The English Sweating Sickness (Sudor Anglicus):
A Reappraisal

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Without warning, first in 1485 and subsequently in 1508, 1517, 1528 and 1551, an epidemic fever, accompanied by profuse sweating, prostration, and death within hours, and of spectacular but highly circumscribed explosiveness, struck parts of England.¹ Only once, during the years 1529–30, did the disease in its classical form, generally speaking, extend significantly beyond English suzerainty. Before and after this one extension into continental Europe, many reports attest that the English alone, at home or abroad, were selectively attacked. The occasional foreigner who did succumb contracted the disease in a mild form. No convincing evidence exists to indicate that sweating sickness occurred in Scotland, Ireland, or Wales.

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Sudor Anglicus, according to contemporary scholars, was characterised by certain features that distinguished it clearly from other pestilences prevailing at the close of the Middle Ages, notably bubonic plague, typhus, and malaria (ague). Plague was clearly recognised at the time as distinct from sweating sickness; it had made many visitations, people were familiar with it, and resigned to its periodical depredations. The sweating sickness of 1485 was 'new' and, in the manner of many 'new' epidemic diseases, disproportionately terrifying. The fact that in some years (in October and November) the plague supervened has led to confusion, although such confusion has usually arisen more from the later compilation of inaccurate indices to texts than to the texts themselves which are quite clear. The disease that we now call influenza was not confused with sweating sickness by contemporaries, but epidemiologists and medical historians, writing in this century and influenced by the great influenza pandemic of 1918–19, introduced this erroneous association.

The five major outbreaks of sweating sickness in England had interepidemic periods of twenty-three, nine, eleven, and twenty-three years. Illness occurred predominantly in high summer (June, July, and, above all, August) and usually ended abruptly in September. The outbreaks were widely scattered and characteristically patchy in their geographical distribution, affecting predominantly rural areas but also London and the university communities of Oxford and Cambridge. Men appeared to have been affected more frequently than women, and amongst men the overall incidence was reported to be highest at the prime of life; young children, the elderly, and otherwise frail tended to be spared—a pattern not infrequently exhibited by some infectious diseases when they first appear in a population hitherto unexposed. John Hoker, contributing to Holinshed's Chronicle, confirms the age incidence of the earliest outbreaks.

It is to be noted, that this mortalitie fell chieflie or rather upon men, and those of the best age as between thirtie and fourtie years. The gredie riddance of life procured by this sickness did so terrifie people of all sorts, that such as could make shift either by monie or friendship, changed their soile, and leaving places of


3. The most important public documents for the reign of Henry VIII (1509–47) are calendared in the Letters and papers, foreign and domestic, of the reign of Henry VIII, 22 vols. (London, 1862–93). References to sweating sickness are generally indexed under the heading 'Plague'; there is little doubt, from the descriptions in the texts as well as the seasonal incidence, when sweating sickness was involved.
concourse, betooke them (for the time) to abodes though not altogether solitary yet less frequented: to conclude, manifold meanes were made for safetie of life.4

The *Sudor Anglicus* seemed also particularly to affect the well-to-do, whose life style enabled them more easily to escape other contemporary pestilences. By contrast with other epidemic diseases, the sweating sickness seems not to have been associated with overcrowding or exceptionally insanitary conditions; indeed, its prevalence was much more marked in the open, sparsely populated countryside than in the densely populated mediaeval towns and cities.

The sweating sickness’s appearance in England in 1485 was first described in print by Thomas Forrestier.

We saw two prestys5 standing together and speaking together, and saw both of them dye sodenly. Also in die proxima we se the wyt of a taylour and sodenly dyed. Another yonge man walking by the street fell down sodenly. Also another gentylman ryding out of the cyte [21 September 1485] dyed. Also many others the which were to rehearse we have known that have dyed sodenly.6

Of the symptoms Forrestier says: ‘And this sickness cometh with a grete swetyng and stynking, with redness of the face and of all the body, and a contynual thirst with a grete hete and hedache because of the fumes and venoms.’ He also mentions ‘pricking the brains,’ ‘and some had black spots, as it appeared in our frere ?Alban, a noble leech on whose soul God have mercy!’ Forrestier avers that the sweating sickness first erupted on 19 September 1485.7 On the basis of Forrestier’s account of a September onset, Charles Creighton developed the idea that the disease originated in the capital and spread outwards to the provinces. There is evidence, however, to contradict both Forrestier’s account and the later theory that sweating sickness was brought by French mercenaries in Henry Tudor’s army, and to suggest that the disease was already present in the north of England in the summer of 1485. No record has been found in the National Library of Wales, or in the archives of Pembrokeshire of the disease’s having broken out either in the army or among the Welsh people at any time during the twenty-one days between Henry’s landing at Milford Haven and the Bat-


5. Early confirmation of the susceptibility of the clergy, whose social status, then as now (but only of the Church of England), was more exalted than their emoluments would suggest.


tle of Bosworth on 22 August 1485—surely a long enough period of incubation for symptoms to have appeared? The first recorded reference during the Wars of the Roses to any combatant’s being affected by the sweating sickness concerns Thomas, Lord Stanley, who, biding his time upon his extensive estates in the counties palatine of Lancaster and Chester, in the process of turning his coat, gave as his excuse to disobey Richard III’s summons to rejoin the Yorkists that ‘he feared that he had contracted the Sweating Sickness.’ This letter was written before Henry Tudor had even made landfall on the Pembrokeshire coast. How came it that Stanley knew of, and feared, the sweating sickness even before Henry’s arrival and before the first case was recorded in London? May it have been because the north of England was already familiar with, and afraid of, the disease? Such a possibility is supported by an entry in the York Civic Records dated 5 June 1485 (far too early in the year for bubonic plague to have been the likely cause), that refers to a shoemaker who had licensed his apprentice ‘... for fere of the plage of pestilence that reigned, to depart from his service unto his proper frends. ...’ Here the word ‘fere’ might well indicate that the disease was an immediate threat, not necessarily that it had already broken out, within the city. What the entry of 5 June confirms is that early in the summer of 1485 the citizens of York were fully acquainted with, and terrified of, the pestilence prevalent in their region. The York contingent never reached Richard III’s army and it is legitimate to ask if their failure to do so were on account of fear of sweating sickness? Such an interpretation is supported by an entry dated 1 August 1485, twenty-one days before the Battle of Bosworth: ‘Wherein – all aldermen and such as were sojourning outside of York, on account of the plague, should repair to the city [Nottingham] for council with Richard III against the invader.’

None of the London-based chroniclers seem to have looked far enough north for the origins of the sweating sickness and the disease may have spread from there southward. The Yorkshire evidence contradicts Creighton’s belief, based on Forrestier, that the sweating sickness first occurred at

8. Dictionary of national biography, s.v. ‘Stanley, Thomas, first Earl of Derby (1437–1504).’
9. Ingulph, Rerum Anglicarum scriptores veterum; historia Eboracensis, continuat ii (Oxford, 1684), p. 570. This passage is written in polished and sophisticated Latin; it is confidently ascribed to Bishop John Russell of Lincoln (d. 1490) writing in 1486, within a year of the events (personal communications from Miss Margaret Condon and Dr. Bertram Wolfe). This is, therefore, an uniquely valuable source.
10. Francis Drake, ‘Eboracum’ or the history and antiquities of the city of York (London, 1736), Book I, 120.
London on 19 September 1485. There is also evidence from ecclesiastical sources that indicates that the disease occurred elsewhere in the provinces before its appearance in the capital. The archidiaconal court records of St. Alban's indicate that the disease afflicted Hertfordshire. Gottfried, in an important demographical study of the effects of the plague and the sweating sickness, deals only with the 1485 evidence for the latter. These data show that a patchy and, excluding London, a predominantly rural distribution was characteristic. In 1485, according to Gottfried, the disease did not extend to East Anglia. One may also note that on 14 September 1485, Alicia Comalard, abbess of St. Editha of Wilton, Sarum Diocese (Wiltshire), died and a congé d’élire (a royal permit to elect) was petitioned on 20 September for the office of an abbess and a prioress. Similar circumstances prevailed at the monastery of St. Mary de Fratis, Leicester, not far from Bosworth. The abbot, John Shepished, died also on 4 September, and William Stoughton and John Perry transmitted a petition from Gilbert Manchester, prior of the monastery, to the King for a congé d’élire for his successor. Since all these entries were made on 20 September, the deaths listed occurred before that date. Also on 20 September the King granted John Manwaryng, chaplain, custody of the Hospital of St. John the Baptist at Stafford, vacated by the death of Richard Coldwiche, clk. (clerk in holy orders).

If the men in Stanley’s entourage had been exposed to sweating sickness, some may have been incubating it and may have been the carriers who brought it to London, having meanwhile gone over to the side of the victorious invaders. When the disease reached London it hit the capital and, apparently, those in high places, especially hard. Two lord mayors died, one within five days of his appointment; four aldermen also perished.

Before the publication in 1891 of Creighton’s influential treatise, it had been accepted, on the basis of Polydore Vergil’s account written half a century after 1485, that the sweating sickness was brought from northern France via Milford Haven by Henry VII’s mercenaries. The impression of a northern French origin was sustained by the appearance, nearly two centuries later, of a much milder illness in which sweating was a conspicuous feature, in Picardy, Flanders, and the southern Rhenish provinces.

Such occurrences imply that sweating sickness was already endemic in France in 1485. However, the evidence already cited indicates that sweating sickness was rife in northern England before August 1485, and such evidence points to towns on the Yorkshire-Lincolnshire coast, handling trade from Scandinavia and Russia, as the more probable points of entry. In 1530 the sweating sickness spread into eastern Europe via Poland and Livonia and was lost. Today a disease symptomatically very like the English sweating sickness is endemic in east European Russia and periodically extends westward in epidemic form to Czechoslovakia and Denmark. The symptomatology of the sweating sickness in all four outbreaks subsequent to the one of 1485 was, in every important respect, precisely similar.

In 1508 Sir Thomas More drew Cardinal Wolsey's attention to the severe depredations of the sweating sickness among the young gentlemen of Oxford and Cambridge. In 1507 an outbreak of an unknown disease occurred at Chester, but the only feature of the Chester epidemic that was in any way akin to the sweating sickness was the massive preponderance of male over female morbidity. The description refers to grass growing in the streets of the deserted city, suggesting an epidemic of long duration, more likely to have been the plague than the sweating sickness. In 1806 Alexander Jenkins made a similarly doubtful claim for the disease's having ravaged Exeter in 1485. Jenkins merely copied from Holinshed's Chronicle, which is descriptive of London and the country as a whole.

The epidemic of 1517 is the first epidemic of sweating sickness to be documented in substantial detail and two passages so vividly describe the explosive onset, rapid course, and often fatal issue, as well as the apparent susceptibility of those in high places, that they deserve quotation.

...suddenly there came a plague of sickness called the Swetying Sickness that turned all his [the King's] purpose. This malady was so cruel that it killed some

15. Sir Arthur Salisbury Macartley, 'More as a student of medicine and public health reform in science,' in E. Ashworth Underwood, ed., Science, medicine and history: essays on the evolution of scientific thought and medical practice written in honour of Charles Singer, 2 vols. (Oxford, 1955), 1, 418-436, pp. 428-430. The sweating sickness has also been linked with Erasmus at Cambridge in 1510, but this, since there is no record of an epidemic in this year, seems highly improbable.
within three hours. Some within two hours, some merry at dinner and dide at supper. Many died in the Kynges courte, the lorde Clinton, the lorde Gray of Wilton, and many Knightes, gentelmen and officers. For this plague Mighelmas Term was adiorned. . . . 18

On 6 August 1517 the Venetian ambassador reported: 'This disease makes very quick progress proving fatal in 24 hours at the furthest and many are carried off in 4 or five hours. The patients experience nothing but a profuse sweat, which dissolves the frame, and when once the 24 hours are passed all dangers are at an end.' The ambassador went on: ' . . . many of his [King Henry VIII's] household are sick and Ammonio, his Latin secretary died. Few strangers are dead but an immense number of natives.' 19

The fourth epidemic of 1528 is of interest in two respects only: the disease extended to Calais, then a British possession, where it afflicted, it would appear, only Englishmen; it did not spread to the rest of France. Also, the susceptibility of the institutionalised clergy is again emphasised by an outbreak in mid-July 1528 among nuns in a convent at Wilton, and in Lincolnshire the sweat is reckoned to have killed four priests and two lay brethren. During the summer of 1528 the Charterhouse in London experienced heavy mortality from the disease, and many of Cardinal Wolsey's household were infected, though probably not Wolsey himself. Du Bellay, the French ambassador, reported that the Lambeth household of William Warham, Archbishop of Canterbury since 1503, was severely afflicted with eighteen deaths among his domestic staff. Du Bellay himself, one of the few foreigners before 1529 to develop the disease, had a mild attack, which he attributed to his visit to the archiepiscopal palace. 20

The last epidemic to afflict England was in 1551, when parochial records of baptisms, marriages, and burials had begun to be kept. This epidemic is, therefore, better documented than any predecessor. In 1552 John Caius 21 wrote a monograph on the sweating sickness and claimed that the epidemic started at Shrewsbury in June. June is an improbably early onset for the sweating sickness, but it may be significant that in 1485 Henry Tudor's army had sojourned at Shrewsbury. Moreover, it has been doubted wheth-

20. Dictionary of national biography, s.v. 'Warham, William.'
21. John Caius, A boke or counsell against the disease commonly called the sweate, or sweating sickness (London, 1552), p. 40.
er Caius ever visited Shrewsbury.22 That the disease did break out in Loughborough—significantly with few deaths—cannot be gainsaid; a note dated 24 June occurs in the parish register which reads as follows: 'The Swat called New Acquaintance alias Stoup! [i.e., Stoo] Know thy Master.'23 A similar note occurs in the parish register of Uffculme in northeast Devon, in the first week of August 1551. Parish registers studied by Slack24 and Oswald25 make clear that locally and over England as a whole the most consistent characteristic of the disease was the unpredictable patchiness of its distribution. In 1980 Wylie and Linn, in a detailed study of the 1551 epidemic in northeast Devon, identified a sequential extension of the disease along an established trade route.26 They also found that the class-specificity, a feature so uniformly emphasised by contemporary historians, was not so evident in this outbreak. The absence of clergy from among the dead might partly be explained by the sharp decline in their numbers after the dissolution of the religious houses in 1529, but neither were the gentry especially singled out in the Devon epidemic. The few victims among the gentry suggests either that the upper-class aspect of the malady may have been exaggerated by earlier writers, a not improbable explanation, or that by 1551, after four previous epidemics, the character of the disease may have started to change. What is instructive is that Slack has conclusively shown that the English sweating sickness, for all its flamboyant symptomatology and supposedly frequent fatal issue, exerted only a marginal influence upon national or provincial demography.27 There was, at most, a very slight overall increase in deaths in the late summer of 1551 and only in the dioceses of Exeter and Lichfield could this marginal effect be observed. At a parochial level, however, at Colyton, the sweating sickness did briefly

22. F. C. Webb, 'The sweating sickness in England,' Sani. Rev., 1857, 3, 105-124, p. 121. Caius’s account is nevertheless valuable in that it prescribes a regimen of management: retirement (fully clothed if necessary) to bed in well-ventilated room with fire. Absolute protection from draughts, especially to the armpits. Light bedclothes, frequent, small, tepid drinks and, above all, abstinence from copious potations of cold water to assuage raging thirst; a procedure now known to aggravate electrolyte imbalance.


reversal the prevailing demographic trends in a quite striking manner—the number of burials rose to equal the number of baptisms, which fell sharply. Even so, whilst appreciable, this reversal was on a much smaller scale than in 1642, a plague year.

The European Epidemic of Sweating Sickness, 1529–30

Scholars have often associated the epidemic of 1529–30 on the Continent directly with, and derived it from, the 1528 epidemic in England. But there is no evidence for such an origin and it is discussed here separately from the English outbreaks and in some detail, because the illness on the Continent, whilst clinically so typical—it was universally called "The English Sweat"—displayed some important epidemiological differences from the English malady. The 1528 epidemic in England had, characteristically, subsided abruptly by September of that year. The very suddenness of the end of the epidemics lends support to the view that an unidentified rodent reservoir of the virus was susceptible to an epizootic and was killed off, ending a rurally orientated small mammal population explosion that, under favourable environmental circumstances, usually reaches its zenith in August. On 25 August 1529 a German ship with a German captain, Hermann Evers, and German crew arrived at Hamburg with twelve men already dead. The incident makes it extremely improbable that they were infected in England; rather, they were incubating already the disease which they had contracted two to three weeks earlier, probably in north Germany. Immediately after their arrival an epidemic erupted at Hamburg, reportedly killing thousands. The disease broke out almost simultaneously in cities as wide apart as Bremen, Lübeck, and Copenhagen, where members of the Danish royal family succumbed. Hecker, the principal German authority on this epidemic, favours the view that the initial outbreaks in the ports on the North and Baltic Sea coasts were quite independent of the English outbreak of 1528 and would have occurred irrespective of Captain Evers’s experience. As with the English disease, however, a bewildering territorial discontinuity was observed when the disease extended to the hinterland, although, in terms of time, central and southern Germany

were affected after Bremen, Hamburg, and Lübeck, indicating a general north to south extension.

In view of the harsh experience at Oxford and Cambridge, it is interesting to note the alleged effect upon the small university town of Marburg-an-der-Lahn, wherein, at the behest of Landgrave Phillip of Hesse, delegates from the fractious sects of the newly emerged Protestantism had been summoned to settle their Eucharistic differences. The colloquy was planned to last eight days, beginning 1 October 1529, but, in fact, was adjourned after three days. The cause of this premature adjournment has been attributed to the prevalence in the town of sweating sickness. This must be doubted, although fear of the disease that was prevailing elsewhere in Germany was such that this alone might have sufficed to persuade the delegates to disperse. It is, however, probable, that since Luther and Zwingli, the principal disputants, clearly never were to agree, the threat of the English sweating sickness provided excuse enough for the delegates to abandon an unwanted agenda. Hecker was able to find evidence at Marburg of only one case of the disease, which was not fatal.

From Bavaria, Württemberg, and Alsace in the south, the sweating sickness spread, possibly via the Rhenish trade route, northward again to the Rhine Palatinate and to Cologne, as well as further southward to the mountain fastness of the northern and eastern Alpine valleys in Switzerland and Austria. The principal export from Switzerland in the sixteenth century was mercenaries, who fought in the religious wars of central Europe. It is not improbable, therefore, that Swiss soldiers on their return home from their martial exploits brought with them the infection of the English sweating sickness, which survived the winter of 1529–30 among the scattered and isolated Swiss villages. M. B. Shaw claims that the appearance of sweating sickness at Vienna forced the besieging Turks to loosen their grip on that city on 22 September 1529. The significant feature, which distinguished the European epidemic from the summer disease as it occurred in England, was its persistence, albeit in a subdued manner, throughout the winter of 1529–30. In the summer of 1530 the sweating

sickness spread eastward to Poland, Livonia, and European Russia, into whose vastness it seemed to disappear. This epidemiological difference notwithstanding, the European descriptions of the clinical course of the sweating sickness tally precisely with those of Forrestier and Polydore Vergil for England before 1529, and John Caius after.\textsuperscript{35} The emphasis of them all is upon the susceptibility of the young male in good estate. The susceptibility of young men is mentioned in two separate and independent German sources, Reimarus Kock at Lübeck and Joannes Rennerus at Bremen—cities some 120 miles apart, between which overland communications were poor.\textsuperscript{36} Both observers give very similar descriptions at about the same time. Kock stated that 'rich young men died but children, the aged and the poor, in wretched conditions are spared.' What is interesting and significant is that France and the Italian and Iberian peninsulas were spared.

\textit{Some later Sweating Sicknesses}

Epidemiologists in the nineteenth century suggested an aetiological relationship between the 'English' and the 'Picardy' sweat.\textsuperscript{37} The latter gave rise to a number of epidemics after 1717, over 150 years after the last recorded outbreak of the English sweating sickness in 1551. The disease was not, despite its name, confined to northeastern France; it extended to Normandy, south almost to the Mediterranean and eastward into Bavaria. Polydore Vergil and John Caius associated the English sweat's first appearance in Britain with the landfall of Henry Tudor's army; that the Province of Picardy seemed to form a \textit{cordon sanitaire} protecting the rest of metropolitan France and the Iberian and Italian peninsulas in 1529 from the spread of the English sweating sickness, suggests an immunological relationship between the two 'sweats.' However, northeastern Europe rather than northern France was the probable origin of the sweating sickness in England in 1485.

The Picardy sweat was a disease that behaved as if it were endemically well established, but subject to periodical epidemic eruptions. Were it a precursor of, or indeed a successor to, the English sweat it would, in either case, have been milder and less explosive, and so it was. Also, when epidemics of the Picardy sweat did occur, it was noticed that it was the more salubrious villages that stood at greater risk and the bourgeoisie were the

\textsuperscript{35} Forrestier, \textit{Tractatus} (n. 6); Vergil, \textit{Anglica historia} (n. 13); Caius, \textit{Sweat} (n. 21).
\textsuperscript{36} Henricus Haemer, ed. \textit{Scriptores de Sudore Anglico} (Jena, 1847), pp. 443-444. 448.
\textsuperscript{37} Czeighton, \textit{Epidemics} (n. 1), i. 256-259.
more usual victims. Even a French equivalent of Stop Gallant, trousse gallant, was current. The Picardy sweat did display certain differences from the Sudor Anglicus; the suette milaire, as the Picardy sweat was also known, erupted again in 1821. As its name suggests a miliary rash (that is, spots of the size of a millet seed as in miliary tuberculosis) was a constant feature, but this sweat was attended neither by a spectacularly brief clinical course nor by high mortality. Exanthemeata were only rarely described in the English sweating sickness.

If the Picardy sweat were an attenuated form of the English sweating sickness, how did the germ get to Britain if it did not come with Henry Tudor’s mercenaries? Infected rodents may have entered the Port of London or the south coast ports. Alternatively, because reliable evidence suggests that the north of England experienced the English sweating sickness before the south in 1485, northeastern ports trading with Scandinavia and Russia may have been more likely points of entry. Ticks, mites, and other parasitic arthropoda can also be carried by birds; and it is just possible that ornithological migration could have brought the sweat to England in 1485.

An exanthematous disease similar to the Picardy sweat was described amongst Allied troops in the trenches during World War I by Sir Michael Foster and later by Sir Henry Tidy. A variety of clinically similar disorders, including leptospirosis, were called trench fever, a sobriquet which covered a rag-bag of diagnoses. What is quite certain is that the disease that Sir Michael Foster and Sir Henry Tidy saw, whatever it may have been, most certainly was not influenza, which contemporary epidemiologists were soon to equate with the sweating sickness. But might it have been an attenuated form of the English sweating sickness? Sir Michael Foster certainly took account of the latter possibility.

Features of the English Sweating Sickness

The clinical manifestations of the disease are well known. The earliest descriptions all agree that men in the prime of life and well-to-do appeared most at risk. This magnificat tendency to ‘put down the mighty from their seat,’ and, if not exalt the less fortunate, then certainly to spare them, is revealed in the nickname applied to the English sweating sickness: ‘Stop

38. Ibid., p. 272.
[Stoop or Stoup] Gallant, or Stop Knave.\textsuperscript{41} according to the social hauteur and the commercial rectitude, or otherwise, of the sufferer’s fisc. The relative well-being of the institutionalised clergy is reflected in the high incidence of the English sweating sickness among monks, and even female religious, before 1351, by which time the monasteries had been dissolved. The vulnerability of members of the universities of Oxford and Cambridge is notable. This might be expected since undergraduates then, as now, were in the prime of life and socially privileged. In those days they were all male and the collegiate foundations had, by the sixteenth century, largely forsaken their original eleemosynary idealism. In the English pattern of town and country incidence, London stands out as exceptional. It suffered in every outbreak. This may be seen, however, as an epidemiological clue. The mediaeval plans of cities like Exeter and Chester, which were untouched by the illness, reveal a great density of buildings, public and private, crammed behind a city wall with ingress and egress limited by gates at each primary point of the compass.\textsuperscript{42} There were virtually no open spaces and the inhabitants lived cheek by jowl; an environment inimical to incursions by small mammals of rural habitat—voles, shrews, wood mice, and others—into human dwellings, animals that may well serve as reservoirs of the infecting agent.\textsuperscript{43} London presented a totally different aspect. Only the City itself was circumvallate, and incompletely, by the sixteenth century; the rest of the London environs amounted to a collection of scattered villages, a synkómiēsia,\textsuperscript{44} more or less widely separated one from another by tracts of countrysid. Bromptom, Chelsea, and Ken-sington, even St. Marylebone, were then isolated country villages. Busy ports, great and small, frustrate circumvallation and are thus vulnerable; Hamburg, Lübeck, Bremen, Copenhagen, and Barnstaple, were comparable to London in this respect.

There are three serious hindrances to the formulation of a theory about the aetiology of the epidemics of sweating sickness: first, their remoteness in time; second, the nonspecific nature of the signs and symptoms; and third, the well-known tendency of infectious diseases to change their characteristics, sometimes over a few decades, so that no exact modern coun-

\textsuperscript{41} ‘Knave’ in this context could also have meant a servant (cf. French = valet on playing cards). Hiredlings in rich households tended to be far better provided for than their social equals who had to fend for themselves.


\textsuperscript{43} Wylie and Linn (n. 26), p. 112.

\textsuperscript{44} Synkómiēsia from the Greek: συν = together; κόμη = a village.
terpart may exist. In the sixteenth century clinical descriptions were primitive and there was an understandable propensity to dwell upon the most prominent features of an illness and to overlook others that, although less obvious, nevertheless may be essential for accurate diagnosis. The main clinical signs of the sweating sickness—fever, sweating, and headache—are common to many infections caused by the whole gamut of microbes from protozoa to viruses. Faced with these obstacles, the best that can be done is to marshal the somewhat scanty information about these epidemics and then, as much by exclusion as by positive inference, to arrive at a postulate that may have the merit of plausibility.

The five major outbreaks in England had interepidemic periods of twenty-three, nine, eleven, and twenty-three years. They occurred during the summer and autumn and were widely scattered and patchy in terms of geographical distribution, affecting especially rural areas. London and the universities of Oxford and Cambridge excepted, provincial towns and cities seem largely to have been spared. Men were consistently reported to have been more frequently affected than women and among them the incidence was said to be highest in the age group of twenty to forty years; there seems to be no special mention of a high incidence in children. As for mortality, the case ratio varied considerably from place to place.

Clinical Aspects

The dominant features were fever, sweating, thirst, headache, prostration, and syncope; 'pricking of the brains' is also mentioned. The prominence accorded in all accounts to the profuse sweating suggests a high degree of pyrexia. Rashess seldom are mentioned and do not seem to have been a particularly notable sign. In a proportion of those affected, death might ensue well within twenty-four hours of the apparent onset, although it is possible that earlier prodromata passed unremarked.

Possible Aetiology

In trying to assign a possible cause for sweating sickness, we make the assumption that all the epidemics possessed a common aetiology. The first step is to decide which of the epidemic infectious diseases should be eliminated from consideration. The summer incidence and the absence of respiratory symptoms appear to rule out influenza. Epidemic typhus is primarily a winter infection and the paucity of references to a rash makes a diagnosis of typhus unlikely. The manifestations of bubonic plague were all too well known for fifteenth-century people to have confused plague
and sweating sickness. The same is true of smallpox, which in any event does not seem to have been prevalent in England much before about 1514, that is, after the first two epidemics of sweating sickness in 1485 and 1508.45

Among epidemic diseases caused by bacteria, only cerebrospinal meningitis seems to be a possible cause by virtue of its ability to spread quickly from person to person under conditions of overcrowding, its rapid onset, and its high mortality if untreated. Like sweating sickness, cerebrospinal meningitis has an acute onset characterised by high fever and headache, but the usual presence of a rash militates against its identification with the sweating sickness, as does its tendency, at least in Britain, to occur mainly in the winter and spring.

It seems most likely that the epidemics of sweating sickness were caused by a virus; three categories merit consideration.

There are about seventy members in the enterovirus family, including the three types of poliomyelitis virus and the less well known but widely prevalent Coxsackie and echoviruses. These agents can cause epidemics, particularly in the summer months. Because they are transmitted by the faecal-oral route they flourish in conditions of poor hygiene: under such conditions, infection is almost universal in young children who in most instances undergo an inapparent or trivial infection that nevertheless confers lasting immunity to the infecting strain. Poliomyelitis itself is, however, an unlikely candidate, because the consequent paralyses surely would not have gone unnoticed. There are, however, a number of other enteroviruses capable of causing acute febrile illness, with or without meningitis, and with or without a rash. Such infections are not nowadays associated with a high mortality; so, to account for the long and irregular interepidemic periods of the sweating sickness, one would have to postulate the occasional appearance or introduction of new strains of enterovirus that differed sufficiently from their predecessors in terms of antigenic composition to overcome the existing herd immunity, but like enough in pathogenicity to cause clearly similar or identical epidemics many years apart. Such a mechanism is a possibility; but the curious epidemiological behaviour of the sweating sickness may be explained more plausibly in terms of a virus infection that had its reservoir in a mammalian or avian host, from which it emerged at irregular intervals to affect man. Two families of viruses behave in this way: the arenaviruses and the arboviruses, the latter through the mediation of insect vectors.

45. Creighton, Epidemics (1914), t. 436.
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The first of the group of arenaviruses to be isolated was the agent that causes lymphocytic choriomeningitis (LCM); this virus has a world-wide distribution and causes a persistent symptom-free infection in house mice; humans occasionally become infected, possibly by contact with mouse excreta. In man, LCM causes a comparatively benign febrile infection with or without transient signs of meningitis. Other arenaviruses cause much more severe diseases: the South American haemorrhagic fevers; Lassa fever which occasionally appears in West Africa.

Argentinian haemorrhagic fever (AHF) and the Bolivian variety (BHF) both have their reservoir in wild rodents and like LCM seem to be transmitted from them to man without the aid of an insect vector. AHF is most frequent in the summer and autumn when agricultural activity is at its height; by contrast, BHF is 'house-associated' and is most prevalent among the poor. 46 Neither infection spreads directly from man to man. Lassa fever, however, has a propensity for person-to-person spread, although like the other arenavirus infections, the primary reservoir seems to be a small rodent, in this case Mastomys natalensis.

The chief claim to our attention of AHF, BHF, and Lassa fever lies in their epidemiology and their clinical features: all three infections are characterised by fever, headache, and high mortality. Nevertheless, their onset tends to be insidious and exanthematous or haemorrhagic signs are frequent; by and large, the clinical pictures do not conform very closely with the known features of sweating sickness.

The arbovirus family derives its name from the fact that these viruses are all arthropod-borne, being transmitted between their mammalian or avian hosts by a variety of mosquitoes, ticks, and flies. More than 300 arboviruses have so far been identified, of which the most notorious, but by no means typical, member is the virus of yellow fever. 47 In terms of pathogenicity, variety of clinical syndromes, and epidemiology these viruses vary enormously, but it is possible to describe a general pattern of behaviour. The reservoir of infection is a vertebrate, often a small mammal or a bird. The infection may circulate for long periods among such mammalian or avian hosts without spreading to cause overt disease in man. The degree to which humans are affected depends on a highly complex interplay of factors among which are the pathogenicity of the virus concerned, the feeding habits of the insect vector, the opportunity for contact with...

47. Ibid., p. 79.
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man, the distribution of the reservoir host, and the state of immunity of the human population. With some of these viruses, epizootic infection in the reservoir host has been observed to precede an epidemic in the human population of a given locality. In man, some arboviruses may cause endemic or sporadic infections, whereas others give rise to epidemics, often at irregular and unpredictable intervals. The persistence of an arbovirus in a given locality may depend not only on the movements and distribution of the avian or mammalian reservoir, but also upon the ability of the virus in question to overwinter in the arthropod vector, which then, strictly speaking, becomes a reservoir as well as a vector. This is sometimes accomplished by transgamet passage to the next arthropod generation.

Yellow fever has been cited as a particularly well-known arbovirus infection, but the yellow fever virus is atypical in that it is primarily hepatotropic. The great majority of arboviruses pathogenic in man cause febrile illnesses of varying severity, sometimes accompanied by a rash, haemorrhages, or inflammation of the joints; sometimes by encephalitis, which may vary from a benign episode with complete recovery to a severe and disabling illness followed by paralysis and/or death. Subclinical infections are common. Age, sex, and occupation may also influence the clinical course and incidence of arbovirus infections. Thus in the United States, western equine encephalitis and St. Louis encephalitis tend to cause more severe disease in young and older persons respectively. Men may be affected more often than women, perhaps because their occupations or recreations take them into wooded areas where the risk of insect bites is increased. With these considerations in mind, we are of the opinion that the descriptions of the epidemiology and clinical aspects of sweating sickness, tantalizingly lacking in detail though they are, at least could be plausibly explained in terms of arbovirus infection. In the fifteenth and sixteenth centuries, England was much more extensively wooded than it is today, and such an environment offered ample shelter for large numbers of wild birds and mammals. Mosquitoes were certainly not lacking, as evidenced by the prevalence of malaria found, there are still enough anopheles mosquitoes in Britain to account for the cases of malaria by transfer from persons who contracted the disease; a number of such cases were reported when the troops returned from the war after the 1939-45 war.

The long and irregular interval between epidemics of sweating sickness accord with the behaviour of arbovirus infections, but it is impossible to say whether the irregularity of sweating sickness was due to changes in behaviour of the reservoir host, the virus
itself, or to successive induction and waning of immunity in the human population. We have mentioned the sudden collapse of the epidemic in September 1528. Even more striking was the suddenness of some of the declensions of the outbreaks in east and north Devon parishes in 1551. In all the villages where the outbreak was typical and accepted as being, without question, one of sweating sickness, the epidemic ended abruptly.48 The degree of immunity in an animal reservoir will depend, in large measure, upon the proportion of the animal population that is immunologically virgin at any given time. If there has been an explosive growth in the population of the animal reservoir, large numbers of young animals will not have been infected, therefore will lack immunity, and the conditions for an epizootic are created. From the earliest times it was noticed that a massive, sudden increase in the numbers of dead black rats (*Rattus rattus*), once the principal reservoir of bubonic plague, was the harbinger of an epidemic of the plague in man. The immune rats were massively outnumbered and numerous deaths supervened among young rats hitherto unexposed to plague. As the rats died the rat-fleas, for lack of a better home, migrated to man, and the man-flea-man cycle thereby started. The brown rat (*Rattus norvegicus*) has now supplanted the black rat ecologically and as the main reservoir and this animal is basically much more resistant to plague with consequently different patterns for plague. One of the few factors certainly known to influence the population of mammals, especially small mammals living in woods and fields, dependent for their food upon small insects, grubs, and worms, is climate. Climatic records, except when there has been some spectacular abnormality, tend to be the more vague the further back they lie in history. However, weather records are likely to survive in greater detail in farming communities than in cities. According to Griffin, during the years in which sweating sickness occurred in England, the spring and early summer were wet, warm, or both; conditions conducive to the explosive growth of small mammal populations.49 Whereas there can be, and clearly often are, changes in weather patterns, which may account for small mammal explosions earlier in the year than usual, the peak populations of field vole, bank vole, and wood mouse normally tend to occur in August.50 If, as we think most probable, this type of small mammal were the most likely reservoir of the arbovirus of the English sweating sickness, then epidemics would have occurred in man when there

49. Griffin (n. 31).
was an high enough proportion of susceptible young mammals of the reservoir species to create first an epizootic in the reservoir and then to trigger an outbreak of the same disease in man. The epidemic thus caused would tend, if our hypothesis be correct, to be explosive at the outset and to decline suddenly.

The apparent predilection for male adults may reflect a greater risk of exposure to an insect vector, but an alternative explanation may be advanced. The English sweating sickness was in the sixteenth century not only an unfamiliar malady but its clinical course, age, sex, and upper-class incidence include some of the characteristics of certain types of infections when first they appear in an immunologically virgin population—characteristics that significantly differ from those of epidemic outbreaks of already well-established endemic diseases. Sir Macfarlane Burnet vividly draws attention to the strikingly different behaviour of poliomyelitis in remote and immunologically isolated communities. The disease, popularly called infantile paralysis in countries familiar with it, because of the apparent vulnerability of children, seemed to avoid young children in the Canadian Arctic epidemic of 1949. Similarly, smallpox, considered a new disease in England in the sixteenth century, afflicted well-placed adults alarmingly, and ravaged their appearance cruelly. By the latter part of the eighteenth century, smallpox had become predominantly a disease of children. The 1769-74 incidence in Manchester, England, strikingly demonstrates this. By contrast, in the unvaccinated community of Sydney, Australia, as late as 1913-15, smallpox again behaved as a new disease in immunologically virgin territory, giving rise to a large majority of cases in the age-range twenty to thirty-five years. Dengue, which is caused by an arbovirus, may give rise to a syndrome known as dengue shock; this is the result of an immunological reaction between the virus and antibody already present in the serum as the result of a previous infection with a serologically different strain. The relatively sudden onset and collapse would accord in part with what is known of the sweating sickness; but, on the other hand, the frequent haemorrhagic manifestation in dengue shock argue against its identification with the English sweating sickness.

New diseases do, however, become modified in severity and age-incidence as they become endemic and there is evidence of such change having occurred in the 1551 epidemic of sweating sickness. Few diseases display

this tendency to adapt, becoming at first chronic and finally self-limiting, more impressively than syphilis. In fifteenth-century Europe, syphilis was an acute fulminating infection, which killed its victims in what we now call the second stage, and became, in due course, the chronic indolent condition, giving rise to morbid anatomical memorials—gummata, aortic stenosis, and general paralysis of the insane—which today adorn the older and better equipped museums of pathology. In the experience of the Norwegian mercantile marine, syphilis seems to have become a much less fell disease than of yore which dies out after a brief period of malaise—all that remains of the once dreaded phase two—without effective treatment. A series of untreated or incompletely treated cases of syphilis were studied in Norway by Boeck and later published by Bruusgaard. They found that 60–70% of cases of syphilis were self-limiting at the secondary stage, and other investigators have subsequently confirmed their figures. While the publication of these results has led to some deplorable neglect of the completion of treatment, the figures do suggest that the virulence of Treponema pallidum in man may have been on the decline before effective treatment with penicillin was introduced. This observation was made before penicillin became available in therapeutic quantities after 1941.

An arbovirus is the most probable aetiological agent of the English sweating sickness, and the prevalence of sweating sickness in northern England before the landfall of the future King Henry VII’s largely mercenary army in Pembrokeshire on 1 August 1485 favours the view that the northeastern ports, rather than those of the south and west coasts, were the portals of entry for the disease. The northeastern ports received merchandise from Scandinavia and Russia, and in 1530 the last cases of the English sweating sickness, as classically understood, occurred in European Russia and thereafter the disease seems to have disappeared. It is, therefore, of the greatest interest to learn of the prevalence today in western Russia, the Crimea, Poland, Romania, Hungary, Czechoslovakia, Denmark, and Finland of certain arbovirus infections that have their reservoirs in mice, muskrats, and hedgehogs and that are tick-borne: namely, group B tick-borne or Russian spring-summer encephalitis, and Omsk haemorrhagic fever. These diseases in man are commonly very severe and bear striking

55. E. P. Abraham et al., ‘Further observations on penicillin,’ Lancet, 1941, ii, 177–188.
resemblance to the English sweating sickness in its most florid form. Virae-mia, always associated with heavy sweating, is constant, but violent; prostrating headache, rapidly rising fever, nausea, vomiting, and photophobia are also frequent. Haemorrhagic enanthemata are always observed. The occurrence of haemorrhagic spots does not conflict seriously with the English sweating sickness, because fear so dominated the scene in the sixteenth century that there can be no surprise that rashes were rarely sought for, although 'black spots,' probably haemorrhagic in origin, were described. Fear would certainly have prevented the even more careful clinical examination necessary to find enanthemata. The significance of this physical sign was, in any case, not recognised in any disease before 1896 when Koplik described his palatal and buccal spots—the pathognomonic prodromata of measles. So, the epidemiological and demographic records of the English sweating sickness, exiguous though they be, are, together with abrupt onset of fever, headache, rapid course, and high mortality as described in the historical accounts, characteristic of some arbo-virus infections known to us today.

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57. Forrestier, Tractatus (o. 6), Chap. 7.