partly because it is in a Darwinian sense in the 'interest' of short survivors not to kill their hosts. Thus smallpox, which kills up to one in ten of its victims, 'can survive for more than a decade outside of the host.' The 'agents of tuberculosis and diphtheria can survive for months and are correspondingly severe.' The 'remaining pathogens tend to survive for a period of hours and days and tend to cause lethality less frequently.'[1]

A second feature of viruses is that because they are so simple in structure and replicate so fast, they are constantly mutating and taking new forms - as has been discovered with influenza or measles. This is important to bear in mind when we try to account for the sudden fluctuations in viral diseases. The relationship in this case is the relatively simple three-way one of organisms - host - environment, which is constantly changing over time.

The viruses, however, do not act alone. They also 'enter into relationships' with the relatively large micro-organisms, for instance the bacteria. This added complexity is well described by Burnett. In the case of the influenza epidemic of 1918, 'It seems as if a very active virus swept over the whole world, finding almost all individuals susceptible to it, and in its passage made all sorts of temporary alliances with pathogenic bacteria spread by the same respiratory route. The virus initiated the illness in every case, but when a fatal outcome resulted it was almost always the bacteria which were finally responsible.'[2] There is thus in any relatively dense human population 'a constant interchange of the viruses and bacteria which can occupy the upper respiratory tract.'[3] It is often impossible to specify what is the 'cause' of death; a pack of causes is at work.

When we move to the micro-organisms that cannot live in the air, it is useful to distinguish three actors - the **carrier** (the infected individual), the **vector** (the agent which bites or deposits the micro-organisms) and the **vehicle**, for instance the water or food through which it is transmitted.[4]

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[1] Ewald, Infections, 63

[2] Burnett, Infections, 122

[3] Burnett, Infections, 133

[4] Harding, Epidemiology, 156

Another way of looking at this as a three-way relationship between host, parasite and environment.[5]

Now these relationships are extremely complex and also contentious. Take the relationship between parasite and host. It was one of the triumphs of epidemiology in the first half of this century to show that it was not in the interest of the parasite to kill its host. Indeed, the best situation was, as in slavery, a form of mutual accommodation. A dead slave is a bad slave and a dead host is a tragedy for a parasite. As Burnett concluded, 'It is very evident that when a parasite and its host have lived together for very many generations the association is a balanced one with little evidence of damage to the host.'[6] Consequently 'In a well adjusted host-parasite relationship, subclinical infection is the rule, disease the exception, and death a rarity.'[7] The corollary of this was that many diseases would start as very serious for humans and then grow milder. Mankind would blunder into a disease chain which had become balanced in some other species. Various 'stories of human intrusion into rickettsial ecosystems illustrate the frequent finding that many of the most lethal infections of man are ecologically infections of other vertebrates - or of insects - which reach man only by accident.'[8] After a while host and parasite would come into balance. This view has, however, been challenged and a less benign, Darwinian, hypothesis suggested. Ewald writes that Dubos 'concludes that "given enough time a state of peaceful coexistence eventually becomes established between any host and parasite" (Dubos 1965). In this book I have argued that even if enough time is given, warfare between a vicious parasite and a defensive host may sometimes be a normal manifestation of the constant interplay between host and parasite.'[9] What the two views have in common is that there is a deep mutual bond and an always-changing relationship between hosts and parasites.

There are thus commonly five major actors in any disease drama. There is the micro-organism, there is the host in which it lives, often an animal (human or otherwise) also known as the carrier. There is the **vector** which transmits the disease, for instance a flea or tick, there is the **vehicle**, water, food, dust or whatever, through which disease is transmitted, and there is the environment in which they exist. Not only are the relations between these complex, but each is more complicated than it looks. For example 'vectors' are not merely small insects or animals, but there are 'cultural vectors' as well as material ones. This idea is developed by Ewald. 'I refer to this process as **transmission by a cultural vector**. Specifically, I define **cultural vector** to be a set of characteristics that allow transmission from immobilized hosts to susceptibles when at least one of the characteristics is some aspect of human culture.'[10] He gives the example of waterborne transmission of diarrheal pathogens, which is clearly

[5] Kiple (ed), Diseases, 1029

[6] Burnett, Infections, 50

[7] Burnett, Infections, 120

[8] Burnett, Infections, 147

[9] Ewald, Infections (xerox), 189

[10] Ewald, Infections (xerox), 68

deeply affected by such 'cultural vectors' as drinking, cooking and sanitation habits.

In terms of hosts, the most important hosts of disease are animals. Here it is worth noting that research suggests that 'It may well be that animals, domestic and wild, form a more important reservoir of disease than has been realized.'[11] This is one of the reasons why the domestication of animals about twelve thousand years ago led to the emergence of many new diseases among humans.

Given these sets of relationships, the alteration of any one can alter the whole balance. The major efforts by human beings have been directed at three points in these chains. 'There are: first, preventing the entry of the parasite by quarantine measures, secondly, interruption of the chain of transmission by what can be broadly called environmental sanitation and thirdly, protection of the susceptible individual by immunization or chemoprophylaxis.'[12] The success of the method will depend on the nature of the chains in question, since each is different. With a slight realization of how complex a web of causation we are analyzing, I would now like to try to distinguish some of the major chains of disease.

The virulence of infective organisms and their hosts.

Another possibility is a change in the 'virulence of the infective organism and its host...'[13] Two types of argument could be put forward here. The first suggests that autonomous changes, for instance in the virulence of a virus or bacteria, may alter the pattern of mortality. This argument has been suggested most frequently to explain the sudden and inexplicable disappearances of diseases, for instance of leprosy in western Europe from the fourteenth century, of the English sweating sickness in the middle of the sixteenth, of plague from most of Europe after 1666, or of a number of diseases in Europe in the later part of the nineteenth century. The mysterious disappearance of a number of major diseases was alluded to by Creighton and more recently by Greenwood who wrote of the mysterious disappearance of tuberculosis and scarlet fever in the later nineteenth century.[14]In relation to bubonic plague, as we have seen, it has been suggested that its sudden disappearance may have been due to changes in the behaviour of the rat, or flea, which had nothing to do with human intervention. As Chambers notes, 'If this is true, it is perhaps the most gigantic example of good luck in the recorded history of mankind: the dietetic peculiarities of the free-ranging flea, apparently enabled the industrial Revolution to proceed on its way.'[15] Zinsser writes 'It is not easy to account for the decline of great epidemics in Europe after

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[11] Harding, Epidemiology, 169ff; table p.167

[12] Burnett, Infections, 159

[13]McKeown and Brown in ed. Heer, 37

[14] Creighton, Epidemics, i,280; Greenwood, Crowd Diseases, 65.

[15]Chambers, Economy, 151

1850. One might assume an unaccountable cyclic change in the characters of prevalent diseases.'[16]

While not discounting the possible changes in viruses and other micro-organisms due to a number of complex factors, it is probably safe to leave this "X" factor on one side for the moment. In general, McKeown and Brown are probably right in arguing that 'although there have undoubtedly been changes in the character of individual infections, it is unreasonable to attribute to this alone the progressive decline in mortality from infections as a whole after many centuries in which mortality remained high.'[17] Likewise, as Kunitz argues, 'Certainly there were adjustments between parasites and hosts, but it is unlikely that either the waning of virulence of the former, or the rapid selection for resistance of the latter, are adequate explanations of the decline in European mortality.' In relation to inherited resistance, Kunitz states that there is 'very little evidence from recent epidemiological studies that inherited resistance is significant in any infectious disease, with the exception of the association between the haemoglobinophies and malaria.'[18]

It seems unlikely that changes in virulence of infective organisms and their hosts will solve many of the particular problems in relation to England and Japan. Like the climate, we need to keep this factor in mind, and to realize that it may well have been important. The more we learn, the more we appreciate the complex and constantly evolving situation in the relation between mankind and the surrounding world of micro-organisms.

There are inter-actions between different diseases so that it is possible, for instance, that as one increases it may lead to a decrease or increase in others. This was a point noted by Creighton on several occasions. He showed for instance that as typhus declined, typhoid rose, or as measles increased, smallpox declined. [19] This synergy of diseases has recently been noted by Cohen, who shows how the spread of malaria and hookworm increases measles. [20] The implication of this is that we have to study all the major diseases alongside each other since, for example, the absence of malaria is not just important in itself but influences many other diseases. Also, a long time perspective is needed in order to notice the patterns. As Creighton wrote, 'In the long period covered by this history we have seen much coming and going among the epidemic infections, in some cases a dramatic and abrupt entrance or exit, in other cases a gradual and unperceived substitution'. [21] This leads him to his principal theory when

[16]Zinsser, Rats, 292

[17]McKeown; Brown in ed. Heer, Population, 38

[18]Kunitz, Speculations, 364, 250

[19] Creighton, Epidemics, ii,202, 629,659.

[20] Cohen, Health, 54.

[21]Creighton, Epidemics, ii, 631.

trying to explain the mysterious disappearance of diseases like sweating sickness or plague, namely 'the only law of extinct disease-species which our scanty knowledge points to - the law of succession, or superseding, or supplanting of one epidemic type by another.[22]

The classification of diseases.

Our success in explaining why mortality assumed an unusual pattern in England and Japan will depend very much on a satisfactory classification of diseases. The explanation of why certain diseases became more or less virulent will clearly rest on understanding how they are spread or contained. For instance, since smallpox is a virus which spreads by way of the respiratory tract, it is unlikely to be affected by changes in clothing, whereas epidemic typhus is a bacillus whose vector is a human flea and hence changes in clothing and washing habits will have an enormous effect. The general point is made by Post. 'Human infections can be grouped into two major categories: those derived from the external environment and from other species, like bubonic plague; and those caused by primary human pathogens, like small-pox. Infections falling into the first category can be prevented in most instances by environmental sanitation and control of vectors.'[23] This is the right approach but the classification needs to be subtler. Each of these categories has several different branches.

In the following analysis I will follow the classification suggested by Macfarlane Burnet in his 'Natural History of Infectious Diseases', when he considers in chapter eight 'How infections spread. He distinguishes between:

A: Digestive tract diseases, spread by faecal matter: cholera, typhoid, dysentery.
B: Bites of animals, insects or ticks: plague, typhus, malaria. C: Respiratory route, or 'droplet' infection: smallpox, measles, tuberculosis.

[22] Creighton, Epidemics, i, 280.

[23] Post, Modernization, 35