practice to increase the potency of the original vaccine by occasionally adding variola virus. This derivation could also result from vacccination in smallpox hospitals.

Razzell's statement (p. 228)'. . . it has been impossible to infect cows with smallpox virus *i.e. produce cowpox*' (my italics)—his incorrect interpretation of Downie (p. 225), is hardly scientific. An objective experimenter would first try to infect cows with variola, and then if successful would investigate the properties of the virus produced. Jenner himself said:

> They who are not in the habit of conducting experiments may not be aware of the coincidence of circumstances necessary for their being managed so as to prove perfectly decisive.
>
> (*Inquiry*, . . . 2nd ed., 1800, p. 44)

Razzell has 'not disputed the power of cowpox to protect against smallpox.' That being so, and assuming he does not dispute the power of vaccinia to so protect, one questions the reason for the plea for inoculation in his last paragraph (p. 229). If he is advocating arm-to-arm inoculation, then conventional vaccination is simpler, at least as safe, less objectionable to vaccinees, and removes any danger of variola becoming attenuated by arm-to-arm passage. If he is advocating conventional 'vaccination' using variola, he forgets the danger involved in producing such a vaccine and the difficulty entailed in producing large quantities of a virus with such a limited host range.

To re-place the emphasis on Razzell's last sentence: a *great deal* of both observation and experimentation is necessary before smallpox inoculation can be used in certain limited circumstances.

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A CONSIDERATION OF THE NATURE OF THE ENGLISH SWEATING SICKNESS

Professor Patrick has put forward the interesting idea that the 'sweat' was not an infectious disease but the result of mass food poisoning by fungi or some other contamination of cereals (*Med. Hist.*, 1965, **9**, 272–279). This explanation admittedly fits the descriptions of the symptoms of the fever, but many difficulties are thereby left unresolved.

The first problem raised by this idea of food poisoning relates to the weather and harvests, because the years of the 'sweat'—1485, 1508, 1517, 1528 and 1551—show no common factor likely to cause such contamination. The weather of 1485 was wet, but the harvest was good; in 1517 an equally good harvest followed a mild winter and a dry spring. The years 1528 and 1551, on the other hand, witnessed bad harvests owing to heavy rains.[1] Even if it is accepted that these diverse conditions could all have caused the same sort of contamination, it is still difficult to see why the incidence of the 'poisoning' should have been limited to periods of a few weeks on each occasion, and yet spread so rapidly from one area to the next. Poisoning surely would have led to sporadic outbreaks which were scattered but lasted as long as the year's food supplies. Indeed the outbreaks would be expected to show a clear seasonal relationship with particular harvests, whereas in fact there was considerable variation in the time of onset; the sweat of 1551, for example, began in early spring, whereas that of 1485 began in the autumn. Then there is also the question of the complete absence of any reference to the contamination—a point clearly made in the reply to Willan which, incidentally, was signed 'Critical' and not 'Inquirer'! The quality of the harvest was the most important concern of the year to the majority of people at that time and someone would surely have noted any mould or fungi in the years of the 'sweat' as was done for example, on the Continent in the years 1500–1503. In fact such contamination was always fearfully watched for, as the 'blood spots', as they were called, were regarded as harbingers of disaster.[2]

Thus the evidence in favour of the 'poisoning' explanation appears to be weak, and the fact that it was based on a logical deduction from the descriptions of the symptoms does not alter this conclusion. The five outbreaks on which this deduction was based have, historically, been treated in a highly selective way in order to make them fit into what was in effect already a 'poisoning' theory.

In the Tudor period diseases were attributed to the effects of some sort of harmful miasma, and the difference between diseases was taken to be little more than a difference in outward symptoms. Under such a system, a disease such as the 'sweat' could only be peculiarly English if there were some particular local conditions not found elsewhere. Hence Erasmus came to the conclusion that the dirty state of English houses, which he had always disliked, gave rise to exhalations which caused the 'sweat'.[3] Caius on the other hand saw the cause in a combination of strange circumstances, such as conjunctions of certain planets and unnaturally dry winter weather in a country usually so damp.[4] Popular superstition often added to these 'localist' theories the idea that only Englishmen were affected, and so the 'sweat' remained 'English' and limited to the five traditional outbreaks in spite of the fact that the whole of Northern Europe suffered from a disastrous epidemic in 1529.[5]

There has, however, always been evidence that this traditional view was inaccurate. Observant contemporaries such as Fernel and Jordanus, for example, spoke of the 'sweating' fevers which swept not only England but also the rest of Britain, the Low Countries, Germany and even France, and not just in one year but throughout the period 1525–1530.[6] There is also some evidence that Ireland suffered again in 1543 and the Low Countries in 1551.[7]

This idea that the English 'sweat' had swept the whole of Western Europe in a series of outbreaks was accepted by the early historians of epidemics who in the eighteenth century attempted to make a meaningful synthesis of scientific observations of the weather and the literary descriptions of diseases of the past.[8]

At the same time as this meteorological approach to a rational history of disease,

there was a similar tendency to look for the causes of disease in simple poisons which acted directly and could be identified, rather than the miasmatic poisons which were more diffuse. This approach seems to have been popular in Northern Europe where the action of ergot was well known, and it was of particular usefulness in explaining those diseases of the nervous system like encephalomyelitis which appear to have accompanied influenza epidemics. Thus Rothman after the influenza of 1762 came to the conclusion that radish seeds were the cause of the stuporose, delirious, paralytic and convulsant fevers which were henceforth distinguished by the name of Raphania; and Kerner in 1820 in the middle of a series of influenza outbreaks, often described as 'sweating sicknesses', attributed similar symptoms to poisoning by Swabian sausage and in this way helped begin the legend of a widespread disease known as 'botulism' (properly allantiasis, not 'botulism' due to *Clostridium botulinum*).[9]

Both of these interpretations were to receive their fullest elaboration in the influenza epidemic of the 1840s at a time when the supporters of the rival 'miasmatic' and 'contagionist' theories were locked in battle. We are lucky that Prus's review of plague (1846) is almost matched by Corradi's compilation for the Italian influenza epidemic of 1842. Being merely a part of a larger work this compilation is not very critical but it does illustrate the continuity of medical ideas: Dr. Agostinacchio thought that the form of influenza that he saw was the 'English sweat', whereas Dr. Semmola thought that it was Raphania![10]

With the rise of the germ theory of the disease the 'miasmatic' and direct poisoning interpretations generally lost support but the idea that the 'sweat' was peculiarly English and restricted to five outbreaks has lingered on despite the attempts by Hamer and Crookshank to fit the 'sweating sickness' into the known pattern of influenza epidemics. The reason for this is the continuing influence of the standard works on the history of epidemics which were written in the nineteenth century before the fullest development of the germ theory. The collection of references to the 'sweat' by Gruner and Haeser remained the basis on which later writers like Hirsch and Hecker relied, and these German sources largely determined the framework within which Creighton was to treat the outbreak of 'sweating sickness' in his standard *History of Epidemics in Britain.*

The common feature of all these influential German writers was their firm adherence to localist-miasmatic explanations for disease.[11] Indeed in order to bolster the uniqueness of the 'English' sweat they were not only obliged to emphasise the peculiarities of the English climate (in distinction to that of Scotland and Wales!), but they were also encouraged to innocent racialist and mystic ideas; thus even Hecker, who realised that the 'sweat' occurred at the same time as other ill-defined epidemics in Europe, spoke of the 'gluttony' of the English, the 'spirit of the mist' and 'mysterious agencies on the domain of organised being'.[12] Creighton's own interpretation was more rational than this but he accepted the uniqueness of the 'English sweat' the cause of which he found in the peculiarities of behaviour of ground water in England'.[13]

Today there is a tendency to accept the facts given by Creighton and his German sources and lightly dismiss their theories without realising that the theory determined the choice and treatment of the facts. It was not until there had been full acceptance of the germ theory, and indeed some appreciation of its over-simplifications, that Hamer and then Crookshank began to question the choice and treatment of the facts of the history of the 'English sweat'. There is no no need here to relate all their arguments

which were part of a wider thesis on the 'epidemic constitution', but their explanation of the 'sweat' can be summarised briefly:

> The 'sweating sickness' was but one form taken by influenza which was sweeping across Europe in epidemics at that time; thus the Venetian Ambassador referred to the 'sweat' of 1551 as 'questo influsso'.[14] There is no reason to suspect that there was anything peculiarly English about the 'sweat', and we have already seen that under the name of either English sweat or 'sweating fever' it did attack other parts of Europe. Even countries like France which are reputed to have escaped the continental epidemic of 1529 did in fact then suffer from a disease known as 'Trousse galant'—a term almost equivalent to the nickname of 'stop-gallant' used for the 'sweat' in England in 1551.[15]

If this identity with influenza is correct there is no reason to suspect that the 'sweat' could have struck only five times. Thus in 1511 Erasmus referred to the influenza of 1510 with the words 'a sudore illo', and in 1558, seven years after the last outbreak of the sweat Dr. John Jones considered that the fever he had caught was the 'sweating sickness'.[16] Also in Italy in 1507 there was influenza which caused death more rapidly than usual for such outbreaks.[17] Similarly in the influenza epidemic in England in 1775 Glass spoke of the 'plentiful and easy sweat', and he called it a diary fever just as Caius had done two hundred and twenty-four years earlier.[18]

On the one hand, then, there is this evidence of relationship between the 'sweat' and influenza assembled by Hamer and Crookshank, and on the other hand there is the selective interpretation of events by the German school and even by Creighton which leaves us with a disease peculiarly English and limited to five outbreaks. On comparing these two interpretations the weight of historical evidence seems to me to be strongly in favour of that of Hamer and Crookshank. The attraction of the food poisoning interpretation is that it gives an easy and complete solution to a problem which otherwise will always remain open to some doubt. The easy way out of a problem, however, is not always the best and we should remember the courage of Scheffelius and Schleger who in the eighteenth century opposed the growing tendency to invoke food poisoning theories as the explanation of disease at a time when they had nothing as well attested as a germ-theory and influenza viruses to put in its place.

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