AN ABSTRACT OF THE DISSERTATION OF

Edwin Del Wollert for the degree of Doctor of Philosophy in History of Science presented on March 15, 2017.

Title: An Assessment of the "Sweating Sickness" Affecting England During the Tudor Dynasty

Abstract approved: _____
Paul E. Kopperman

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While historiography and interest in Tudor England at both the popular and specialist levels presents few signs of diminishing, there may nonetheless exist a sense that we have little left to learn about this period and its culture. A notable gap in our knowledge, however, remains regarding the mysterious disease known only as "sweating sickness" or *sudor anglicus*. This dissertation addresses and evaluates this disease from the perspective of the history of science, and in doing so, it makes three key arguments. First, this project examines how the early modern science and medicine known and practiced by Tudor subjects influenced their perceptions of this new disease, leaving them in a mostly helpless position from which to combat it and indeed often wondering if the unknown illness might represent a divine judgment, especially in the form of questioning a dubious claim to monarchy made by the first Tudor ruler, Henry VII. Second, the dissertation offers a detailed and layered thesis concluding that the disease was ultimately caused by an earlier version of the louping-ill virus, or LIV, a virus and accompanying illness which continued to affect parts of Western Europe, with its own unique strain still extant within Britain. The third argument will return to the opening statement of this abstract, and reveal how this more thorough and unique treatment of Tudor historiography does much to further our understanding of the Tudors and their citizens, all the more relevant since the "Sweat" even now is typically either mentioned in passing, or not at all, but those who write about this period of history.

An Assessment of the “Sweating Sickness” Affecting England During the Tudor Dynasty

by
Edwin Del Wollert

A DISSERTATION

submitted to

Oregon State University

in partial fulfillment of
the requirements for the
degree of

Doctor of Philosophy

Presented March 15, 2017
Commencement June 2017

Doctor of Philosophy dissertation of Edwin Del Wollert presented on March 15, 2017.

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I understand that my dissertation will become part of the permanent collection of Oregon State University libraries. My signature below authorizes release of my dissertation to any reader upon request.

Edwin Del Wollert, Author

ACKNOWLEDGEMENTS

The Author expresses his sincere appreciation to the following:

Professor Paul Kopperman, for accepting me as an advisee, and also my committee members:

Professor Jacob Hamblin, with the Department of History, Science, and Religion;

Professor Rebecca Olson, with the School of Writing, Literature, and Film;

Professor Tara Williams, with the School of Writing, Literature, and Film;

Professor William Todd Jarvis, with the Water and Watershed Institute, and also for acting as my Graduate Council Representative.

Ms. Anne Bahde, with the Oregon State University Special Collections and Research Center at Valley Library.

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I. Introduction: The Historical Context: Why Study the Sweating Sickness?

All historiography must seek a balance between offering too little explanation of a time and place, leaving a superficial treatment, and too much, such that a reader may feel lost in the minutiae. To summarize as briefly yet essentially as possible, England during the late fifteenth century was a kingdom seeking greater political stability. The Hundred Years War with France was finally past, as was the Black Death, which itself afflicted multiple continents, though conflict with England's traditional enemy as well as further outbreaks of plague would continue. Even the period of English civil conflicts known as the Wars of the Roses had been largely resolved, finally leaving Kings Henry VI and Edward IV in their graves, and Edward's brother, Richard III, on the English throne. Yet his own short reign remained hardly secure, and by 1485 he would face a new threat from a returning exile with a less legitimate royal claim, but one he was clearly willing to test in battle.

The actual battlefield would be known as Bosworth, and the drama alone of the ensuing conflict would offer inspiration for much subsequent literature and theatre and simple wonderment that the contending returning exile, Henry Tudor, succeeded at all. His forces were outnumbered, and some may have been ill with something they brought with them. The army of the future Henry VII contained a mix of British and Continental troops, the latter of whom would unwittingly contribute one of the key questions motivating this current research, since disease apparently invaded England with them. Yet what follows is not a military history. It will offer no assessments of late medieval battle tactics nor the political strategies which motivate war. Instead, this will be an examination behind one of the major factors which enabled Henry's victory. Two chief allies of Richard III, Thomas Lord Stanley, and his brother William, who had already given their oaths of loyalty to their sovereign, simply waited with their forces in full

view of what transpired, and when it seemed that Henry actually had a solid chance of prevailing, they switched loyalties (making them guilty of treason, but since their monarch would soon be dead this point would be mooted). They then charged into the melee to side with Henry. Richard was slain, the first grandiloquent narrations of the battle began with the alleged presentation of his very crown to Henry as victor, and the start of a new dynasty, using Henry's Welsh-inspired family name of "Tudor," had commenced.

The reason the Stanleys ostensibly gave for their disloyal caution, meanwhile, was fear of contracting a disease, the one which may have been imported into England with Henry's hired troops. And that is where this dissertation begins. Had matters gone otherwise, the Yorkists might have remained in power for far longer, while the Tudors might have been relegated to a more anonymous listing in the history texts of the sixteenth century, merely one of the endless "what-ifs" which sometimes receive more historical consideration than may be their due. Yet this strange disease, known variously as "sweating sickness," *Sudor anglicus*, or simply the "Sweat" occurred almost exclusively in England and only during the first half of the Tudor dynasty, seemingly vanishing in 1551. Attitudes to the illness, which has never received a more official scientific classification, ranged widely, with some believing it was divine punishment for an undeserving monarchy, or a nation led into sin via its own reformation, or both. Others tried to address it more rationally and scientifically, with some realizing that it presented in such a way that it must have been something different from the likes of plague or leprosy or other diseases that had been studied during the Middle Ages, although genuine prevention, much less actual treatment, remained elusive.

Yet, the simple truth is that no one really knows or knew just what the disease was, and even now hypotheses abound about it, emerging in a mix of academic disciplines as well as lay

interests. Interest in trying to establish what the disease was has intrigued historians for five centuries already, and more recently has also tested the argumentative mettle of physicians and various types of scientists. Perhaps attempting to solve a mystery, even a historical mystery, offers its own enticements, whether or not the mystery under study can ever reach a definitive conclusion. With that sense of the unknown in mind, and with the briefest but hopefully coherent explanation of the relevant historical setting now established, it is crucial to indicate what the following hopes to accomplish.

First and foremost, this dissertation attempts to earn its place within the historiography of science. While this will not entail an attempt to comprehensively explain Tudor science, with its newer understandings of anatomy and medicine while remaining true to outdated bodies of knowledge which included humoral theory, astrology, and alchemy, it will consider such disciplines only within the context of how each might have helped contemporaries attempt to explain or deal with this frightening new disease. While early modern sciences could each receive book-length treatises of their own (and indeed they have), addressing how each might help ourselves to understand the "Sweat" will play a part in reaching a particular conclusion. The first task in reaching this historical conclusion is to explore a scientific and medical understanding of this part of English history.

Second, the main conclusion this dissertation will reach regards the identity of the sweating sickness itself. This is the first academic work to argue that an earlier variant of what is now classified as louping-ill virus (LIV) was indeed the culprit, a form of arbovirus still found within Britain, even though sweating sickness seems to have thankfully otherwise vanished from the historical record, an issue which has helped confound its study ever since. This particular virus "probably emerged in the British Isles less than 800 years ago and most LIV dispersal

occurred in the last 300 years," [1] as one recent article notes. It will also be argued that this virus has changed during these intervening centuries, and since "there is, theoretically, scope for the evolution of transmission cycle-specific subtypes or strains of LIV," [2] this enables the accompanying explanation as to why we do not find sweating sickness outbreaks still occurring. In the process of making this claim, the relevant historiography about the Sweat will need to be considered in more detail, which will take the form of systematically considering every disease which has previously been suggested as the cause, from Tudor times and into our own, and this careful examination will necessarily come before the conclusion favoring LIV and the shorter consideration of Tudor science and medicine, though some background about LIV will be offered initially. The argumentation will thus begin as a synthetic explanation of all these prior arguments and hypotheses, and end via a process of logical elimination: briefly, louping-ill virus is the most plausible source of the Tudor period sweating sickness because it offers the greatest explanatory prowess among all the various contenders. This is not to say that the retrospective diagnosis of LIV is something worth etching in stone. Rather, this current work is unique for making this particular argument, and further explains why, at least at present, making a retrospective diagnosis is in truth the closest we can come to ever knowing what the disease was.

Third and finally, this new work has to address the most glaring question of all, one that any researcher and writer of something as intensive as a doctoral dissertation must consider: why bother with what might be more safely relegated to the heading of "unanswerable historical mysteries," and just leave it alone? This question becomes even more pressing simply when one considers that the Tudors, to put the point bluntly, have been done to death, so to speak. Clearly they have marvelous longevity as subjects: even during the reign of Elizabeth I herself historiography and literature were already exploiting the grand story of this dynasty, sometimes

accurately, sometimes for political justifications, sometimes to make money. And audiences continue to read Tudor historical fiction as well as genuine history, and still savor watching plays and films and documentaries alike with a huge and intriguing cast of characters which never seem to get boring, partly for themselves and partly for their time: in addition to conflict and entertainment, the Tudors also represent forays into North America, they pose the troubling question of religious reform in Europe, and they occupy a rough historical division between medieval and modern. So again, why bother, since there exists a sense that with the Tudors, "it's all been done?" And the answer is that, no, it has not all been done, and more specifically, Tudor medicine in general and the reactions to the sweating sickness in particular have the potential to reveal not just more about residents of early modern England but more about ourselves as well. By this is meant the sort of illumination described by a more modern historian who addresses the very question of retrospectively diagnosing a disease: "research based on historical sources will not only help us to understand the spread of diseases around our planet during the course of human evolution, it can also help us to plan for future unexpected health events." [3] A gap exists within otherwise well researched and written Tudor historiography, but when it comes to describing a key recurring event which clearly affected personal decisions as well as politics, historians and others simply bypass it as quickly as possible and note that some new disease broke out, but we cannot say much about it. This current work exists largely to fill that gap.

The understanding of disease has surely improved since the Tudor period, yet so too has the taxonomy of disease, such that some of those cited herein will acknowledge that in some cases, as with the potential disease-causing viruses already classified into taxonomical groups, even the experts may not know exactly how many such microorganisms in each category may exist. But understanding how a time and place and culture responded to a disease, especially

when the people involved clearly did not even know what it was that plagued them (no pun meant, especially since sweating sickness was not plague) helps us understand them and ourselves better, since we are partly their cultural descendants. History can do little if anything else if it cannot explain something of the past within its relevant context while also showing something of a connection to ourselves, and this dissertation intends to follow that tradition.

So, since this project has several tasks: to explicate some of Tudor science and its understanding; to reveal a new and unique interpretation of a historical mystery in the form of a disease and its several visits to England (and, on one occasion, to parts of Continental Europe as well); and to consider Tudor history from a perspective not often used (and never in this combination), first we have to consider some other background questions. The first has to do with the strangeness of this new disease itself. To put it succinctly, the sweating sickness is famous mainly for being mysterious. Plenty of writers have left it ambiguous or undefined, whether to avoid getting into side debates or perhaps wondering if researching it is even worthwhile at all. To some extent this makes perfect sense. When engaged in historiography, after all, no matter one's expertise and knowledge of one's subject, no one can presume to answer absolutely any question even within one's own specialty. The sweating sickness conforms well to this frustration, such that, depending upon whom one reads, the "Sweat" itself may simply be described in passing, with no genuine consideration of what it may have been, or in some cases really not mentioned at all. The problems are manifold. Henry Tudor's own eldest son died of tuberculosis, for example. Or was it actually sweating sickness? Or was he perhaps poisoned, considering how quickly he developed symptoms and perished? The first answer is the likeliest, though the questions point, not to some scholarly conspiracy, but to simple confusion among the writers of history, even those attempting to keep royal records at the

time. As for Henry's surviving second son, the second Tudor monarch, Henry VIII may have had type-II diabetes, McLeod Syndrome (apparently from being Kell positive, which may in turn have helped explain why he never had grandchildren), and was certainly obese, at least during his later years. Did he also have a head injury from either or both of his documented jousting accidents (in 1524 and 1536), and did either or both of those cause complications from his ulcerous leg which sometimes reopened and had to be drained and the pain of which has been cited as part of the reason for his becoming tyrannical during his last decade? And as for Catherine of Aragon, was her fate related to a series of heart attacks, perhaps liver or stomach cancer, or even poisoning for political reasons? These are some of the other medical mysteries, and if we cannot know for sure even with the most documented of Tudor-period personages, then how can we hope to address a separate illness which is seemingly unknown in our own time?

These cases entail different approaches. With famous individuals, we have various signs and symptoms and documents and can argue from there. In the case of the sweating sickness, we have the benefit of numbers on our side: the disease clearly affected many thousands each time it erupted, and it afflicted England five times. This leads us to the second consideration, and one issue which might help the overall case here is that even centuries later we know at least a few of the disease's victims. It seems enticing to learn that Anne Boleyn and her brother George contracted it yet survived it, and that Thomas Wolsey may have actually survived the disease himself, twice no less (making him unique in the annals of sweating sickness investigation). Less well known to non-specialists are the sons of Charles Brandon, Duke of Suffolk and childhood friend of the future Henry VIII, both of whom died in one of the Sweat outbreaks, or the wife and children of Thomas Cromwell, who may or may not have perished at the same time as the Brandon boys (even the fates of Cromwell's family have become part of this historical

dispute). While single-factor causality within history is both irresponsible and intellectually lazy, this work will never suggest that Richard III lost his crown to the man who would shortly become Henry VII because of a disease. It would be even worse in this case since even if such could be plausibly argued, the logical structure and support of such an argument would quickly collapse since we do not even know for sure what the sweating sickness truly was. Further, it is presumed throughout that a "disease" is something one contracts, from a source ultimately traceable to a microorganism or other type of infectious agent like a toxin. However, to fully understand a disease, one has to gain a sense of it within a particular context: so while the single-factor causal explanation remains too easy, contemporary fears about this disease appear to have played a notable part in just who actually showed up at Bosworth, and even under what circumstances and with what loyalties.

Next, there remains the fascinating question of just how many scholars over the years have in fact grappled with this disease, whether they seek to mention it as quickly in passing as possible (as "plague," "ague," or the like), or perhaps opt to give it its due attention, sometimes leading to other elaborate and often finely argued assessments of what it must have been. This always entails at least some degree of retrospectively diagnosing a disease: even though we know some "famous" victims, whether they survived the illness or not, there exist notable problems with trying to identify it further, through, say, dealing with their bodily remains. Additionally, whether diseases can truly be diagnosed this way is itself a debated issue, and many experts in these different fields, including many sciences, have begun with the (usually unstated) assumption that such can be done, even if it perhaps may have to remain tentative, a historical "best guess" based on the available evidence. That some of the evidence is only partial or often unreliable, or both, hardly helps, and at least one of these two unfortunate features

applies to the usage of the likes of old parish records and changes in people's wills, to take two examples which will be treated herein regardless. One may just as easily make the logical case that this notion may extend to any writing of history, at least if one starts from the equally basic rule that, at least within history, written records are usually all we have from which to proceed. Herein the reader will find this assumption continued, and expanded: that *sudor anglicus* indeed can be argued for, rationally and plausibly, and that, like any good history, the process necessitates borrowing from other subjects and fields while also in the process understandng one's chosen time and place becoming clearer and more approachable overall. So to gain our first understanding of the sweating sickness, we need to evaluate the conflicted political situations at Bosworth, and how the Stanleys could come to feel justified in making such a blatant betrayal.

First, tenuous family ties are worth considering. Historian Alfred Rowse notes the strange politics of how the first husband of Lady Margaret Beaufort "died when she was only a girl of thirteen, leaving her pregnant with the child who became Henry VII. She married twice more, Henry Stafford and then the Early of Derby," [4] so Henry Tudor was actually the stepson of Thomas Stanley. Dynastic family ties notwithstanding, more recent historian Michael Jones confirms that "the Stanleys were a rising force... determined to protect their landed estates and influence." [5] Even more tellingly, family "self-interest saw the pursuit of a kind of insurance policy where the family tried to back both sides in a conflict." [6] Even step-relatives might be perceived as valuable allies during a period when kinship mattered, yet this practical if amoral approach to getting ahead must have proved difficult to justify and maintain. Still, "the extraordinary juggling act was to complicate the forthcoming battle... the prevarication of the

Stanleys shows us how difficult it is to interpret Bosworth simply on moral grounds," [7] including our assessments of Richard and Henry.

Shortly after the landing in southwest Wales by Henry and his forces, William Stanley had permitted them to pass "along the borders of his jurisdiction." "Suspicious and alarmed," as Richard might be expected to feel, he summoned Stanley to explain this apparent kindness and perhaps dereliction of duty, but "Stanley replied that he was ill of the sweating sickness." [8] A key part of the answer must lie in a secret meeting at the village of Atherstone, Warwickshire, on 21 August, the day before the battle. Rowse describes this fateful meeting as follows: "Richard sent a message to Lord Stanley ordering him to join in against the enemy without delay," [9] threatening to kill a son of Stanley's held hostage. Henry, meanwhile, "was also kept in anxiety as to what Lord Stanley would do," though "after the conference at Atherstone, Henry was in better heart," even if he received a more dubious reply the morning of the battle.

For historian Michael Jones, "the most likely member of the family to take positive steps in Tudor's support was Stanley's younger brother Sir William, but following a tense meeting at Atherstone... neither man committed himself directly." [10] Still, even with the aid of Stanley, Richard came close to winning: these historians agree with the old claim that the king really slew Henry's standard-bearer, thus getting within what must have been mere paces of his enemy. And "as it was so hard to comprehend the battle's outcome, the only explanation some could find was one of treachery. Sir William Stanley's intervention could rightly be seen as a betrayal of Richard III by that powerful, self-interested family." [11]

Noble egos aside, Gladys Temperley confirms that "the attitude of the Stanleys was of the utmost importance... but they preferred not committing themselves to either party until they saw how things were going." [12] This must have seemed callous then, as it likely still does now.

So, a key question for this background becomes, did sweating sickness really play any part in the decision of Stanley to forego his loyalty and thereby switch sides, or did other factors play a part in this? Rowse describes Henry's arrival in London clearly, with its implications: "as soon as he could Henry dismissed his foreign mercenaries. He made his ceremonial entry into the City on 3 September 1485, though an outbreak of sweating sickness postponed the coronation till 30 October." [13] This is Rowse's second and final mention of the disease, though it seems quite telling. These were of course the same mercenaries who may have unwittingly brought the disease with them. Temperley confirms this basic scenario, describing how Henry "was busy preparing for his coronation when the 'sweating sickness,' hitherto unknown in England, appeared in London. The disease was very virulent," [14] killing but then vanishing by October. Further, "many have thought that the disease was brought to the crowded streets of the capital by Henry's foreign mercenaries. The visitation was popularly regarded as an omen of a stern rule and trouble reign." [15]

Biographer Sean Cunningham offers among the more succinct yet powerfully explanatory summary of what was at stake. "Unfortunately for Henry," he tells us, describing the then-upstart's coming into England to stake his claim, "his army carried a virulent new disease, known at the time as 'sweating sickness', which began to kill hundreds of Londoners within a month of Bosworth." [16] Cunningham points out that Henry was already in trouble for this, even if it was hardly his fault. "The violent change of ruler and the outbreak of disease were linked as an omen of disaster," [17] with some likely questioning whose side had received divine favor after all. The outbreak thus "tested the organisational and propaganda skills of the fledgling king," who even worked to censor public news about this aspect of his very new reign. Yet Henry should not have felt especially picked upon in this regard. Writing about his

immediate predecessor's apparent bodily deformities, Mary Ann Lund observes how "in Richard's case, this purported link between physique and character was frequently underlined, and as the Tudor regime became established, his image became more distorted," [18] becoming eventually the malicious crookback of Shakespearean drama. This speaks to an ancient superstitious link between the health of the ruler being tied to the health of the reign, as with the Grail King from Arthurian legend: only a healthy and justified ruler could enjoy a good term on the throne.

Despite all the possible influences of diseases in general and of one strange disease in particular, part of understanding a key player in the ensuing drama of Bosworth lies in knowing that the Stanleys perceived themselves as kingmakers. Earl Thomas also receives credit from Aisling Byrne and Victoria Flood for helping to thwart the royal designs of pretender Lambert Simnel (whose true name remains unknown, but who claimed to be a Yorkist Earl of Warwick, even undergoing a farcical crowning ceremony as early as 1487), and for playing a "crucial role" at the Battle of Flodden in 1513 during Henry VIII's first military excursion to France. Even alleged kingmakers could falter, though: William Stanley would be "executed in 1495 for supporting Perkin Warbeck," [19] another impostor to the throne, this one falsely taking on the role of the Duke of York. So they remained quite powerful before and after Bosworth.

Yet the best summary and most appropriate place to leave this historic battlefield is with the appraisal by John Norwich, describing the chaos in northern England. "(F)rom this point onward the picture (of the battle) becomes hopelessly confused... It seems too that Henry, determined to make one last appeal to the Stanleys, suddenly rode off towards them; and that Richard, recognizing his banner, led his men against him in a direct attack... Henry found himself, for the first time, fighting for his life... He was saved by Sir William Stanley." [20]

Norwich makes no mention of the disease either, but whatever it truly was, it may well have generated enough anxiety to help shape the outcome at Bosworth.

As for the influence of the Sweat upon Bosworth, it is curious that with the literature consulted herein, the writers describing the disease mention the battle either in passing or not at all, while those addressing the battle mention the disease either in passing or not at all. Again, no one wishes for the simplistic causal explanation, and yet the Tudor dynasty would not have resulted without Henry's victory at Richard's expense. So "Henry's reign therefore began with an urgent need for command of the crown's relationship with the population," [21] as Cunningham relates the immediate need of the new and young monarch to be perceived by his subjects as in control, justified with his royal position, and sympathetic to the sufferings of those same subjects. Winning at Bosworth, regardless of his allies and favorable circumstances, Henry had to address the question of what to do about a disease that no one could possibly understand.

Some contemporaries understood it as divine judgment, itself a possible explanation for illness for probably as long as humans have been reporting their afflictions. Occasional epidemics of actual plague certainly took far more lives, and even though plague itself would not be more thoroughly understood until a working germ theory of disease emerged so much later, plague remained more of a known factor. Fleeing it, and indeed escaping from any disease outbreak, always seemed the best course of action, and while some Tudors might still decry plague as God's wrath upon a sinful populace, at least there was some predictability of when and where it might occur, how it would affect victims, and how to perhaps avoid it in the first place. But Sweat was much less certain: who was most vulnerable, contemporaries wondered? Where did it come from? Did it affect some types of persons more than others, and if so, then why?

Historian Danae Tankard writes that "the epidemic of the sweating sickness was perhaps the first real test of a Protestant nation," [22] though the focus of her work in this context is the final Sweat epidemic, and the first four outbreaks, during the reigns of Henry VII and Henry VIII, occurred within a kingdom that even during the 1540s was still taking tentative steps toward religious reform. Still, maybe the specific year 1551, on which Tankard concentrates, should be regarded as such a test, with young Edward still on the throne, before his very counter-Reformation-minded Catholic half-sister Mary occupied it, with the essentially Protestant and more conciliatory Elizabeth coming to power later. Tankard describes how by April of that troubling year, rumors of uprisings in London, and coin debasements, with curfews, appeared, and this was still almost three months before the illness itself returned. Sufferers and typical citizens alike wondered what might have led to such trouble, and in the case of their health, typically continued to "interpret sickness and death in providential terms as being sent by God." [23] As for popular historian Peter Ackroyd, 1551 would be recalled as a "year of horrors." As he summarizes, "the debasement of the currency... had the natural consequence of inflating the prices of the basic and most necessary foodstuffs. The harvest of 1551 was poor, the third such harvest in a row; and the European market for English woolens had diminished... Money had lost half of its value since the last days of Henry." [24] The latest epidemic would simply compound "all the distress and woe." When Parliament resumed in early 1552, religion and its proper practice headed the list of crucial issues, above economics and health care.

Nükhet Varlik, an historian who has helped illustrate links between diseases and potential causes, speaks some of moral causality in this context, since "plagues and moral decay were considered to be signs of the apocalypse (well, on a more grandiose scale, anyway). Moreover, moral decay was seen as the cause of plagues." [25] This returns us to the issue of whether the sick

somehow deserved their fates, which appeared in the context of not just plague (mainly due to its utter devastation, especially in the mid-fourteenth century), but also of leprosy. The other interesting detail at least in this one case is that Varlik keeps his own interest limited to the early modern Ottoman Empire, which under the rule of Suleyman I proved an occasional counterpoint to Holy Roman Emperor Charles V, who of course in turn might pursue an alliance with Francis I of France against Henry VIII of England, or with Henry against Francis (or he might even find himself facing Henry and Francis; the first half of the sixteenth century had entertaining international dynamics). The tripartite Christian powers often get described together, with the Muslim empire to the east as more of an afterthought, but considering that medieval Europe had already benefitted so much from philosophical and medical texts translated in Muslim lands and then brought back to Europe translated again into European languages, the Ottoman perspective seems worth investigating. And while "apocalyptic" seems a bit hyperbolic in this context, here again is the unfortunate notion that those suffering the most deserve their fates. And effects of plague certainly compelled the reevaluation of priorities, since "plague was a reminder of the transience of everything connected with life; it stopped work and destroyed wealth." [26]

There is surely more to it than that, and for all the trepidation than any disease outbreak may elicit, the inhabitants of Tudor England might be said to have taken at least a slightly more rational approach generally. Of course there were exceptions, and our own time hardly proves any exception either, such that future historians may wonder just how many of us allowed ourselves to succumb to the potential fear-mongering of reports of strange diseases like avian influenza, Ebola viruses, acquired immuno-deficiency syndrome, or hantaviruses. This author clearly recalls the bigotry and alarmism during his high school years over just how deadly and apocalyptic AIDS was supposed to become, but again, it is vital to consider disease in context, to

truly understand it. Diseases do not occur in vacuums: this is not mere scientific quipping, but a literal truth, since diseases vanish without the continued interplay of hosts and vectors and, ultimately, victims, whatever their fates.

Greater rationality, despite the fear, which has lingered through the ensuing centuries into scholarly curiosity, is hinted at early in the Tudor years by Thomas More. "For if you suffer your people to be ill-educated," he writes in *Utopia*, "and their manners to be corrupted from their infancy, and then punish them for those crimes to which their first education disposed them, what else is to be concluded from this, but that you first make thieves and then punish them." [27] More could still be frightfully judgmental, as with his willingness to burn heretics, but even this decision came from the rational interpretation of a religious argument, even if built on faulty premises (heresy was dangerous, and the most appropriate punishment for non-recanters was a preview of the fires of hell, and fire itself has a cleansing as well as destroying aspect to it). This makes it similar actually to humoral theory and astrology: valid but unsound arguments built upon initial falsehoods, whether the idea that all that exists is reducible to four primordial components or that celestial bodies much further away than early peoples could have imagined actually influenced their overt behaviors. Nor did More given any indication of recognizing the great irony of his life, probably craving the very martyrdom that he enabled in his harsh judgments of those Lutherans (who may have fit his own notion of the "ill-educated" in his earlier idealistic treatise, making it a double irony). More "was convinced by the time he began *Utopia* that a liberal arts curriculum involving languages, history and philosophy was better suited to the creation of a good society than a vocational one based on law," [28] or even traditional theology, or the ways of war favored by the medieval nobility, whose children would likely remain illiterate. Further, More "believed it met the needs of girls and boys alike." And in the

case of his daughter Margaret, there is a link to the Sweat, too, since she contracted it but survived, thanks to a rationally minded treatment. "Of all pleasures," More writes later, "they (the residents of the dreamy Utopia) esteem those to be most valuable that lie in the mind; the chief of which arises out of true virtue, and the witness of a good conscience." [29] There were times in Tudor England when once might blame the victim, as with heresy, but in the case of disease, the earlier medieval thinking about deserving to be ill was fading.

As a final note on this antiquated idea of just desserts, Victor Vaughan notes that "the claim has been advanced that the infectious diseases have benefitted the race by the destruction of the unfit," [30] though this highly prejudicial occasional trend in historiography presents a straw man fallacy of both evolutionary theory as well as what "fitness" means in the context of evolution and natural selection. Vaughan himself uses typhoid fever as an example to try and make this point, but such a claim could perhaps be forwarded in reference to any disease with a historically high human body count, and plague itself is a popular candidate from a longer-term perspective, since its destruction during the Black Death years of the late 1340s contributed, decades later, such thinking goes, to slightly improved rights and standard of living for the masses of peasants who could by then demand more from the nobles as the fields of the latter lay unworked and unproductive. This represents a largely economic and political oversimplification, but arguing that horrific disease epidemics might have a metaphorical silver lining years later does have a certain appeal, like maybe the suffering and death might have eventually been worthwhile. Still, Vaughan evinces an often all too typical historical bias as well, popular to invoke about the Middle Ages, in that they allegedly "were indeed dark physically, intellectually and morally." [31] And yet "the history of medicine is that of mankind," [32] as he states, and it has offered, he maintains, more to the advent of science than any other field of inquiry.

Whether we have an obligation to be intelligent, as Vaughan insists, might sound either Kantian in moral tone or Galtonian in its condemnation of the "unfit," though the moral issue keeps appearing in the literature, about plague and sweating sickness and other diseases as well. Another summarizer of the alleged link between morals and diseases is historian Patrick Wallis, writing about how "epidemics throw into question people's moral responsibilities to the communities in which they live." [33] By the sixteenth and seventeenth centuries, Wallis argues, there appears a clearer consideration touched on during prior epidemics of "whether a person should flee from disease, or stay and help the sick." This represents a moral shift from culpability of those afflicted to how the unafflicted should respond. J. F. C. Hecker also speaks to this moral issue, and even though he writes decades before the judgmental Vaughan, describing how "morals were deteriorated every where, and the service of God was, in a great measure, laid aside," [34] so sufferers and neighbors did not become morally blameworthy until their behavior in the midst of plague was considered. Further, "repentance seized the transgressor," [35] as sinners might well become more aware of their dubious actions during outbreaks, and in what might be interpreted as almost a concurrence of thinking of the greater good along with Vaughan, Hecker describes how human fertility would increase after plague strikes, and rather than evidence of a wrathful if omnipotent deity, such would instead signal "the prevalence of a higher power in the direction of general organic life. Marriages were, almost without exception, prolific," [36] at least after the outbreaks.

Plague had been understood historically in this context, too, with something as horrific as the Black Death leading countless sufferers and survivors alike to wonder what they must have done to so anger God. The two key logical differences when we advance in time two centuries are that while the plague of the fourteenth century killed exponentially more people, the sweat of

the sixteenth century seemed to only pick English victims. Among the early modern commentators on plague is historian William Kelly, who studied its effects specifically in and around the city of Leicester. While he never connects the disease to sweating sickness, his writing emerges while more detailed research into the nature of disease and associated microorganisms was also appearing. Contemporary authors like Daniel Defoe and Walter Scott even contributed stories about plague, though they shifted the action to London. Slowly vanishing from such tales, as well as from historiography, was the notion that disease in general and plague in particular represented divine judgment, and that victims, surviving or not, deserved their punishments. At the much more recent end of plague historiography, that trend can still be traced, something noted clearly in the work of Varlik. Even in the early modern period, religious causality was understood similarly to contemporary Christian and Jewish traditions, in that "plagues were inflicted upon humans by God, and God alone had the power to relieve humans from this ill." [37] The early modern Muslim perspective within the Ottoman Empire as described by Varlik differed a bit, as it also had with more medieval medical interpretations considered via observation more than religious influence. Part of this means that after prior medieval treatises on disease and medicine, "the mainstream Ottoman historical narratives composed in the sixteenth century gradually eliminated the theme of devastation" [38] and divine retribution. More of a notion of early public health was developing instead, something which would not be remotely attempted by the British until at least the first Poor Law under Elizabeth I. Paul Slack in particular remains unimpressed by this initial effort, mainly the *Act for Relief of the Poor* of 1597, partly since "the machinery of the poor law was not designed as an economic regulator, but as a moral, social and political one." [39] The moral component may be more clear in recognizing that "the presence of able-bodied paupers in Tudor society caused great

consternation. Charity was meant for the infirm, the malformed, and the helpless." [40] The interesting part for commentators like Varlik and Slack, however, is that the early modern period seems to have witnessed a shift in attitudes about what one should receive in life, and whether a person truly deserved to be poor, or to become the victim of a potentially fatal illness. Sometimes it takes a major assault on social sensibilities to enable such a change in thought. Physiologist Archibald Sloan, for one, notes how "the sweating sickness was no respecter of rank. Unlike the plague, which was typically a disease of the poor, it affected the highest classes of society and appeared to single out young, previously healthy men." [41] Whether Sloan's reporting in this context is accurate will be considered in due course, since it does seem rather "unfair" for a disease to target the supposedly strongest members of a society, but even if only the perception of this at the time was broadly true, then it must have led at least some to speculate about who deserved what, at least in some contexts. And a greater exposition of Tudor values and thus their morals will appear later, some initial comments deserve to appear in this context, since dealing with disease, especially plague in all its historical destructiveness, has not just killed indiscriminately but also posed medical and moral questions.

Even during the fourteenth century, fleeing was known to lessen the likelihood of contracting plague, as Wallis explains, but this could be easily offset by obligations to the ill, as well as to other possible victims, like the poor, or one's own neighbors. Urban officials and clergy might even be held to similar standards of rendering aid like physicians, and yet "authors (of moral treatises) worried about poverty and disorder, not medical desertion." [42] Only those physicians employed "civically" would really be expected to remain, though an irony existed in that the same physicians might well be those hired exclusively for the service of wealthier nobles who could further afford to escape areas struck by plague, thereby saving the doctors as well as

themselves. For Wallis, the moral component shifts, by the seventeenth century if not earlier, from blaming victims to demanding more dedicated behavior from those educated the most highly in medicine. This is the more remarkable for Wallis keeps his focus strictly both on plague and on early modern England. Contemporary hyperbole may also reflect perception of the Sweat being understood "as an instrument of divine correction," [43] to quote historian Alan Dyer, who will have much more to say about viruses with regard to the Sweat. As for the poor, they could be understood at the time "as objects of charity," writes Slack, as "targets for the pity, sympathy, generosity and sometimes admiration of (their) betters," [44] in another ironic twist. Additionally, and of more immediate concern, "they might appear as a threat," to be removed from consideration for charity and sympathy, or even as a "productive resource." Slack has little use for the possible conscience-based assistance argued for by earlier historian Hecker, though, insisting that during times of disaster human behaviors mostly remain "rational," though this seems to pose more questions about human psychology and morality. Some respond and assist in crises; others flee or at least look to themselves, and even the same person might alter such behaviors depending on the exact circumstances. And even Slack concedes that victimization could result from prejudice as much as from disease. While early Tudor identifications of the poor included "beggars" and "vagabonds," soon were added to their ranks the "dangerous poor," with growing concerns about them as well as "new or newly virulent, infectious diseases of the early sixteenth century: syphilis and the sweating sickness." [45] Such afflictions might explain the abject "horror at the 'incurable diseases and filthiness of body' and the 'maladies tedious, loathsome or abhorrent to be looked upon' which were now associated with the poor." John Wylie and Leslie Collier refer similarly to a dearth of clergy as victims, which "might partly be explained by the sharp decline in their numbers" [46] after the Dissolution had run its course.

Clergy were on the decline as an educated group, and yet contributing to the numbers of poor, a double societal loss and social problem. Wylie and Collier will turn out to be those who bring sweating sickness research back into the academic spotlight in the late twentieth century, which continues into the twenty-first.

As we leave Bosworth behind, then, the main idea is to remember that the new king, founder of a new family dynasty, had to justify his reign and he already had two major issues facing him: how to justify that reign via alleged right of conquest, and how to resolve the question of whether his rule was already manifesting divine sanction in the form of a new illness. If no one deserved to become sick on their own, then something, or someone, else must have been culpable.

To further summarize, this dissertation asserts that we must content ourselves to argue in this case as historians more than as scientists or other specialists, using plenty of evidence, to be sure, but in the form of written and testamentary evidence, and not in the form of physical specimens of any kind which might be submitted to some form of laboratory testing. Since the focus herein is on disease, ultimately on one disease in particular, this initial claim may frustrate the more scientifically-focused, and yet it will be shown that the period of Tudor history within England can still surprise us, that there are still early modern historical mysteries in need of the best resolution possible, and that the likely inability to subject the existing evidence to more scientific testing should at no point dissuade anyone from considering what follows.

While the next section will commence with what is known of "the Sweat" from primary sources, it remains essential to begin to consider how more recent researchers and scholars have attempted to address it. The most compelling work emerges within the last half century, though some of the sources herein will reach as far back as the earliest secondary sources, appearing

during the seventeenth century. Interestingly, writing about the sweating sickness becomes minimal after the final known outbreak in 1551, though occasional comparisons appear later with the "Picardy Sweat" which developed later in France. That disease will also be discussed later, since it may have an unusual connection with English sweating sickness. Additionally, scholarship regarding the sweating sickness more recently (from the latter twentieth century into the present), rather than suffering from narrowness or a sense of frustration in making a retrospective diagnosis, has actually flourished, with scores of scholars offering their own insights and perspectives into this historical mystery. While some commentators have reluctantly conceded that perhaps the ultimate culprit behind this disease must remain unknown, even they have acknowledged that additional work might be done, and indeed often offer their own suggestions as to what else might yield results, ranging from the practical, to the time- and effort- and resource-consuming, to the far-fetched. Still, prior to considering the actual work of these scholars, it is crucial to address some background questions, the answers to which will further justify and clarify the research behind this dissertation.

To begin with, there is the just mentioned phrase regarding a "retrospective diagnosis." Lester Little may have described this issue best, referring to the academic "Cunningham debate," which within the history of medicine explains the notable and crucial difference between identifying diseases by recorded symptomology, as with our previously discussed primary accounts, and identifying them via some form of actual laboratory testing, yielding empirical confirmation. As historian Little puts the matter, "the days of diagnosing past illnesses solely upon the basis of written sources are numbered if not entirely gone." [47] That indeed sounds encouraging for history generally, but it does pose a question for this current project which will have to be addressed. And matters are even more complicated than that quotation might suggest.

A century ago, for example, historian J. T. C. Nash noted how "the evolution of disease implies and involves the evolution of *ideas* of disease as well as of causes... Hence the importance of careful observation of phenomena objective and subjective." [48] The implication here is that even our notions of what constitutes disease, seemingly a straightforward idea, do not necessarily yield specific working or binding definitions. [49] And while writing mainly about plague, Nash referred to his own earlier work, in which he noted "that in comparing the 'sweating sickness' of the later Middle Ages with modern influenza we gain an insight into evolution in thoughts and ideas as well as in actual disease processes." [50] While influenza itself will be evaluated as a contender for sweating sickness, it seems that with this retrospective notion, the effort is becoming more complex. This dual evolutionary process highlighted by Nash is also referred to by more recent historian Nancy Siraisi, among the many historians herein describing some of the implications for what can be described as a general shift during the early modern European period from medieval thought about disease, often itself rooted in much older theories, to a more recognizably scientific approach. There will be no discussion within the body of this dissertation of whether or not a true "scientific revolution" overall took place, though for now it is enough to note that approaches and understandings were altering during the Tudor period. Part of that process of change entailed recognizing that it had become more challenging to arrive at "contemporary concepts of evidence and its interpretation in works relating to disease, diagnosis, and treatment." [51] The need to advance the known *materia medica*, which at the time included the work of physicians, surgeons, apothecaries, midwives, and even gardeners, also "connected medicine closely to an expanding world of commerce and craft." [52] In essence, what qualified as a working diagnosis and its accompanying treatment could and did vary, not just through comparing ourselves to our ancestors, but also in comparing them to each other. Politics and

economics were also helping to change ideas of disease, in addition to science and medicine. We already know the sweating sickness was almost entirely English, even if it apparently had a Continental origin and was inadvertently exported back across the Channel during one of the outbreaks. How to classify it, even how to think about it, much less how to render a *post hoc* diagnosis of it, must raise their own problems.

Little himself noted how even a term like "plague" might be specific or vague, depending on context and which medieval or early modern writer one consults. It might be a more generic term, "a label for a disease of extreme virulence," [53] as researchers John Theilmann and Frances Cate describe it, though they opt to focus on a stricter biological definition of plague in their own work. While Tudor commentators, particularly physicians and historians, understood the sweating sickness to be something distinct from actual plague, the latter term nonetheless has appeared various times since at least the Middle Ages as a catch-all description of any disease leading to an epidemic, sometimes including the sweating sickness itself. More specific definitions of relevant medical and biological terms will appear below with some help from modern texts, but for now the important point is that our understanding of and even definition of disease has often been as evolutionary as some diseases themselves have been. For now, continuing in the tone of Nash and Siraisi, the work of Jon Arrizabalaga is worth noting, as he reminds us that "the idea gradually spread that those phenomena labeled as diseases are not merely biological events essentially continuous in space and time or, at most, subjected (in the case of infectious diseases, resulting from microorganisms) to bio-evolutionary changes linked to the host-parasite interactions." [54] This does not mean that definitions need be fluid or vague, but rather that "they are also, and above all, human constructs resulting from specific socio-cultural contexts and, as such, only understandable within these specific coordinates." This dissertation

could actually not exist without disagreements about diseases in general and one historical disease in particular, part of the reason that this point needs emphasizing. As Arrizabalaga continues, "a real understanding of disease always goes far beyond its mere biology." [55] One of his own examples is sweating sickness, and he compares its historical understanding to that of typhus, which also took time to be comprehended fully.

Interestingly, sweating sickness is hardly the only disease from the Tudor period to offer its own potential ambiguities. Theilmann and Cate note this too, and working on the background of plague, decide that "checking symptoms is one way to diagnose a disease, (but) the chroniclers are decidedly unhelpful in this area," [56] so some other sources may be required for such research. Historian Charles Volcy notes recently how diagnosing was hardly without its problems for the time, either, since a Tudor medical diagnosis "se limitaba a la observación pasiva de los pacientes y no aplicaba métodos activos como la palpación, la percusión y la auscultación." [57] Such basic "vital signs" as palpation (for heart rate, as an example), percussion of extremities, or auscultation (often to check respiration), did not become routine until later.

In her own book covering royal physicians during the Tudor and Stuart periods, historian Elizabeth Purdell describes the issues surrounding our knowledge of Henry VIII in particular, who, while escaping the Sweat during the 1528 outbreak, has been himself retrospectively diagnosed with "malaria, gout, alcoholism, and syphilis. He had bladder trouble as early as 1528 and himself recommended a cure for tumor of the testicles." [58] The King had no shortage of the finest physicians around him, some of whom would be professionally inherited by his three children who became the later Tudor monarchs, and while one wonders what his alleged tumor cure may have been, this already proves quite a list of illnesses. [59] While not myself arguing for or against any of them here, biographers and other historians have attempted to explain some of

Henry's apparent behavioral changes based on some mix of illnesses and injuries and how such may have affected his psyche as he became less active, morbidly obese, and continued to work his way through wives while trying to steer a course as head of the new Church of England. This is not to serve as a return to the "great persons" tradition of historiography, but rather to point out that an understanding of one historical detail, be it whatever altered Henry VIII into more of a despot or what a certain disease truly was, can genuinely affect our understanding of history. Knowing what this disease likely was, to the best of our ability, offers a better understanding of the Tudor dynasty.

Second, beyond this issue of retrospective analysis, it is legitimate to ask if it would not be simpler and more satisfying to just access human remains, if possible, and test them by whatever scientific means might be necessary in order to ascertain just what the sweating sickness really was. This was the ideal espoused by Little, for instance, and an ideal hinted at by Siraisi, as well as by Theilmann and Cate, and after all, it seems the whole point might be simplified and thus rendered academically moot if some mix of archaeologists and infectious disease specialists could study actual remaining tissue samples from the Tudor period and offer a working present diagnosis. This is the other half of the Cunningham debate. And this notion would seem even more appealing since we indeed know the identities of several contemporaries who either survived the sweating sickness or died from it, and in the case of parish records (which can include causes of death, and changes in wills to reflect implications for families from disease, and which, as will be shown below, some scholars indeed prefer), we might be able to ascertain still many more who acquired the disease, whether they survived it or not. Yet as it turns out, this question has a resolution which can only necessarily leave us relying upon the aforementioned retrospective diagnosis.

To begin with, on the topic of possible testing of human remains, the Department of Anthropology at Oregon State University has staff specializing in such, even a medical anthropologist in the person of Professor Melissa Cheyney. When I contacted Dr. Cheyney in early 2015, she put me in touch with one of the university graduate students, Dawn Alapisco, who was serving as an adjunct instructor and who graciously agreed to an interview about these types of questions. I was initially quite hopeful, if a bit naive: if some kind of testing could be done, I thought, then my dissertation research could perhaps extend to some kind of field work. Yet I was soon availed of my prior optimistic delusion. The first problem, as Ms. Alapisco pointed out, was a mix of biology and time. Simply put, different microorganisms have differing degrees of survivability, and even these variables will change based on how and where bodily remains are disposed of. Burials compared with cremations, for example, or even burials in different types of containers and in different soils, will affect how tissues decompose and what else might be found among such tissues years or centuries later. And again, without knowing what to test for precisely as in this case, the problems get compounded. Hypothetically, and without such practical concerns as costs, I wondered about the possibility of simply testing different specimens from the same bodily remains for known diseases: why not test for all the various bacteria and viruses considered in more detail below (all of which have been offered as explanations for sweating sickness), until we arrived at some more definitive matching among those already identified as having had that disease? Yet "disease alters DNA," Alapisco further explained, and not just of the remains but potentially of the very microorganisms I dreamily thought of studying, briefly fretting whether I might need another graduate degree in biology or anthropology to proceed. Ultimately, unless sweating sickness left some type of indicator behind in osseous tissue, "medical anthropology is unlikely to help much, if at all." [60] Soft tissues,

meanwhile, after four to five centuries, should have sufficiently decomposed to be useless in this context, typically regardless of whatever containers in which they may have been buried. Royals might have received lead-lined caskets; commoners could typically expect wood caskets, or, more often, linen shrouds, and both lumber and linen likewise tend to decompose more thoroughly and quickly, especially in the often damp soils of Britain. Considering all this information, Nash seems appropriate again, having shifted his focus to sanitary practices, including burials. "Very little sanitary provision was made" [61] during Tudor times, he tells us, and not until 1543, well after the medieval plague (at least in its most destructive former outbreaks), but during the time before the fifth outbreak of sweating sickness, the earliest known English plague law appeared, "and provided for notification, quarantine, destruction of infected straw, etc." [62] While this starts to outline the haphazard development of improvements in burial practices, it also means field researchers now would have to be quite careful in determining which persons from the time really might have died from sweating sickness. Historian Mary Dobson is also aware of Tudor recognition of how different environments could contribute to not just the preservation of bodies, but of hygiene in general. "Outstanding among these concerns in the early modern world was the idea that the environment and the atmosphere," Dobson writes, "the lie of the land, the nature of the terrain, the type of soil, the smell of the place, the proximity to stagnant, salt or fresh water sources, the vagaries of the weather – all appeared to influence and impinge on the health and well-being of a locality and its residents." [63] Dobson's historical research will be of even greater relevance when we consider the possibility of some kind of poisonous agent being responsible for sweating sickness, but for now we have to accept that Tudor burial practices, shaped by medieval tradition, changes in land usage (most obviously in

the form of enclosures for grazing), and competing religious demands, tended overall to not leave much in the form of human remains for us to study presently.

The potential good news, Ms. Alapisco assured me during our interview, was that "osteo tissue remains," [64] so long as it is not exposed to an environment in which it too would tend to degrade. Some such samples, even discovered after centuries, can offer telltale signs, such as her own example of how thinned cortical bone and perocity indicate malnutrition. Ms. Alapisco continues to work on categorizing an assortment of skeletons found from the heyday of the Byzantine Empire, considerably older than anything Tudor, and yet in many cases likely also better preserved in a warmer, drier climate. As for particular diseases affecting subjects of Tudor England, since different microorganisms vary in their requirements in terms of seasonality, whichever disease I found myself arguing in favor of, there would remain the issue of trying to match what is currently known about it with descriptions from primary written sources (even knowing the concerns of writers like Little, or Theilmann and Cate), and the latter might suffer from various inaccuracies, at least in a more modern context, perhaps part of that shift in focus that the likes of Siraisi and Nash described. Yet even this would still not resolve the confounding issue of knowing just what biological marker or markers to look for, nor could it hope to address the open question of just how many sixteenth century bodies, in whatever condition by this time, might have to be exhumed for such invasive study and testing. As Nash himself concludes, we may have to deal "with complicated *historical* processes related to a far-reaching past, and hence only to be *approximately* estimated. Hence we have to proceed by *induction*, with proportionate confidence from the accumulation of detailed observations." [65]

The next issue, which I had already gained some notion of, reduced to simple politics. As some scholars point out, notably microbiologist Edward McSweegan in his work about

anthrax as a contender for sweating sickness, while gaining access to human remains in some English parishes might be as simple as explaining one's reason for exhumation to the local religious officials and gaining their written permission, the remains of the more famous persons to have had the "Sweat" remain inaccessible without royal consent. Such a political statement hardly seems likely to be forthcoming for a foreign graduate student who might want "to dig up Anne Boleyn and Thomas Wolsey" to perform some type of testing which itself must remain elusive at this point. And it is, after all, rather difficult to "test" any type of organic tissue specimen for a disease when one does not ultimately know what the disease actually is in the first place; as will be seen, whether sweating sickness was bacterial, viral, toxic, or something else, is itself a largely contested question in the academic literature, something McSweegan himself and many other scholars acknowledge. Indeed, attempting to resolve that dispute forms a central part of this current project. So the logic surrounding the possibility of biological testing has in fact become circular: any form of consent to disturb the long dead would need information which could only really be obtained post-exhumation, and even then might not be forthcoming. Usable specimens might indeed prove impossible to obtain and test even if exhumation could proceed, though that is a separate issue. If such testing were ever to occur, it would have to be justified with a better understanding of what sweating sickness most likely was. Perhaps some kind of testing might become possible at a later time, but for now we must content ourselves with historiography rather than microbiology or anthropology.

So until then, logic itself remains. And yet that scholars have disagreed so sharply, for so many years, about what the sweating sickness was, means that we have plenty of clues and other types of evidence. This dissertation, accordingly, is in no way a treatise about anthropology, nor a collection of biographical sketches, and certainly not a work on microbiology, virology,

toxicology, or epidemiology, either, even though it must consult work done by experts in each of these fields. Nonetheless, we should actually feel reassured by the very diversity of not just opinions and conclusions about the sweating sickness offered by so many scholars, but also by the diversity of their own backgrounds which have shaped their findings. Having addressed the concerns of those who might argue about the impracticality of retrospective diagnoses, I can now point out that, as so often within the discipline of history, we typically have incomplete source material, which tends to make us more thorough arguers. And in this case, there is in fact a wealth of material, and when historians wish for more, they simply have to argue the most cogently and strongly that they can. Arrizabalaga can help once more in this regard, explaining that "the fact that there has been no consensus among them about its nature makes it a good idea to explore some of the multiple labels that sweating sickness has been given during the last one hundred and fifty years or so." [66] So what we need is a systematic evaluation of all the agents suggested for the sweating sickness, but this time from the perspectives of more modern researchers, almost all of whom have made their cases just within the last several decades. First, we need to revisit the Tudors themselves, and consider what else they had to offer toward understanding this disease, and how its multiple outbreaks affected them. Next, louping-ill virus will be explained more fully, and then the other disease candidates will be explored in more details, with the argumentation revealing that LIV really is the most appropriate candidate as an explanation.

Notes for Part I.

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55. Arrizabalaga, 56.
56. Theilmann and Cate, 378.
57. Charles Volcy, "A Propósito del Enigmático Sudor Inglés." *Iatreia*, 23, 4 (December, 2010), 428.
58. Elizabeth L. Purdell. *The Royal Doctors, 1485 – 1714* (Rochester: University of Rochester Press, 2001), 30.
59. Henry VIII indeed could call on various specialists for health care reasons: "besides a handful of physicians, there were also serjeant-surgeons, principal surgeons, surgeons-in-

ordinary and to the household, court herbalists and botanical physicians, chemical physicians and apothecaries, midwives, oculists for the royal eyes, and aurists (Purdell, 10)."
60. Dawn Alapisco (graduate Anthropology student and adjunct instructor, Oregon State University) in discussion with the author, May 15, 2015.
61. Nash, 29.
62. Nash, 19.
63. Mary Dobson. *Contours of Death and Disease in Early Modern England* (Cambridge: Cambridge University Press, 2003), 9.
64. Alapisco, discussion with the author.
65. Nash, 21.
66. Arrizabalaga, 63.

II. Sweat and the Tudors

A. Primary Sources

Since this is a history treatise first and foremost, the appropriate place to begin is with primary written sources, and fortunately we do possess an abundance of such, even if their interpretation will raise additional questions. While contemporary physicians and other specialists would attempt to address the disease as well, the accounts of contemporary historians offer broader perspectives. Several in particular have left us with works describing numerous details of the period in question and the effects of this previously unknown illness.

The first comes from Italian physician Girolamo Fracastoro, who also displayed keen interests in other sciences such as astronomy and geography. His work probably most often arises now in the context of the ancient natural philosophy known as atomism (different from our own atomism in that the earlier model suggested indivisible atoms, and the void or space between them, as constituting all reality), which in turn shaped his own theory pertaining to infectious diseases. Fracastoro claimed that tiny "spores," of what we would understand as having microscopic dimensions, could infect living creatures with diseases, even over long distances and through minimal contact. This was an intriguing perspective for someone without access to microscopes or a working germ theory of disease, especially considering that his own time still tended to prefer medical explanations immersed in theories like miasma or humors. His treatise, *De Contagione et Contagiosis Morbis et eorum Curatione*, is important to understand, partly to get a sense of contemporary interpretations of the nature of disease, but more directly to consider what he reveals about the sweating sickness. His twentieth-century translator and editor, William Wright, further describes part of Fracastoro's interest as arising within his native land: plague afflicted his home city of Verona in 1510, and the city was struck

further by typhus in 1528. As Wright summarizes, Fracastoro even argued "that the public authorities ought to pay more attention to sanitary methods," [1] an idea which would not gain much credence in most quarters for centuries to come.

It is the fifth chapter of the second book of Fracastoro's chief work that concerns us here. As he relates matters,

> "in Britannia insula, quae nunc Anglia vocatur, genus est pestilentis febris et contagiosae, quae ad ephemeras referenda videtur, quod die una aut hominem perdit, aut liberat." [2]
>
> "In the island of Britain, which is now called England, there is a kind of pestilent and contagious fever, which apparently must be classed among ephemeral fevers, because, in a single day, it either kills its victim or lets him escape."

Prevention remains more important than any unknown or unreliable cure, Fracastoro goes on to explain. Once a person succumbs, however,

> "nullus victus exhibeatur: medicamentum autem ullum utrum sit afferendum habet dubitationem et, an in Britannia recte faciant nullum aliud auxilium adhibendo nisi situm, et decubitum per quem sudent." [3]
>
> "no food must be given. It is a question whether any drug should be administered, or whether they act wisely in Britain where they give no remedy and only keep the patients in bed and lying down, to make them sweat."

Above all, Fracastoro implores readers to not permit sufferers access to wine, since he believes it makes fevers worse. While alcohol consumption does not directly increase fever, both encourage dehydration, hardly the best combination for a person potentially already sweating profusely and thus experiencing at least some water loss.

Perhaps more known in our own time as a general historian is Fracastoro's fellow Italian and also fellow graduate of the University of Padua, Polydore Vergil, a humanist scholar and Church official who arrived in England as early as 1502 in the service of the Catholic Church, even becoming Archdeacon of the Cathedral of Wells several years later. He lived until a bit

after the final Sweat epidemic of 1551, so that he could serve as a (perhaps less than willing) witness to most of the outbreaks. His impressive *Anglica Historia* would become a sweeping account of the first half of the Tudor dynasty. He was also the first to write any surviving record of the sweating sickness itself. Like Fracastoro, he returns initially to the earliest Tudor years.

> "Eodem anno (1485) nouum morbi genus inuasit totum regnum, sub primum Henrici in insulam descensum, dira quidem lues, et quam nulla sit aetas antea experta. Subito enim sudor laetalis corpus corripiebat, ac simul dolor caput *et* stomachum uehementi sudoris ardore affectum infestabat. Quo morbo homines correpti quia alii caloris impatientis si in lecto erant, pannos summouebant, si uestiti, uestes deponebant, et alii sitibundi frigidum potum sumebant, alii demum patientes caloris, foetorisque (nam sudro foedissime olebat) additis pannis, sudorem prouocabant, aeque omnes aut illico aut non multo post quam sudare coepissent, expirabant, ita ut ex omni languentiium numero, uix centesimus quisque mortem euaderet. Et qui post uiginti quatuor horas (tanto temporis spatio uis eius morbi saeuiebat) abeunte sudore, seruabantur, non eo tamen ita expurgati erant, quin iterum atque denuo in morbum reciderent mutique inde perirent." [4]
>
> "In the same year (1485), immediately after Henry's landing in the island, a new kind of disease swept the whole country; it was a baleful affliction and one which no previous age had experienced. A sudden deadly sweating attacked the body and at the same time head and stomach were in pain from the violence of the fever. When seized by the disease, some were unable to bear the head and (if in bed) removed the bedclothes or (if clothed) undressed themselves; others slaked their thirst with cold drinks; yet others endured the heat and the stench (for the perspiration stank foully) and by adding more bedclothes provoked more sweating. But all alike died, either as soon as the fever began or not long after, so that of all the persons infected scarcely one in a hundred escaped death. And those who survived twenty-four hours after the sweating ended (for this was the period when the fever raged) were not then free of it, since they continually relapsed and many thereafter perished."

The sweating and thirst appear here quite dramatically, with the practical consideration of dealing with someone who sweats sufficiently to become unpleasant to be around for the additional olfactory reason. Vergil notes, though, that a potential cure might well occur to those who had already survived a prior outbreak, though he maintains that no resistance or immunity seemed to confer to such survivors. Simply put, he advocates encouraging, even compelling, the patient to sweat as much as possible, and that the most direct way to accomplish such was to

keep wrapping them in as many clothes and as much bedding as they could possibly handle. Similarly, patients were to be kept awake by whatever means necessary, even if they complained of exhaustion. If these did not work (and Vergil, like most contemporary commentators, remains frustratingly vague about fatality statistics, though in fairness such were exceedingly difficult to come by), disease in general and Sweat in particular might instead be comprehended as an omen, reminiscent of the earlier notion of somehow even being deserved:

> "Sudor iste duriciem principis aduersus populum significasse fertur, sub quo quasi cuncti duriter tractati et in initio et in fine eius regni sudare hoc est multa incommoda subire compulsi sint. Caeterum fuit credo ostentum mortis cum labore regnare desineret." [5]
>
> "This sweating sickness was claimed to portend the harshness of the monarch towards his people, by which almost all were heavily oppressed, and under which they 'sweated', that is to say were forced to undergo many discomforts both at the start and finish of his reign."

This is still the reign of Henry VII, however: Vergil is reporting here on the concerns and potential undermined political stability which marked the conclusion of the Wars of the Roses and the inauguration of a new dynasty, founded upon a rather tenuous royal claim. Henry might have had stunning success at Bosworth, yet ruling by conquest seems a shaky basis for government. And Vergil in particular could pose as an aloof judge. At least with the first two Henrys, there often appears in his writing a sense that perhaps Tudor subjects suffered as their monarchs made questionable decisions. Two issues which stand out from his account include the effects of enclosing land for more private usage, and the dissolution of the monasteries, which, like other landowners, varied widely in terms of how sympathetic they may have been to those persons who relied on their responsibility. Yet whether disease or some social rather than medical ill, Vergil's descriptions often leave one feeling sympathetic to the plight of these poor English and their scary new ruling family. Granted, his first loyalty must have been to the

Catholic Church, itself already under attack and criticism during his life, and certainly not limited to the English. But Vergil also gives an immediacy to his reporting, having worked as an ambassador on the far side of the Channel. In between the sweating epidemics of 1508 and 1517 he found himself in political trouble for angering Cardinal Thomas Wolsey, and between that and the disease itself, may have felt fortunate to eventually have been returned home.

Another helpful chronicler to emerge at this time was Englishman Edward Hall, completing his history covering the Wars of the Roses, and trying to account for the lineages of monarchs from Henry IV to Henry VIII throughout a highly detailed if sometimes wordy and cumbersomely titled work. In covering such a brief amount of time in terms of actual years, his attention to minutiae is profound, and he offers something new about sweating sickness each time it appears in his record, published not long before the final 1551 epidemic. Writing about the "second year," for example, from the reign of Henry VII, Hall describes how

> "a newe kynde of sicknes came sodenly through the whole region cuē after the first entryng of the kyng into this Isle, which was so sore, so peynfull, & sharp that the lyke was neuer harde of, to any mānes remembraūce before that tyme: For sodenly a dedly & burnyng sweate inuaded their bodyes & vexed their bloud with a most ardēt heat, infested the stomack & the head greuously: by the tormentyng and vexacion of which sicknes, men were so sore handled & so painfully pangued that if they were layed in their bed, beyng not hable to suffre the importunate heat, they cast away the shetes & all the clothes liyng on the bed." [6]

This is noteworthy for two reasons. First, the amount of detail surpasses that of the other primary accounts appearing thus far; and second, this account also comes from someone with no training in medicine, apothecary knowledge, or surgery, the three most current and sometimes competing approaches to Tudor health care, at least for those not opting to rely instead upon more ancient and generally less regulated means. Not even Doctor John Caius, the most prolific physician to document the Sweat, offers this specific an account of it. The same, in truth, can be said of other contemporary physicians. Doctors Andrew Boorde and Thomas Forrestier dealt

with the Sweat, and others may have too, including Henry VIII's later personal physician, William Butts. Fracastoro himself was also a physician, and some of this account by Hall should seem familiar, including especially the common desire of sufferers to feel cooler rather than have to endure such personal heat that they would sweat even more. Also of interest in Hall's description is how what was coming to be recognized as the same affliction could yet be experienced by different patients in varying ways:

> "Other were so drye that they dranke the cold water to quenche their importune heate & insaciable thirst. Other that could or at the least woulde abyde the heate & styntche (for in dede the sweate had a great and a strong sauoure) caused clothes to be layed vpon theim asmuch as they coulde beare, to dryue oute the sweate if it might be. All in maner assone as the sweate toke them, or within a short space after, yelded vp their ghost. So that of all them that sickened ther was not one emongest an hundreth that escaped." [7]

Terrifying this must have been, and not only because of the apparently tremendously high fatality rate, though this too is another issue with plenty of contention. And the potential treatments also varied, from what might be loosely called common sensical approaches to plans which at best might have offered some amount of placebo effect. Whatever the genuine fatality rate may have been, some died and many survived, and as will be shown, a notable part of what so affected Tudor perceptions of this disease was its very mystery.

Hall mentions slightly different attributes in his depictions of the later outbreaks. For "the X. Yere," moving ahead to the reign of Henry VIII, which describes the next epidemic and its frightful speed, "this malady was so cruell that it killed some within three houres, some within twoo houres, some mery at dinner and dedde at supper." [8] Such an extremely aggressively rapid dissemination of any disease is tricky to account for even now, since most simply have longer incubation and developmental times. And it was beginning to affect even politics. Hall speaks of the Michaelmas term of Parliament being adjourned, as the disease "continued from Iuly to

the middes of December." And a decade later, upon the Sweat's return, England's ambassador to Spain died of it, along with various courtiers and influential persons.

While assessing and studying and even diagnosing this disease is a major task of this whole project, we at least are fortunate in that so many primary accounts of the disease do exist, which may hopefully help remove some of the guesswork. The final of these primary sources comes in the form of a major collection of letters, only several of which will be considered here. These are the works of merchant John Johnson, a stapler and draper (a trader in wool and merchant of cloth, in other words), and his family and acquaintances. Their testaments offer us a glimpse of how the final Sweat epidemic, during the summer of 1551, was interpreted by a mix of persons, none of whom in this case were physicians or natural philosophers, or especially high on the Tudor social scale, though this family did comprise a successful gentry clan, part of the "middle class" which had slowly developed since the Middle Ages. Johnson himself owned his own company, and traded "in cloth and herring, grain and wine... sheep and cattle," [9] based on the descriptions of historian Barbara Winchester, who takes on the huge task of transcribing many hundreds of these "Johnson Letters" from the years 1542 through 1552, just after this final epidemic, that "ill-omened year of the sweat and financial chaos (which) took a toll of life and fortune that ended in the ruin of the family firm." In this case it was not just disease but some of its lasting effects that led to catastrophe, although the family suffered as well: Johnson's own brother Otwell would succumb to the Sweat that fateful year.

As a source, the Johnson family documents may well comprise "the most magnificent collection of sixteenth century letters," [10] offering a "revelation of Tudor family life" which had become for many "more comfortable and civilized in the middle years of (that) century than it had been even fifty years before," [11] during the reign of the first Tudor monarch. A previous

historian comments about this often overlooked feature, too, such that "in some respects the labourer was very well off," working a standard day of eight hours with frequent, usually religious holidays, and that "house rent and fuel were cheap, and the average cost of necessaries was about one-twelfth of their cost to-day." [12] This historian, Temperley, also speaks of numerous pastimes and entertainments, and opportunities for work advancement and recognition, though she concedes that relocation remained probably as difficult as it had during the Middle Ages generally. Also, direct comparisons of cost of living adjustments between then and now (or between now and previous times in history anywhere) are exceedingly difficult, made more so since the availability and desirability of goods and services varies widely by time and place. In the meantime, the Johnson letters invite careful readers into the sometimes intimate details of the family's collective life, though countless other letters provide simple, even mundane, summaries of transactions and any news which might prove of consequence for a successful business. Even by midsummer of 1551, as an employee writes to his boss, Johnson, who is himself involved in financial affairs in Continental Europe, there is nothing of concern in his letter. The first known outbreak for the year dates to July 8, and Johnson's underling Henry Garbrand reveals nothing noteworthy, merely that "Ower proffyt is vere bare thys yere, God sende us better fortune the next yere. The best remede is paciens. No more to yow at this tyme, but Cryst presarve yow." [13]

Younger Johnson brother Otwell, unaware of his impending fate, completes a letter to John the same day, detailing recent transactions for several paragraphs before offering grimmer news. Indeed, the tone suggests that the item of greatest concern will be the as yet unknown fallout from rumors of a currency debasement. Henry VIII had instituted such problematic practices late in his own reign, most notoriously to fund his final and dismal quest for glory at

the expense of the French, but now his teenaged son occupied the throne and with his advisors was attempting more rational economic practices. Many of the early statutes of his reign deal with trying to offset the negative effects of land enclosures, for example, [14] though Royal Proclamation 373 from 1551 tries to do more, entitled sweepingly, "Ordering Reform of Coinage, Engrossing, Enclosures." The idea was to control inflation via alteration of the value of the royal coinage, and Otwell touches on that just prior to even more anxious news.

Finishing up his report, Otwell notes that "even this Cite (London) within thies x dayes with Swete and sodain death, Yea, and that of the yongest and lykelyst men and women to lyve, not leving yong children to escape if thay be not well looked to, so as moost paasse not 8 howers laying after thay be taken, the Lord be mercifull unto us." [15] The "Swete" had apparently visited "xx howses within thies 6 dayes," though the letter ends not on an alarmist note, since only three persons were believed dead of it by then. Otwell finishes with an appeal to John to keep the faith, and, as commentators on the Johnson Letters remain far fewer in number than those familiar with the works of Fracastoro and Vergil, Tankard describes how "for both Catholic and Protestant 'healing' was dependent on remission of sin." [16] In this case, she tells us, "it is notable that not once in the Johnson letters does the correspondent refer to the possibility of securing intercessory provision in an attempt to divert sickness and death." And if that was not enough evidence of divine judgment, or in the belief in such, "the disease was believed only to strike down the English," [17] a curious notion also observed by Caius at the time.

This dual notion of religious interpretation and how contemporaries comprehended diseases and other disturbing events will also receive due consideration, and represents a huge and multi-faceted subject in its own right. The Tudors tended to still adhere to ancient humoral theory, with its logically simple yet evidentiary highly problematic explanations of four-fold

understandings: four elements could explain four basic personality temperaments, which would further affect a person's health through the interaction of four corresponding bodily fluids or "humors." Alternatively, a "miasma" might explain matters, by which invisible agents in the air could infect otherwise healthy souls, though the "air" portion of the quadruple humoral format also accounted for this possibility. Still, that did not mean that more practical and direct responses might not be justified, regardless of one's metaphysical beliefs: "the Johnson Papers," Tankard continues, "reveal both the constancy of plague (and other ailments) and the strategies that were used to cope with it. The usual response was to try to avoid all contact with it, if possible by vacating the area of infection." [18] John Johnson himself, and his wife Sabine, who often traveled with him, went to Bruges and finally to Calais upon reading the news from London. Sabine herself contributed numerous letters to the vast posthumous collection, and both partners made business decisions and spoke and wrote at least French in addition to English. Ambrose Saunders, brother to Sabine, caught the sweating sickness on 10 July back in London, complaining of prolonged weakness, though he survived. John and Sabine, too, contracted the Sweat and survived. By 19 July the disease was abating, and here this incident raises some other questions regarding the ongoing historiography of this Tudor mystery. The Johnson Letters indicate the business couple became ill in Calais, for example, though the 1528 epidemic is the only one which has more general records as having crossed the Channel to inflict its misery upon the Continent, a feature not otherwise believed associated with that in 1551. Further, Bartholomew Warner, brother-in-law to the recently deceased Otwell Johnson, reported that thirty souls had perished in London on 19 July, compared to one hundred-twenty just the day before, so while Warner's reporting might give hope to those interested in early modern mortality statistics, "how Warner came by his information is unclear." [19] Indeed, "Caius put the

total number dying in the city between 9 and 30 July at 903," while other sources approximate that estimate, but as will be shown in a later section dealing with other mortality records, especially church parish records and legal instruments such as wills, the true mortality of the Sweat is yet another problematic issue for its study.

These contemporary writers, even with their diverse cultural, educational, and professional backgrounds, each understood that the sweating sickness was something *other* than those diseases with which they already had at least some familiarity. Fracastoro knew that the plague and typhus afflicting the place of his birth were different diseases from each other, and also different from the Sweat. Caius was unsure how to categorize sweating sickness, but never suggested it was a variant of any known illness. And Hall sometimes described the Sweat as a "plague" yet also knew that it differed quite notably from actual plague (indeed, some of these issues regarding genuine medical terminology, and how it affected understanding of disease both then and now, is another topic which will receive due consideration).

Even the apparently unique features of the Sweat got the attention of physician John Caius, who argued "that this disease is almoste pecular vnto vs Englishe men, and not common to all men." [20] As for signs and symptoms, "this disease is not a Sweat onely... but a feuer," [21] and that this fever "for the feruor of burning, drieth sweating feure like; of one naturall day, for that it lasteth but the time of xxiiij houres." [22] This particular feature would become a core part of the disease's growing mythology: since it apparently lasted, at least in its most severely feverish form, for approximately a day, then surviving that first terrifying day became of paramount concern. Once the victim accomplished that, the overall prognosis seemed much more encouraging. As for causes, the investigating doctor described "as night to dwelling place, merishe muddy groundes, puddles or donghilles, sinkes or canales, easing places or carions,

deadde ditches or rotten groundes." [23] Some basic hygienic concerns appear here, though one could just as easily say that avoiding places with any amount of putrefaction was known to be a good idea for many centuries, as Tankard has also noted. A second explanation relied on the also ancient humoral theory: "thimpure spirites in bodies corupt by repletio," as Caius describes matters. Humoral imbalances could still be used, as mentioned, to explain any illness or other basic disruption to health, and in this case, "cold and dry" persons were typically safe, since the sweating sickness was described more in terms of hot and wet from the telltale fever and sweat.

Fracastoro, as did Caius, offered his own interpretation of the necessity and practicality of prevention. "Keep the house clean and well ventilated," Fracastoro advises, "and avoid becoming heated, lest, by opening the pores of the skin, you contract the contagion." [24] While such advice is intended for one's health generally, and the logic is valid in terms of trying to avoid sickness in the first place, some of the precise methods might seem more dubious to us now: "that the air which you breathe may be purer, always keep in your mouth either juniper berries or gentian root or galanga root, or cassia bark, or macer, or the seed of a citron." Plenty of options emerge, though the potential social awkwardness of regularly appearing to others with a mouthful of prophylactic bark or other heavy plant fibers might prove offsetting, especially in the presence of a general populace which might be as likely to favor magic or prayer as a more reliable apothecary or physician, or at court, where judgments were constantly made about physical attire and grooming and overall appearance as much as on courtly etiquette.

More recent interpretations may offer more options for social tolerance, accompanied also by greater understanding of the nature of disease, in terms of both causality and underlying microbiology. Writers during the twentieth and twenty-first centuries have sometimes displayed less interest in discussing the sweating sickness, perhaps due to its ongoing frustratingly

mysterious nature. Alternatively, they may simply wish to ignore or bypass it, and plunge instead into the perhaps more immediately enticing, or at least more thoroughly documented, minutiae of Tudor history and society. What tends to unite these more recent commentators is hardly agreement regarding etiology or identification, but rather general confirmation of descriptions, filtered through their own differing perspectives. Each of these researchers will be found within this current work, so the purpose here is to help offer a more comprehensive introduction to the sweating sickness before delving into the murky question of attempting to ascertain what it ultimately was.

Virologists James Carlson and Peter Hammond, who will, as their professions suggest, offer a viral interpretation and argument for the nature of the illness, note that the Sweat "remains one of the most interesting mysteries in medical history... (it) was characterized by specific clinical and epidemiological features that distinguished it from other epidemic diseases that prevailed at the end of the Middle Ages, including bubonic plague, typhus, and malaria." [25] This attitude is typical, such that "the English sweating sickness is perhaps the best example of that interesting group of diseases which appears suddenly, wreaks havoc among a populace, and then vanishes." [26] Further, like any "newly emerging epidemics, it caused disproportionate fear," [27] a crucial issue in our understanding and further evaluation, since the primary sources, too, confirm that emotional reaction to this disease can probably be said to have truly outweighed its actual effects in terms of lethality. True plague, and the diseases of warfare (such as typhus and dysentery) would each dispatch far more souls during the sixteenth century, but at least they were better known, if still quite poorly understood according to our own more stringent demands. In the spirit of such, Carlson and Hammond offer perhaps the most comprehensive and clinical assessment of what is known of the symptomology, definitely worth repeating here since it

includes elements of the primary accounts. "The clinical features of the sweating sickness," they tell us, "included:

- 1) an extremely sudden onset and violent course;
- 2) unique clinical symptoms, more serious than plague (though these will have to be elaborated on elsewhere);
- 3) a three- to fourteen-day course with a diaphoretic crisis point followed by asthenia (the telltale sweating with later general weakness);
- 4) prodrome with fever, rheumatic pain in the back and extremities, abdominal pain, tachycardia, and flushing (these symptoms and signs overlap with other diseases);
- 5) vomiting, bleeding, and diarrhea (again, these are common in many diseases);
- 6) neurological signs with mental status changes (typically delirium);
- 7) signs of multiple-organ failure (though such would have been the most difficult to assess during a time when internal medicine can hardly be described as truly existing, and when autopsy was just beginning to gain more prominence)." [28]

These scientists correctly point out that the greatest attention during the past five centuries has been given to the diaphoresis component, which of course gave the disease its moniker, yet this feature also was typically rather short-lived. For them, the most distinguishing attribute of the disease was "a unique combination of 'brevity and intensity'," [29] as they borrow the phrasing of Dyer, who agrees with a viral interpretation but with a different conclusion than Carlson and Hammond. Dyer's own work will use a quite different type of scientific reasoning, based largely on statistical analysis, and he maintains that the first four Sweat epidemics simply do not leave us with enough data, for a mix of reasons, but primarily for a general dearth of parish records and bills of mortality, his preferred source of information. "Evidence from the other sweat epidemics," as Dyer reports, "is too fragmentary to support any theory as to their origin." [30] So here already is a very typical hopeful yet quite cautious tone, common to most if not all later researchers: what kinds of data might be useful, they ask, and how might we best analyze them to arrive at a conclusion about this historical mystery? This dissertation is the first work to comprehensively consider all of them: historical, scientific, literary, as well as medical.

That same hopeful call to action appears a full century ago, coming from Nash, though he was studying disease epidemics more generally and only includes scant references to the sweating sickness. For further research into any disease, he insists, account must be taken of evolution and the mutable traits of so many diseases. To properly understand a disease necessitates "(a) field work epidemiology – based on painstaking investigation and accurate clinical observation apart from laboratory aid (an ideal facing major problems in the case of a historical illness which is not only unknown but does not appear to be afflicting anyone in our own time). (b) Systematic, day-to-day bacteriological observations in the laboratory (though this suffers the same problem)... (and) (c) Philosophical logical deductions based on (a) and (b)." [31] While complexity and adaptation both contribute to evolution, and at a microscopic level can make diseases more difficult to diagnose and combat, the point about including Nash at this stage is to begin considering the logic at our disposal for this research in the first place. How can we hope to reach conclusions about this early modern illness when all we have to go on is, apparently, a bunch of old documentation?

Sometimes the historians get things wrong, too, which can hardly help. More popular histories can prove misleading also, even when otherwise well written. Author Jane Bingham confronts this by trying to simplify matters, claiming that the Sweat remained between the first and second epidemics, posing a threat to the new dynasty's royal family. In this case, "Catherine (of Aragon) and Arthur (whose reign would be quickly replaced by younger brother Henry) both contracted the deadly sweating sickness, and while the sturdy Catherine survived, her more delicate husband died." [32] There are no known primary sources suggesting that the young Spanish queen on the English throne actually had the disease, however, and as for Arthur, he did perish while in his teens, but historians generally favor either tuberculosis, or some unknown

ailment, or the medieval and early modern catchall term of "ague" as the culprit. Ackroyd says "consumption or the sweating sickness," [33] not otherwise speculating either way. Bingham herself suggests "a form of typhoid" as equating to sweating sickness, and she blames a mix of poor hygiene, especially within the cities, along with poor knowledge of medicine, combining to exacerbate the disease. Still, she follows the general summation of signs and symptoms, with early headache and extremities pain, with subsequent fever, sweats, and tachycardia, though she also adds a notable thirst to the list, along with "an irresistible urge to sleep," which contemporary observers also commented on, sometimes insisting that if the patient could be forcibly kept awake during the critical first day, likelihood of recovery improved hugely.

Finally, among the recent historians who gets the basics accurate and does a plausible job of keeping matters in perspective is biographer Alison Weir. Alas, her initial reference often does commit the ongoing problem of using "plague" interchangeably with other diseases, including the one under study: "The plague that had hit London in July of 1517 was of a type known to be extremely deadly – the sweating sickness, a scourge prevalent only in Tudor times." [34] Weir makes no mention of the first epidemic of 1485, though in fairness she writes mainly about Henry VIII, and his various wives and children. While young "Bluff King Hal" (a much later nickname) at that time was "horrified and disgusted" by "illness in any form," the Sweat even more effectively "reduced him to a state of abject fear." [35] Such fear motivated him to flee on more than one occasion, typically taking just a few vital courtiers and government officials with him. The next outbreak in 1528 would almost take the lives of some of those close to him, including Lady Anne Boleyn and Cardinal Thomas Wolsey, yet both survived it. One curious detail of that incident is that, first, Henry may not have known about Wolsey coming down ill (though he certainly knew of the fate of his beloved Anne), and second, a record taken to

indicate his growing frustration with his Chancellor and the ongoing "Great Matter" to secure a divorce from his current yet no longer fertile first wife, actually seems to refer instead to this same trepidation of the King's regarding the Sweat. Wolsey had prepared to meet his monarch at his palace of the More in Hertfordshire, though the latest epidemic complicated matters. Stephen Gardiner, still serving as secretary and not yet Bishop of Winchester, relayed that Wolsey described "howe glad ye wolde be to receyve the kinges highnes at the More," while the royal response, as recorded by Gardiner, reflected

> "that synnes his determination to go thither he was aduertised howe at Rikemansworth (the nearest community to the More) and other townes aboute the more certain this yere and of late had the swet... the oonly name and voyce wherof is so terrible and fearrul in his hignes eeres that he dare in noowise approche vnto the place where it is noysed to have been and therfor his highnes wil not goo thither but in the stede of that goo to Titennehanger (a large manor house, also in Hertfordshire) and take suche chere of yor grace there as he shuld haue had at the More mynding according to his former gists to departe from Barnet vpon Saterday come sevenight and after dyner to goo that night to Titenhanger," [36] and there to stay at least through the weekend. (Even at the time, many understood that the danger seemed to be past once the crucial first day had been surpassed, and a little more time might give sufferers a chance to recover some, or at least give the impression they were no longer contagious).

This planned meeting seems to have never happened, however, leaving us to wonder less about the disease this time and more about what might have been resolved, though not long after, Wolsey truly did fall from favor at last, as visiting Cardinal Campeggio kept stalling a papal decision about the divorce and returned to Rome. As Bernard further reports, "there is then no definite evidence that any meeting took place at Tittenhanger." [37] Henry appears to have gone, but not Wolsey, though whether the Chancellor had a viable excuse in being ill, or in recovering from the Sweat, goes unrecorded. So the King was again trying to not become sick, partly for simple survival and partly in recognition of his royal duty to provide his realm with a viable, preferably male, heir. Such lay behind his desperation to rid himself of a now barren wife and

obtain one who promised a son, though of course it would be his third wife who finally came through in that regard, even if the first two wives had each left him a surviving daughter. Finally blessed with a surviving son, Edward, free of the doubts of illegitimacy as his elder half-brother Henry Fitzroy was not (and who had already died young by then, though of consumption and not Sweat), Henry's caution might instead be interpreted as obsession or perhaps paranoia, since the baby prince was kept in an environment for most of his childhood coming as close to sterile as Tudor society could create. Not just sweating sickness, but other ailments like true plague, had to kept at all costs from the one whom Henry thought might well be his only heir worthy of the crown. He could not have known that all three of his surviving children would wear that crown, nor that after 1551 none would have to contend with this disease. And now we already have more information than most Tudors had about the sweating sickness, and it is time to fully and comprehensively consider and evaluate just what it may have truly been.

B. Tudor Science and Medicine

Such are the witnessed or at least contemporary accounts of the disease. It is curious how, as with veterinary sweating sickness, no more "official" name has ever truly appeared for this affliction over the centuries. The writers of the time referred to the Sweat or English sweat or sudor anglica, or sometimes pluralized any of these, and perhaps this offers some indication of our own ongoing discomfort with the disease even now. What should we label the things which appear to exist but which we have trouble confirming, or at least describing more fully? Yet since it has already been shown how understanding a disease entails understanding something of those who had to deal with it, we can consider how the Tudor understanding was equipped to deal with it. "Tudor medicine has an undeserved reputation for being entirely barbarous and

ineffective," opines Tracy Borman in an account of the more intimate details of the leading figures of Tudor England, and yet caregivers were "advised to pay attention to (their patient's) mental state, daily habits and diet, and to observe the body as a whole." [38]

The outbreak of a disease and its impact upon victims and witnesses alike is an event that never occurs in isolation, even when we may have trouble knowing what the other effects of the outbreak were. While some consideration has thus far been offered of how contemporaries tried to understand the sweating sickness, including those with medical educations, it remains essential to more fully explore the Tudors themselves from the perspective of appreciating their own attitudes to disease in general and this disease in particular. It has already been mentioned that we can, of course, not literally or ultimately know what another person or other people thinks or feels or believes, an ongoing psychological question that historians must typically confront whenever we ponder the meaning behind the words of whatever documentary evidence they may leave behind. We so often must either take recorders at their word, or compare them to other writers from the most direct proximity in both time and place that we can to evaluate potential trends; this latter issue surely applies to what sixteenth writers had to say about this new disease, too. Similarly, and more directly, we must also consider the gradual shift from ancient and medieval explanations and justifications of disease to a more recognizably modern perspective. This can be seen if we divide contemporary understanding into its relevant fields of humoral theory, improved anatomical and physiological knowledge, astrology, and alchemy.

Before considering each of these fields, though, key questions remain: as Arrizabalaga and Dyer, among others, remind us, understanding a disease, any disease, is partly (and in some cases almost exclusively) an act of interpretation within a given historical context. That means studying the given culture and attempting to understand what its populace knew, or thought they

knew, about disease and medicine and science in general, and about the specific disease in particular. For this early modern period, Siraisi notes the side effects of the changes, so that while the time would enjoy vernacular texts, the medical "marketplace" really did emerge, with rivalries among physicians, surgeons, apothecaries, midwives, even veterinarians, with exclusions of various former practitioners, especially if they were women or not members of guilds. Madeleine Cosman describes how women themselves worked in hospitals, health spas and leprosaria, and in gardens for medicinal herbs and plants. Some "medical women treated chronic diseases," [39] and indeed had been doing so since the Middle Ages, and it has been noted how "women physicians (also) compounded medicines and salves," [40] and sometimes even had their own laboratory settings. Such a market mentality would encourage that growing sense of individuality regarding each person's own healthcare to yield a "largely unregulated medical marketplace (which)... attracted a diverse array of practitioners." [41]

What ultimately caused disease, then, was the key part of this issue, such that the notion of "balance," that relic of humoral theory, remained so intractable even for humanistic minds. Restored balance, via appropriate attention to one's humors, enabled cures. In actual practice, then, each medical patient would have to alter her or his behavior to restore that balance, to reacquire health, which represents quite a different approach from attempting to combat an illness more directly, typically along medicinal or surgical lines. With this in mind, it might be easier to appreciate the notion of health as pertaining to virtue. If one behaved according to higher moral precepts as discussed previously, then illness should remain preventable, and the link to the possible moral component underlying disease, at least as formerly understood, is now clear. We also have to appreciate the cultural context, and the historical setting, of disease, partly since we have to address two issues: "on the one hand the double 'translation' implied in

labeling pre-modern infectious diseases which were written before the germ theory; and on the other, how far conjectures by historians of disease with the aim of retrospective diagnosis could sometimes go." [42] If this is somehow not enough, there exists the additional consideration of "how intriguingly close their proposed disease labels are to the nosological concerns of medicine at their precise historical times." [43] And yet, as hoped for at the onset here, the research into the mystery of the Sweat remains as strong as ever, perhaps even stronger currently due to the number and diversity of those writing about it. Still, context definitely matters. While Nash oversimplifies the details when he claims that for medieval Europe generally, "the feudal system was responsible for such misery" [44] as various plagues, literal as well as when the term indicates other diseases, his input regarding what "disease" really refers to is of far more help. We have an ontological responsibility here. For our present purposes, a "disease" is a condition of disrupted health in the sufferer who has become the recipient of some foreign body or agent. While the cycle of disease transmission varies widely in complexity and indeed differs among those caused by pathogens (viral and bacterial alike) or toxins and other chemical agents, these involve details more appropriate fields other than history. Thus, the explanation of disease necessarily includes social factors, such as politics, economics, and religion. Such factors influence how individuals and groups respond to disease, regardless of who may survive.

Once we commit to understanding "disease" in its proper social contexts, in addition to scientific, medical, and biological, then we can more fully appreciate the details and implications of the English Sweat. For example, diseases are often understood as living things, at least when the offending pathogens are bacteria, while the notion of viruses as truly living has proven problematic for taxonomists within microbiology. Regardless of this potential additional semantic concern, the spread of a disease might be more fully comprehended as analogous to a

living organism. It needs "fuel" (hosts) to continue, can reproduce itself, and can certainly "move" in the sense of afflicting ever more victims over wider areas. And yet "highly lethal diseases are not successful pathogens." [45] This seems quite ironic, considering the apparent "purpose" of a disease as it attempts to continue replication, yet the "rapid slaughter of the host cannot ensure longevity of the (pathogenic) organism." [46] Whatever causes a disease, including sweating sickness, might thus become "too" successful if it wipes out too many potential additional carriers and thereby spreaders of the disease that it creates in its victims. Programmed dormancy periods might help offset this, in the tradition of anthrax spores so favored by McSweegan, as another example. And researcher Eric Bridson continues from his assessment of the limits of a disease. Pathogens might adapt to restrict the speed and ability of "species-hopping," like the "emerging property" description of microbe strength discussed by Casadevall et al., which will be described below. And "the new host has defences but humoral and cellular immunity takes time to be activated fully. Viral load, virulence and host factors determine whether recovery or death is the result of infection." [47] "Humoral immunity" might seem an inside joke in the context of this dissertation, but just refers to immunities found in or traveling via bodily fluids, which might include blood, phlegm, and bile (though of course the fanciful subdivisions into yellow and black bile have long since been revealed as false entities). What is more important here is how "none of these three factors (viral load, virulence, host susceptibility) fully explain the sudden appearance of the 'sweate' or its equally baffling disappearance." [48] At least we can say that it behaved as other diseases, still fitting "the established spectrum" of disease behavior, as noted by Dyer, though the evolution of what diseases are and do remains ongoing, like Nash reminded us, already a century ago.

Renowned historian F. Smith Fussner confirms a similar continuity, however irrational it may have been, though shifting from pathogens to human society. "Most characteristic, perhaps (for the Tudors, and definitely including their attitudes and beliefs about science and medicine), was the persistence of medieval ideas and attitudes." [49] As for history, it was still largely understood as the unfolding of God's divine plan, such that its "characteristic purpose... was to uphold and justify religion, law or country." [50] There was nothing, really, by way of historical tradition, at least not in the sense of specializing in particular periods or fields: "no history of learning and the arts. This meant no history of science, of technology, of philosophy." [51] It is against this old trend that we have to work, in order to continue to justify this new sojourn into Tudor history via a retrospective medical diagnosis.

For humoral theory, the original four Greek elements at the root of all existence and metaphysical hypothesizing eventually yielded the four corresponding personal dominant characteristics of melancholic, choleric, sanguine, and phlegmatic, and finally with the equally corresponding bodily fluids, or "humours," of black bile, yellow bile, blood, and phlegm. These elements, so named since they allegedly could not be divided into anything simpler, included earth, fire, air, and water, and were proposed during the time of the presocratic natural philosophers, differing somewhat from the just as ancient Asian five elements of earth, fire, water, wood, and metal, though having just as much influence upon astrology. Each person tended to have one major character trait (and thus one humor) dominate, but too much or too little of any of them would prove unhealthy. And the most effective manner by which to maintain one's own humoral balance was via careful attention to the six "non-naturals."

Influences [52] upon the humors had a variety of sources, though the core non-naturals included adequate air, rest, exercise, appropriate diet and good digestion, hydration, and emotion.

For air, as Louise Curth begins, "Hippocratic writers believed that plagues or epidemics were the result of breathing in noxious air," [53] and we of course know now that there can be some genuine truth in this ancient assertion, which also found support particularly in the Middle Ages with the idea of miasma, or "bad air." Galen was known to advocate isolation of patients in environments which would hopefully remove them from further (mainly airborne) risks, either to themselves or to others who were otherwise healthy. Nash notes that during the reign of Elizabeth I, an unusual order in 1563 tried to "shut up the foul air of infected houses," [54] which was revoked in 1564 as too unworkable. Caius had previously mentioned the need for "good" air as part of generally preventive medicine. And Fracastoro reported that "we must suppose that (the Sweat) comes into being chiefly from some taint in the air," [55] and we already have witnessed his overt attempt to purify what one inhales. The Johnson family seems to have understood the risks of "infected" air or "miasma" as well. John received a letter from brother Ambrose with quite a specific warning: "if it please God to visit you or any of your friends with this sweat observe these three things... first let no breath of air come to your bed, drink very little and at no hand sleep not for if they be suffered to sleep by the space of 12 hours but one paternoster while death follows incontinent." [56]

With exercise, "then, as now, social forces exerted a great deal of influence upon contemporary ideas of appropriate physical activities." [57] Archery and some melee weapons work would be practiced by mainly the lower class, while tennis and jousting and swordplay were still preferred by the upper class, though they too would hone their bow skill for hunting, as with those men who may have unwittingly exposed themselves to disease-carrying ticks, which

become important players in the thesis about LIV. Even simple walking might be prized, especially during winter, or by nobles who might find themselves wearing more confining and elaborate, if fashionable, clothing. Regular attendance at church services remained mandatory until after the Tudors had vanished, regardless of the Catholic – Protestant clashes, and if the village church was miles away, then such was the distance to be covered by typically strong and often quite well nourished bodies, despite the occasional poor harvest as described by Carlson and Hammond, and Slack, or the price and availability of quality foods, which varied. Boorde emphasized exercise as part of his "Dyetary," along with all the other non-naturals, and was known to exchange knowledge with Butts in order to offer the proper "physic" to patients (the term itself still referring mainly to the Greek "physis," or "nature.")

"The healthiest diets were supposed to change with the seasons," [58] Curth writes, both in quantity and kind. And while social constraints could limit availability of dining options, some foods were considered hazardous in themselves, which, along with food prices during the period, will be explored more by Andrew Appleby. But for all classes, "wine, consumed in moderation, was thought to be an important ally in the fight against disease." [59] Hippocrates and Galen recognized wine for its external cleansing antiseptic qualities, too, and seem to have understood its application as a topical antibiotic, even if germ theory and understanding of the behavior and even the very existence of microbes would have to wait many more centuries. Yet the early modern writers understood that wine carried its own risks, potentially beyond the mere concern of intoxication. Fracastoro admonishes readers to "not give the patient wine... because it raises the fever to the highest pitch," [60] so its medical usage deserved a reevaluation. In the meantime, wine seemed to positively influence digestion, and not just because water could be risky.

An aid to diet was the newer more refined gardening, done for social status and decoration as well as to supply food, herbs, spices, and medicinals, and which remained largely the domain of women. The ancient tradition of a local wise woman remained alongside this trend, typically understood as a healer, and historian Sandra Sabatini confirms how "women were also taught the use of medicinal herbs and became skilled in simple first aid, though they were not allowed to practise outside the home as doctors or surgeons." [61] She makes no mention of sweating sickness, though a researcher like Dyer might find the reference fascinating, a result from his own hypothesizing about rotten pine nuts playing a part in the disease's dissemination. The problem for Sabatini has more to do with taboos: plants used in medicine might have logical connections to magic, witchcraft, and astrology, even if such practices might have limited or conditional approval in certain circumstances, and indeed, astrology often benefitted from official sanction and even sponsorship. Still, for the time, "most men agreed that all women were inferior beings," [62] while Alison Sim notes how the role of women as healers became gradually more downplayed as early modern physicians emerged from universities. And Sim reminds us further that "medical treatment was centred around restoring the balance of the humours, bearing in mind a person's basic constitution." [63]

So, each individual largely conformed to one of the four basic humoral types, so some customization of treatment would follow, but the old idea of balance remained in force. The sweating sickness was unusual for making a patient both hot and wet, overly sanguine in other words, and corresponding to air. This would correspond to the miasma thesis, as well as seeming to suggest bloodletting as the most obvious treatment. Phlebotomy remained the dominant method of releasing tainted blood to achieve balance, usually in a direct effort to regularize the patient's humors, though the reasoning might have to be stretched to make patients

comprehend how the likes of phlegm, black bile, and yellow bile could be drained or at least balanced in addition to the obvious blood. Fracastoro describes his own concern with phlebotomy as well, since with high fevers, which might be a sign of sweating sickness, "I do not approve of phlebotomy, because (such fevers) very seldom have their origin in the body; they come from without," [64] suggesting a more direct external influence in the acquisition of disease than unbalanced humors. "Venesection can do very little good," Fracastoro warns us, "because the germs (his "seeds of contagion," not actual microbes as we know them) cannot be removed; and it may do great harm," [65] partly since loss of blood of course will start to cause weakness. There also exist no known accounts of bloodletting being used for sweating sickness, interestingly. Perhaps patients simply did not survive long enough, or maybe surgeons fretted about getting too close to them. Phlebotomy was not going anywhere for some time, though. Writing during the early Stuart years, physician Henry Stubbe could note that "but if thofe *Humours* be evacuated, which are the caufe of the difeafe, and the Patient *bear it well*, fuch evacuations are not hurtful; whether they be *greater* or *leß*." [66] The sweating sickness as he describes it, based on accounts which by then were over a century old, as having an "affect of robust nature," such that the actual diaphoresis of victims "muft have been violent," led to physicians advocating phlebotomy.

The humanist approach, thriving during the Tudor years through the likes of such disparate writers like Erasmus and More, represents a return to the much earlier *humanitas*, or classical culture. For a more specifically medieval source of influence on Tudor medicine and science, as well as gaining a segue into the newer knowledge of both anatomy and physiology (and of even understanding each of these as its own field of inquiry), the best place to start is

with the College of Physicians, begun in 1518 under royal decree and largely under the guidance of Cardinal Wolsey. This was the year after the second epidemic.

In addition to sanctioning this new College, in 1518 Henry VIII "supported programs of sanitation for the public health," [67] though historian Marjorie Boyle writing about it devotes little space to detailing just what such programs may have entailed in practice; Jørgensen and Lee are of more help here with their appraisals of water sources, as is Poore for his critique of London from the perspective of the history of sanitation. Prior to then, "the general state of sanitation in the Middle Ages (summarized mainly in the forms of polluted water in towns, problems in urban waste disposal, close proximity of rodent and human populations, and absence of a working germ or other infectious agent theory) meant that, at any time, epidemics could break out and spread." [68] Alas, we unfortunately "don't know very much about the College of Physicians during its first years, and it appears not to have been especially effective." [69] And while "the common good" was part of the rationale behind the College's establishment, "both Puritans and advocates of Paracelsian medicine, emphasizing the spiritual aspects of disease, maintained that the government orders were ineffective and uncharitable." [70] Part of the tone of these accounts might give a reader the impression that our own efforts to rework health care coverage, even if insurance is much more a part of the structure now, are actually based on old and contentious themes. So, it is difficult to avoid the conclusion that the College, while surely well-intentioned and having official sanction and some funding, could not accomplish its purpose of concretizing issues like education and status, and the professional divide remained, which meant too that health care standards remained highly subjective and personally interpreted, as with each person taking responsibility for his or her own humoral balance above. Also, blending of theory with practice, for Chamberland meant that surgeons were sometimes "at odds with the College of

Physicians, who defined their work as the learned branch of medicine in contrast with the craft origins of surgery, but physicians rarely adopted public roles or civic duties (they still tended to either practice on their own or aspire to noble or even royal patronage), and they failed to meet their goal of exercising unmitigated authority over the practice of surgery." [71]

From a wholly different cultural perspective, William Kerwin considers where developments in medicine and science in England might intersect with developments in English theatre. There was a "crisis of 'medicine' (Kerwin admits to having trouble defining just what that term might mean, rather like the previously discussed ambiguities with "plague") and not just of 'health' (the same definition issue, apparently)... evident in the failure of the College of Physicians to retain public and royal support." [72] There might have been just too many internecine conflicts among the practitioners. During the more "flexible structure" of late medieval incorporation and growing urban life, "the medical arts were not sharply distinguished from associated crafts and trades." [73] Historian Charles Webster further reports how during the same period of transition, notable parts of medicine were highly influenced by barbers (which often included actual surgeons), grocers (for production and trade of medicinal plants, part of the *materia medica*), and spicers (for access to more exotic items sometimes sought by apothecaries but typically not by grocers). And each of these occupations already had organized and powerful guilds, especially within London. Each such company had its own "powers of regulation," built upon a "fellowship" with "a perpetual commonality," so while "physicians may not have felt any strong identity of interest with the merchants and tradesmen... they were obliged to enter into competition, for the privilege of tending the sick." [74] In such an ongoing spirit of competition, the attitude of anyone anxious about obtaining the best diagnosis and treatment might earn our sympathy. In the case of Henry VIII, his "practical and academic interest in the art of medicine

is... creditable. He concocted drugs and medications for members of his household and friends," [75] though just how he went about serving as his own apothecary has left us quite scant records. One source refers to the "over 230 prescriptions for Henry VIII" [76] created by his physician John Chambre. And during the 1517 outbreak, the King "sent his own recipe to friends and relatives," [77] though sources disagree about what this sweating sickness treatment allegedly contained.

The person who offered more to a sixteenth century understanding of the human body, in England or anywhere else, was Doctor Andreas Vesalius, a Belgian physician whose work would be published in Switzerland, France, and Italy within his own lifetime, and who made the effort to respond to criticisms. In the context of anatomy, Tudor understanding was becoming far more modernized, and not only because of the attempt of Vesalius to offer a comprehensive view into the human body. The struggle between old and new, easy as it is to misrepresent (reactionary versus progressive?), may best be witnessed through the work of this doctor who knew Greek, Latin, and Hebrew and eventually became physician to Emperor Charles V. His principles "did not lead him immediately to see and observe correctly all the structures of the body, and in those instances where he believed Galen correct, although he had not yet tested all of them, he was content to refer the students to the appropriate Galenic passage." [78] Yet it was one thing to admire an intellectual ancestor and something else to give in to hero-worship and slavishly follow what that person said and did. This is a huge theme in all the scientific sections herein: when to bow to tradition and older knowledge (even if it had become suspect or could be shown, as with some of Vesalius' drawings and observations, to be simply wrong. Galen himself had largely worked with non-human primates in his own studies, after all), and when to pursue new ideas, wherever they might logically and empirically lead and regardless of whomever might take offense, as Sylvius would oppose Vesalius, though for intellectual more than artistic

reasons. And "the essential point about the *Fabrica* is its emphasis that it is the first proper account of *human* anatomy." [79] This meant that medicine could now be built upon anatomy much more fully and rationally than previously. Yet perhaps the most curious feature among physicians like Caius and Boorde in England, an anatomist like Vesalius in Italy, and an alchemist like Paracelsus in Germany, lay in their efforts to get past the old inflexible humoral theory, despite its attractive logical structure, and move more into the domain of direct experience. What patients and witnesses reported about sweating sickness could not be much accounted for, at least not accurately, by the old model, however. And while medical knowledge was becoming greatly enhanced via revised anatomical studies, there exist no known cases of sweating sickness victims being dissescted to learn more.

C. D. O'Malley studies Thomas Linacre and John Caius, both physicians to the Tudor court and so closer to the events of the Sweat epidemics. Linacre partly made a name for himself by continuing the tradition of translating older texts, "to combat this situation of medievalism, ignorance, and superstition that Linacre now devoted himself... to the translation from Greek into Latin of a number of Galen's medical writings." [80] This refers only to Galen, the same focus of so much yet then current medical understanding, and going only so far as Latin would leave such a work still unavailable to many, including even some new physicians and, as a general rule, all other practitioners as well. And yet the legacy of Linacre lies in "his introduction to English physicians of a series of classical medical texts... essential to any reputable physician," [81] which O'Malley maintains were also superior to similar publications available in England. Indeed, "a crucial factor in inducing receptivity to Linacre's ideas was public concern over the serious threats to health in the capital." [82] If Webster in this context can be accepted directly, as he notes that "rivalling plague (along with influenza, typhus, smallpox, and measles) as a cause of

mortality during this period was the 'new disease', sweating-sickness," [83] then Linacre would have had some notable influence, as he appears as one of the founders of the College of Physicians. Additionally, the Sweat appears here as a major contender for local mortality rates, though considering that actual mortality from the disease throughout its epidemics remains highly contested, we are likely better off at this point understanding the newness of professional medical organization, at the same time that concern over a variety of potentially devastating diseases was encouraging a more systematic approach to their study and to the means of enabling specialists to combat or at least hopefully prevent them.

By the later Middle Ages, the "divine chain of being," while still naturally keeping God at the top of the hierarchy, remained under influences like astrology so that celestial bodies still weighed more heavily than the actions of mere mortals themselves. Varlik, for one, even considers toxic agents as part of the chain of being, but unfortunately does not otherwise elaborate in a way which might further elucidate the prior section on toxins as sources for the sweating sickness. Sabatini includes astrology along with phlebotomy and urinalysis to summarize the potential and idealized repertoire of medieval women's skills. And a full medical preparation might unashamedly manifest as magic, since "the choice of healing ingredients was sometimes dictated by the symbolic considerations of perceived magic... (and) medical procedures often involved explicit or implicit attention to the effects of heavenly bodies." [84] Whittock, summarizing the English medieval approach, notes that "medieval medical care was a complex mixture of Christian theology, Greek and Roman medical concepts, astrology and traditional practices." [85] One may get the sense overall from these commentators of the medical Middle Ages that almost anything went, and while there might be little consensus, there was yet

a logical structure to each. In the meantime, the faith of Whittock himself seems low, as at least "in a pre-scientific environment, actual medicine was a mixture of tried-and-tested herbal remedies and others ranging from the bizarre to the dangerous." [86] Unfortunately, this attitude oversimplifies, and does a disservice to those at the time who found themselves at an intellectual crossroads of ancient, medieval, and early modern, with issues of religion and politics and economics at work with their practices and research as well.

Keeping the medieval approach in mind just a bit further, physician Leonard Rosenman writes of questioning attitudes within medicine which had begun during the time of Guy, and how urinoscopy, astrology, witchcraft, and alchemy all played major parts in healing, though Guy himself reveals a dubious attitude regarding much of these common practices. And Eustace Tillyard describes how the "moving forces of history were Providence, fortune, and human character," [87] in that order, though externals also had their influences, and astrology belongs to this latter category. Since human character could thus act upon the natural world (obvious to some, and a widely held perspective in much of the ancient world as well, though reinforced partly through the ideas emerging within humanism), another helpful feature was the increase in printed works in vernacular dialects. Printed manuals might number up to 1000 copies, and "gave a few people more to talk about," [88] with readers seeking details. The ancient and medieval ideas remained very much in force, even though no evidence exists postulating a link between what celestial bodies displayed with their alignments and influences and the onset of sweating sickness, either for particular persons or with English society at large. This must have actually added to the confusion and fear, since previously, astrology had been used for some major events in conjunction with other sources of knowledge. Studying the Black Death, John Kelly writes how "like many contemporaries, the Paris (university) masters believed that the

extraordinary upheavals of the 1330s and 1340s – the succession of earthquakes, floods, tidal waves, heavy rains and winds, and unseasonable weather – played an important role in the plague," [89] any of which might be postdated by those professing astrological insight. Other events deserve mention on this list of hazards, too: "comets, meteors, extremes of heat and cold might presage some terrible epidemic such as bubonic plague, smallpox, influenza or epidemic fever... Seasonal fluctuations might explain the waxing, waning and distribution of some epidemics," [90] and of most concern were when such "fearful scourges" might come "without warning." Yet in the upcoming consideration of weather, within the section about toxins, only the last two of these have been put forth as having any correlation, much less causality, with the sweating sickness. Tudor science helps us understand the thinking of the time, but could never explain the Sweat much beyond superstition and confusion. Previously known diseases simply remained better understood, albeit themselves still not fully treatable.

A physician like John Dee possessed "the dignity, the sense of operational power, of the Renaissance Magus," [91] for biographer Frances Yates, though her own focus is the work and life of Giordano Bruno, an Italian polymath who spent time in England but eventually succumbed to the Inquisition, accused of a mix of religious beliefs likely to irritate Lutherans as well as Catholics. Dee and Bruno probably never met, though both recognized the reactionary undertones of their times. Dee required "the mediaeval traditions" upon which his work was heavily based, and "the destruction of the monastic libraries caused (him) great anguish and he tried to rescue as much of their contents as possible." [92] This had the unfortunate effect of others often perceiving him as either a "conjuror" or, probably worse by late sixteenth-century England, a "papist," the same sort of bigotry Caius had encountered earlier. As for Bruno, he likewise recognized that England was "divided, finding himself at home" in some ways yet "antagonistic

to some aspects of the Elizabethan world." [93] Summarized succinctly by Yates, starting with the execution of Thomas More (for purely political and not religious or scientific reasons), "a chapter in the history of thought which had only just begun was prematurely closed." [94] Far more than just penning *Utopia*, More appreciated scientific endeavor, unafraid of physicians trying new practices nor viewing the heavens through his own telescope with his King, perhaps pondering how accurate Copernicus had truly been.

Terms like "bloody flux" (usually but not necessarily dysentery), "ague," "fever," "surfeit" (typically having moral and religious connotations, in that it speaks to gluttony or greed) do little to assist with our understanding. Regardless of actual cause, the overall tone for the early modern European period is that the ill tended to remain so at home, since "from home they could decide on what type of medical practitioner to pay," and this list, of course already elaborated on before, gets expanded here to include "wise-women, astrologers, herbalists, uroscopists, empirics, apothecaries, barber-surgeons, physicians, or specialists like tooth-drawers or lithotomists." [95] The only inherent limitations were the financial resources of patients, who could benefit further from the comfort of house calls from many of these workers, and this is the most comprehensive listing of early modern health care thus far. Those without the means would typical find themselves limited to treatment by immediate family, especially women, and some of these specialists, including astrologers, tended to be available only in larger cities. Whatever one thought of astrology, it clearly entailed detailed education, and astrologers might enjoy the benefits of courtly patronage as physicians might, or earn enough from consulting to keep their own practices. Those with the requisite funds could have their horoscopes cast, whether to attempt to ascertain part of the future or perhaps even to evade a bit of personal responsibility for decisions.

Celestial bodies, their positions and motions of course in need of proper interpretation, "were thought to exercise a profound influence on the weather, the harvest, the humours, and the tranquility of the soul." [96] Still, theological questions remained, certainly made no easier with the religious upheavals in England at the time, and Allan Chapman cites the Sorcery Act of 1541 as a notable source of concern for practitioners, the same year Caius became a physician as well as when the botany text by Fuchs first was published. This marked the first time that the College of Physicians in London "began suing non-physicians for practice without its license in the Court of Exchequer." [97] While this Act would be repealed several years later, sorcery, despite its own problematic definitions (how much "magic" was truly a concern for authorities, and what qualified as such?), became a capital offense. In the meantime, Chapman has found plenty of contemporary skepticism, especially in notions of human will and its potential limitations, and the ambiguous (hazardous?) concerns of casting horoscopes. Indeed, horoscopes themselves could no longer be really discussed with regard to members of the royal family, since any prognostication suggesting their deaths regardless of cause had also become illegal. The moon, Chapman suggests, remained the most influential body for assessing and even predicting diseases, though the bodies associated with the zodiac signs remained in vogue, as they do into our own time. And yet while clearly highly erroneous, astrology "was no more fallible than Galenic medicine," [98] and still widely influential.

Perhaps the most mysterious, or at least the most hazardous, of the early modern natural philosophies was that which might be illegal in some areas or officially sanctioned in others. Such was the case with the risky partial precursor of chemistry, alchemy, perhaps as ancient as humoral notions and astrology, though not as refined and directly experiential as anatomy, and

often regarded with more suspicion and awe than any of these. In addition to its fabled account of the Elixir which could grant a disease-free immortality to the user, the practitioners of alchemy often likewise confessed to interest nearing obsession associated with its other chief artifact, the Philosopher's Stone, able to transmute "lesser" metals like lead into gold. Such mystical objectives ensured that alchemy "carried with it an aura of secrecy and mysticism," although practicing alchemists themselves "placed a new emphasis on observational evidence." [99] While the introduction of so much gold and silver from the New World into the early European colonial powers of Spain and Portugal had the unfortunate if ironic effect of actually weakening their economies (leaving aside the moral issues of colonialism, forced conversions, and accidentally introduced devastating diseases), an "impetus to practical alchemy was supplied by the measures taken under Elizabeth I to diversify the economy" [100] of England to make it more financially competitive. The Queen was apparently willing to underwrite actual alchemical research, and "alchemical 'secrets,' like medical 'cures,' were eagerly sought." [101]

As with Vesalius for human anatomy and Fuchs for botany, and even Dee for astrology, the star of the time regarding alchemy has to be Philippus von Hohenheim, who insisted upon being addressed as Paracelsus. Whether he truly went "beyond" Celsus (thus the nickname), an early Greek opponent of Christianity and contemporary of Galen, must remain the subject of a separate treatise, as his own logic is what matters to us. Pagel describes Paracelsus as exhibiting two key traits: "restlessness and aggressive criticism," [102] such that perhaps the man perceived himself as much of a Socratic gadfly as scientist. In his own writings he admits to displaying his own bias, at least culturally, as "I can well realize that my prescriptions may turn out to be ineffectual among the foreign nations." [103] And current physician Michael Kennedy describes his having advised physicians to "seek out old wives, gypsies, sorcerers, wandering tribes, old

robbers, and such outlaws and take lessons from them." [104] Paracelsus' own list is just as revealing: "I went not only to the doctors, but also to barbers, bathkeepers, learned physicians, women, and magicians who pursue the art of healing. I went to alchemists, to monasteries, to nobles and common folk." [105] While such interactions must have enabled quite a mixed anthropological sampling from his time, which included traveling as far east as Constantinople, careful readers may wonder just how he might have gone about locating such souls, especially since some of those on this list had also appeared on the lists of Europe's wanted, though for reasons other than edification; further, it is probably impossible to assess how his growing antipathy toward many such individuals may have colored his interactions with them. His chief legacy, other than trying to be a foil to physicians and other university authorities generally, lay in how he "taught rather that diseases were often due to external causes and that they were localized in particular origins," so that diseases could become understood as "entities in themselves which may be distinguished by specific changes and causes... (and also as) local processes which may be defined in chemical terms." [106] There had been hints of these ideas from other traditions, especially in the work of Fracastoro, but to unite them like this, and ultimately take the logic of a chemical explanation of the world, was what proved so revolutionary. Whatever the case, Paracelsus probably would have highly approved of More's clyster treatment, which we meet later, though there apparently is no evidence of their having communicated.

A more cynical student of this period might simply conclude that funding was all, and one is logically compelled to admit that any spiritual background of the pursuits of alchemy or the tawdry trade that much of astrology had become, added to often competing medical attitudes "which encouraged the careers of heterodox practitioners, some of whom... practised physic based upon Paracelsian natural philosophy," [107] it may become difficult to see the forest of

medical advances for all the greedy trees of those willing to exploit knowledge new and old alike. Yet the question of how else to potentially allocate finite resources, having finished with this foray into Tudor science, becomes a worthy sub-topic with which to complete this section, since there are moral issues at work as well, another part of studying disease in context. Each of these early modern sciences, if they may be labeled such, could only offer clues as to the explanation of a new disease. Their failure to address sweating sickness, and indeed all disease to some extent, has been often cited as part of their intellectual downfall, though they remain essential to comprehending the Tudor mentality regarding disease.

C. Tudor Experience of a New Disease

The best way to avoid dying from a disease is of course to not acquire it in the first place, but since Tudor citizens remained divided in their assessments of just how it might be spread, so did their interpretations of what might be done about it. The basic scientific grounding discussed above left plenty of intellectual room for personalized interpretation, and this background lay behind what were regarded as the most promising (or, really, the only) efforts at treatment once prevention failed. Still, some creative approaches emerged, mostly based upon prior knowledge and belief. The sweating sickness would actually become a test of much of the scientific grounding discussed above.

Vergil insisted that no resistance or immunity seemed to confer to survivors of the *sudor anglicus*. No reference appears to wine consumption, itself among the non-naturals, as Curth reminds us above. Vergil also suggested a higher fatality rate. He advocated encouraging, even compelling, the patient to sweat as much as possible, and that the most direct way to accomplish such was to keep wrapping a patient in as many clothes and as much bedding as possible.

Similarly, patients were to be kept awake by any means. Even if these efforts did not work, disease might instead be comprehended as an omen, though perhaps on a more personal than political level. "This sweating sickness was claimed to portend the harshness of the monarch towards his people," wrote Vergil, "by which almost all were heavily oppressed, and under which they 'sweated', that is to say were forced to undergo many discomforts both at the start and finish of his reign." [108] One may wonder further if this portended disaster for Henry VII's successors. Vergil reported on the concerns and potentially undermined political stability which marked the conclusion of the Wars of the Roses and the inauguration of a new dynasty, with its tenuous royal claim.

Fracastoro recommended a possible medical treatment for the Sweat, though it is also intended for several diseases and seems more of a desperate panacea, such as "sphragis 1 drachm, unicorn's horn 1 scruple; vinegar ½ oz.; rose-water 1 oz." [109] This is an example of spagyric medicine, or herbal medicine using practices found within alchemy, yet even obtaining the proper ingredients for such a prescription is laden with its own difficulties, as one might imagine. It is intriguing, too, how Fracastoro may have been onto more than perhaps even he thought, since, as with the above noting of how ticks can infect grouse with the likes of LIV without even biting them. He identified two ways by which contagions could infect: by direct contact only ("alia enim contactu solo afficiunt"), and also from some distance ("sed et ad distans etiam transferunt contagionem"). [110] He even gave a quite modern-sounding interpretation of what might be at work, even if unseen. A pestilent fever, he reported, "esse febrem sordidae, et profundae putrefactionis, includentem seminaria acustissimae contagionis per se, proper quod et lethalis est, et ad alium contagiosa" ("(such) is a fever of foul and deep-

seated putrefaction; it contains germs of the most acute contagion per se: hence it is a deadly disease, and is contagious for another person"). [111]

Meanwhile, the potential treatments of the time also varied, from what might be loosely called common approaches to plans which at best might have offered some amount of placebo effect, something people of the time would not have identified as such, and may have simply offered prayers of gratitude instead. Whatever the genuine fatality rate may have been, some died and many survived, and as will be shown, a notable part of what so affected Tudor perceptions of this disease was its very mystery. Some contemporaries simply understood it as divine judgment, itself a possible explanation for illness for probably as long as humans have been reporting their afflictions. Occasional epidemics of actual plague almost certainly took far more lives, and even though plague itself would not be more thoroughly understood until a working germ theory of disease emerged so much later, plague remained much more of a known factor: fleeing it, and indeed escaping from any known disease outbreak, always seemed the best course of action, and while some Tudors might still decry it as God's wrath upon a sinful populace, at least there was some predictability of when and where it might occur, how it would affect victims, and how to perhaps avoid it in the first place. But Sweat was much less certain: who was most vulnerable, contemporaries wondered, and why, and where did it come from?

Caius maintained that treatment might best include "the bred, of swet corne… the drinke of swete malte and good water kyndly brued… no wine in all the tyme of sweatyng." [112] Like Fracastoro, he clearly admonishes against excessive drinking, all the more interesting during a time when fresh water was typically distrusted, and often for good reason, but disagreed with Fracastoro as to the benefit of enforced warmth of the patient. Pores had to remain clear for Caius: while contagions could enter them, they might also leave the same way. Also for Caius,

various foods, cleanliness (both of self and clothing), and good air could also help, those these are equally old notions, part of the so-called non-naturals for medicine: "al these be to be don a litle before ye end of spring, that the humours may be seatled, and at rest, before the time of the sweting, whiche cometh comonly in somer, if it cometh at al." [113] Mostly one just had to wait out the Sweat and hope for a speedy recovery, and again, it is worth noting that this account by a physician remains much more concise than those written by those who did not work in the burgeoning health care trade.

Historian Frederick Holmes, whose work pertains to the viral rather than bacterial explanations, yet refers to divine intervention in the allegory (apparently quite true) of Thomas More saving his daughter Margaret, a victim of the 1528 Sweat epidemic. This entailed the judicious if awkward usage of a clyster (enema) to treat dehydration, though Holmes phrases the incident as a case of "a lawyer saving his dying daughter with a physiologically sound treatment prescribed by God: surely a unique event in the annals of medicine, theology, and law." [114] Divinely inspired or not, Geoffrey Marks and William Beatty confirm the utility of such a treatment in their history of epidemics, describing this sadly underused option as "a more rational method of therapy." [115] Margaret's husband, William Roper, apparently wanted to keep his wife from sleeping: the Tudors still feared that Sweat patients had to be kept awake for at least that all-important first day, and yet Margaret may have actually slept through the treatment. Regardless, "the objective was clearly fluid replacement," [116] and while in our own time enemas are intended mainly to empty bowels, "nutrient clysters" such as this were recommended, so Marks and Beatty relate, at least as far back as the time of Celsus. The sad part of this historical reference though is not its apparent veracity but rather that more of the afflicted either did not know of it or proved unwilling to try it, though in fairness, some of the ingredients would have

been offsetting to many. It is not known if father and daughter regarded her recovery from the illness via the uncomfortable and scary clyster an act of God or the fortuitous result of a weighed medical decision, though it does not matter either way. It was based on a treatment of Galen himself, consisting of "oil of violets with the leaves of red roses dried in an oven and the yolks of two eggs, or else of crushed aloes, saffron and myrrh blended in a thick astringent syrup." [117]

Still, perhaps the most revealing example of an effort at both prevention and treatment is the *Oratia Contra Infirmitatem Sudoris*, the prayer given in a number of churches, especially in and near London, during the time of the 1528 epidemic. Its precise authorship is unknown, and has been attributed to Wolsey, More, and Henry VIII himself, and perhaps others, though authoritative credit is less important than what it imparts to listeners.

> Sub tuam protectionem confugimus ubi infirmi acceperunt virtutem et propter hoe tibi psallimus dei genetrix virgo: "ora per nobis beata mater Christi ut liberemur in praesentia sudore tristi:" oremus.
> Domine I(es) hu Christe qui nostrarum animarum pro salute in monte oliveti genibus flexis, sudore effudisti concede propitius, ut tuae dulcissimae matris interventu a magni sudoris specie pestifera salvemur, omnes tibi supplicantes sudoris infirmitate ut vexati per virtutem beatissime Marie Virginis celebriter liberentur per Christum dominum nostrum Amen. [118]

Some details are worth noting. The Latin of the poem means that not just would only the educated understand it, but it also remains much closer as to Catholic rather than Lutheran tradition in its tone and appeal. Its beseeching of the Virgin Mary also gives this away. Only the vaguest hints of religious reform could have been perceived in Britain by the late 1520s, since this was still a bit ahead of the monastic Dissolution and the English *Book of Common Prayer*. It is also intriguing how this mentions the Sweat by its most common name, thrice, and that the prayer is not aimed at relief from diseases in general, but only this one, though it does have some similarities to anti-plague prayers from earlier centuries. Even though sweating sickness outbreaks must have been scary at the time, they yet must have seemed so more for their mystery

rather than their actual destruction. And politics were such that during the final outbreak, in 1551, the prayer certainly would have been amended to be in more in line with what the reformist Edward VI had in mind, probably even composed in English, though if such a revision was created, no record of it appears to remain.

To gain a greater sense of that anxiety, perhaps one of the more dramatic and heartfelt understandings of Tudor attitudes to the Sweat itself, we can also turn to the words of the prime figures of that society. Such emerge in the correspondence between Henry VIII himself and his paramour, Anne Boleyn, during the 1528 epidemic. In the background still lurked the King's "Great Matter," his continually fruitless attempt to officially divorce his first wife, Catherine of Aragon, mother of the future Mary I, and pursue not just another wife but a possible source of a still needed male heir. But while "the pope has decided to delegate the judgement over the divorce to (Cardinals) Campeggio and Wolsey," [119] Henry, after removing himself from his court to flee from the disease, could write and inform the Lady Anne that

> "There came to me in the night the most afflicting news possible. I have to grieve for three causes: first, to hear of my mistress's sickness, whose health I desire as my own, and would willingly bear the half of yours to cure you; secondly, because I fear to suffer yet longer that absence which has already given me so much pain – God deliver me from such an importunate rebel!; thirdly, because the physician I trust most is at present absent when he could do me the greatest pleasure. However, in his absence, I send you the second, praying God he shall soon make you well, and I shall love him the better, and then I hope to see you soon again." [120]

The "second" best physician in question was Doctor William Butts, whose other patients included "Wolsey, the Duke of Norfolk, and Chancellor Cromwell," [121] and also Henry Fitzroy, Henry's bastard son through Elizabeth Blount. Butts "must have had the best practice of any man of his time," [122] and would go on to listen to his king describe marital issues with Anne of Cleves, and even defend Thomas Cranmer during the reign of Mary I. [123] One may also be

struck by the refusal of the King to actually attempt to visit his sweetheart, especially compared to such romantic gestures during his often disruptive, uncertain, and mercurial love life, such as galloping to Rochester years later to surprise bride-to-be number four, Anne of Cleves, despite his unfortunate response to her upon arrival. Still, we must keep in mind the King's notorious (justifiable?) attitude toward diseases in general and the Sweat in particular. Even trusted but increasingly desperate Chancellor Thomas Cromwell "tried to have the duke exiled from court on the pretext that there was a case of the sweating sickness" [124] nearby (the Duke of Norfolk, Thomas Howard, the third such, uncle to both Anne Boleyn and Catherine Howard, who was working to discredit and endanger Cromwell for his part in Henry's hopeless fourth marriage). This seems exceedingly callous, and not only because Cromwell himself probably lost his wife and children to the 1528 epidemic, and "must have been grief-stricken by the loss of his 'well beloved wyf'." [125] Biographers Borman and Hutchinson both describe Cromwell having had to endure such losses, perhaps an explanation of sorts of his later apparent callousness. But the chancellor knew his audience: this was the monarch who, when finally presented with a living son through his third marriage to Jane Seymour, would order the baby, the future Edward VI, quarantined under quite zealous disease-preventing conditions.

Thus, Henry seems years earlier to have felt that all he could do was write and pray, his curious disposition toward unconventional medical prophylaxis not including anything to ward off the Sweat. His next letter to Anne that summer of 1528 mentions an assortment of persons from his own household who had fallen ill, though that "none of our court and few elsewhere have died of it," [126] makes him sound hopeful, despite the apparent fates of Cromwell's loved ones. A bit later, in July, various other persons are described more fully by Henry in another letter to Anne, though he again ends on a more upbeat note, returning to his flirting with his

future second queen. As for Anne herself, she wisely pens to a fellow survivor of that outbreak, Cardinal Wolsey, knowing he is still expected to play a major role in the Great Matter:

> "As to your grace's trouble with the sweat, I thank God that those who I desired and prayed for have escaped, namely king and you. I much desire the coming of the legate, and, if it be God's pleasure, I pray Him to bring this matter shortly to a good end, when I trust partly to recompense your pains. In the which I must require you, in the mean time, to accept my goodwill in the stead of the power; the which must proceed partly from you, as our Lord knoweth, whom I beseech to send you long life, with continuance in honor." [127]

Attitudes and reactions to sweating sickness thus appear across a spectrum of English society, from royalty through new and growing gentry, and down into the lower classes, even if reports from the latter are second-hand within the primary sources. So far we have considered initial reactions to the disease, especially as experienced and described by individuals, as well as how the Tudor general mindset was prepared to encounter a new disease. Clearly there existed several discrepancies and disputes for the time, all affecting medicine and medical interpretations of the Sweat. The first is conservative Hippocratic and Galenist traditions mingling with more progressive and accurate anatomical studies and research. Next come physicians educated at the Oxbridge schools as well as on the Continent, competing for influence and pay from surgeons, who themselves learned their trade as a craft, and benefitting likewise from guild protection, in turn reflecting the professional classifications of the Middle Ages, and both similarly competing with the comparatively untrained, be they midwives, apothecaries, or whomever might live nearby who seemed a reliable source of traditional training and treatment options. And finally, we have religious questions, more at the beginning of the Tudor period, trying to assess who might deserve illness or health, and political questions more at the climax of the time dealing with the earliest versions of a welfare state. Each of these intellectual and social conflicts has its own important considerations, though any attempt to resolve each has either already been tried

elsewhere or remains outside the scale of a dissertation attempting to retrospectively diagnose a single disease amidst all this other historical context, but a good summation comes from Cook, who argues that "in the long run, then, English society increasingly came to value professional authority based on educated judgment less and to value one based on empirical experience and efficacy more." [128] Centuries earlier, the original natural philosopher Bacon (Roger, not Francis), summarized a major portion of the Western philosophical approach in a similar way, writing that "there are two modes of acquiring knowledge, namely, by reasoning and experience. Reasoning draws a conclusion... but does not make the conclusion certain, nor does it remove doubt... unless the mind discovers it by the path of experience." [129] This is an extension of traditional Aristotelian thinking, such that "medieval logic was mainly concerned with how things are described and to what extent those descriptions are real." [130] Reality was rational, then, more so than empirical. As a final example, Fussner can return us to Caius, that "outstanding English scholar-physician." [131] Despite a willingness to study with a progressive thinker with Vesalius, Caius' "conservatism in preferring Galen to Vesalius (which was ultimately Caius' attitude) only illustrates a sixteenth-century paradox: intelligent men often united credulity with skepticism, and contributed to progress while holding firmly reactionary beliefs." [132] This is among the most concise explanations of the problem, and we can only imagine how cognizant the Tudors themselves may have been of occupying something of a historical crossroads between medieval and modern, even while the pre-medieval seemed often secure (though it could often nonetheless be demonstrably wrong), and while the post-medieval was exciting yet perhaps giving a sense of changing too rapidly for the comfort of some.

The world, indeed the whole universe, was still understood as divinely and teleologically ordered, with chaos in any field representing "cosmic anarchy" and thus a threat to the divine

plan and the linear unfolding of history. Tillyard goes further than that, though, by reminding us that for the Tudors the medieval notion of the great chain of being remained, yielding "an ordered universe arranged in a fixed system of hierarchies but modified by man's sin and the hope of his redemption." [133] Yet just how humans were to achieve such redemption had itself become a source of conflict, and how much modification humans might be capable of via their ever more experimental and questioning understanding had become itself more questionable. Tillyard describes the later Tudors as "terrified" that the ancient order, however divine or random or human-influenced, might become upset: thus the clinging of some to the problematic notion that those who suffered from the likes of the sweating sickness might deserve such a fate, or that an epidemic of this disease might itself represent direct divine intervention. For such a group, the "moving forces of history were Providence, fortune, and human character," [134] even when these exhibited clear signs of conflict among these in enabling explanations. What makes study of the sweating sickness the most interesting, then, is not just its elusiveness and mystery, but also how it could possibly be understood by those who lived through it.

As a final example of Tudor perspectives, Bryan Tuke, secretary to Henry VIII, composed a letter to Sir Thomas Heneage, himself in service to Cardinal Thomas Wolsey, and it illustrates so many of the issues at work here that it is worth repeating.

> "So most humbly thanking His Highness, I read forth till it came to the latter end, mentioning Your Grace's good comfort and counsel given to His Highness for avoiding this infection, for the which the same, with a most cordial manner, thanked Your Grace; and shewing me, first, a great process of the manner of that infection; how folks were taken; how little danger was in it, if good order be observed; how few were dead of it; how Mistress Anne (Boleyn) and my Lord of Rochford (George Boleyn, brother of Anne) both have had it; what jeopardy they have been in, by returning in of the sweat before the time; of the endeavour of Sir (Doctor) Butts, who hath been with them, and is returned; with many other things touching those matters and, finally, of their perfect recovery. His Highness willed me to write unto Your Grace, most heartily desiring the same, above all other things, to keep Your Grace out of all air where any of that infection is, and that if

> in one place any one fall sick thereof, that Your Grace incontinently do remove to a clean place; and so, in like case, from that place to another, and with a small and clean company: saying, that that is the thing whereby His Highness hath purged his house, having the same now, thanked be God, clean. And over that, His Highness desireth Your Grace to use small suppers, and to drink little wine, namely that is big, and once in the week to use the pills of Rasis; and if it come in any wise, to sweat moderately the full time, without suffering it to run in; which, by Your Grace's physicians, with a possetale, having certain herbs clarified in it, shall facilly, if need be, be provoked and continued; with more good wholesome counsel by His Highness in most tender and loving manner given to Your Grace than my simple wit can suffice to rehearse; which his gracious commandment I said I would accomplish accordingly..." [135]

Within this one letter, we have an account which offers gratitude for the King having survived the 1528 Sweat, and that Anne Boleyn and her brother George both acquired it but survived it. Further, apparently very few actually had the disease, and indeed that there seemed little overall risk from it. The emphasis remains on the mixed influence of humoral theory and miasma theory, as the writing warns to stay in clean locales with good air, and there is even dietary advice, including the "pills of Rhazes," as well as to sweat the sickness out, literally, if the need yet arises. What we are mainly left wondering is what the pills contained: they apparently were the homemade recipe of Henry himself.

Notes for Part II.

1. William C. Wright, trans. *De Contagione et Contagiosis Morbis et eorum Curatione, by Girolamo Fracastoro* (*Hieronymus Fracastorius*) (New York: G. P. Putnam's Sons, 1930 (1546)), xii.
2. Wright, 96.
3. Wright, 240.
4. Denys Hat, ed. and trans. *Anglica Historia, by Polydore Vergil* (London: Offices of the Royal Historical Society, 1950 (1537, 1513)), 6.
5. Hat, 142.
6. Edward Hall, *The Vnion of the Two Noble and Illustre Femelies of Lancastre & Yorke*. (New York: AMS Press, 1965 (1809, 1548)), 425.
7. Hall, 425.
8. Hall, 592.
9. Barbara Winchester, "The Johnson Letters, 1542 – 1552" (doctoral dissertation, University of London, 1953), 1.
10. Winchester, 7.
11. Winchester, 32.
12. Gladys Temperley, *Henry VII* (Westport, CT: Greenwood Press, 1971 (1914)), 194.
13. Winchester, 1233.
14. Paul L. Hughes, and James F. Larkin, eds. *Tudor Royal Proclamations, Volumes 1, II, III* (New Haven, CT: Yale University Press, 1964), 309. Among the very first pieces of Edwardian legislation was called "Announcing Enclosure Inquiry," from 1548, and noted "that of late by the enclosing of lands and arable ground in divers and sundry places of this realm many have been driven to extreme poverty and compelled to leave the places where they were born and to seem them living in other countries, with great misery and poverty." That was proclamation 309, and the subsequent ones 311, 327, 333, 334, and 338, all deal with trying to deal with the issues, economic and legal alike, which grew during the enclosure period. Enclosure itself would revisit Britain in the early seventeenth century and remain a sensitive issue for centuries to come.
15. Winchester, 1235.
16. Danae Tankard, "Protestantism, the Johnson Family and the 1551 Sweat in London." *The London Journal*, 29, 2 (2004), 9.
17. Tankard, "Protestantism," 10.
18. Tankard, "Protestantism," 4.
19. Tankard, "Protestantism," 6.
20. John Caius. *The Sweating Sickness: a Boke or Counseill Against the Disease Commonly Called the Sweate or Sweatyng Sickness* (Memphis, TN: General Books, 2010 (1552)), 5.
21. Caius, 7.
22. Caius, 6.
23. Caius, 9.
24. Wright, 241.
25. James R. Carlson and Peter W. Hammond, "The English Sweating Sickness (1485 – c. 1551): a New Perspective on Disease Etiology." *Journal of the History of Medicine and Allied Sciences*, 54, 1 (1999), 23.
26. Michael W. Devereaux, "English Sweating Sickness," *Southern Medical Journal*, 61, 11 (November, 1968), 1191.

27. Carlson and Hammond, 24.
28. Carlson and Hammond, 10.
29. Carlson and Hammond, 32.
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31. Nash, 21.
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50. Fussner, 251.
51. Fussner, 237.
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72. William Kerwin. *Beyond the Body: The Boundaries of Medicine and English Renaissance Drama* (Amherst: University of Massachusetts Press, 2005), 252.
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74. Webster, “Linacre,” 202.
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81. O'Malley, "Humanists," 17.
82. Webster, "Linacre," 206.
83. Webster, "Linacre," 207.
84. Sabatini, 306.
85. Whittock, 122.
86. Whittock, 123.
87. Eustace Tillyard. *The Elizabethan World Picture* (New York: The Macmillan Company, 1944), 48.
88. Paul Slack. "Mirrors of Health and Treasures of Poor Men: the Uses of the Vernacular Medical Literature of Tudor England," ," in Charles Webster, ed. *Health, Medicine, and Mortality in the 16th Century* (Cambridge: Cambridge University Press, 1979), 260.
89. John Kelly. *The Great Mortality: an Intimate History of the Black Death, the Most Devastating Plague of All Time* (New York: Harper Collins, 2005), 170.
90. Dobson, 20.
91. Frances A. Yates. *Giordano Bruno and the Hermetic Tradition* (Chicago: University of Chicago Press, 1964), 150.
92. Yates, 187.
93. Yates, 290.
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107. Frances Dawbarn, "Patronage and Power: the College of Physicians and the Jacobean Court." *The British Journal for the History of Science*, 31, 1 (March, 1998), 2.
108. Hat, 142.
109. Wright, 243.
110. Wright, 9.
111. Wright, 86-87.

112. Caius, 12.
113. Caius, 15.
114. Holmes, 43.
115. Marks and Beatty, 104.
116. Marks and Beatty, 104.
117. Guy, 187.
118. William Snell, "A Prayer Against the Sweating Sickness: Oratio contra infirmitatem sudoris." *Geibun-kenkyu: Journal of Arts and Letters*, 73 (1997).
119. Tim Coates, ed. *Letters of Henry VIII, 1526 – 29: Extracts from the Calendar of State Papers of Henry VIII* (London: The Stationery Office, 2001), 55.
120. Coates, 56-57.
121. Young, 87.
122. Young, 86.
123. Among the more intriguing revelations offered by Purdell has to do with how "monarchs themselves deliberately influenced the evolution of the medical profession." (Purdell, 9) She further cites Henry VIII, and also Linacre and Wolsey, and their influence upon the College of Physicians, a key issue for which would be "investigating unlicensed practitioners," yet another move toward more specific education (we might say "accredited" now). A generation after the founding of the College, a somewhat cooperative "Act of 1540 defined medicine to encompass surgery, but most physicians did not perform operations of make medicines, preferring the tradition of working consistently with the same surgeon and apothecary." (10) Perhaps more tellingly with regard to politics, "under the Act of Union (of 1536, officially bringing Wales into the royal domain of England), the new Barber-Surgeons Company had full parliamentary authority," (33) which even granted them an annual allotment of four bodies of convicted and executed felons for dissection. Purdell refers to apothecaries dating to the early thirteenth century in England, surgeons to the fourteenth, though their antecedents are considerably older.
124. Tracy Borman. *Thomas Cromwell: The Untold Story of Henry VIII's most Faithful Servant* (New York: Atlantic Monthly Press, 2014), 335.
125. Borman, "Thomas Cromwell," 71.
126. Coates, 57.
127. Coates, 59.
128. Cook, 25.
129. James B. Ross, and Mary M. McLaughlin, eds. *The Portable Medieval Reader* (New York: Penguin Books, 1977 (1949)), 626.
130. James Hannam. *God's Philosophers: how the Medieval World laid the Foundations of Modern Science* (London: Icon Books, 2009), 47.
131. Fussner, 243.
132. Fussner, 243.
133. Tillyard, 3.
134. Fussner, 48.
135. M. St. Clare Byrne, ed. *The Letters of King Henry VIII*. New York: Funk & Wagnalls, 1968 (1936, 1509 – 1546), 73.

III. The Curious History of the Louping-Ill Virus.

As briefly alluded to thus far, the louping-ill virus and the disease it causes (typically known by the same name) have existed within Britain since the Middle Ages. No one has previously suggested any link between the virus known by its acronym of LIV and events of previous centuries. While the subsequent section will discuss other viral diseases, as well as bacterial ones and some other sweating sickness candidates, among the most intriguing works in relation to this dissertation is already a quarter century old, and describes a "forgotten disease," though without making any reference to Tudor history. As physician Marilyn Davidson describes how louping-ill manifested, "the most commonly reported (infection in human patients) has been an influenza-like illness which has resolved in about a week. This illness is characterised by fever up to 39.5 C (37.0 C is most normal), headache, anorexia, dizziness and muscle stiffness." [1] Thus far the symptomology reads like the primary accounts from centuries ago, though in fairness, this list is hardly conclusive, and some of these traits overlap with other diseases. In some patients, LIV infection was also apparently bi-phasic. "The febrile phase, after a short period of improvement, is followed by an encephalitic phase which is marked by fever up to 39.5 C, severe headache, vomiting, drowsiness, neck stiffness and tremor of the head and limbs." [2] One case out of eighteen reported in this study turned out to be fatal. This offers a closer match, such that, at least with the bi-phasic patients, the report much more closely matches what we know from the primary accounts. The drowsiness and fever are the most interesting of all, though what seems missing so far is perhaps the most important sign of all: the sweating itself, so perhaps that simply got overlooked. Someone suffering these symptoms would very likely present with diaphoresis also.

Having established some context as well as considering some primary accounts, we can make the initial return to one of this project's key tasks: establishing that a particular virus and a particular disease associated with that virus together offer the most plausible explanation of this disease. Thus far mentioned is its known existence within Britain for enough centuries to reach back to the Tudor period, and one notable feature of LIV in particular is that "louping-ill in Britain is genetically distinct compared with similar viruses that occur in Spain, Greece, Ireland, and Turkey." [3] In its more recent history, the ability to vaccinate livestock (with a chemical compound called acaracide) "first became available in the 1930s," [4] and has since enabled a reduction in the virus' prevalence in many areas, though the problem remains in some regions. Particularly afflicted areas currently are described in Scotland, which escaped the Sweat during the fifteenth and sixteenth centuries, so assuming the virus has altered since, it has also migrated some, though as P. Hudson et al. already have indicated, this remains separate even from the other strain of LIV known to reside in nearby Ireland.

Still, part of accounting for LIV as a candidate entails explaining how it yet remains within Britain, and that same study by Hudson notes how "antibody resistant escape mutants of louping-ill virus with reduced neurovirulence for mice have been identified." [5] This is an essential possibility to explain the present combination of LIV still infecting and killing a mix of grouse, hares, and livestock, mainly sheep, in parts of the United Kingdom but without apparently killing or potentially even infecting humans. Studying and trying to manage the virus and its resulting disease "is of particular importance in the UK in terms of economy and rural livelihoods because of its effects on sheep, but mainly because of its perceived effect on the economic sustainability of managing large areas of upland for red grouse hunting." [6] Domestic sheep (*Ovis aries*, though various breeds exist), red grouse (*Lagopus lagopus scotica*), and

brown hares (*Lepus europaeus*) have been "identified as the most ecologically important transmission hosts for LIV." [7] Sheep and grouse can both produce what is known as post-infection viremia, enabling them to infect other hosts, and "grouse alone may not be able to maintain LIV because they alone cannot maintain a viable tick population." [8] Hares do not enable the viremia, so LIV will vanish unless it can move to other species. Grouse can preen each other, like some other animal species do, which helps remove parasites like ticks, though consumption of infected ticks can lead to the disease as well. And the grouse suffer by far the highest mortality from LIV, even with such preening. Still, two of these host species, grouse and deer, are capable of spreading LIV along with ticks, and there have been documented cases of humans contracting it in turn from them. The most plausible explanation to fit in with what we know of Tudor history, including who among the Tudors were most susceptible, lies in focusing primarily on the deer, the species most able to transmit, via the ticks, the disease to humans, and also the prey species most in demand by the Tudor social elite. Hunting of hares throughout the Middle Ages and into the early modern period was an option typically available to all. Hunting of grouse could be restricted according to medieval Forest Law, while hunting of deer was the most restricted, usually only allowed to the aristocracy.

Perhaps the strangest parts of LIV's British history is reported by Davidson, who describes how certain labor groups have been exposed much more recently than Tudor times, with cases dating to 1934, 1948, 1961, 1962, and 1966. And "the most unusual manifestation of the disease reported in 1963. This described a haemorrhagic fever in a laboratory technician working with Korean haemorrhagic fever samples." [9] Two viruses were isolated from this man's blood, both identified as variants of LIV, and Davidson refers to the possibility of the disease being something known as Omsk hemorrhagic fever. In the following section, the connection to

Korea will be explored further, as it has a particular relevance to hantaviruses, which are similar to arboviruses like LIV except that they do not require arthropod vectors, and hantaviruses also rarely are found in humans, even though hantaviruses are encountered on multiple continents, as will be shown. The hemorrhagic fever described in the previous passage is a typical manifestation of hantavirus infection, however.

Since Davidson was writing in 1991 and has not contributed to the discussion about LIV since then, she reports that "since 1968 (up to 1985) eight human infections (of LIV) have been diagnosed." [10] This hardly suggests an epidemic, and of these eight, six developed encephalitis, and in four of those six the disease presented as bi-phasic, matching sweating sickness symptomology more closely, as summarized above. The occupations of these eight patients, all men, were likewise interesting. Five were shepherds, one a butcher, one a forester, and the last a medical practitioner known to spend much leisure time rambling. All eight therefore had exposure to animals wild and domestic, though whether they contracted the disease from tick bites does not appear in the records. Virologists Carlson and Hammond will also highlight the link to the Omsk-named disease, though they will abandon that explanation in favor of their own thesis regarding a slightly different hantavirus explanation.

So this comprises the basic summary of LIV. One recent paper even explains how the LIV genome has been deciphered, [11] though of what service this knowledge might prove to the scientific and medical communities goes unremarked. There already exists a useful inoculation, after all, at least for sheep, and the same article discusses how TBE viruses are more dangerous for humans, while the LIV risk for our kind is quite minimal. This laboratory work comprised "the second LIV genome sequence reported and the first prepared from a clinical sample," [12] from a donor sheep that died unexpectedly in England in 2009. The good news is that

agriculturalists in Britain have a weapon to use to continue to prevent the spread of LIV. The not so good news is that other species suffer from the disease, which in turn continues to create ecological and economic problems within the same parts of Britain, in land already set aside for protection from other development. And the fascinating news is how the background details continue to grow throughout this work to get us closer to a working retrospective disease diagnosis and its implications for this otherwise quite well explored time and place in history.

While the application of the likes of genome data entails a separate research question, especially if the use of such may be uncertain or may not even be perceived as necessary, another fairly recent piece of academic research by Gilbert, working with a different cohort of colleagues, notes how ticks do not even need to bite their potential hosts to infect them with LIV. While "adult ticks prefer mammalian hosts," [13] the larvae and nymphs of the tick *Ixodes ricinus*, the vector in question, may target a mix of species. In the case of the red grouse, the birds may unwittingly and ironically consider the ticks, in any of their three basic life stages, as potential food sources, thereby acquiring the disease by ingestion instead. The work by Hudson et al. had also been confirmed several years earlier. There apparently exists no instinctive warning for the grouse to avoid such meals, which must be relatively easy to acquire as the ticks may simultaneously seek out the grouse. In other words, the grouse are truly ignorant of the danger.

All of this needs to be compared with other disease candidates, of course, which will occupy the bulk of the next section, with some appearances by other potential disease-causing agents as well. In the meantime, Mitchell can once more assist with the logical isolation of LIV. In addition to his warnings of what can lead to a poor retrospective diagnosis, there are also textual clues which can "improve reliability in retrospective diagnosis, (including):

1. Eye-witness testimony (we have many such accounts)
2. Clear description of symptoms and signs of disease (while these vary according to

each writer, they can be streamlined, as Carlson and Hammond have done)
3. Combination of both nature of lesion and its physical location described (such symptoms do not really apply to the Sweat)
4. Minimal evidence for modifying description to match medical views of period (as with item 2, above, this has to do with consistency)
5. Presence of one or more virtually diagnostic symptoms or signs (the diaphoresis and febrile period are the places to start)
6. Record of any epidemiological observations given" [14]

Sometimes "imperfect data from historical sources (are) still better than no data at all," [15] as he notes, and he emphasizes again that such study helps to understand a certain period and culture more fully, and actually, we have most of the requirements he emphasizes on this list. Again, it matters little whether this dissertation offers something worth etching into marble for the ages. The point is instead for the reader to note that Candidate Wollert argues that sweating sickness as experienced in Tudor England resulted from a prior version of the virus now known as LIV, and he also argues that studying the history of this particular disease will get us closer to understanding Tudor society as well as the history of science generally.

Notes for Part III.

1. Marilyn M. Davidson, "Louping Ill in Man: a Forgotten Disease." *Journal of Infection*, 23, 3 (1991), 244.
2. Davidson, 244-245.
3. P. Hudson, et al., "The Epidemiology of Louping-ill, a tick-borne Infection of Red Grouse (*Lagopus lagopus scoticus*). *Parassitologia*, 39, 4 (December, 1997), 322.
4. M. K. Laurenson, et al., "The Role of Lambs in Louping-ill Virus Amplification." *Parasitology*, 120 (February, 2000), 97.
5. Hudson, 322.
6. Gilbert, 364.
7. Gilbert, 365.
8. Gilbert, 365.
9. Davidson, 245.
10. Davidson, 246.
11. Denise A. Marston, et al., "Louping Ill Virus Genome Sequence Derived from the Spinal Cord of an Infected Lamb." *Genome Announcement*, 1, 4 (2013), 454.
12. Marston et al., 454.

13. Lucy Gilbert, et al., "Ticks Need Not Bite Their Red Grouse Hosts to Infect Them with Louping Ill Virus." *Proceedings of the Royal Society*, 271 (2004), 204.
14. Mitchell, 86.
15. Mitchell, 86.

IV. Current Historiography and Literature Review of the Sweating Sickness.

A. Possible Culprits: Bacteria.

The case for a bacterial source of sweating sickness has a far longer history than that for a viral one, even predating the actual discovery of microorganisms. While some of the ambiguities of the term "plague" still must be addressed more fully, actual plague itself remains among the contenders for sweating sickness. Joining that disease will be fellow bacterial diseases rheumatic fever and anthrax, and each has been chosen by different researchers at different times based on different reasoning: some writers have preferred trying to match the better known signs and symptoms of these diseases as they are understood in our own time with those reported in the primary source materials; some have made historiographic connections with other aspects of Tudor society than the health of its people, such as economic factors, legislation, or trends and sometimes new knowledge in science and medicine; and others have opted for a less stringent process of deductive and eliminative logic as that evinced by this dissertation. The same tactics have indeed also influenced decisions made by those seeking links between sweating sickness and viruses as causal candidates, as will be revealed below, while still others have decided that trying to identify this mystery illness as a specific known disease may be a way of missing the historical point, and instead consider sources other than microorganisms. While each of these options has its own evidence and conclusions, the requisite first step is to consider these three mentioned bacterial diseases and why they have been offered as explanations.

Just before that, however, it is also worth showing that sometimes, in both academic and popular literature, other common names appear which may mislead writers and readers alike into suspecting sweating sickness when something else in truth is being described. This happens

more often with plague, though two other such examples really stand out, the first being sleeping sickness, or African trypanosomiasis, caused by the protozoan *Trypanosoma brucei* and using the tsetse fly as its vector, within all three species of its own *Glossinidae* insect family, all three of which in turn are located throughout central Africa. [1] Searching for information about the Tudor sweating sickness may inadvertently lead a casual researcher to this disease instead, perhaps due to a superficial similarity of names which might inadvertently emerge from the likes of automatic correction features of internet search engines. An even more direct link is with the second such example, something actually known as sweating sickness, though this "other sweating sickness" is strictly a veterinary disease. This illness, which interestingly does not have an accepted scientific name like sleeping sickness does, is a form of toxicosis, or poisoning, presenting with fever, eczema, and hyperemia of skin and visible mucous membranes. [2] It uses ticks within the *Hyalomma truncatum* family as its vector instead of *I. ricinus*, and hyalomma ticks are found on multiple continents, but again, it is veterinary, almost entirely limited to cattle, especially calves, though other large ungulates can contract it, mainly domesticated ones. Sources of these diseases, and research and data about them, need not concern us here; they only make an appearance at all to show that the name of the chief subject of this dissertation has manifested elsewhere, albeit with very different meanings.

That brings us to plague. The disease itself is highly infectious, and part of the problem with offering retrospective diagnoses of it is that plague appears to have historically manifested in bubonic, septicemic, and pneumonic variants, seemingly able to mutate as needed to continue finding new hosts. The bubonic form has sufferers presenting with headache, fever, chills, fatigue, malaise, plus the telltale "buboes." These are often warmer and black fluid-filled pustules, usually extremely sensitive, which appear most often in the groin, armpits, or neck, and

are the result of the severe swelling of lymph nodes. Bubonic plague is spread via the bites of infected fleas, and can easily cross species lines. The septicemic or blood-poisoning version also exhibits fever with chills, abdominal pain, shock, any combination of nausea, vomiting, and diarrhea (in our own time these are so often found together that the acronym "NVD" may be used to collectively list them), and also bleeding from various orifices (potentially any of them), plus gangrene in the extremities as the patient becomes starved of blood. Septicemic plague requires contact with infected tissues of a creature with the disease, from ingestion of its flesh or other direct exposure. Finally, the pneumonic variant offers cough with bloody sputum, breathing difficulty, nausea and vomiting (though typically without the diarrhea this time), weakness, and fever (usually higher than with the other variants). [3] The pneumonic variant has proved the least common of the three yet also the deadliest, nor does it require non-human vectors like fleas or contact with infected hosts, but can be spread via "droplet" form, simply through breathing, coughing, or sneezing. Recovery from this variant in particular was extremely unlikely in centuries past, though those fortunate enough to survive any form of plague did seem to acquire immunity, which already contradicts primary source accounts of sweating sickness. [4] Clearly these symptoms differ enough from each other as well to lead some researchers to question whether all can have manifested from *Yersinia pestis*, and even some of the researchers consulted herein have little or even nothing to say about sweating sickness itself, and instead have focused their attention onto plague and its potential varying diagnoses. "*Yersinia pestis* is able to mutate readily and has done so in the past," as Theilmann and Cate report on matters. And such "mutations can introduce a more virulent form of the plague." [5] This debate about plague remains beyond the scope of this current project, however: no attempt will be made here to argue as to whether the Black Death of the mid-fourteenth century, or other

outbreaks such as the sixth-century "Justinian Plague" in the Mediterranean region, were mainly one or more of these three types of plague or even another disease entirely. Theilmann and Cate prove themselves helpful once more here, not worrying about the hazards of retrospective diagnoses for either plague nor even sweating sickness, even if the latter "was not the killer that the great pestilence had been." And for the former, "descriptions of physical symptoms neither confirm nor disprove the presence of plague, particularly given the scant details that the chronicles provide." [6] Very interestingly, there actually exists more conformity of reported symptomology with sweating sickness than with plague, as will become clear, though in fairness, that three forms of plague exist and apparently only one form of sweating sickness did, should make this discrepancy more understandable.

Historian Vaughan, like Kelly, mentions sweating sickness just once, and then in passing, and notes the issue already encountered about taxonomy: the major epidemics of the medieval period were typically classified as *pestilentia* or even the more dramatic *magna mortalitis*. Wallis never mentions the Sweat at all. And for Slack, sweating sickness was a "type" of plague in this more general sense of pestilence. The myriad forms of true plague led to various purported prophylaxes and attempted cures, which ranged from the rational to the creative to the supernatural. Some treatments, like fumigation, might have ironic if misunderstood benefits, while the idea of making victims as warm as possible, even to the point of smothering them, might just finish them off instead of the Sweat. Yet by the Tudor period, "the most striking aspect of the English debate about plague policy was its lack of intellectual vigor, and hence of clarity." [7] Slack further describes how the reciprocal bond between sovereign and subject would reach its greatest strain during an epidemic, and that practices like quarantine probably had little genuine effect; the best way to avoid the disease, as with sweating sickness, was to avoid

contracting it in the first place. William Shakespeare may have had the bawd in *Measure for Measure* decry that, "Thus, what with the war, what with the sweat, what with the gallows, and what with poverty, I am custom-shrunk," [8] and those who were poor or otherwise unable to escape must have felt the futility of both prejudice based on class as well as genuine ignorance about diseases and their effects and transmissions. Whenever we remain ignorant of causality, we tend to succumb more readily to fear; sometimes the quest for knowledge itself represents simply the desire for control, or at least the sense that our fates are not wholly arbitrary or perhaps up to the apparently random or capricious decisions of a deity whose own reasoning we supposedly cannot begin to comprehend. Trying to evade contagious and destructive diseases appears to have always inspired anxiety, with occasional forays into more rational understandings.

Hecker, too, describes the essential features of plague transmission, sounding almost like Fracastoro in so doing. "The *pestilence*," he tells us, "or epidemic constitution, is the *parent of various kinds of disease... the pestilence* bears the same relation to contagion, that a predisposing cause does to an occasional cause." [9] And Vivian Nutton gives a wider summary of Fracastoro's views, since his "explanation involved possible planetary influences, bad air, humoral imbalances, earthquakes, and fleeing rodents, as well as contagion and seeds." [10] These comprise many of the "traditional data of the plague investigator," she tells readers, and in a letter composed during the 1551 sweating sickness outbreak Fracastoro describes the disease though without the notion of seeds, but rather with principles of contagion, while trying to ascertain what enabled the disease's spread. Clearly some contemporaries continually attempted to comprehend various diseases, including plague and sweating sickness, and understood importantly that these were discrete illnesses, even if true prophylaxis and treatments lay beyond

their own knowledge and resources. Yet while these commonalities were shared by the two diseases, certain features help ensure that sweating sickness was not plague. Simply put, the symptoms which appear in primary sources just do not match very closely. There are some overlaps, though: indeed, elevated fever, for example, is a known symptom shared by sweating sickness with all three types of plague. But fever is part of the inflammation process, and is common to so many diseases that it actually takes multiple additional symptoms to discriminate among them at all. Also, in fairness, primary sources do not typically call attention to insects like fleas except for within the context of how annoying they could be to human and non-human animals alike, along with occasional writings about how to keep them at bay within the home, almost regardless of social class. The work of Guy de Chauliac, for instance, as well as the treatises by Tudor-period physicians like Caius, Boorde, Vesalius, Paracelsus, Fracastoro, and various others, simply make no mention of insects in relation to diseases. Some might argue that since fleas were not understood, even during the sixteenth century (and certainly not before then) as having such an overwhelming correlation with the transmission of *Y. pestis*, maybe their non-appearance in medical writings of the time means that plague, having been spread by them, might accordingly be a contender for sweating sickness, as we have witnessed. Even this does not help resolve the surviving descriptions, though, and even if it could, it would only account for bubonic plague, but for neither septicemic nor pneumonic plague. And in the case of bubonic plague, the telltale buboes emerging from a diseased lymphatic system never appear in any descriptions of the Sweat, and neither do the later symptoms of bubonic plague like bacteremia, subcutaneous hemorrhage, or necrotized tissues. The pulmonary distress with or without bloody sputum found with pneumonic plague also does not develop in sweating sickness, either. As for septicemic plague, while the fever and chills and even abdominal pain might also be encountered

in sweating sickness sufferers, the bleeding from bodily orifices and the possibility of gangrene in extremities which is also found sometimes in bubonic plague, just do not appear from the Sweat. In truth, more recent scholars understand that while Tudor medicine may seem "primitive" (always a prejudicial historical adjective) by our own standards, the Tudors seem to have definitively known that these diseases were discrete, and the primary sources go out of their way to describe the "newness" of sweating sickness, and their authors picked through earlier documents, ancient and medieval, to seek precedents, concluding that they did not know how to classify this newer affliction. That almost no scholar from our own time has seriously argued in favor of plague for sweat should also reveal something to us. Sloan, for example, notes that with the 1508 sweat outbreak, London was also struck that same year by plague, measles, and diphtheria, clearly suggesting that contemporaries knew to discriminate among them. That Sloan wrote this almost fifty years ago and that most scholarship on sweating sickness has appeared since then should help remind us that comparing sweat to plague is so largely discredited that no one really takes it seriously any more. "It is unlikely too," Sloan continues, "that the sweating sickness would be confused with the ague (malaria, at least according to him, though "ague" is perhaps even more ambiguous than "plague") or with gaol fever (typhus), both of which were common at the time," [11] so while the Sweat was taken as distinct, "many feature (of it) are still unexplained: its strange geographical localization, its recurrence after long periods... its final disappearance, and its predilection for young men and for the upper classes of society." [22] Historian R. S. Roberts, though, sounds more like Arrizabalaga in his response to Sloan, whose interpretation "was basically the traditional one as developed by Hecker and Creighton in the 19th century, and as such makes more of a mystery of the disease than more recent workers find necessary." [13] Hecker, for examples, and sounding reminiscent of the condemnatory attitude of

Boorde, pointed out that "if we consider that the disease mostly attacked strong and robust men... while women, old men, and children, almost entirely escaped, it is obvious that a gross indulgence of the appetite must have had a considerable share in the production of this unparalleled plague." [14] Finally, though, regarding this comparison of sweating sickness and plague, by the time of the sixteenth century eruptions of sweating sickness, the notion that victims were somehow receiving their just desserts had largely shifted. This switch in attitude may have partly resulted from realization that victims of the Sweat were often the wealthy, though in the case of plague, it, too, would certainly strike down rich and poor alike when it came to those who could not escape the latest pandemic. More appropriately, the early modern period generally helped enable an understanding of medicine and disease alike which shifted away from blaming victims and toward trying to understand what these diseases ultimately were and how they spread. One could well cite a general scientific approach to medicine that was more lacking, or at least less systematic and less empirical and experimental, in earlier centuries; this would help satisfy those who emphasize the traditional model of a Western scientific revolution during the sixteenth and seventeenth centuries.

Next on this list of bacterial diseases comes rheumatic fever, a result of streptococcal pharyngitis permitted to develop further. Consequent inflammation from this disease will typically cause cardiac damage. Bauman notes a causal curiosity with the disease, in that "though the exact cause of the damage is unknown, it appears that rheumatic fever is not caused directly by *Streptococcus*, but instead is an autoimmune response," [15] yielding antibodies which target streptococcal antigens and then "cross-react" with cardiac antigens. Those with the disease exhibit symptoms of inflammation, with fever and often pain and tenderness in joints. Strep throat or scarlet fever may precede the disease, and the fever remains if either of these

occurs. Other symptoms may include the appearance of small, painless subdermal nodules (though not discolored or painful or even affiliated with lymph nodes, as are the buboes in bubonic plague), chest pain, fatigue, uncontrollable motions (usually in the hands, feet, or face, and often diagnosed separately as either Sydenham chorea or the more medieval "St. Vitus' dance"), carditis (heart inflammation), erythema (skin redness from hyperemia, or increased perfusion of blood to tissues), abdominal pain, and nose bleeding. This is a highly varied list, even overlapping with other diseases, so diagnosis often entails various steps, such as blood testing (complete blood count, blood sedimentation rate, strep test), and electrocardiogram. [16] Because of this myriad of symptomatic options, a retrospective diagnosis of rheumatic fever may be the most difficult of all the diseases on this list; still, various writers have rendered promising explanations of why this may be the best case to connect to sweating sickness.

Rheumatic pain, by definition in the joints or other connective tissues, might correspond to dozens of diseases, including autoimmune disorders. Such pain does appear in the sweating sickness research of Carlson and Hammond, though Hecker emerges in the literature as the single strongest advocate of this. While Hecker has been heavily critiqued since, and his own work admittedly is quite dated now, he did nonetheless consider the primary materials from a solid understanding of the medicine of his own time, the mid-nineteenth century. He in turn seems almost critical of Tudor-period physicians, who "could do little or nothing for the people in this extremity. They are nowhere alluded to throughout this epidemic," [17] which is simply untrue, though frustration at those same physicians also sometimes appears in primary accounts. And he is definitely harsh about their contemporaries, prepared almost to assign at least a correlation if not an outright cause to "a gross indulgence of the appetite" of young and strong men, whose nutritional greed "must have had a considerable share in the production of this

unparalleled plague." [18] Aside from yet another misuse of the word "plague" here and another instance of the often morally condemnatory assessment of disease, Hecker alludes to the effects of multiple years of poor weather, and how such could have not only affected the soil and its agricultural yields but also the resistance to disease of the human populace. Those in Tudor England may simply have been more susceptible to disease, including otherwise unknown diseases. His conclusion is among the more direct ever offered for the Sweat, which according to him "was inflammatory rheumatic fever, with great disorder of the nervous system." [19] For him, rheumatic fever had to be sweated out, via "a profuse, sour, and offensive perspiration." [20] He also argued that extremity pain described by some accounts of the Sweat could match rheumatic fever, along with a tendency to develop dropsy (though dropsy was understood by Tudor writers, yet does not appear in their accounts of sweating sickness). And the curious smells emitted by sufferers continued for Hecker as well, who noted that rheumatic fever patients also would typically present with "volatile acids of a strange odour" which become "prevalent in the sweat, and urine, and animal excretions." [21] While his initial focus is on the first 1485 outbreak, Hecker argues that the same combination of excessive rain and other factors enabled the additional epidemics.

While "the English Sweating Sickness was the theme of discourse everywhere," [22] especially when it attacked Continental Europe in 1529, it became common and received knowledge that sufferers must be encouraged to sweat and not sleep for a full day, in a "rehearsal of hell," though the problems with possibly overheating patients is a topic in need of fuller addressing later, as will be shown. Yet it was descriptions like this that helped draw Hecker to his own conclusion. First, rheumatic fever marks a susceptibility to chills: "in no known disease does this irritability of the skin (especially to cold) show itself in so prominent a degree as in

rheumatic fevers." [23] Second, rheumatic fever, he thought, had to be sweated out, via "a profuse, sour, and offensive perspiration." Third, there developed in sufferers a "peculiar alteration in the fundamental composition of organic matter in rheumatic diseases, in consequence of which volatile acids of a strange odour are prevalent in the sweat, and urine." [24] One could detect, perhaps, a genuine sweating sickness victim by a particular scent, and not just that of excessive perspiration during a period marked by infrequent bathing. Fourth, the radiating pains in the extremities matches a common symptom of rheumatic fever. And finally, rheumatic fever had an alleged tendency to develop into dropsy, though this does not appear in the primary accounts, and dropsy itself often appeared more often than might be expected in Hecker's own time.

What we know about rheumatic fever now, compared to a century and a half ago, is that it arises from untreated streptococcal pharyngitis, specifically group A, "in which inflammation leads to damage of heart valves and muscle." [25] While a comprehensive author like Bauman reminds us that the ultimate etiology of this damage remains mysterious, the resulting damage to heart valves may indeed become excessive, and the disease was far more lethal prior to the development of antimicrobial drugs. The point to keep in mind here though is that rheumatic fever, for all its apparent former appeal as a contender for sweating sickness, really does not appear in much more recent academic writing with any connection to the sixteenth century. Carlson and Hammond hint that perhaps the problem has been an overlap with symptoms of other diseases; indeed, one might well imagine a Venn-diagram sort of approach to all these various diseases, with a reader trying to visually assess how symptoms overlap with what is known about the Sweat. Carlson and Hammond, meanwhile, appear in far more detail in the upcoming section about viruses, since their own arguments lie in that area. In summarizing sweating sickness, they do note, though, how "rheumatic pain in the back and extremities" [26]

numbered among the clinical manifestations of the Sweat, but that such should be understood as part of the symptomology rather than as cause to argue for a different disease. Such pain will appear within their own conclusion in favor of a particular viral illness, but for them, a major part of sweating sickness assessment lies in considering as many details, including symptoms, as possible.

Finally on the list of bacteria-borne diseases appears anthrax. From the perspective of our own time, this one might elicit the most interest, simply due do its more recent associations with potential "weapons of terror," with even an ability to mail samples of spores to victims, and indeed it has been weaponized by several nations. For the Tudors, however, anthrax was the "wool-sorter's" or "wool-gatherer's" disease, since the spores which carry the bacteria are often picked up by domestic livestock, especially those with thicker hair, like sheep. Wool was the most valuable of the Tudor exports, and the only way to harvest it at the time was entirely by hand, so whatever sheep had come into contact with could easily in turn contact humans handling and shearing them, perhaps especially via small cuts to skin which were common among both the sheep and the shearers. Sheep agar solutions are even often used presently to grow anthrax spores in laboratory settings, such as those associated with the Centers for Disease Control, though of course very few individuals have access to such testing. Yet anthrax occurs in profusion throughout the world, and is most often associated with ungulates who ingest the spores or pick them up in their hair, unwittingly, and become carriers, so that their own flesh becomes a vector for any creature ingesting it in turn, but typically only if it is consumed raw or undercooked. [27] Additionally, like plague, the disease can be contracted in three different ways, manifesting with different signs and symptoms. The gastrointestinal version develops from the

meat vector, while spores can also be inhaled, or simply contacted directly, resulting in the respiratory and cutaneous variants, respectively.

Respiratory or pulmonary anthrax presents usually with flu-like symptoms, including sore throat, mild fever, muscle aches, fatigue, but then typically develops into pneumonia with respiratory collapse, when the disease becomes far more lethal. [28] Other symptoms may consist of chest discomfort, dyspnea, nausea, difficulty swallowing, and bloody sputum, and even if it does not lead to pneumonia, it may later entail higher fever, respiratory distress, shock, and may instead develop to meningitis. Cutaneous anthrax needs at least a partial break in skin to enter, which will initially manifest as itchy bumps similar to mosquito bites, altering into painless sores with a black center considered a hallmark indication of the disease. Such bumps differ notably from those described for rheumatic fever. Swelling then develops in the proximal lymph nodes, but this is far less severe and painful than the telltale buboes from bubonic plague. And gastrointestinal anthrax will present with nausea, vomiting, abdominal pain, headache, loss of appetite, fever, bloody diarrhea, and sore throat. [29] This last variant of anthrax may seem like dysentery, having numerous symptomological overlaps with it, and testing may be required to confirm either or both. Also, gastrointestinal anthrax and inhalational anthrax are both rare in humans. Non-humans are more likely to contract spores via inhalation, while humans remain most susceptible to the cutaneous form. Considering the proximity of humans to sheep during the Tudor years, one may easily imagine inhalational and cutaneous anthrax as certainly possible, perhaps likely under the right circumstances. Cutaneous anthrax does not closely match sweating sickness symptoms, though. The fever, achiness, and fatigue from inhalational anthrax seem a bit closer, though sufferers of the Sweat do not appear to have reported trouble swallowing, nor evince bloody sputum from coughing. The same applies to most of the

symptoms of gastrointestinal anthrax, plus this disease is also rare. Yet anthrax overall cannot be dismissed just yet.

By far the most dedicated proponent of anthrax to explain sweating sickness is McSweegan. His initial interest in the subject had nothing to do with the Tudors, but rather from attacks on Americans using a variant called the Ames Strain in 2001 (dozens of actual strains exist). That a disease-causing agent might be synthesized and even mailed through the Postal Service proved quite newsworthy. Yet McSweegan's own research soon turned up reports of sweats, night sweats, and drenching sweats in England from centuries earlier, and even Tudor physicians Caius and Forrestier appear briefly in his article. "Wool and animal hair are common sources of anthrax spores," [30] McSweegan notes, and the Tudor period experienced a major growth in wool production, often via the practice of land enclosure, as noted. This effect was felt far more in some areas than in others, and almost exclusively in England instead of Wales or Scotland (likewise, Scotland and Wales never appear on lists of sweating sickness epidemics as having been infected), though from 1455, to 1607, more than half a million acres of land in more than thirty counties in Britain were enclosed. As historian Lacey Smith concludes, this amounted to still less than three percent of total possible arable land, with roughly 50,000 persons total displaced. [31] The timing of the known sweating sickness outbreaks were chronologically and geographically "scattered," as McSweegan describes the matter, making specific diagnosis difficult, and he admits he would find the issue helped along with accompanying climatic data which we will likely never have with sufficient accuracy (though climate will be discussed below). And yet, "anthrax as the etiological agent of the Sweat has the virtue of not requiring an insect or rodent accomplice," [32] which could simplify matters greatly. Any disease diagnosis indeed requires an understanding of all culpable parties: microorganisms

need far larger host species to get anywhere, and hosts can either fall victim to the relevant disease or act as vectors in its further transmission. This must be the case for communicable diseases generally, whether the actual transmission mode is airborne, droplet (very short-term airborne, as with a sneeze or cough), tactile contact, or contamination of some seemingly more inert source like soil or water.

While McSweegan recognizes that inhalational anthrax is not really considered contagious among humans, part of the appeal for him is that "durability of anthrax spores and proximity to contaminated agricultural fields and products could have provided numerous opportunities for infection." [33] These spores "are forever," as he describes them, and he also clearly favors the potential of exhumation: some who fell victim number among those Tudors whose "graves have not been disturbed" for centuries, and he even has communicated directly with physician Guy Thwaites about some of these issues, though ultimately the two still disagree over fundamentals, as McSweegan favors a bacterial solution while Thwaites prefers a viral one, as will be shown. McSweegan does agree with Thwaites' work highlighting the rural aspects of sweating sickness, though for the former, the possibility of the kind of testing described above continues to hold tantalizing promise.

Among the stronger systematic arguers described herein, following a similar tactic of elimination logic, are the epidemiologist Paul Heyman, and parasitologists Leopold Simons and Christel Cochez, who have worked together trying to unite sweating sickness (and, curiously, the later Picardy Sweat) under the category of hantaviruses. While such work will thus be considered more in depth below, in the meantime their attitude toward the work of McSweegan is that it may be worth further study, but they collectively remain neutral to both it and other bacterial diseases. Another concern with anthrax as the culprit emerges in passing from the work

of Theilmann and Cate, who, while still mainly working with plague and its own certitude in medieval England, write that specifically "pneumonic plague can be misdiagnosed as several diseases, among them influenza, anthrax," [34] and others, though such others do not number among sweating sickness contenders. Anthrax may remain popular with some writers: Bauman notes that "historically, mortality rates have been high" [35] with this disease, though attempting to conclude with actual death tolls from sweating sickness has its own historiographical concerns. Bauman also points out that the early signs and symptoms of anthrax, at least the inhalation manifestation, "are common to many pulmonary diseases." Still, though, Bauman also points out the sureness of how *Bacillus anthracis* can only cause one disease, even if it can take a mix of clinical manifestations, and McSweegan makes a powerful case for the historical as well as current presence of anthrax spores throughout so much of England. That these spores have impressive longevity and could be tested with present techniques that have almost become routine makes further study of anthrax in this historical context quite appealing. Before coming to more specific conclusions, though, it is time to segue from bacteria to viruses, and how the latter have influenced sweating sickness research.

Table 1: Bacterial Candidates for Sweating Sickness: Plague, Rheumatic Fever, Anthrax

	Author(s)	Similarities of Symptomology Benefits to Sweat Research	Issues with Matching to Sweat
Plague (*Yersinia pestis*)	Kelly; Slack	Pneumonic matches Sweat descriptions most closely of all types of plague. It also requires no non-human vectors.	All three variants of plague (bubonic, septicemic, and pneumonic) were known to contemporaries as discrete.
Rheumatic Fever (*Streptococcus pyogenes*)	Hecker	fever, fatigue, chest pain, joint pain	Those favoring this as an explanation for Sweat appear chronologically earlier in the literature, and more recent researchers do not support it.
Anthrax (*Bacillus anthracis*)	McSweegan	fatigue, malaise, fever, muscle aches; Residual spores have high longevity and offer perhaps the best source for testing. Cutaneous eschars (sloughed dead tissue) might have been mistaken for plague buboes or vice versa.	Cutaneous anthrax is the most common of the three variants. Inhalational and gastrointestinal anthrax are both rare in humans.

Signs and Symptoms for Sweating Sickness (based on primary accounts of Fracastoro, Vergil, Hall, and Caius, and summarized by Carlson and Hammond):

sudden onset; full course from roughly one to fourteen days (diaphoresis followed by asthenia); fever; rheumatic pain (typically in back and extremities); abdominal pain; tachycardia; possible vomiting, diarrhea, bleeding; possible delirium; possible severe headache; possible multiple organ failure

B. Possible Culprits: Viruses.

As with the bacterial agents, the viruses which have been suggested as explaining sweating sickness seem initially to constitute an equally short list, as another three diseases arising from three different viruses have entered the historical discussion. On further examination, however, we will find that considering viruses is actually less straight-forward than considering bacteria, since two of the three viruses discussed here are actually members of rather large viral families. There is also far more detail to wade through, partly for this complexity with the study of viruses, and partly since academic writing about viral candidates for sweating sickness notably outweighs that about bacterial ones. Accordingly, we will have to confront matters even more specifically than in the preceding section, but in the meantime, it should prove advantageous to again consult Bauman and his text in particular, to get an overall sense of these diseases and why they are contenders for sweating sickness at all. Starting with these simpler definitions, we can then proceed into the sometimes surprising complexity evinced by the description and categorization of viruses.

First up for consideration is influenza, or simply flu. Bauman informs us that it consists of two species of orthomyxoviruses, and that cytokines (secretions from certain immune cells which can affect other bodily cells) released through the immune system and immune responses cause the signs and symptoms of disease, in this case usually including "fever, malaise, headache, and myalgia." [36] While this will come to seem the simplest classification of viruses in this entire section, it is also true that "genomes of flu viruses are extremely variable," [37] which is actually the reason why those seeking flu vaccines from pharmacies and other health care practitioners have to do so annually. Decisions about which particular strain will prove the most frequent culprit each year have to be made by government agencies, including the CDC in the

United States and the PHE in the United Kingdom, often in turn working with international groups like the World Health Organization. Since such predictions must evaluate huge numbers of variables, sometimes the wrong vaccines will get ordered, resulting in larger epidemics of flu in one country, though a repeat of the much wider pandemic from a century ago seems far less likely now. That misnamed "Spanish Flu" pandemic is in turn a useful place to start, as Bridson and Arrizabalaga each mention how that global crisis which killed some 40 million persons must have motivated work not just on the disease itself but on its history, at least as much as could be gleaned. Arrizabalaga describes early twentieth century commentators like F. Graham Crookshank describing sweating sickness as "but one form taken by influenza which was sweeping across Europe in epidemics at that time." [38] This hardly simplifies matters, however, since whichever other epidemics of "that time" possibly referred to are not otherwise specified. Crookshank himself also never mentions sweating sickness, and his interest lay in not just diagnosing but classifying diseases. There were, during the early modern centuries, "many severe catarrhal, 'sweating,' of influenza-like epidemics... associated with the prevalence of cases of illness affecting the brain and spinal cord in the manner of" [39] diseases of later times. Polio is especially cited in this context. For Bridson, Crookshank's work from then probably was partly driven by an urge to link influenza to other disease outbreaks which might remain otherwise unexplained, including sweating sickness, though this connection, even if it accurately describes Crookshank's thinking (which we can hardly confirm), leaves us with the same problem. Motivation to write history is not the same as reaching a historically useful or rational conclusion. Wylie and Collier also mention the global crisis of 1918 to 1919, similarly observing that confusion of sweating sickness with influenza likely only occurred during the

twentieth century, as epidemiologists and historians alike have since accounted for the flu pandemic. [40]

As it turns out, supporters of influenza as the leading candidate for sweating sickness comprise a perhaps surprising numerical majority, sufficiently so that later writers favoring other viruses or other causes entirely often feel compelled to offer their own explanations about why influenza must not be the same or necessarily even very similar to sweating sickness. Still, there is a rough chronological flow to the work with viruses in this context, with some similarity to that evinced in the prior evaluation of bacteria, with accounts of plague as a culprit tending to be older, even if it was dismissed even by the writers of primary Tudor accounts. In other words, emphasis in the scholarly literature about influenza does seem to have been largely inspired by both an increased understanding of it, along with a truly catastrophic outbreak of it, as recently as a century ago. Since then, though, and especially within the past half-century, the gradual shift has been made to consider various other viruses for the Sweat instead. One writer who may have unintentionally predicted part of this shift is Creighton himself, who recognizes even before the twentieth century pandemic that influenza "appeared comparatively late in the history" [41] of epidemics, especially those possibly pertaining to sweating sickness. He describes how "as early as the year 1554 the Venetian ambassador in London called the sweating sickness of 1551 an *influsso*," [42] the Italian form of an older term, *influxio*, which refers to symptoms (he mentions a humor, or a catarrh, the latter of which includes the mucus discharge so often associated with flu). If this is true, and Creighton believes that this Italian-based terminology to have perhaps "got that sense by popular usage," then it may be easier to tell how potential confusion might have arisen. That influenza was not understood by the Tudors might account for influenza emerging as a candidate for no other reason than linguistic ambiguity. Persons in the sixteenth

century also do not appear to have been able to differentiate its symptoms from, say, a common cold, and of course knew nothing of viruses. Disagreeing with such assessments is Theophilus Thompson, though he writes from the mid-nineteenth century and focuses almost solely on influenza, albeit during a time which we ourselves may associate with less developed medicine. He describes how "in 1510, the first well-described and widely-prevalent epidemic of Influenza appeared." [43] He also insists that "Sweating-sickness ravaged England in 1506," which is clearly a wrong date, though that hardly reduces the need to elaborate more with viruses.

Prior to the continuation of this study of viral candidates, then, it should prove helpful to explain that "while some microbial threats seem to be frequently emerging or re-emerging, others seem to wane or attenuate with time," which in this case may also include "the disappearance of 'English sweating sickness'," [44] as microbiologists Arturo Casadevall, Ferrir Fang, and Liise-anne Pirofski describe matters. Their focus is not with Sweat as such, and indeed this reference to its vanishing is their only mention of it. Rather than even deal with specific diseases at all, their explanations are about virulence, the ability of microbes to successfully invade hosts, whether disease results or not. The ability to cause disease is essentially its virulence, and this reveals another etymological curiosity, since the term clearly has the same root as "virus," even though viruses of course are just one broad category of infectious and potentially disease-causing agents. As Casadevall et al. further explain matters, "critical to our understanding of virulence as a property that can only be expressed in a susceptible host is that both the microbe and the host bring their own emergent properties to their interaction." This enables the same microbe, regardless of type, to express as disease in one host and yet possibly have no discernible effect, advantageous or detrimental or neutral, on another host of the same species. Additionally, it means that our understanding of host-microbe

interactions, with virulence thus described as an emergent property or properties, "implies that the outcome of host-microbe interaction is inherently unpredictable." [45] And yet patterns do emerge: without them, disease prediction and most of the field of epidemiology would either not exist or become meaningless, and Casadevall and his co-authors further recognize that human behaviors, on both individual and societal scales, can certainly affect such outcomes.

Robert Hope-Simpson cites a publication about the history of flu, and of Britain, which sounds like it might resolve the issue all by itself. An additional interesting feature of his work emerges in his plan to use parish registers to help with the overall analysis, which will be discussed further below. For him, various influenza "key years" were selected "in which an influenza epidemic or an epidemic almost certainly of influenza was recorded." [46] And the work he refers to as hopefully assisting in this endeavor is a piece finished in 1852 by Thompson in Great Britain purporting to account for all outbreaks of influenza from the early sixteenth to early nineteenth centuries. This would cover the periods of the third, fourth, and fifth outbreaks of sweating sickness, missing the earlier ones from 1485 and 1508. The immediate problem, however, has to do with this timing: while the data and charts in the book are indeed useful for studying influenza itself, the first outbreak described dates only to 1558, when Elizabeth I first came to power, and this is seven years after the final known Sweat occurrence from 1551. Perhaps of more direct use is an anecdotal case described by Hope-Simpson regarding a physician named Jones, who in the account by Thompson "proceeds to compare the sweat, almost certainly the epidemic mentioned in St. John's (then governor of the Isle of Wight) despatch of 6th September 1558, with the sweating sickness of 1551." [47] However, while this comparison sounds fascinating, nothing else is mentioned of the case, neither by Hope-Simpson nor Thompson; considering that this is the only potential link between the Sweat and the gap

between the two years discussed, we have nothing to really work with here to help the case for influenza.

One feature in favor of influenza is its similarity in virulence, or contagiousness, as compared to primary accounts of sweating sickness. And while they will eventually come to favor arboviruses as discussed below, virologists Carlson and Hammond remind readers that "person-to-person transmission of sweating sickness is strongly suggested." [48] More importantly, "evidence of transmission by close contact is further supported by the observation that there was a tendency for multiple deaths within families," [49] a feature of the disease which would also tend to support a disease like flu, which thrives in close quarters among multiple roughly simultaneous sufferers. Sometimes logical leaps come too easily, however. Purdell, for example, while describing the physicians in service to Henry VII, quickly decides that they "had to handle medical affairs in the castle during an epidemic of the sweating sickness, probably a form of influenza with pulmonary complications." [50] Even the timing of this assessment is dubious: if Henry was already king, then it cannot refer to the 1485 outbreak, and so must indicate instead that of 1508, not long before Henry's own death. The descriptions of medical staff in Purdell's work are outstanding, but this summary of the Sweat feels jumpy and uncertain, or at least simplistic, though in fairness she is hardly the only one to have encountered the historical curiosity in the sources, realized something must be said of it, and then moved on to the next topic. Contentious or mysterious issues may sometimes be best addressed so abruptly, at least in order to continue the flow of one's writing.

A similar perhaps hasty conclusion is reached by Wylie, "a retired pathologist and theologian," and Collier, "a professor of virology." [51] Their article in 1981 is referred to in much of the more recent literature as prompting the increased interest in sweating sickness since it was

published, and to their credit they, like Arrizabalaga and Bridson after them, among others, do a fine job of summarizing both the history of the disease and its various commentators, both primary and secondary. [52] Among their more powerful contributions to the discussion is their noting how three major obstacles exist in arriving at a working theory about sweating sickness etiology and its outbreaks: "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," [53] the last of which is considered effectively by Casadevall, Fang, and Pirofski.

Yet detailing the hazards of this research and actually concluding with a working retrospective diagnosis are clearly two different tasks. Still firmly on the metaphorical side of influenza remain Nash and Roberts. Nash continues to seek definitions of disease and how it relates to the study of other sciences. He argues that "in the evolution of medical science two distinct tendencies diametrically opposite are in evidence: – 1) A tendency to differentiate as distinct affections diseases which had been included under one common designation (such as with the already discussed ambiguities in clarifying what is meant by historians and sources when they mention 'plague'); 2) A tendency to determine a persistency of type running through a long series of disorders of various designations." [54] For his own case, Nash refers to potentially different causes, and therefore understandings, of pellagra, and then contrasts this with leprosy, the latter of which seems more scientifically and epistemologically secure: throughout most of history, it seems, leprosy has only quite rarely been mistaken for something else. Perhaps some diseases are simply easier to know and thus differentiate from others. Nash has already been cited for comparing sweating sickness to influenza, and for him there exists another comparison, which will have to wait for now, with Picardy Sweat of later centuries; such examination must be delayed since whether to classify Picardy Sweat as bacterial or viral or something else is itself an

unknown for the moment. Yet for now we are left with Nash's awkward comparison suggesting that influenza itself "is essentially a protean disease, showing changes of type," [55] with subsequent name variations. However, one feature shared between influenza and the Sweat is the former's "tendency to relapse." One must not lose sight of his focus: Nash is not arguing about sweating sickness as such, but for the "specialized types" of some disease, and he proceeds from this triple comparison (Sweat, influenza, Picardy Sweat) to similar logical issues in knowing measles, smallpox, and malaria, three others, perhaps like leprosy, which seem a bit more straightforward in recognition and thus diagnosis.

Roberts, meanwhile, remains entirely focused on sweating sickness. His initial effort is mainly to answer Sloan, and who criticizes not just him but also Creighton, albeit a century after the fact. For Roberts, "evidence has always been available (and ignored by Creighton) that proves that there were other outbreaks of the sweating sickness." [56] Such alleged extra outbreaks have their own historical problems, mainly in that Roberts is the only source who ever seems to mention such, but the key note in his work is that "thus sweating sickness was so restricted neither in time nor in locality and it was often equated with outbreaks of influenza." But this is all: he gives us no further assistance in equating sweating sickness and influenza, and while most works considered in this section about influenza are more recent than his own, his too-brief summation would seem to leave readers eager for more or frustrated and prepared to disagree, if for no other reason than that, as with Purdell (although under more forgivable circumstances), we are left with just a quick statement and nothing else to really use. We cannot even determine who (nor in which century) really claimed that the Sweat and the flu were one and the same.

Perhaps the most unusual consideration of influenza in this context is not so much how often it has been cited, but that it is easy to offer as an explanation, and yet also, interestingly,

comparatively easy to deny as the culprit. Robert Gottfried plays a key part in explaining this, as he cites current scholarship as generally indicating "a variant form of influenza" [57] for sweating sickness. He also notes how "seasonal mortality patterns take on special significance in studying the effects of the Sweat," [58] so he is likewise in tune with how some researchers emphasize the effects of climate on diseases appearing and infecting, in many cases having comparative dormant periods which might last years. Such questions will be analyzed further below with regard to the Sweat. And yet before we get there, it is important to note how Gottfried himself refers to how if sweating sickness truly was influenza, then it would likely have continued to spread and infect and kill through the winter, though primary accounts regularly and reliably refer to the Sweat being a summer disease. This hardly resolves the influenza question by itself, though it does show how Gottfried initially seems open to the flu explanation, only to reject it. Still, he did this work prior to the more recent appearances of hantaviruses, and one can only speculate if he might have changed his mind at all in light of the later theories.

So, influenza can arise just from either of two main types of orthomyxoviruses. Type A is the one accounting for human pandemics. Type B can also be contracted by humans but is not nearly as deadly, and can also infect avians and pinnipeds. There actually is also a Type C, though it often receives less consideration, its virulence limited to humans and other mammal species, in this case just the families *Canidae* and *Suidae*, dogs and pigs, in other words. [59] The various strains within Types A and sometimes C comprise the targets for the makers of annual vaccinations. And while this categorization may seem tricky to keep straight, it simply pales in comparison to the other two groups of viruses for consideration herein. The arboviruses, by comparison, named for having arthropod vectors, are grouped into four families, with a total of at least twenty-eight genera. They become spread via mosquitoes and ticks into larger animal

hosts, typically small mammals and avians, making them zoonotic. "Arboviruses enter target cells through endocytosis (using active transport to pass cell membranes) and replicate within them. Most cause mild, flu-like symptoms in humans," [60] though they are also the culprits behind the likes of encephalitis, dengue fever, and yellow fever, diseases having notable febrile stages. These result when such viruses invade key organs or blood vessels. The hantaviruses, meanwhile, are a genus of the *Bunyaviridae* family of viruses, which number "about 300 different viruses," [61] according to researcher Eric Bridson. They are named for the region in Uganda where they were first isolated. Hantaviruses are the only members of this large group to not use arthropods as vectors, and can lead to other, less well known diseases in humans.

Hantaviruses will be saved for final consideration, partly for their complexity and partly since they comprise the main competition to my own conclusion in favor of arboviruses. In the meantime, we can consider the potential merits of arboviruses as candidates for sweating sickness. As they do for influenza, Wylie and Collier continue to invigorate interest in the virus hypothesizing, and while they end up not favoring influenza directly, they turn instead to arboviruses, the first to do so within sweating sickness literature. Based on their already mentioned trio of logical pitfalls for this research, Wylie and Collier systematically proceed to rule out not just influenza but also typhus, plague, and smallpox, and even meningitis (though this last is bacterial instead of viral). A similar approach is utilized later by Casadevall, Fang, and Pirofski, though they reach different conclusions. And interestingly, after leaving influenza behind, Wylie and Collier then consider whole families of viruses, only to decide that these too cannot stand up to scrutiny as contenders. Enteroviruses and arenaviruses thus make brief appearances in their influential article, but they feel the most promise lies with arboviruses. A basic distinguishing feature of these different taxonomical groupings is the vector: enteroviruses

are part of an even larger group with the potential to affect vertebrate species generally, and arenaviruses rely on rodents, occasionally affecting humans and sometimes reptiles. As for arboviruses, again, they by definition require arthropods, specifically some insect species, to get about. [62] Arrizabalaga offers a useful summary of them: "an arthropod-borne large order of RNA viruses which can cause four different sets of diseases, namely encephalitides, diseases with fever and rash, diseases with hemorrhagic manifestations, and mild fevers." [63]

Interestingly, some of the very diseases Wylie and Collier briefly mention at the conclusion of their piece number among those considered later, and include some that may typically only even be recognized by specialists like microbiologists, virologists, and infectious disease physicians. I had certainly never heard of such afflictions as Russian encephalitis or the even more obscure sounding Omsk hemorrhagic fever, also mentioned by Davidson, but the idea that either or both might be contenders for the Sweat will be taken up by later writers. It is worth noting that both of these are members of the *Flaviviridae* group of viruses, and not the *Bunyaviridae* group, the latter of which includes hantaviruses. Again, a writer like Arrizabalaga, arguing more than twenty years after Wylie and Collier, agrees with their assessment of both these exotic sounding illnesses, pointing out as well that in the often changing field of viral research, Russian encephalitis itself has undergone a name change, now known as both Russian spring-summer encephalitis, or as TBE, tick-borne encephalitis.

While these researchers all have offered worthwhile contributions to the ongoing dialogue, work by virologists Carlson and Hammond offers the most comprehensive explanation and support for the arbovirus solution, as well as giving excellent summaries of the sweating sickness and its strange historiography. In their own research another strange-sounding disease (at least to non-specialists) becomes the focus of their research: Crimea-Congo hemorrhagic

fever, or CCHF. Arrizabalaga approves of their work in this summary, too, noting that this is one of a quite short list of arboviruses that can be transmitted person-to-person, even without the otherwise requisite arthropod vector (though insects may yet be necessary for initial outbreaks), and the "only one among them that has been associated with epidemics not restricted to Africa." [64] In a manner reminiscent of Arrizabalaga, Carlson and Hammond present their summary of sweating sickness symptomology, even while they lament that we have few primary descriptions by physicians. Tudor doctors might have compared some symptoms with plague, noting how both diseases were "violent" and had high fatality, though more specifically, "the signs described by Caius and Forrestier are typical of those now recognizable as a severe viral prodrome, including a sudden onset fever, headache, myalgias with backache, pain in the arms and legs, abdominal pain, and heart palpitations or tachycardia." [65] Caius recognized that plague in its later stages can be hemorrhagic, and Forrestier understood that "black spots" on some patients with sweating sickness would also seem to support some internal bleeding aspect. Forrestier also noted how some of them might present as "red and yellow," which Carlson and Hammond easily link to liver failure with its distinct symptom of skin jaundice. Renal failure, meanwhile, is suggested by "turbid dark urine... passed in small quantities," [66] along with halitosis and odorous perspiration, perhaps from uremia.

Hemorrhagic fever as a symptom is the key, however, for Carlson and Hammond, much more so than for its simply comprising part of the name of their concluding disease. After summarizing potential agents, including some bacterial ones as well as other viral contenders, they note that the listed criteria "all indicate a viral infection with a source within a zoonotic life cycle." [67] The "epidemiological clues" offered tantalizingly within primary Tudor accounts, as well as the "sudden and violent clinical course" which also appears to have been the expectation

with sweating sickness, "point to a diverse group of viruses that cause hemorrhagic fevers." Four families of such are then identified, with their own backgrounds in six continents, yet they vary widely with regard to reservoir, vector, transmission, and of course, resulting illness. As for CCHF itself, its cycle includes ticks as a vector (possibly as a reservoir as well), with the *Hyalomma* genus able to exploit small vertebrates for immature ticks, and larger vertebrates for adult ones. They also cite twentieth century research for CCHF, in which hares and cattle were especially affected, though avians and many other mammals, including humans, are known to be able to carry the virus. Even more appealing, "the geographic range for the CCHF virus is the most extensive for the tick-borne viruses associated with human disease, and... CCHF virus is the most widespread of all medically important arboviruses." [68] For these researchers, this particular virus can match both the known symptomology and the necessary biological cycle. They still have to fit this cycle into Tudor England, though. The significant issue for CCHF in this case, despite its other merits for sweating sickness, ultimately entails placing *Hyalomma* ticks in Britain, and there is simply no compelling evidence to argue this way. Quite recent research into this question concludes that just obtaining data on ticks infected with CCHF (sometimes abbreviated as CCHFV, the "V" simply for "virus," in the academic literature) requires "(1) the total number of *Hyalomma* spp. ticks entering (Britain) on migratory birds per year, (2) the proportion of those *Hyalomma* spp. ticks originating from CCHFV-endemic regions (in this study, the authors cite "Asia and sub-Saharan Africa," as well as "some areas of southeastern Europe and the Balkans"), and (3) the prevalence of CCHFV in *Hyalomma* spp. ticks in endemic regions." [69] This more recent study by virologist Marion England and her team tries simply to judge the relative risk of such ticks hitching rides into the British Isles from the Iberian Peninsula, and these researchers mainly recommend continued vigilance on such ticks, though

otherwise do not really delve into history, thinking that it is unnecessary. Such ticks are not found in Britain.

That monitoring research is described more fully by disease specialists Lisa Jameson and Jolyon Medlock, who point out initially that "before 2005 there existed no formal tick surveillance program in (Britain) and limited contemporary data on nationwide tick biting," [70] including earlier history as well. For them, twenty species of ticks "are considered to be resident in (Britain)," and *Hyalomma* is among the "exotic" of them, when it is found at all. (On an aside, *H. truncatum* also numbers among these exotic species within Britain, the vector for veterinary sweating sickness mentioned quite briefly earlier). The most important part of their claim from this perspective is that "whether *Hyalomma* ticks would be able to survive and establish in (Britain) is currently under investigation," [71] so the contention of CCHF is unfortunately not as promising as it initially seems. The point is that if English sweating sickness truly was distributed by *Hyalomma* ticks, then it must be plausible in turn to describe these arthropods as living in Britain; yet this does not appear to be a coherent case after all. This in turn poses a major point against the otherwise fine case argued for by Carlson and Hammond, who clearly favor CCHF as the culprit.

Nonetheless, parts of the case made by Carlson and Hammond can still help us in our quest to pin down the precise culprit. To return more to the historical issue, as they summarize, roughly speaking, rapidly shrinking forests in England and increasing human population both led to large increases in agriculture, an issue the Tudors certainly recognized. One example is legislation from the reign of Edward VI, a 1548 statute "Prohibiting Encroachment in Waltham Forest," which partly reads, "and in the forest of Waltham in our county of Essex... command that none of you our said loving subjects do at any time from henceforth enclose with any such

unreasonable hedges or ditches and of the said closes and pastures." [72] The enclosure issue has been touched upon before, albeit mainly as an economic concern: here it becomes more of an environmental notion, with possible side effects regarding the transmission of disease. Carlson and Hammond outline matters further: large non-human species began to have their habitats encroached upon as a result. They mention swine, beavers, lynx, and bears as examples as victims of such encroachment, but these were not often the focus of hunting by humans. And in a curious display of land preservation, albeit to satisfy royal demands, reserves of deer parks dating at least to the eleventh century remained, and could be acquired by wealthy nobles and perhaps even newly rich merchants as well. Such hunting grounds "preserved high densities of small and large mammals that could have fulfilled the requirements of CCHF virus to establish enzootic cycles (pertaining to certain areas or seasons or both)." [73] This model does account for the tick life cycle, and the virologists will eventually try and account for the increased likelihood of upper class men falling prey to the disease, as mentioned previously by Sloan in reference to the past and by Gilbert in reference to the present, and now more firmly based on the pastime of deer hunting in such exclusive locales. They thankfully question this contentious notion about the Sweat, too, among the few writers to do so. Primary sources sometimes do mention an apparent preponderance of the disease among healthy and generally younger men, including courtiers, clergy, and university students and faculty, and during the first four of the five outbreaks. "The mortality pattern," writes Bridson in this context, "suggests person-to-person contact," and yet "the young and the old were spared, suggesting that males in the 15 – 40 age group were most likely to congregate in ale-houses, etc., and spread the infection between themselves." [74] Dyer shows that burial records indicate a roughly 3.5 to 1.0 ratio of male over female deaths from the Sweat, at least within London, though he also suggests there may have

existed "a female majority in the countryside and a male one in the towns," [75] and insists that longer burial runs tend to be dominated by one gender. He does not otherwise specify the alleged masculine disease dominance in this case. And such reports are "fascinating," yet "susceptible to the criticism that the disease was over reported in these classes of high distinction." [76] This almost leaves a careful reader wondering if we have returned to the old prejudices of "great persons" history narratives, in which the "little people" simply do not appear, even when their presence might help explain some useful cultural detail, as with the history of diseases. Yet, "the persistence of reports from the earliest epidemics (of sweating sickness) has given them added credibility," as these two report. Also, with the final 1551 epidemic, susceptibility of these same privileged males "was diminished," and children even appear in the reports from that year, "an age group that had not previously been commonly reported." [77]

While the strength of their case will, along with these other candidates, be returned to later, it will prove useful to summarize Carlson and Hammond and their details of CCHF, all the more so since of all the diseases contenders thus far, this one is likely the least known among non-specialists, indeed is probably not much known outside their specialty of virology. To summarize briefly, Crimean-Congo hemorrhagic fever has an incubation of three to six days, with a "high ratio of disease to infection (1:5)," [78] as they indicate. It has the three typical clinical stages for a hemorrhagic illness: prehemorrhagic, hemorrhagic, and convalescence. There is usually an ongoing febrile component, with apyrexic periods in approximately half of patients. Other signs and symptoms include chills, headache, rheumatic pain (not be confused with bacterial rheumatic fever, however), lumbar back pain, epigastric pain, the unpleasant "NVD" combination mentioned in the bacteriological section above, adynamia (decreased

strength or vitality), loss of appetite, hyperemia in the face, neck, chest, or all three, congested sclerae, conjunctivitis, and early bradycardia and low blood pressure yet with tachycardia (but no reported hypertension) later. This is quite a list, including some signs and not just symptoms, since medical signs by definition include attributes verifiable by witnesses, perhaps more reliably than by the patient. Death, if it comes, usually occurs during the hemorrhagic stage, during which hemorrhages "from the size of petechiae to large hematomas may appear on the mucous membranes and skin; intestinal hemorrhages and uterine hemorrhages occur" [79] also. Finally, the hemorrhagic stage may also present with pneumonia, hardly making matters easier for sufferers (and part of disease diagnosis, including of a retrospective historical sort, becomes more complicated when multiple diseases may be at work). Bleeding in various organs and tissues can be confirmed via autopsy, though of course this is hardly possible with sixteenth century decedents, even ones whose remains could perhaps be exhumed; organs and soft tissues simply do not last long post-mortem without specific preservation techniques, which the Tudors either did not have or did not use. Mummification was hardly a practice in the British Isles during any period, with a very small number of accidental exceptions such as "Lindow Man." To conclude, the "overall disease course" with CCHF can be expected to last approximately fourteen days, and during the convalescent stage survivors are likely to experience sweating, headache, dizziness, nausea, and perhaps weakened breathing, vision, hearing, along with possibly memory lapses and tachycardia. Again, the purpose is not to be exhaustive at this stage with any particular disease, but rather to call attention to the minutiae of one which many may find unusual. Also, there is no way to confirm every sign and symptom on this list with documentation in the primary Tudor sources, so the logic and argumentation of the overall case will remain what matters most here.

Speaking not of any particular viral illness but about the sweating sickness, "it was a unique disease," Bridson tells us, "in that it did not respect princes, high nobles, rich merchants or poor men," [80] echoing those sources which try and account for whom it struck. It seemed to vary "from most infectious diseases that slaughtered the poor whilst leaving the rich relatively spared," he continues, concluding that males roughly from the ages of 15 to 49 appeared to have been most at risk. That this admittedly broad age range largely coincides with the typical late medieval into early modern penchant of making boys and men from 15 or 16 up to 50 or 60 eligible for some kind of military service, even if just the outdated medieval annual muster, goes unnoticed by all the researchers, including Bridson himself. This seems strange, considering especially the initial 1485 outbreak and its military contexts: imported from the Continent (allegedly) with or from foreign mercenaries, and the military implications it had during that same epidemic, notably at Bosworth.

The other issue should be clear here, also: every disease, literally, is of course unique, strictly speaking, but one gets the sense from Bridson (and from others, too, particularly Heyman et al.) that the reference here is more to sweating sickness having been so unusual and so uncharacteristic that it presented with its few historic outbreaks and then vanished into some realm of mystery and speculation. While these issues represent more of Bridson's own outlook, and while his own conclusions are more in line with hantavirus, he raises some logical questions for the arbovirus argumentation. To begin with, after he, too, reviews the old reports, including those of Caius and Forrestier, Bridson writers that "surprisingly, the physicians (of the time) did not report excessive fever; and whilst sweating was a significant symptom, it was most likely caused by acute pain and high anxiety." [81] Pain and anxiety will have to await further assessment, and while he is correct about the unanimous reporting about diaphoresis (it would be

odd for this disease to have received its moniker otherwise!), the contention about the febrile component would be troubling if true, since the arbovirus explanation seems to really need fever to remain coherent and plausible.

Table 2: Sweating Sickness in England

Year of Sweating Sickness Epidemic	Environmental Conditions	Intraepidemic Period	Interepidemic Period
1485	1480 – 85; wet years	June – October	
1508	mild winter; hot / wet spring and summer	July – August	twenty-three years
1517	dry summer	July – December	nine years
1528	wet spring; crop failure with famine	June – August	eleven years
1551	dry / cold spring; hot and and humid summer; crop failure with famine	April – July	twenty-one years

(taken from Carlson and Hammond, "The English Sweating Sickness (1485 – c. 1551): a New Perspective on Disease Etiology," p. 29, and reproduced with permission)

While we have already met Bridson's disagreement with influenza, he also targets Dyer, and the potential issue with human-to-human contact for the spread of sweating sickness. Dyer admits, as we have witnessed, that there are indeed few such contenders in that regard, "however, Dyer was convinced that (human-to-human) contact was the only explanation for the explosive spread of the disease in local communities." [82] And moving on from there, he proceeds to critique the work of physicians who have thus far not appeared in this work, but whose articles will play a prominent role in the forthcoming hantavirus section. These are mainly Guy Thwaites, Mark Taviner, and Vanya Gant, and while they do not favor arboviruses either, they appear in the summary of Bridson since, while "agreeing that the sweating sickness was caused by a virus with a rodent vector, dismissed the arbovirus theory. They argued that

most arbovirus diseases are associated with cutaneous haemorrhagic signs, and, notably, these were absent from all accounts of the disease." [83] This research writing team does come to favor hantavirus instead, and while their potential agreement with Bridson is not precise, the point is that while arboviruses have their merits, these other writers maintain that they remain insufficient overall as an adequate explanation.

There exist additional points of contention among these ideas. Despite the excellent summary and interpretation by Dyer, for one, he nonetheless concludes partly by arguing that "we must assume... that sweating sickness was not generally transmitted by an insect vector but rather through close personal contact," [84] which clearly goes right back to the issue questioned by Bridson, among others. And while the other contenders for arboviruses find no general problem with this direct contact, presumably either tactile or at least within exhalation range, if Dyer is right about the lack of an insect vector, then arboviruses, by definition, would have to be removed from consideration as the culprit. The research they refer to which links CCHF particularly to species like hares and cattle may also be problematic: both groups have of course existed throughout the British Isles for thousands of years, yet no other scholarship, then or now, has ever suggested that infected ticks might move from hares or cattle to humans for the purpose of disease transmission, nor indeed of any known disease. Also, while the dwindling forests and growing human populace of Tudor England are well established historical details, a causal link to more intensive agriculture seems problematic. Indeed, human population growth may have actually inspired increases in both of the other factors: thinned forests and more livestock, with populations of the likes of hares and ticks otherwise unaccounted for, unless we return to the more recent scholarship offered by Hudson, Gilbert, and Laurenson in particular, and their explanation of LIV. They account more comprehensively for the ecological interactions among

hares and ticks as well as other woodland species relevant to sweating sickness historiography. And while Carlson and Hammond themselves admit that person-to-person transmission of these types of viruses is rare, they make the logical leap to CCHF since it is one among only four known viruses which meet their two essential criteria of being transmissible in this direct manner while also manifesting with hemorrhagic fever as a key symptom. And yet as Wylie and Collier point out, most arboviruses which can lead to human epidemics simply require bites from arthropods. Perhaps the encouraging news is that, with the Sweat, "its various manifestations may be fitted into the established spectrum of disease behaviour." [85] This is also true of LIV, a virus which interestingly never gets mentioned by Carlson and Hammond.

Of equal frustration is the claim that "evidence from the other sweat epidemics (those taking place in years other than 1551) is too fragmentary to support any theory as to their origin," [86] which might threaten to throw out the whole project. Dyer, having said this, prizes other types of research tools, especially parish records, to help narrow the issue, and yet with no origin story, even a tentative one, a retrospective diagnosis seems all the more difficult, perhaps leaning towards the implausible. Knowing, or at least postulating, where a disease came from, could also help with that diagnosis, since part of medicine and epidemiology in our own century entails an understanding that different illnesses really do have different sources. As for an ultimate etiology, "when examined at parish level we see a chain of infection which appears to be very fragile, easily broken to terminate the outbreak or to await further re-infection from outside." [87] Fortunately at this stage, geographic and organismic origins have yet to be considered but will be later, including an appraisal of where this "outside" might have been. LIV, for example, already existed within Britain, yet sweating sickness apparently arrived with Henry Tudor's troops, so since there are no prior accounts of the Sweat prior to the Tudor years, something must have

enabled it to return or to alter itself in such a way that it could become sufficiently prevalent and virulent to begin afflicting humans.

The final commentators to consider regarding arboviruses are the previously mentioned Heyman, Simons, and Cochez, who will also offer their own insights into the possibility of hantavirus. What is most attractive about their article is the systematic logic of elimination utilized herein, and also by writers such as Arrizabalaga and Bridson. They acknowledge that whatever the sweating sickness may have been, it was clearly infectious, "but different... from the plague, influenza, smallpox, typhus, scarlatina or malaria." [88] While plague may have been the only one on this initial list known to the Tudors, these writers then continue to deduce, based on both primary and secondary writings, what the Sweat apparently was not. It was not typhus, they argue, "because of the speed with which the symptoms appeared and the extremely short course of the disease." Likewise, influenza gets ruled out due to "the absence of any respiratory symptoms or secondary cases of pneumonia." [89] Food poisoning, considered below and according to Heyman et al. as most likely manifesting as botulism, seems easy to discount due to its geographical implications: as they note, the Sweat's appearance, as Dyer helps confirm with his mapping, had its strongest influences in particular areas, and food toxicity would seem more likely to have a more even distribution. Additionally, famine does not appear to have ever followed sweating sickness outbreaks (though there may exist evidence for the reverse of this, as we shortly consider toxins). Finally, this is the first mention of the possible connection of sweating sickness and Picardy sweat of a later century, a link that cannot be as easily dismissed as some may believe, including, previously, the author of this dissertation. Viruses have to be completed first, however.

So, the works of Dyer, and of Carlson and Hammond, together help to give the arboviruses probably the greatest strength as candidates thus far. Interestingly, it may be Arrizabalaga once more who offers the best summary of the implications of the work of Carlson and Hammond (Heyman, Simons, and Cochez published more than a decade after Arrizabalaga), as he describes them as feeling so strongly about their conclusion regarding CCHF that they became willing to claim that the Tudor sweating sickness was a historical one-off, a unique biological occurrence, a perhaps logically safe yet academically less than satisfying statement since it allows us to "never know definitively and also since RNA (for potential testing) degrades quickly." [90] This was the same lesson I had already learned from my discussion with Dawn Alapisco at Oregon State University: having nothing to test, even if consent could be obtained, we would seem left with a mysterious conclusion. To try and resolve the mystery, then, we can turn to the final viral category appearing within the scholarship of sweating sickness: the hantaviruses.

As with early twentieth century analyses of influenza (especially in its globally devastating pandemic form just after the Great War), perhaps part of the scholarly appeal of hantaviruses is their apparent newness. It may even be common knowledge by now that they, or at least one such virus, was isolated and identified as recently as 1993, and as close as the United States Southwest. It is important, however, to illustrate that this family of viruses was actually identified and initially categorized with other similar viruses forty years before then, and in East Asia instead. Indeed, to understand the scholarly utility of hantaviruses, and in particular their potential relevance to the study of sweating sickness, it is crucial first to evaluate their history and what is actually known about them, and partly since North America and East Asia are hardly close to England. Theilmann and Cate offer a useful segue from arboviruses to hantaviruses,

although it does curiously pose as many questions as it tries to answer. "Although the sweating sickness was not the killer that the great pestilence (the plague) had been," as they report the matter, "it clearly confounded the medical practitioners of the day." Far more tellingly, and directly pertaining to this section on various viruses, "One explanation is that its causative agent was similar to an arbovirus, perhaps a hantavirus." That gets us closer to the current discussion, yet "when subjected to closer scrutiny, the hantavirus diagnosis falls apart." [91] That seems a less encouraging way to begin this section, and yet, frustratingly, these authors do not say why this must be the case: arboviruses may be contenders, but they dismiss hantaviruses out of hand with no explanation, after having already explained the details of why the Sweat was not plague. Stronger is the claim made by Heyman et al., which indicates "a strong possibility based on the resemblance of Sweating sickness with HPS (or HFRS)," [92] but they otherwise bow out of the debate at this stage, having offered all the logical considerations they feel they can. And their claim in this regard feels suspect: while they acknowledge hantavirus as the most recent scholarly contender (other than anthrax, perhaps), "however, if the English sweating sickness had been caused by a hantavirus, at least isolated cases would have been reported over time in England and this has not been the case." [93] The second part of this is surely true: there is no extant case within the United Kingdom in any way comparable with the 1990s incidents recorded in the United States. But this apparent contradiction is frustrating, and it should prove far more beneficial to instead delve into understanding hantaviruses more fully, as well as why they deserve to be considered as the culprit for the Sweat.

So, to begin with, we need to understand what separates hantaviruses from arboviruses, especially since the scientific nomenclature easily becomes layered and confusing for laypersons. Officially hantaviruses are a form of bunyaviruses, typically zoonotic pathogens coming from

arthropods, but they do not need those same arthropods for disease transmission, as the arboviruses of course do. They "are transmitted to humans via inhalation of virions (the infective encapsulated forms of a virus outside a cell which can invade the cell to hijack it with foreign DNA or RNA) in dried deer-mouse urine or feces." [94] That is a typical microbiology textbook summary, which may include another detail, frustrating for the non-specialist researcher, about how "diseases caused by bunyavirus infections (which by definition may or may not include hantaviruses) are indistinguishable from illnesses caused by several other viruses." [95] This hardly helps to narrow the subject down.

Of just as little help is the otherwise fine writing of Arrizabalaga, who troublingly offers his interpretation of the relevant classification: "the hantaviruses are a genus of arboviruses belonging to the family *Bunyaviridae*, which is transmitted by means of mites and mainly hosted in small animals and humans." [96] This is the only reference to mites anywhere in the scholarly literature about sweating sickness, so we can thankfully dispense with that reference. Also, while the general note about "small animals and humans" is accurate if unusably vague, his note on the precise taxonomy is off. Hopefully more clearly stated is that bunyaviruses are in truth types of arboviruses, with one exception, and that is the hantaviruses. In fairness, Arrizabalaga gets most of the details correct, and indeed is one of a quite small number of writers who also summarize the work of others into this strange disease, the others including Bridson, and Carlson and Hammond.

Despite these early issues, however, there exists potent research into the hantavirus group, including attempts to narrow down matters sufficiently that only a precise virus within that genus can perhaps be identified as the best candidate for sweating sickness after all. The key works are those by the international team of Colleen Jonsson (an American microbiologist), Luiz

Figueiredo (a Brazilian virologist), and Olli Vapalahti (a Finnish infectious disease specialist). Their combined expertise lies in explaining hantaviruses more succinctly, and while they admittedly devote very little to British history, their work is essential to understanding this virus group. The other key piece in this section is that of physicians Thwaites and Taviner, with microbiologist Gant, all working in Britain.

Since even the taxonomy of certain viruses pertinent to this discussion seems to have its own issues, the place to start is with Jonsson et al., then, since their work can help make the classification more approachable. While the key issue is to ascertain what ultimately lies behind sweating sickness etiology, and while such is most likely traceable to a single virus, and while how that particular virus is classified within the whole larger world of viruses, not knowing how to classify it as such can only leave serious readers wanting. Accordingly, this first article fortuitously serves as a definitive history of hantaviruses, and the first striking detail is their apparent newness, or at least the rather recent time period in which they were first identified, isolated, and more fully described.

Hantavirus hemorrhagic fever, with renal symptoms (HFRS), is one of the two key diseases appearing in the context of hantavirus research. The other is hantavirus pulmonary syndrome (HPS). Noteworthy already is how different the expectations of these diseases will be based just on their names alone, even diverging in terms of the quite different bodily systems targeted. Both have rodent hosts, though the relevant species also differ, and each emerged in entirely different parts of the world. To begin with HFRS, then, it used to be known as Korean hemorrhagic fever, since roughly 3000 Americans and their allied Korean troops were exposed to it and became ill during the Korean War from 1950 to 1953. Even this piece of the historical puzzle often goes overlooked: the only other writer to even mention the war as playing a part in

understanding viruses is someone who up until now has seemed more associated with arboviruses instead: Bridson. The illness contracted by those military personnel was "a haemorrhagic virus disease, leading to kidney failure and a 10% mortality rate. It took 20 years (1956 – 1976) of work before the causative virus (HFRS) was isolated from the lungs of striped field mice (*Apodemus agrarius*, the rodent host) that were common in the Hantaan River region of Korea." [97] Indeed, that region would give the genus of viruses its name. Bridson further refers to the roughly 150,000 cases of infection which occur annually in East Asia, citing an overall mortality average of approximately 5%, and that the disease is typically associated with rice planting, though Bridson makes no account of how nearby mice must spread the disease, nor in what form (tactile, aerosol, contact with droppings, or something else), nor whether infected humans pass it on to other humans, nor if all these infections arise strictly from some contact with *A. agrarius*. What proves even more interesting is his subsequent direct claim that "this specific virus (HFRS) has no connection with the English sweating disease." [98]

Jonsson et al. maintain that 1978 actually marked the discovery of the connection to *A. agrarius*, and that in the 1980s, the rat-borne Seoul virus (SEOV) was also isolated, another hantavirus. Getting closer to Britain, they refer to nephropathia epidemica (NE), a European disease which is a milder form of HFRS and which was first described earlier, in the 1930s, though this is caused instead by Puumala virus (PUUV), carried by the bank vole, *Clethrionomys glareolus*. In this last case, the "cyclic population variation of bank voles in the region (mostly Scandinavia, and other parts of western Europe, but, interestingly, not within the British Isles) had a profound effect on the risk for humans to acquire PUUV infection," [99] to cite one fairly recent study of these rodents and their related hantavirus. So already there are at least four hantaviruses, none with clear links to the British Isles, and moving about with the help of

different species of rodents. To obtain a clearer picture, then, it will help to consider the map used by Jonsson et al. Fortunately, this map is global and also account for the three key diseases discussed in this article: HFRS, HPS, and NE, the latter of which has never received any scholarly advocates as having been responsible for sweating sickness. HFRS has a high preponderance in China, with some outbreaks also in South Korea and in Japan, but the disease remains restricted to East Asia, and nowhere else. HPS, meanwhile, appears only in the Western Hemisphere, clearly matching the notion of "New World" viruses and diseases, with the largest rate of infection found in Brazil. Finally, Europe is only afflicted by NE (though the Russian Federation appears with a mix of HFRS and NE), and while the northern nations of Sweden and Finland have the highest rates, the British Isles have no shading. They are gray in this map: if the information herein is accurate, then England seems wholly free of hantaviruses. Even the extensive classification chart included in the article by Jonsson et al. has no branch at all extending into Britain. So while their work offers non-specialists likely all they would ever wish to know about hantaviruses, the initial conclusion can only be that sweating sickness clearly either arose from something other microbe, or that it somehow managed to completely disappear during the ensuing centuries.

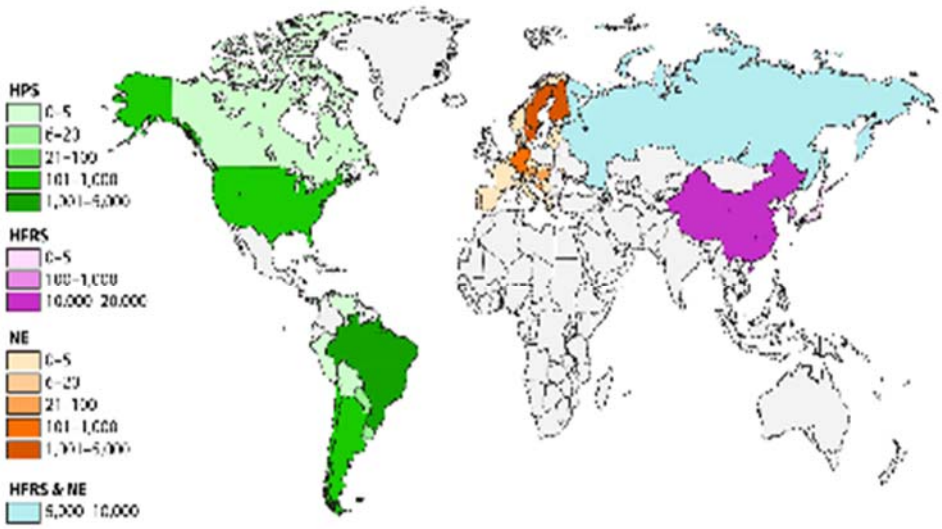

FIG. 3. Geographical representation of approximate hantaviral disease incidence by country per year. (Courtesy of Douglas Goodin, Kansas State University; reproduced with permission.)

(Map 1: Global Incidence of Hantavirus Diseases, from Jonsson, Figueiredo, and Vapalahti, "A Global Perspective on Hantavirus Ecology, Epidemiology, and Disease," p. 416, and reproduced with permission)

Thus far, most of the explanation has focused on the disease HFRS. Moving to HPS, then, we may be on slightly surer as well as clearer grounding, since "the etiological agent of HPS, Sin Nombre virus (SNV) was identified within weeks of the Four Corners outbreak." [100] This marks the main divide between HFRS and HPS: not just different diseases brought on by different viruses, but quite different geographic sources as well. The incident referred to occurred as recently as 1993 in the "Four Corners" area of the southwestern United States. It also benefits by having been considered by a mix of researchers: McSweegan, Thwaites et al., Arrizabalaga, Bauman, and Bridson, in addition to Jonsson et al., refer to it and try to place it within the appropriate historical context, regardless of whatever conclusions they draw about sweating sickness. To summarize the 1993 incident, a "rapidly fatal illness was reported in 17 young adult Navajo Americans." [101] These men presented at a local hospital with fever, myalgia (muscle aches or pains), and typically some respiratory distress, which would not seem to make sense with something that actually had a mortality rate of 70%. This alarmingly high fatality

"was caused by a catastrophic failure of the lungs, in which increased capillary permeability allowed fluid to fill the alveolar sacs." [102] Various species of rodents were trapped near Navajo lands, with the deer mouse strongly suggested (genus *Peromyscus*, a strictly "New World" rodent species, and thus not found in Britain), as some 30% of these mice were infected. The environmental effects helping to enable this outbreak are explained below, since such are part of a broader consideration of climatic effects and the possibility of toxins rather than microorganisms lurking behind the Sweat. In the meantime, the number of cases in 1993 in the area reached 110, with the overall mortality rate dropping notably, but remaining still high at an overall 51%. And there is still no notion yet of even this disease, despite its similarities to sweating sickness, having either come from or becoming able to return to Britain.

Broadly speaking, and after initially considering these two diseases, the genus of hantaviruses divides into New and Old World viruses "due to the geographic distribution of their rodent reservoirs and the type of illness (HFRS or HPS) that manifests upon transmission to humans." [103] Even so, these viruses "share high homology in the organizations of the nucleic sequences and exhibit similar aspects of their life cycles." They display quite similar structures and infect healthy cells in equally similar ways, and the further philosophical question regarding whether viruses are literally living things or are mere bits of RNA and DNA in need of hosts to "live," will remain outside the topics raised in this dissertation. Regarding the viruses themselves, Jonsson et al. show and describe the actual structure, including how these viruses invade healthy cells. The current consensus is that "for all of the viruses in the family *Bunyaviridae* that each genomic RNA forms a circular molecule that forms by base pairing between inverted complementary sequence at the 3' and 5' end of linear viral RNA." [104] Hantaviruses are able, then, to infect a wide variety of cells, including epithelial (cell lining of

vessels and body cavities) and endothelial (cell linings of blood and lymphatic vessels, and the heart), as well as macrophages and lymphocytes (all are white blood cells tasked with immunity, with the former engulfing and digesting invaders and the latter killing them and appearing as NK, T, or B cells), and finally follicular dendritic cells (also part of the immune system, found mainly in the lymph follicles). These are all the types summarized by Jonsson et al., and the preponderance of victim cells which comprise essential parts of the immune system should make hantavirus-related diseases seem quite disconcerting. The viruses infect "via the attachment of the viral glycoprotein to the host's cell surface receptor(s)." [105] The resulting mortality rates cited include 12% for HFRS and a much higher rate of 60% for HPS.

While this wealth of detail intended mainly for microbiologists may seem overwhelming, the work by Jonsson et al. becomes most revealing when it shifts from microbiology to geography. The distribution and accompanying epidemiological considerations of diseases pertaining to hantaviruses "have been considered a consequence of the distribution and natural history of their primary rodent (or insectivore) hosts." [106] These researchers cite work into how those living hosts can maintain the viruses, as well as circumstances which enable and promote transmission and infection to other living organisms, prevalence of the viruses around the world and how other potential host species may be affected, and the possible evolution of these viruses. They also agree with a key detail of the work of Bridson, in that at least with the "Old World" variants, hantaviruses are quite largely dependent upon "rodent reservoirs," which includes not just their populations but how those populations in turn depend on climate and habitat. Further, both Old and New World hantaviruses share more in common in that "the dramatic fluctuations in rodent reservoir populations and the prevalence of hantaviruses in these reservoir hosts give rise to localized, sporadic, and unpredictable HPS outbreaks." [107] Outbreaks of HFRS may be

more predictable, yet this passage about localization and the sporadic nature of HPS occurrences may help the case for linking one of the HPS viruses with sweating sickness: after all, the Sweat was indeed largely localized, not just to England (and not the rest of Britain) but also to particular communities (although this does not resolve the dispute between those who argue that it was mainly rural and those who hold that it tended to be urban); and the Sweat also erupted rather sporadically, since the dates of the five epidemics was frustratingly irregular, as expressed above in the Table compiled by Carlson and Hammond. These two factors together give the sweating sickness a high degree of unpredictability, both for those who lived through it and those of us who study it.

Such is our introduction to hantaviruses. The others who advocate them as candidates for sweating sickness can now be more fully evaluated. "The exact cause of death is not entirely clear," [108] historian Paul Hunter reminds us, summarizing the four earlier outbreaks before emphasizing the 1551 occurrence. Dyer, too, emphasizes this final epidemic. For Hunter, what matters about 1551 is that the last visit by the Sweat "occurred in an England that in many ways was rather different from the nation affected by previous epidemics." [109] His main emphasis in this regard has to do with recent yet crucial social changes which "had serious effects on the quality of life." This is not to say that England had become somehow more susceptible to sweating sickness, using similar logic to that sometimes employed to argue how poor harvests and famine had, for example, apparently helped the Black Death of the fourteenth century have such widespread and disastrous effects. Rather, Hunter refers to how sweeping social changes "had serious effects of the quality of life" [110] in England, more so than in other parts of the British Isles. This entails some drastic oversimplifying, but in essence, the economic effects of the likes of land enclosures and a growing populace (which itself suggests lower death rates from

sweating sickness during the sixteenth century than from plague in the fourteenth), along with unintended effects like inflation, displacement, and impoverishment of the former residents of the monasteries, may have encouraged at least one disease to revisit an old target and run rampant again. This does not explain its apparent final demise, however. And interestingly, Hunter is the only writer to suggest a different genus, that of enteroviruses, as perhaps the explanation. Such viruses (which include those responsible for polio, for example) can lead to diseases which present with sweating, muscular aches, and with a tendency to operate more during warmer months, and to affect adults more than children (though this may reverse the stereotype some may yet have about polio). Enteroviruses also thrive when able to achieve transmission via the charmingly named fecal-oral route, typically in locations which may give less attention to personal hygiene and which are also associated with food. Yet for Hunter, the Sweat remains "as mysterious today as it has been for more than 500 years." [111]

Enteroviruses prove a distraction here, then: while hygiene will receive its due course below, the main advocates for hantaviruses really include Frederick Holmes, and Thwaites et al. Various other writers have surely alluded to them, and offered suggestions about what might make a hantavirus and sweating sickness logically meet, so to speak: Arrizabalaga, Bridson, Heyman et al., even Jonsson et al. While Holmes, too, admits that "the cause of this interesting sickness... has never been identified," [112] that does not mean that its identification must be impossible. Like so many of these contributors, he also summarizes those who have gone before, both during the sixteenth century and his own, and notes that whatever the Sweat was, it came on suddenly, and "was characterized by fever, headache, malaise, limb pain, flushing, rapid and forceful heart beat, delirium and profuse sweating throughout the illness." [113] Writing similarly, Thwaites et al. refer to the work of Caius during the final epidemic, and his six "signes

or tokens (sic)" to identify the disease: myalgia or headache, abdominal pain, vomiting, further headache or delirium, cardiac palpitations, possible paralysis with agonal breathlessness (which may have seemed the most frightening of symptoms to the Tudors, since they may have known no other diseases to present this way, nor of any methods to encourage the resumption of breathing, which might have reminded them of the older idea of disease coming from miasma, or tainted air); and likely death, with sweat, assumedly, occurring largely throughout the disease's course.

Both works then return to twentieth century scholarship. For Holmes, there is an appeal in how "hantaviruses are uniquely associated with small rodents... (and) apparently, particular hantaviruses have long associations with particular rodent species." [114] Also, transmission of hantaviruses and their associated diseases "is horizontal, not vertical, with the viruses spreading in excreta and infection documented in the laboratory between caged rodents up to four metres apart." This would certainly help explain the apparent ease of transmission. Likewise, Thwaites et al. concur that a non-human mode of transmission was likeliest, pointing out that the summer preponderance of Sweat epidemics would seem to strongly suggest an infectious agent with a mammalian or perhaps avian reservoir. Because of this key attribute, they contend, "viral hemorrhagic fever therefore seems most unlikely." [115] If that is true, then HFRS would be ruled out by definition, leaving HPS as the only hantavirus disease possible. Indeed, that potential may offer a strong case for the reconsideration of the arbovirus argument instead.

Holmes maintains that hantaviruses likely migrated during the previous century, the fifteenth, from Siberia into Russia and then into the Baltic nations. As for England some decades later, "increasing trade in timber and furs brought infected rodents to England through its eastern ports... Wylie and Collier, and Creighton, state that spring and summer weather was wet, warm,

or both in England before the five epidemics, favouring the growth of rodent populations." [116] An interesting side effect of this, he argues further, is that the poor in England, "living in miserable rodent-infested conditions," would have a better chance collectively to avoid contracting the Sweat during each of its epidemics, with the rich suffering in a greater ratio, matching the old reports of how well-off men seemed most at risk. This might further seem to suggest some form of immunity, which has not been discussed by modern researchers (one has to know what a disease is prior to assessing whether one might develop immunity to it or whether an inoculation might be developed, after all), though this does disagree with some primary source reports, perhaps most famously with Cardinal Wolsey, who is known to have contracted the sweating sickness more than once. Even during the nineteenth century, Hecker noted that "there was no security against a second attack (of sweating sickness); for many who had recovered were seized by it, with equal violence, a second, and sometimes a third time, so they had not even the slender consolation enjoyed by sufferers in the plague and small-pox." [117]

Similarly, Thwaites et al. consider the rodent thesis as well. While they do not evaluate the question of an ultimate geographic source of the sweating sickness, they argue that a rodent vector is likeliest. Further, if it was truly a viral pulmonary disease, "then its clinical and epidemiological features seem most closely to resemble those of the hantavirus pulmonary syndrome," [118] though they recognize how new such an identification really is: their first article emerges just four years after the outbreak in the United States in 1993. What is also interesting about their work also is not just that are they the only ones who publish a follow-up to it, they also do so very quickly, with a second article, specifically about sweating sickness, appearing just a year later. [119]

There are other details worth mentioning regarding this work by Thwaites, Taviner, and Gant, as well. First, while previously discussing bacterial diseases, it was noted that McSweegan, in his favoring of anthrax, communicated with Thwaites in particular, and that both agreed on the basically rural modeling that seemed to have existed for sweating sickness. This notion also shares much in common with Dyer, who himself sticks to his contention that road progression would have been encouraged during the busier summer travel, including the summer of 1551, more so than during spring. There is also some agreement with Dyer in that Thwaites et al. favor "a viral disease with a rodent vector," [120] the very passage Dyer quotes in his own work. Additionally, and noteworthy for its attempt to combine elements of the major arguments, they refer to Dyer and his argument "that the causative agent of the sweating sickness was spread by human-to-human contact *as well as* initially through a zoonosis or an environmental vector." [121] They also show some inclination to agree with Dyer, who in turn agrees with the earlier work of Wylie and Collier, that a virus via arthropods with rodent hosts was the explanation. Finally, the focus with Dyer on case-by-case studies from parish records "might equally be interpreted as reflecting a simultaneous clustering of small mammal populations," [122] as would typically be found easily in smaller communities. While Dyer ultimately remains hedgy about hantavirus, Thwaites et al. show themselves much in its favor.

As for remaining issues with the hantavirus argument overall, Heyman et al. clearly state that the early modern virulence of sweating sickness "cannot be explained by genetic variation in present-day hantaviruses." [123] They do briefly consider one "Old World" hantavirus, though the technical problems with virus taxonomy have already been considered, including its confusion even for the specialists. Their attitude would also preclude the possibility of an "older" version mutating into a "newer" version, perhaps including one to afflict Tudor England, which would

also contradict the argumentation of Casadevall et al., who insist microbial virulence is essentially an emerging, changing characteristic. Another problem is the reminder offered by Holmes himself, who, while noting how wet weather enables more food which enables increases in rodent populations (indeed often in a logarithmic expansion, which will then typically entail them invading human habitations), yet "human-human transmission of hantaviruses has never been documented." [124] This would clearly limit the spread, if sweating sickness indeed traces back to a hantavirus, which in turn could help explain that regardless of however deadly the Sweat may have been, its ability to infect large numbers of the Tudor populace apparently was never nearly as profound as plague or influenza. Further, "that asymptomatic hantavirus infections occur in humans is quite likely," [125] as Holmes adds: this would make some persons innocuous carriers, which, while it might reduce apprehension about the disease (and perhaps did five centuries ago, too), does not resolve the retrospective diagnostic question yet. Finally, Jonsson et al. note that while early modern England indeed suffered "a rapidly fatal viral infectious disease" known as sweating sickness, "a review of these epidemics suggested that HPS does not match the English sweating disease completely." [126] As already revealed via an examination of their excellent work about hantaviruses, HFRS also does not emerge as a contender for them for the Sweat, even with the near global geographic distribution of the relevant viruses and the organisms able to act as vectors for them. As they put matters quite succinctly, "we note that hantaviruses have not yet been detected in Great Britain." [127] And yet strangely, "the emergence of zoonotic pathogens remains one of the great unsolved mysteries in biology," [128] even though currently a pathogen gets identified on average roughly every eighteen months. Mutation, as Casadevall et al. indicated, is the main trait of many microorganisms, though that very mutability, which typically comes via basic environmental adaptation, hardly

helps the current task, since it logically leaves the possibility of the Sweat indeed having been unique and thus unrepeatable historic event, as Carlson and Hammond suggest.

It is issues like these which work together to keep matters murky for those of us seeking the most rational and plausible arguments and conclusions. Sometimes historians have already noted the difficulty in retrospective diagnoses simply due to the terminology, as with "plague" and "pestilence" and "ague," as Slack, Theilmann and Cate, Hecker, and Sloan have all pointed out within their own writing. Blending the themes of taxonomy with divine retribution, John Kelly notes how "in the Middle Ages, both plague, a biblical term used to describe an affliction associated with divine displeasure, and pestilence were applied to all kinds of epidemic disease," [129] not making matters easier. Continuing in a similar line of thought, historian J. A. McSherry writes that "theories of aetiology have been the subject of much conjecture and debate," [130] which should hardly seem surprising at this point. And yet sometimes the theorizing becomes almost too easy, as though sweating sickness should be expected to either remain dismissible, other than within occasional references to Tudor medicine, or easily filed into a convenient category. McSherry continues: "there is little doubt that Sweating was a specific entity, the absence of respiratory symptoms making confusion with influenza unlikely, the absence of a rash eliminating typhus or smallpox from the differential diagnosis and bubonic plague being too well known at that time to go unrecognized." [131] This reads like a simplified version of what this entire dissertation is indeed supposed to be about: elimination of less likely candidates via the examination of all known primary and secondary sources until the most plausible logical case is left, but this description by McSherry is too easy. He seems unjustifiably confident in making such claims, even if he uses the same model of eliminative deduction. As he concludes matters, "the manifestations of Sweating resemble nothing so much as an acute encephalitis, leading

modern authors (he mainly cites Wylie and Collier, though in fairness, his own article appears well before much subsequent scholarship) to suggest an arbovirus as the likely cause." The arbovirus notion also remains in how Thwaites, Taviner, and Gant respond to the roughly concurrent work of Dyer, since they all cite rodent hosts, but arthropods as a crucial necessary ingredient for the spread of the disease.

Table 3: Viral Candidates for Sweating Sickness: Influenza, Arboviruses, Hantaviruses

	Author(s)	Similarities of Symptomology; Benefits to Sweat Research	Issues with Matching to Sweat
Influenza (*Orthomyxoviridae*)	Creighton; Gottfried; Purdell; Hope-Simpson	fever, myalgia, headache	It is difficult to match known outbreaks with Sweat epidemics.
Arboviruses (*Arbovirus*)	Wylie & Collier; Carlson & Hammond; Arrizabalaga; Dyer; Theilmann & Cate	flu-like symptoms (fever, headache, malaise)	Infection may lead to other distinct diseases, including encephalitis, dengue fever, and yellow fever. These viruses require arthropods as vectors.
Hantaviruses (*Bunyaviridae*)	Thwaites, Taviner & Gant; Jonsson, Figueiredo & Vapalahti	fatigue, fever, muscle ache; possible nausea, vomiting, diarrhea, abdominal pain; HPS generally is considered the closest match to sweating sickness etiology.	Hantavirus pulmonary syndrome only attacks lungs. There is no known evidence of hantaviruses in Britain. It has a longer incubation time.

Signs and Symptoms for Sweating Sickness (based on primary accounts of Fracastoro, Vergil, Hall, and Caius, and summarized by Carlson and Hammond):

> sudden onset; full course from roughly one to fourteen days (diaphoresis followed by asthenia); fever; rheumatic pain (typically in back and extremities); abdominal pain; tachycardia; possible vomiting, diarrhea, bleeding; possible delirium; possible severe headache; possible multiple organ failure

Note: Other authors favoring viruses generally but who do not necessarily take specific positions on exact types include Bridson; and Heyman, Simons, and Cochez. Another issue for viral explanations generally is that in the case of arboviruses and even more so of hantaviruses, many such viruses and their related diseases are known and categorized, so the usage of any in particular as explanations of sweating sickness must necessarily become more detailed.

C. Possible Culprits: Toxins and Climatological Effects.

Thus far all traditional disease models have been explored, even in the light of changing notions of what constitutes disease and how it is defined. More specifically, the previous two sections about bacteria and viruses have attempted to explain every existing microorganism which has been offered as a plausible candidate to explain the sweating sickness. In order to account for every conceivable logical explanation, though, we must now turn attention to a possibility not considered by scholars nearly as often as microorganisms, though it can be another source of illness. The main question now becomes whether the cause of the sweating sickness might be traced plausibly and convincingly to some form of toxin instead of to a microorganism, and whether such a toxin was introduced intentionally or not.

Since unintentional poisoning of some kind may overlap with some of the prior research into bacteria and viruses (both broad types of microorganisms have numerous species, of course, which can prove quite toxic or even fatal to more complex species), let us consider intentional toxicity first. The most appropriate place to start then is with Tudor attitudes to poisoning, and as historian Krista Kesselring describes matters, such could be rather alarmist. Her own article is quite succinct, including a reprint of the precise text of the *Acte for Poysoning* of 1531, which, taking earlier medieval attitudes into account, made poisoning treasonable and punishable by boiling. One wonders if the more established traitor's death of drawing and quartering might have been preferable, since boiling was apparently deemed most suitable if it could be made to last as long as possible. So now the act was deemed a greater offense than before (not just homicide but a crime against the crown), and would accordingly be met with a harsher response. Two details stand out with this torturous practice, however. First, as Kesselring explains, only two persons are known to have been put to death in this manner, boiling, during the entire Tudor

period. The first was Richard Roose (or Rice), a cook in the employ of John Fisher, Bishop of Rochester, who allegedly was attempting to eliminate the bishop for his ongoing theological defense of Queen Catarina of Aragon, still married at that time to Henry VIII (in February, 1531, just before passing of the *Acte for Poysoning*). Two persons did die from this attempt, and while Fisher would later be executed along with Thomas More in 1535 for refusing to swear the oath required by the *First Succession Act* which would favor Elizabeth over her elder half-sister Mary as next in line for the throne, he of course survived the fateful dinner. The second person condemned to boil was one Margaret Davy, about whom even less is known today. She apparently was a serving maid punished for poisoning her mistress, though details about either woman remain quite scant.

It might be said, then, that either the Act for Poisoning must have worked as quite a deterrent upon the public, or that poisoning was widely perceived as a counter-productive or unreliable method of dispatching someone. Further, any attempt to link such overt attempts on someone's life must contend with a lack of descriptions of how the victims of Roose and Davy actually died: none appear in Tudor historiography, and these three persons are simply listed as dying of poison. Even a retrospective disease diagnosis at least benefits from primary descriptions of signs and symptoms, and now we do not even have those. Thus the precise toxic agents in these cases thus remain unknown, and there is likewise no known scholarly effort to ascertain these poison's identities. The truly fascinating part of this largely forgotten piece of legislation, meanwhile, having been repealed quite early during the reign of Edward VI, in 1547, is that "a statute of 1512 removed the benefit of clergy from men who committed murder, felony on consecrated ground, or robbery on the king's highway or in houses with people present." [132] So poor Roose, who might have otherwise benefitted from this traditional legal loophole as the

bishop's employee, never had any more chance than did Davy as a less protected commoner. The timing for this is curious, too: if the goal of the legislation was truly to take away a traditional legal protection for religious officials, then 1512 was hardly a time when Henry was combating the Catholic Church, and the removal of benefit of clergy dispute seems more worthy of another royal Henry – Thomas conflict, that between Henry II and Thomas Becket in the twelfth century instead. In 1512, Martin Luther had yet to even post his criticisms of the Roman Church, and as late as 1521 Henry would be named *Fidei Defensor* by Pope Leo X in large part for writing against Luther in favor of the Catholic sacraments. Ultimately, the reasoning behind the act about poisoning almost two decades after that 1512 statute and then its repeal after the death of Henry VIII seems to have been more of a move to reinforce secular authority at the expense of religious authority, so that "by declaring fatal poisonings treason, Parliament could render offenders ineligible for benefit of clergy without creating a new precedent or violating an established, heavily defended custom." [133] This may have helped enable the execution of later religious rabble-rousers, but since poisoning does not appear in their accounts, this is not the place to address them.

Such was the relevant legislation, and so poisoning appears to have never been much of an issue for the Tudors. Thus far, historian Adam Patrick has been the only other scholar considered besides Kesselring to seriously hypothesize some type of toxicity regarding the sweating sickness, as with his notion of food poisoning resulting from bacterial contamination, though his own assessment of it was that it must have been innocent and accidental rather than murderous. And he wrote his simplified summary of sweating sickness a half century ago. Arrizabalaga, for example, referring to such research, notes that "Patrick suggested that the sweating sickness was not an infectious disease but the result of mass food-poisoning by fungi or

some other contamination of cereals." [134] Still, this is merely part of Arrizabalaga's own summary of sweating sickness research: Arrizabalaga does not specifically favor toxins or indeed any other group category for identifying the disease. Similarly, Hunter, who like Patrick tends to favor bacterial explanations, notes that Patrick refers solely to food poisoning, perhaps cereal crops invaded by some kind of fungal-based toxin, but does not otherwise speculate on particular grains, nor on any culpable fungus. Heyman et al. suggest a nineteenth century reference that perhaps a certain fungus, *Claviceps purpurea*, might be to blame, as it is known to affect grasses and cereal crops and "has been linked to cold winters followed by wet summers." [135] Yet rye is a likelier victim of this ergot fungus, and rye was less popular in England than on the Continent during the Tudor years, as they also note. Patrick, weighing in with the toxicity question, suggests that rheumatic fever might have been a plausible result of such, though perhaps since he remains quite cautious regarding a source, he is at least as cautious also with a more specific resulting disease.

Perhaps toxicity arising from another source might be to blame, then. The notion of food toxicity from other than spoilage from age or improper or inadequate storage and preservation appears not to have been a notable focus of Tudor understanding, at least not from a scientific perspective. Fracastoro proves a bit of an exception in this regard, at least briefly, as he warns that "furthermore, there are certain foods whose frequent use causes this or that sort of infection," [136] though alas, he does not offer much by way of specifics. And his noting of the risks of excess was hardly new; even the ancient Aristotelian notion of all in moderation was well understood throughout the Middle Ages and into the early modern period. As for consideration of food toxicity, historian Sally Hickey confronts some of the side effects of such considerations in her own work on agriculture, livestock, and a thus far wholly unconsidered

notion herein: that of witchcraft. While no one has suggested witchcraft as even connected with sweating sickness, much less as its literal cause (and Hickey herself never mentions the disease, even though her writing deals wholly with the Tudor and Stuart periods), "toxic and poisonous chemicals commonly present in the plants of the fields and foods of the community's livestock, eaten unwittingly or through excessive hunger, caused symptoms which led the owners to believe that their livestock had been overlooked or bewitched." [137] Further, as she describes matters, "the changing nature of agriculture not only placed stress on the populace but also influenced the toxic and chemical components of the plants in the natural environment," which she refers to as including "fields, forests, ponds, hedgerows and village gardens." [138] This is the sort of change warned about by both Nash and Arrizabalaga, as well as Heyman et al.: not just alterations in our understandings of disease, but perhaps in diseases themselves, which have been shown in many cases to adapt and even alter to continue infecting new host organisms, which in this case might even be extended to understanding why sweating sickness really only affected England, so far as is known (its brief excursion into mainland Europe after its 1528 return to England notwithstanding). But that is getting ahead of matters at this stage. Again, there is no link known between witchcraft and sweating sickness.

There could be links between witchcraft and agriculture, however, and contemporaries had to wonder at what factors might enable changes like those mentioned above. The masses knew that prices for basic foodstuffs had increased during the reigns of the Tudor monarchs, and they certainly knew years of poor harvests and inclement weather when they occurred. It might take a genuine blight, though, for folks to consider the possibility of witchcraft as culpable. As Hickey summarizes, "perhaps the most common cases of animal bewitchment were those in which the animal gradually wasted away before the eyes of the owner," [139] all the more alarming

since livestock were a principle source of wealth, as they had been throughout the Middle Ages. But this refers to individual cases, not masses cut down as from some spreading contagion. And like these animals, the wasting away of land, particularly arable used for food production, might also elicit charges of witchcraft. The point is not to detail witchcraft and compare it to other magical practices like sorcery, but rather to note how it was widely understood as a possible source of maleficence. And in the case of Hickey, for someone researching the more esoteric aspects of Tudor history, her never mentioning sweating sickness would seem odd only if witchcraft itself was perceived as connected to the disease. Even in a larger compilation of witchcraft practice within legal, political, and anthropological perspectives, historian Alan Macfarlane never mentions sweating sickness either, nor, for that matter, poison of any kind. His focus remains mainly on Essex, and while a reader can review scores of accusations and their resulting decisions, witchcraft appears nowhere in this detailed text as dealing with poisoning. And while the early modern period would witness the witch hunts and purges, Sim reminds us that from a medical perspective during the Tudor period, "the local witch or wise woman would have been the only general practitioner available to most people" [140] for some basic health care considerations. For the present, they might have remained generally trustworthy. Even Paracelsus admired them, claiming no less than tutelage from some of them, though he admits to such without giving names or locations. Witches, or at least wise-women, will be revisited later as in conflict with physicians regarding issues like licensing and education and standards of practice, but ultimately there is nothing within primary or secondary sources to suggest anyone in the sixteenth century believing that the sweating sickness had its origins in the quite old and traditional practices of witchcraft.

Moving on from witchcraft, then, another potential source of toxins receiving little attention in primary accounts is hygiene, and the stereotype is often of unwashed and ripe-smelling Tudor persons of all social classes going about their affairs. Hygiene has already briefly been mentioned in the context of burial practices, but it certainly had effects on more common activities. And despite stereotypes, the Tudors had at least some understanding of preventive measures to help ensure personal health, some of them hygienic. We have met the work of Dobson before, and she addresses hygiene mainly in the form of potable water. Though not among the traditional non-naturals, "water, like air, was a natural resource which varied in purity and quality but it was also a commodity that had to be acquired, fetched and often paid for." [141] It took early modern logistics, too, in order to ensure "a steady flow of fresh and uncontaminated water. Water pipes were often broken; drains, channels, gutters and sewers had to be scoured and cleansed; wells maintained, pumps mended," [142] and this required coordination and funding. The only contemporary physician Dobson refers to is Andrew Boorde, and she quotes from a 1547 treatise of his admonishing readers to rise in the morning, remember (pray to) God, and wash hands, face, and teeth, at a minimum, using cold water. Further, it was advisable that "no common pyssing place to be about the house." [143] Boorde himself wrote his book partly "that sycke men may recuperate theyr health, and whole men may preserue theym selfe frome syckenes (with goddes helpe) as well in Phisicke as in Chierurgy." [144] He seemed to want to give good advice to all readers, and his "Dyetary" focuses on general health, including the six non-naturals, while his "Breuyary" is more about diseases with potential remedies and treatments. He could just as easily condemn, complaining of "seven evils in England," including neglecting of fasting; a prevalence of swearing and heresy; laziness among the young; a specific need of proper training for midwives; the problem of cobblers acting as physicians; the

mutability of too many people's minds; and general lust and avarice, including ill treatment of the poor. As for one disease in particular, when "the swetynge syckenes is in a towne or countree... the people doth fle from the contagious and infectious ayre... a man cannot be to ware, nor can not kepe hym selfe to well from this syckenes, for it is so vehement and so parlouse." [145]

Matters appear to have been worst in and around London. Historian G. V. Poore explains that "the situation was not healthy, because of the marshy surroundings of the city. Ague and dysentery were always present, and were terribly fatal." [146] Additionally, "the streets were filthy without, the houses were filthy within," [147] so there seemed no escape. Yet it was typically felt at the time, via a surprisingly resilient belief which does not appear to have been eradicated even now, that "a most important factor in the causation of disease was the moral conditions of the population, which was very low, and marked by superstition, ignorance and brutality." [148] This is the same sort of attitude already described above in the description of plague, for example, and Tudor ethics as a whole will receive its due attention in a later section about Tudor belief structures. Yet this also exhibits a certain historical bias which goes beyond ethics, and even if one could establish that the Tudors were somehow less moral than other historical groups, ethics and epidemiology remain two quite distinct fields of research. No one deserved to contract sweating sickness simply from being perhaps less clean and socially presentable than might have been hoped, an attitude which was slowly increasing in acceptance at the time.

Beyond that, no water source in England or anywhere else has ever been shown to be contaminated with anything that can infect humans with a disease that matches the described signs and symptoms of the Sweat. There is no locale comparable to the Broad Street Pump studied by physician John Snow in the nineteenth century as he isolated a specific source of

cholera, for example. Water could certainly be quite unhealthy to imbibe during the Tudor years and for countless centuries before then, and Poore recognizes that marshy ground could enable the contamination of wells, but this was about the ability of water to carry diseases, and not due to its being a source of toxins of both a non-bacterial and non-viral nature. And even this too remains often stereotypical, as some recent scholarship has confirmed that open water sources in medieval through early modern England to have actually been quite safe and trustworthy. "Regulations for the use of water," [149] as historian John Lee describes affairs, "like those for the market, made particular reference to protection for the poor," mainly since fresh water really "was considered a drink suitable only for this class." Lee criticizes modern historians for diminishing the importance of particularly urban water supplies throughout Britain, noting that even the grand tour by Tudor antiquarian John Leland in the 1540s found that while many had to still rely on wells, streams, and rivers, what they drew from those sources was often potable. The main exception would typically be rivers running through the larger cities, already showing the effects of pollution even from the later Middle Ages. Another criticism of more modern historiography, as pertaining to the usage and management of water resources, comes from the work of fellow historian Dolly Jørgensen, who observes that even the idea of "resources," including water, already has a certain anthropocentric bias about it. Still, as she describes the situation, "three interwoven concerns dominated medieval concepts of river pollution: obstructions (which could interfere with commerce, sometimes blocking trade ships), disease (the theory of which still tended to favor miasma or "bad air") and proper moral behavior." [150] Beyond natural and occasional human obstructions to navigation and thus access (usually "debris and stones," or "weeds and filth"), she describes how an English city's polluters offer a list that "is quite comprehensive: textile manufacturers..., leather workers..., and brewers" [151] tended to

number among the worst offenders. [152] However, it is important to keep in mind that her own study deals specifically with rivers, and not smaller, typically unnavigable streams, or the other older method of obtaining fresh water, from wells. Also, pollution at the time she recognizes to have been understood and responded to in the broad sense, since there was of course no "bacterial revolution" to influence Tudor thinking, and additionally, she makes no further mention of any diseases at all.

Indeed, with towns and cities, "piped water supplies also enhanced the dignity of public spaces and emphasized civic pride, as did other infrastructure such as guild halls, market crosses and street paving." [153] For Lee, actual piped sources, though they required specialist workers to install and maintain, and did not benefit from any influx of wealth for the purpose from either Crown or Parliament and had to thus be covered by local urban residents, those same residents might recognize the chance to further their own relative autonomy in such a manner. Sometimes this arose from simple necessity: Lee himself notes that major new water sources had to be tapped in London by 1430, then again during the reign of Henry VIII in 1543 to 1544, and he also cites new digs in Exeter in the 1490s and Gloucester in the 1540s. New laws attempted to circumvent illegal "tapping" or "letting," though here the emphasis appears to have been more on getting unfair access to water rather than polluting it. Leland himself "described conduits in Bath, Bristol, Coventry, Gloucester, Lincoln, Newcastle, Stamford and Southampton," [154] plus in many smaller towns, emphasizing that these communities had taken measures to ensure access to safe water. More recently, Jørgensen focuses on York, Coventry, and Norwich. Hopefully this combined work of Jørgensen and Lee helps show how at least one stereotype for the time does not merit the attention it still seems to receive.

As for other hygienic considerations, the most direct potential link between hygiene and contagion seems to have been with air instead of water, as Jørgensen notes, citing the older notion of miasma. Ancient writers "believed that plagues or epidemics were the result of breathing in noxious air," [155] historian Louise Curth explains, and we know now that some diseases, within both bacterial and viral categories, can be spread in aerosol form (like pneumonic plague and influenza), even though miasma as a more general theory has long since lost credibility. Additionally, however, "toxic air could be created through the work of butchers, tanners, or farm workers." [156] Reminiscent of the concerns of Nash regarding burials, Curth argues that decomposition of bodily wastes, or the rotting of dead bodies, "was thought to create very dangerous fumes. Other harmful vapors were said to arise from swamps and muddy areas or stagnant water." [157] Burials of victims of disease might be quick and haphazard, possibly influencing the proper keeping of records of such persons, which will be evaluated more fully below. And the situation described by Poore also leaves one wondering who would intentionally even live in London, where "streets were filthy from constant contributions of slops and filth from animals and human beings." [158] But other than the types of diseases mentioned by him and Curth, including dysentery, malnutrition, and the still vague "ague," nothing indicates that sweating sickness was ever believed to number among them.

Perhaps, then, there could be indirect effects, somehow yet connecting toxicity to the onset and outbreak of sweating sickness. While no one has suggested a link among harvest productivity and food prices with acts of witchcraft (alleged or actual), or hygiene with any of these, Andrew Appleby insists that food distribution in Tudor England was rather comparable to now, with the rich benefitting most, though this curiously meant the rich consumed much more meat, sometimes suffering the effects of a very high emphasis on both protein and fat. The rich

could also afford wheat, while the poor relied on oats and barley and rye. During the sixteenth century, social factors contributed to notable price shifts, so if any particular foods could spread disease, perhaps it would be reflected in such changes. [159] To summarize briefly, during the whole Tudor period wheat rose in price by a factor of approximately 4.6, barley by 5.6, oats by 6.0, pulses (peas and beans) by 4.7. Costs of livestock also increased: sheep by a factor of 5.0, cattle by 4.5, pigs by 3.6, and dairy products for food by 3.4. Meanwhile, wage rates increased during the whole period by only an average of 2.7 times, so the need for poor relief legislation should become clearer. If anything, the rich continued to eat well, while the poor were more likely to face malnutrition, even starvation in especially lean years. Appleby also, agreeing with Curth, points out that some other foods were erroneously generally believed to not only be relatively non-nutritious, but actually harmful if ingested, including various fresh raw vegetables (onions, radishes, and pulses like peas), and also recent New World imports (potatoes, tomatoes, though neither of these arrived in larger quantities until later in the period). Still, people of the time were not wholly nutritionally ignorant, either. In his study of the Black Death, for instance, historian John Kelly criticizes modern historians for not connecting the plague to malnutrition, noting that "the profound malnutrition of the Great Famine (starting mainly with the harvest of 1315, the result of poor weather and a "terrible summer," still three decades before the plague arrived in Europe, but "the worst in living memory") may have left millions of Europeans more vulnerable to the Black Death." [160] He also refers to more modern knowledge of how poor nutrition can interfere with appropriate development of one's immune system, leaving the young often more vulnerable to disease generally, though no one has made a case of malnutrition as affecting rates of sweating sickness contraction, which would seem problematic just since those most likely to become ill were social and financial elites.

But none of this suggests any connection or even description of food spoilage, which might in turn enable sweating sickness instead. These changes were socially troubling, but do not say anything in themselves about disease. If these considerations were not enough, the economics of Tudor England is itself a huge area of often contested research, which, to make sense, must necessarily include components such as debasement of currency, effects of land enclosures in preference for increased pasturage for livestock, income and unanticipated social consequences of the dissolution of the monasteries, international trade, land leases, changes in population mobility, and privateering overseas colonies, though both of the latter really only developed when Elizabeth I was on the throne. Indeed, such considerations will in fact be explored in the context of both primary materials and recent scholarship, since consideration of such factors may help us further in the overall assessment of the sweating sickness and its effects, but in the meantime, the statistics offered by Appleby and implications rendered by Hickey and Kelly must be kept in context of the current theme of toxins. There is nothing in the historiography of nutrition for the period to suggest otherwise, though, that certain foods were connected, accurately or not, with particular diseases. "Another great cause of unhealthiness was the diet," [161] writes Poore, writing as far back as Creighton. His main concern is with hygiene more generally, but he makes no other case for linking food, its lack, or its overall quality, with any type of disease other than malnutrition.

Ultimately, though, perhaps historian R. S. Roberts summarizes matters about this agricultural poisoning hypothesis best, in an anthology about modern understanding applied to the history of medicine edited by fellow historian Edwin Clarke. As Roberts concludes, the idea seems sound and attractive: "consequently attempts have been made to solve the problem in the only logical way available, namely to ascribe the five outbreaks (of sweating sickness) to food

poisoning," [162] though no further indication is offered to differentiate between unintentional and intentional food poisoning. The main problem either way, as he himself admits, is that "when such explanations are scrutinized, it is quickly seen that they must fail for lack of evidence." [163] The problem of studying a "localist" disease, to borrow one of Roberts' terms, is that something local must seem inevitably the culprit, and one can hardly fault Roberts for trying: he critiques Creighton for using evidence selectively (indeed, historians have had trouble with Creighton for this in a mix of contexts, perhaps most significantly in his denial of the germ theory of disease even as late as the end of the nineteenth century), though oddly enough, Roberts himself also tries to insist upon sweating sickness having attacked Ireland in 1491 – 1492, even though evidence for that does not stand up to any scrutiny. So this is one area in which both primary accounts and later scholarship have perhaps proved wanting. Simply put, we have no grounds for concluding that the sweating sickness could have plausibly resulted from any form of intended poisoning. There is no motivation: the early Tudor legislation about poisoning meant that even trying to poison a single individual, regardless of social class, would likely be met with a truly dire fate. There is little to no reliability: as Hickey in particular points out, while Tudor persons, "particularly women, had a reasonable working knowledge of herbs and plants, they were unaware of the components of each plant." [164] Hickey delineates the many different chemical compounds within plants, from acids and sugars, to minerals and starches, to compounds often understood even now typically just by botanists. Plants are also not the passive organisms we sometimes consider them, either, having various defensive measures for their own or collective usage. Sim, too, can contribute to this part of the discussion, and not just for her similar emphasis on women, and often local "wise" women, as providing the bulk of care for the populace, but also since even in this increasingly literate society, "Tudor medical books,

especially herbals, seem an odd jumble of unlikely ingredients with no particular method behind them." [165] She further emphasizes that there was a logical structure to such, but it tended to remain locked in the mentality of the restoration of a balance of the patient's humors. Humoral logic continued to hold sway with most, it seems. These writings help reinforce that even trying to apply particular poisonous compounds for nefarious purposes was most likely a haphazard and unreliable practice. Some plants had been known since ancient times to be deadly: foxglove, nightshade, monkshood, yew, elderberry, mistletoe, and many others were understood as quite dangerous if misused, but their application as possible murder weapons could hardly have been subtle or carefully measured. The upper nobility might employ food-tasters, and the Sweat was notorious for attacking noble men in particular, or "to be particularly fatal to the young adults, who should have been best placed to resist disease," [166] and plants such as these listed above, and their components, tend to be obvious, since they either cannot be disguised as food or have noticeable effects on smell, taste, or both. Hickey describes how "alkaloids, cyanogenetic glycosides, cardiac glycosides and saponins are the most prominent plant defences." [167] And, ultimately for poisoning, there is no point: those truly set upon dispatching someone in Tudor England would likely have been better off taking their chances with something more direct like a fatal violent assault, rather than relying on information about plants which, while in many cases well known, tended to remain the preserve of apothecaries and gardeners, who would hardly want associations made between their occupations and poisoning.

If not intentionally introduced poison, then, one could yet argue in favor of the other idea, that of unintentional poisoning, perhaps including from food sources, arising more independently from human intervention, and to evaluate this idea, we must examine agricultural influences surrounding the times of each of the five Sweat outbreaks, and also consider if anything is

suitably known about the climate at those times. This may seem quite daunting, perhaps itself worthy of yet another research project, but we fortunately do have some material to help with this, the last real possibility of some kind of toxin helping to cause this disease. What emerges in the relevant literature is a mix of agricultural and climatic assessments, themselves more forms of retrospective diagnoses in that trying to find evidence of agricultural productivity, much less of climatic factors, is as problematic and contentious as for treating the history of medicine and disease in a like manner. That leaves the final theme within toxicity to be considered: the possibility of climate influencing agriculture in such a way that perhaps some kind of plant poisoning enabled and perpetuated the sweating sickness. Dobson blends these considerations into a cohesive whole, as she observes that "relationships between mortality, food prices and the extreme peaks and dips of mortality, as well as with runs of bad harvests or the overall patterns of short-term variability, have opened up all sorts of important questions about the nutritional consequences of diminished and inadequate harvests. Searches for statistical correlations between temperature and seasonal mortality levels have generated all kinds of epidemiological speculations." [168]

To begin this consideration of climate and agriculture and disease, despite the aforementioned criticism of his refusal to go along with germ theory, Creighton remains important for posing a simple question. Considering that the 1485 initial outbreak of sweating sickness appears to have featured in the short military campaign beginning with Henry, formerly exiled Earl of Richmond and future first Tudor monarch, landing in Wales at Milford Haven and concluding in England at Bosworth with the death of Richard III, and that Henry was known to have recruited various Continental mercenaries who quickly became associated with the disease, then why has there been less focus on those soldiers as potential, even likely if unwitting, sources

of the disease? And what must have happened to them, since the incubation of the Sweat was supposedly so abrupt? Indeed, the area around Rouen appears in this context of having been one of the sites where Henry was doing his recruiting, and is associated with the later Picardy Sweat in 1717. Tantalizing as this link may seem at first glance, Creighton claims this likely soil-bound disease was not the same as the English Sweat. The city of Rouen itself is regarded as the center point here, very close to modern Picardy, the namesake of the "other" sweat, so maybe the mercenaries described by William Shakespeare in his theatrical rendition of *Richard III* as "a sort of vagabonds, rascals, and run-aways" may have unwittingly become carriers of disease. Creighton uses the "soil poison" analogy, focusing on far better known diseases (even in the late nineteenth century when he was writing), like cholera, yellow fever, and typhoid fever to help explain the time gaps between the various outbreaks of the Sweat. It seemed plausible, in other words, to try and explain the latter disease as analogous to these other diseases in its potential dormancy periods, emerging when whatever environmental conditions it required had cyclically reappeared. And yet he also notes that the circumstances of importation of foreigners as in 1485 did not happen with the later outbreaks: no one from across the Channel is described as playing any part in them. Perhaps the source lay dormant in London, and yet the fifth outbreak began in Shrewsbury instead. And climatic conditions are considered in relation to this: "there is not, on the surface," Creighton maintains, "much uniformity in the weather preceding the epidemics of the sweat in 1508, 1517, 1528 and 1551. In the first of these the winter was mild and the early summer excessively hot and dry; in the second the winter and spring were remarkable for drought, with several weeks of intense black frost in the middle period; in the remaining two the antecedent appears to have been an excessive rainfall." [169] As Wylie and Collier summarize matters, while the epidemic outbreaks always ended fairly quickly, outbreak years themselves

"were wet, warm, or both." [170] Heyman et al. notice this as well, suggesting the arbovirus explanation, and recommending research into excess rain, possibly with flooding, together leading to ticks or mosquitoes or both as carriers, which could further account for cooler regions of Britain (such as Scotland and perhaps Wales, neither of which apparently ever suffered from sweating sickness) remaining unafflicted. A comparison appears between the apparent soil dormancy of sweat and plague, another disease known to lay in wait for its right time. Though when sweat disappeared, something may have had to replace it; Creighton favors influenza in this regard, while Wylie and Collier prefer arbovirus.

Still, Creighton's assessment could only hope to make estimates of whatever conditions the sweating sickness may have preferred for its periodic returns, something impossible to ultimately determine unless the disease itself is known. And while we have encountered the work of many other researchers reappearing in this section, this time it is their insights into climate and its potential effects on agriculture, food, microorganisms, and diseases, which has to be evaluated. Could, in other words, something about climate during or chronologically near to the five outbreaks exist which could help explain the disease itself? A suitable introduction to such, then, comes from the work of Casadevall, Fang, and Pirofski and their explanation of microbial virulence. "A defining feature of infectious diseases is changeability," [171] they write, "with change being a function of microbial, host, environmental, and societal changes that together translate into changes in the outcome of a host-microbe interaction." If any or all of these complex variables get altered, then, an infectious agent may become more or less so. Curiously, while they mention English sweating sickness just once, their very next sentence after mentioning it notes that "global travel in the modern world can rapidly spread pathogenic microbes, but what is less obvious is that travel may also enhance virulence." [172] While it seems

like they had the sort of intercontinental travel we have quick and inexpensive access to now, it might also be interpreted to refer to the high degree of travel nonetheless experienced by the often highly mobile Tudors, maybe even of hired troops who had to travel to their destiny.

Dyer and Bridson have already mentioned how the Sweat may have been spread by human mobility within England. And Slack, writing in a different context from before, mainly economic, concludes partly that "there two powerful forces making for change over most of the (Tudor) period: population growth and inflation." [173] Demographic growth yielded a population that Slack argues was overall younger than previously, and that further, "demographic changes also affected population mobility." [174] He cites increases in vagrancy, the cause of which he also blames partly on the dissolution of the monasteries with monks and nuns left to fend for themselves, but more so on land enclosures to increase British wealth through its chief export, wool. As for the land becoming more fenced in, "once enclosed, such property could be turned into sheep runs or farmed more efficiently without regard for the communal customs of the manor." [175] Health reasons aside, what irritated the commoners about enclosure was how the practice tended to encourage land ownership among fewer hands, with the extra effect of the land divisions leading to evictions of humans in favor of more sheep. Whichever the case, human mortality increased notably after each bad harvest for the period (he discusses from 1500 to the early years of the Stuart period). Also, "grain was certainly scarce in some localities in 1520-21 and 1527-28 (the latter preceding the fourth sweating sickness outbreak)... but we do not know whether it was starvation, malnutrition or some independent epidemic disease." [176]

Only a few scholars have proven willing to consider climatic effects within the sixteenth century. Slack is needed here yet again, this time contributing to the same anthology in which Appleby described major changes in Tudor food prices, and arguing that broadly speaking, the

1530s, and the years from 1566 to 1585, consisted of good harvests. "Bad or disastrous harvests," meanwhile, "occurred in 1520-21, 1527-28, 1545, 1550-51, 1555-56, 1586, and 1594-97," [177] and further, "virtually every bad harvest appears to have been followed by a period of high mortality," though he does not identify the precise sources of those mortalities yet. One can immediately notice that 1527 and 1550 are the predecessor years to the fourth and fifth sweating sickness outbreaks, the ones typically also defined as the worst. Fracastoro, too, notes that "in the year 1528, there was first a winter of south winds and much rain (in England), and in the spring many rivers overflowed their banks." [178] Yet "such variations, as well as the inadequacy of the sources before 1560, make precise comparisons between mortality crises in the 16th century and other periods impossible." [179] Perhaps more importantly, though, "plague epidemics did not follow harvest failures," [180] and yet "within towns plague, like famine, was particularly severe in the poorer suburbs." [181] And one area in which Carlson and Hammond, for example, agree with Dyer, is in the former noting that "a common feature of each epidemic was a summer peak in incidence that occurred in years with moderate winters and heavy rain... in two of the epidemics, 1528 and 1551, crop failure resulted from excessive rainfall," [182] similar to the poor harvests mentioned above by Slack. Additionally, "the 1508, 1528, and 1551 epidemics ended during the summer season, whereas the epidemics of 1485, 1517, and 1529 (really part of the 1528 epidemic: 1529 marked just when it became the only outbreak to escape Britain and invade continental Europe) lasted into early fall and winter." Malnutrition, bad housing, poor hygiene, and proximities of humans, rats, fleas, and ticks could all contribute to illness. Though even Slack concludes that, including within the Tudor period, "the history of climate is scarcely less contentious than the history of plague," [183] so the Little Ice Age, for example, was likely little to blame.

Interestingly, perhaps the most vocal proponent of climatic effects, including a potential influence with sweating sickness, arises from the voice of Hoskins, which may seem odd considering that he was an economist and not directly a historian. Right away, he expresses his frustration by lamenting that it seems "incredible how little attention has been devoted by English economic historians to the importance of the annual fluctuations in harvest yields," [184] which affected disease, mortality, even demography and legislation. For Hoskins, the missing piece is how good or bad harvests would affect economic growth: using the year 1550 as a divider, he argues that 29 out of 70 harvests were "good" from 1480 to 1549, and then presents the same ratio for 1550 to 1619. The other researchers relevant to this discussion of climate have likewise been encountered previously. Among them is Bridson, who actually offers his interpretation in reference to the 1993 event in the United States. While we have already considered that virus in the context of understanding the hantaviruses in general, "this outbreak was explained by the fact that heavy winter rains had led to a record crop of pine nuts and a consequential population boom of the deer mice that fed on them." [185] Tracing the history of pine nut collection as a food source is highly problematic, but several Old World species are known, and they were likely to be growing in the British Isles since ancient times. Regardless, the point here is that if sweating sickness was indeed viral and relied upon a small mammalian vector for its spread, then here is a quite recent account of how that spread could be (and in this case truly was) spread with an increase in the food supply for those small mammals. This "cash crop" of pine nuts was harvested by the local Navajo, who "were infected through rodent faeces, saliva, and urine," found in proximity to the nuts themselves, and further, "there was evidence of deer mice invading houses." [186] For Bridson, then, as he links this recent biological history from North America back to Tudor England, "it seems probable that three factors control the epidemic

cycles of the 'sweate': the prevailing climate; the size of the rodent population; and the level of immunity in the human population." And this last factor in turn will certainly vary depending on overall health and levels of nutrition, affected by the poor harvests reported in the primary sources. Hoskins, while mentioning sweating sickness on occasion, never argues for a link between climate and illness, at least not this illness. After the 1517 outbreak, "between 1519 and 1529 we get a remarkable pattern of three bad harvests in a row (1519, 1520, and 1521), then get good harvests in a row (1522 to 1526 inclusive), followed immediately by three more bad harvests," [187] the last two of which witnessed the fourth outbreak. The year 1528 he argues was a slight improvement over 1527, but a compounding effect may have left a weakened populace such that disease "aggravated the misery." And further, "excessive rain seems to have been the cause of these two bad years." [188] Bridson notes the excess rain, too, citing recent research in the arena of global climate change to help understand sweating sickness, an application that those who study climate probably did not anticipate. As he describes it, evidence from ice cores, enabled by the Greenland Ice Sheet Project, suggests that "falling temperatures persisted throughout the 15th century, and did not rise until the second quarter of the 16th century. The sustained drop in temperature was accompanied by a considerable rise in rainfall, bringing periodic flooding to many parts of the country." [189] For them, this might also explain how viniculture essentially stopped throughout Britain at about the same time, by approximately 1400 at the latest. Independently of such an industry, however, the extra rain would have enabled the larger rodent populations. For Hoskins, however, the main effect of poor harvests was neither malnutrition nor epidemics as such, but rather the effects on the British economy. So while each Tudor monarch he refers to assumed the throne "in a good harvest year," it remained the case "that a failure of the wheat harvest (the central grain at the time) drove up all the other food-

prices," [190] the same effect already noted by Appleby in his study of price fluctuations for the century.

Returning to Carlson and Hammond and their CCHF argument, who maintain that the timing of the outbreaks is further suggested by environmental as well as human social factors, as well as transmission routes, we can note that three factors in particular for them may help explain the final 1551 outbreak which they emphasize (as does Dyer): "(1) the Reformation, with the downfall of the regular clergy and the dissolution of their religious houses and large land holdings; (2) the dramatic population growth in England during the sixteenth century; and (3) periodic famine caused by crop failure." [191] A mix of these, they contend, could offer the appropriate context in which the disease could emerge, though not all of these three criteria can truly account for the earlier epidemics. The Dissolution was underway by the time of the fourth epidemic, in 1528, but would not be finished until some years later, and nor can the English version of the Reformation be seriously held up as an explanation for that epidemic crossing the Channel into, conveniently, other lands which were in fact also loosing themselves from the Roman Church. Also, referring to an increase in human population while also discussing disease in any historical setting easily runs the risk of begging the question, in that the relevant issue might become: did this increase unwittingly encourage the transmission of diseases, since innumerable diseases of course tend to thrive and spread more readily when more hosts and victims are available? Alternatively, would the logic go in the other direction, so to speak, and the issue instead be: if the Sweat (and plague, and any other diseases), which purported to have such high mortality, really broke out in such dramatic fashion in several dated epidemics, then how could that same population grow so much in the first place? Something must be getting left out of the explanation.

Most other considerations of possible climatic influence have already been explored in the prior sections about bacteria and viruses. Some other factors are mentioned in the academic literature in other contexts, such as the work previously mentioned of Kelly with the history of early modern plague and the city of Leicester. The others who have discussed actual plague, Nash, Theilmann and Cate, Slack, and Gottfried, have been mainly interested in describing the disease and its effects, Kelly adds something else to the ongoing conversation, pointing out knowledge we might take almost for granted now but which was likely little understood at the time. "There can be no wonder," he writes, "that when the burning heats of summer and autumn penetrated the decomposing masses of animal and vegetable matter, hundreds of the population should be periodically swept away by the outbreak of a pestilence." [192] Alas, "hundreds" is a very vague estimate, and nor does Kelly identify any particular other pestilence in this context, though there is some sense here of the problems with certain types of hygiene as discussed previously by Dobson and Nash, though they also spoke of burials as well.

If the rodent connection can be elaborated on further with the interspecific transmission of diseases, Bridson contends that extra rain in spring and summer enables increases in rodent populations, and not just the increases in food supplies, as when he discusses the pine nuts in the American Southwest example. These increases in British rainfall "could have happened in the years 1485, 1508, 1517, 1528, and 1551, when conditions may have been particularly favourable." [193] If rain encouraged rodent population growth, it would have "led to rodent invasions of houses and dwellings, increasing the indoor viral load from their excreta." To his credit, Bridson tries to account for problems with his own explanation, in that his assessment of climate for disease years may be "distorted by the fact that two potential epidemics (of sweating

sickness) did not take place" as his data suggested, which would have included approximately the years 1495 and 1540.

Another interesting detail regarding climate and sweating sickness is that some of the other researchers who favor viruses as the culprit, like Bridson, are the other key contributors to the logical dialogue about virus-sweating sickness mutual interaction. Even Caius, in his time, noted in his consideration of prevention that his precautions should be addressed during spring, "that the humours may be seatled, and at rest, before the time of the sweting, whiche cometh comonly in somer, if it cometh at al." [194] Heyman, Simons, and Cochez can be considered anew here as well, telling readers that "the English sweat progressed from West to East, in the opposite direction of influenza epidemics and it appeared in summer, while the plague was typically an autumn / early winter event." [195] The authors further note that other nations, as during the 1529 extension of the outbreak so favored by physician John Christiansen, may have suffered more from outbreaks of typhus and plague. As careful scientists, they try to account for how perhaps various other factors, like English climate (they mention excessive moisture and fog, for example) to English lifestyles to the infamous mercenaries employed by the future Henry VII have all been blamed for the disease.

Similarly, Jonsson, Figueiredo, and Vapalahti explain how the ecology of various viruses, including the hanta- group, in their reservoirs of rodent carriers, "depends upon complex interactions among competing drivers, including climate and landscape / habitat." [196] What could have occurred in the case of Tudor England would have included a series of "mast years," each of which "is a year when abundant nuts of forest trees accumulate on the ground and therefore provide abundant nutrients for forest rodents." A high population density of rodents, and corresponding epidemics, correlates, these scientists write, and even can be predicted, by

higher summer temperatures two years in advance and higher autumn temperatures one year in advance. The two-year gap covers when flower buds develop, while the one-year gap accounts for the development of seeds. Without this masting, rodent populations only vary modestly, not truly enough to enable notable increases in the transmission of rodent-borne diseases to humans. Such minimal variation "does not enable the efficient spreading of the (hanta-) virus." Still, the article considers colder northern winters in Scandinavia (the apparent terminus of spread during the 1529 expansion), as well as rodent losses via predation, but actually makes few references to Britain. And the other factors affecting likelihood of rodent to human disease transmission "are related to the structure of human settlements, occupation, and human activity." [197]

Carlson and Hammond also have an interpretation of this effect on smaller animals, though with a quite different conclusion. For them, hunting by humans, legal and illegal, may have inadvertently contributed not just to the 1551 outbreak but also that of 1528, based on the aforementioned crop failures resulting from bad weather. Desperate human families may have turned to poaching even potentially infected deer, or at least those deer which might have been infected with Carlson and Hammond's culprit of CCHF, or Davidson's and Gilbert's culprit of LIV. "We suggest that poor harvests put extreme pressure on wild animals that could be easily hunted as sources of food, and that it was through this mechanism that the CCHF virus enzootic cycle was broken," [198] as they phrase the issue. They also point to an apparent decrease in British populations of hares and rabbits, perhaps themselves killed off by disease, though these researchers refrain from speculating as to which disease might have been responsible. But then they also refer to more recent work with CCHF which indicates that overpopulation of these smaller mammals seems to increase CCHF transmission. Further, the deer hunting hypothesis might even help explain how the primary Tudor sources often emphasize how sweating sickness

seemed to favor young men from the nobility and emerging gentry. Carlson and Hammond again "suggest that epizootics of CCHF virus in England originated in the upper classes from the popular sport of deer hunting: we propose that primary infection sources included tick bites and exposure to infected meat." [199] Heyman et al. concur with a key part of this, too, noting that the disease affected mainly "wealthy, upper-class males," and generally the more "middle-aged, professionally active section of the population," [200] and Gilbert also describes how deer can maintain higher populations of potentially infected ticks than even brown hares or red grouse can. "Professionally active" is a problematic phrase to apply to the Tudors generally, however: strictly speaking, the "professions" which existed at the time were still considered the traditional medieval European ones of medicine, law, and theology. And as there also existed a much larger merchant and trade class during the sixteenth century throughout not just England but most of Europe, it remains difficult to ascertain just to whom Heyman et. al may have been indicating. Still, they do try and account for the Sweat afflicting certain members of society. This might even also account for the direct person-to-person spread hypothesis discussed by them as well as by Arrizabalaga and Dyer, as members of human social groups tend to prefer the company of members of their own, so to speak. Yet the logical appeal of the deer hunting explanation is that already expounded upon in the previous section on viruses: stretching the case to argue that consumption of diseased meat suffers from both the problem of not being accounted for in any earlier sources (the Tudors knew that food could be poisonous under certain circumstances, as seen), as well as for entirely different bacterial or viral explanations, such as those causing foodborne diseases.

To conclude this broad section considering so many aspects of toxins: intentional or accidental, climatic or chemical, Nutton and her own historical research fit perfectly, as she

refers to a nineteenth century renewal of interest in the early modern ideas of Fracastoro, since his own explanation "involved possible planetary influences, bad air, humoral imbalances, earthquakes, and fleeing rodents, as well as contagion and seeds – in short, all the traditional data of the plague investigator." [201] In a letter in 1551, Fracastoro considered the English sweat but used the notion of *principii* rather than "seeds," and further seemed to approve of explaining infection by means of "extremely subtle vapors." We have now considered all of these "vapors," subtle and overt, and there is one final type of evidence in need of consultation before we can start to make a stronger case for the true culprit behind sweating sickness.

Table 4: Toxins and Climatological Effects for Explaining Sweating Sickness:

	Author(s)	Potential Benefit to Research	Potential Drawback to Research
Toxins			
fungus (*Claviceps purpurea*)	Patrick; Heyman, Simons & Cochez	As with *B. anthracis*, it remains easily found and can be studied further.	No primary accounts suggest toxicity as a source for Sweat.
polluted or poisoned water	Jørgensen; Lee; Dobson		No known waterborne illnesses appear in primary accounts. Quality of water varied throughout Tudor England.
other toxins	Kesselring		No specific toxins or poisons are suggested. Severe penalties for their usage in Tudor England probably acted as a deterrent.
witchcraft or sorcery	Hickey	easy explanation	No known associations or legal ties exist between witchcraft or sorcery with sweating sickness epidemics.
other diseases (dysentery, malnutrition, "ague")	Poore; Curth	Primary accounts describe Sweat epidemics sometimes following years of poor harvests.	As with plague, many of these other diseases were understood in Tudor times as discrete.

Note: The key issue with suggesting toxins as explanations for sweating sickness is that since no specific toxins are suggested in the academic literature (nor in primary sources), no matching can be done with signs and symptoms of the disease itself, making a retrospective diagnosis even more challenging if not impossible.

Climatological Effects	Dobson; Slack; Bridson	Some information does exist for the Tudor years, including years of bad weather and/or poor harvests.	Those suggesting climatic effects as contributing to Sweat pose it as a related factor but not a direct cause.

D. Another Research Option: Parish Records and Wills.

The objective behind trying to utilize sixteenth century personal records like parish data and wills would be to apply another discipline into the study of sweating sickness. Thus far we have examined the work of historians, and also anthropologists, physicians, health educators, and microbiologists, and now is the time to consider the field most often consulted for the study of diseases: epidemiology. Borrowing from the work of Bauman again, it is crucial to keep some basic terminology in mind, regardless of which type of study one opts to pursue. While most types of such studies will be ignored in this work due to their impracticality (many epidemiological studies must be done with live patients and ongoing cases), some may yet remain as genuine options. Before getting there, however, we can consider some epidemiological terms relevant to the study of any epidemic and its effects:

- incidence, the total of new cases of a given disease, with an assigned area, sample population, and timeframe;
- prevalence, the total cases, which included both the new ones represented by incidence, plus existing cases;
- endemic disease, which is one that occurs with some regularity within a particular region or population, often the case with plague, for instance;
- sporadic disease, one which afflicts only a few scattered victims;
- epidemic disease, meaning one which occurs with greater frequency than an already established average; and
- pandemic diseases, which occur on multiple continents at approximately the same time. Thus, even the primary sources indicate that sweating sickness may have been defined as pandemic with the 1528 to 1529 outbreaks. It may also generally be classified as sporadic, since it tended to afflict only comparatively small segments of the English population, and also perhaps endemic, though the intervals between outbreaks admittedly seem a bit random. What is needed truly to pursue epidemiological research is more information regarding incidence and prevalence.

Typically, epidemiologists work much closer to the present, and some of the most revealing studies are ones in which patients can be studied, treated, observed, followed, and even interviewed, but since all the known patients of *Sudor anglicus* are long dead, this would seem to pose some formidable problems. Fortunately, epidemiologists Ruth Keogh and D. R. Cox note

that one particular type of research in their specialty might assist, known as a retrospective case-control study. This approach "provides a powerful method for studying rare events and their dependence on explanatory features," [202] and is used most often to consider incidence of disease, with the added benefit that it can be applied historically. Their usage in studying "rare outcomes" already sounds like a possible connection to something as unusual and mysterious as the Sweat, and such a study also would attempt to determine the number of exposed persons within certain populations at certain times. We would have to establish known populations for England as best as possible for each of the five outbreaks, but if those could be known with some accuracy then we might be apply to rely on a straight-forward approach. [203] As Keogh and Cox continue, "the simplest type of exposure (study) is binary, so that any individual is either exposed or unexposed." [204] Case-control studies compare two groups, then, and with the sweating sickness we might even have two binary options: either compare those afflicted with those who were not, regardless of survival outcome and as suggested by the quote above, or compare those afflicted with to those who died from it.

Fellow expert Leon Gordis also elaborates on case-control studies, which "can be carried out quickly... (and) are also valuable when the disease being investigated is rare." [205] Further, these studies are non-experimental by design, though a drawback exists for studying sweating sickness in that parish and other records from Tudor England do not offer something more comprehensive like actual exposure rates, and these would have to be induced from a mix of known causes of death and populations. While the purpose of such a study is ideally to obtain "information on both exposure... and disease outcome," [206] we would likely have to contend with a relative dearth of relevant data as Thwaites, Taviner, and Gant mentioned previously that would make key scores like case fatality rates difficult if not impossible. Indeed, with these

concerns in mind, it is actually historian Gottfried, and physician Christiansen, who hold out the most hope for the utility of completing some form of cast-control study. Christiansen deals with the 1528 outbreak as it extended into 1529 after crossing the Channel, and he refers to Gottfried, who "presents a thorough discussion of the methodological problems in connection with the use of testamentary records (like parish records and wills) for demographic purposes." [207] As for Gottfried, he also notes that while wills do not list causes of death, neither on the Continent nor in England at that time, they nonetheless offer tantalizing clues. "The major sources," he informs us, "are probated wills and letters of admonition or administration; three lay subsidies and a poll tax; and a sixteenth-century muster." [208] He writes further of women in the registers as typically being single or widowed, while "females are clearly underenumerated in the testamentary population," [209] and while this might offer an interesting link to the allegedly greater fatality of sweating sickness among men, it similarly does not help much with cause of death or population. Gottfried's main focus is with genuine plague, however, and he ends his study at the year 1530, the time of a plague outbreak, curiously right after the fourth case of sweating sickness. As for the latter affliction, however, Gottfried's attitude remains skeptical generally, since "while it (sweating sickness) received considerable attention in the narrative and medical records, its demographic impact was minimal, and actually less significant than the influenza epidemics of the 1420s." [210] He further refers to the chief disease import from the New World, syphilis, which made its debut in England roughly concurrently with the Sweat, but also like it, "had little real effect on mortality differentials." Still, for Gottfried, "later medieval England was afflicted by many diseases, including dysentery, smallpox and influenza, but plague... was by far the most significant." [211] As for Christiansen, an increased study in wills for 1529, especially in Germany, might offer more hope for a case-control study, though such would

offer no new insight for the effects of sweating sickness during the previous year, nor for the other outbreaks. He also favors using a crisis mortality rate (CMR) as a measure, or a ratio of deaths in a particular year, compared to a working mean death rate for "adjacent" years, typically five to ten in each direction in time from the studied year. This would offer a prevalence score, so long as it could be shown how many deaths occurred, and how many of them resulted from sweating sickness. Wylie, Slack, and Dyer have all similarly used CMR rates, which have to be selected by the researchers, though Christiansen insists that no universal consensus exists about this measure, partly "since this parameter is based on a yearly death rate," [212] which unfortunately seems impossible to track for this disease, partly due to the irregular gaps between outbreak years. Dyer also points out, in a detail noted by Christiansen, that a key factor in dealing with a mysterious disease like this one is not cause of death nor even rate of death, but increases in burial rates during comparatively short periods, perhaps just a few days with a disease which kills quickly like plague or sweating sickness.

So, however catastrophic or minimal the sweating sickness may have been remains a topic for later, and for the present application, any epidemiological approach would require plenty of data, including yearly death rates. The potential application of parish records or wills, or even both, accordingly needs just a bit of clarification before delving into statistics. Parish records reveal details about medieval religious history throughout much of Europe, and were created initially to try and account for very basic personal data. Parish churches date at least to the sixth century, and were roughly local or village churches. Towns and later cities benefitted from larger and more influential cathedral churches, and sometimes collegiate churches, which were daughter churches to the cathedrals but which also often developed further as scholastic institutions. Parishes, meanwhile, remained the home center of worship for most, a broad

organizational category lasting for many centuries. The idea of them keeping records was simple, since it would seem that little organization or literacy was truly required: the local parish priest, with perhaps some assistance, could record births, baptisms, marriages, and deaths. By the later medieval period, causes of death also appeared in many parishes, though such would need confirmation by the local coroner, and the accuracy of such indicators varied widely.

Still, since evaluating cause of death would be of great potential usage here, it is worth noting that there has been some fascinating very recent literature created about late medieval to early modern coroners within England, particularly by historians Sara Butler, and Steven Gunn with Tomasz Gromelski. Butler begins partly by delineating the differences between coroners and sheriffs, noting the latter dealt with broader aspects of law enforcement, while the former were those truly working with sudden deaths, though "the records include also deaths by misadventure, the occasional suicide or death by disease." [213] While her own work is mostly within the fourteenth century, Gunn and Gromelski trace this history further: "coroners in Tudor England, who had been appointed since the twelfth century and had some legal but no medical training, were supposed to hold inquests on all sudden, unexpected, or violent deaths." [214] This single passage points out a variety of issues to consider: that coroners already had an established history; that they were supposed to be adept in record-keeping; and that they were simply unqualified to render judgments on actual causes of death, or at least medical ones, despite having to investigate such. [215] They were law enforcement officers answerable ultimately to the Crown, and would assemble juries of local men to collectively offer a plausible narrative of cause of death, and then the coroners themselves would rule such as homicide, suicide, accident (illness or injury), or even act of God. One of the better known coroner investigations from the Tudor period was the local coroner was summoned to choose local jurors to make an official

inquest into the mysterious death of Amy Robsart, the young wife of Robert Dudley, Earl of Leicester, in 1560, shortly after Elizabeth I came to the throne. For all the spectacle and potential scandal (Queen and Earl were rumored to be lovers, and of course could not wed while the Earl remained married), the inquest ruled the death officially accidental.

More presently, Butler remains more concerned with the medical implications behind these judgments. With disease in particular, "community authorities hoped to exploit the spectacle of a coroner's inquest to issue a public warning that another outbreak had begun." [216] A parish or village receiving a visit from the coroner might have to wait days for his arrival, which could pose problems in the care and preservation of a decedent's body, but that arrival would also be newsworthy. A community being liable, usually in the form of a hefty fine, for the untimely demise of one of its members, remained more of a holdover from the Middle Ages, and it was in everyone's interest to explain causes of death as quickly and succinctly as possible. Yet fear of legal trouble could be exceeded by something else: "what frightened people most about epidemic disease was the lack of control they had over its spread." [217] Butler's own lengthy chart lists the ailments that might prematurely carry off English persons living in the sixteenth century, after she, like so many others, briefly explains how a restoration of one's humors was taken as the key to overall health. The diseases included ague (this time a dated reference to fever, usually, perhaps with shivering), aposteme (another outdated term indicating an abscess or other edema), "blood sickness," cachexia (general weakness and wasting away), debilities, dropsy, epilepsy, "fatal spots," "fester," fever, flux, gout, "hasty spots" (one wonders if they could prove fatal, or if fatal ones might come on quickly), "hot sickness," jaundice, "narrowness," palsy, pestilence (either as a catch-all, or a reference to true plague), pox, sicknesses in the head, chest, or extremities, and these are just a partial sampling. Working one's

way through the contemporary terminology can require miniature retrospective diagnoses just to ascertain what these terms meant.

However, writers like Butler, and Gunn and Gromelski, are not alone in questioning whether the field of epidemiology might help a case such as this, and would seem to gain some support by fellow historians Barbara Harvey and Jim Oeppen, who try to assess morbidity ("disease in a population as distinct from disease in the individual," [218] as they concisely define it in later medieval and early modern Westminster Abbey. Morbidity as an actual measure "tends to be an elusive feature of past societies and more elusive than mortality (of individuals, as with, say, parish records)." This would include the CMR measure already mentioned as well, itself a datum of prevalence. Indeed, Harvey and Oeppen sound quite hopeful, concluding early that parish registers may have helped enable "trends and fluctuations in mortality" from the period in question "with a degree of confidence impossible to conceive of earlier." [219] Burial practices, even with the hygienic and religious overtones having to be evaluated herein, still tended to ensure that burials occurred quickly after death, and so parish records might "provide evidence for the seasonal incidence of mortality in this period."

Regarding parish records, then, the attraction seems straightforward enough. After all, if written records exist which accurately list such basic personal data for Tudor citizens as dates of birth, marriage, death, and, even better, cause of death, then the appeal becomes clear indeed. The timing is also promising, at least with the final 1551 Sweat outbreak, since such record-keeping was usefully deemed official policy starting in 1538, a curious side effect of the English Reformation. Still, the problems of relying on parish records can be summarized as follows. To begin with, as just stated, these records did not become a matter of state policy until 1538, and considering that four of the five known outbreaks of English sweating sickness occurred before

that year, we could only use their alleged greater accuracy for the 1551 case. Thwaites et al. favor this approach, too, observing that "for the 1551 epidemic, more extensive records are available from surviving parish registers." [220] Next, there are numerous such registers available: Dyer, in particular, speaks to this, as much as he clearly favors study of the 1551 outbreak. By the mid-sixteenth century, the original roughly 10,000 parish registers done more informally during the Middle Ages had only about 1200 still covering births, baptisms, marriages, and deaths. Dyer cites some 680 registers ultimately offering "a significant coverage of the year 1551," [221] but there are two key modern problems, even if all 680 of these constitute an accurate listing of those relevant to this research and assuming that all of them could be consulted on site by a single researcher, a daunting logistical consideration. "The first is that sweat mortality as manifested in the registers is unlike that created by most of the epidemic diseases," [222] which we might expect in such a record. The main feature to notice with the Sweat "is not the total number of people killed (often modest)... but rather the concentration of burials over a very short period," [223] typically a week or slightly more. Thwaites et al. also note that the "lack of data makes it difficult to estimate the case fatality rate," [224] an essential component of epidemiological studies, including case-control studies. It is not the dearth of records they cite, but their application in this manner: the case fatality rate compares deaths from a disease to a known population with the disease, not to a general population; it is typically expressed as a percentage or ratio to indicate risk, but in this case, we clearly need much more information. This point has also been highlighted by Christiansen. The second issue for Dyer here "is that of the small parish:" typically each would account for less than 250 persons, and with such small samples, accounting for random variation will typically yield "bizarre results." [225] Such sample sizes are too small to be statistically meaningful, in other words. [226] Third, this dissertation assesses the merits of a

variety of specialists who have clearly varied in their assessment of not only the availability but also reliability of such records; these disagreements apply not just to Tudor historiography generally but to the history of medicine, too. A measure, including a statistical measure, is of little use if its potential proponents disagree about what it allegedly even records and tries to measure. And finally with parish records, as the epidemiologists have explained in this section, even with a case-control study, we need more such data, and more reliable data than we actually have, to be able to compute certain statistical measures. Causes of death even within the same parishes turn out to be often contradictory or vague to the point of uselessness, part of the problem also raised in considering how contemporary coroners went about their work. This is noted by Slack, for instance, who disagrees with Dyer in the relative importance and utility of parish records. He notes that few such records indicate precise death, though many record "the sweat," "the plague," "the flux," and "the spotted fever." [227] These are hardly the meticulous causes of death we would wish for in order to accurately study mortality. Even having access to the records has become problematic, since Bridson offers another concern, as it was found well after the 1538 start of these records "that the paper records were not surviving storage." [228] Efforts were accordingly made to recreate them, back to at least the start of Elizabeth's reign, though "unfortunately, the result of this edict meant that many parish registers restarted in 1558, thus losing all traces of (even) the 1551 epidemic in those parishes." Hope-Simpson draws attention to the problem as well, and while the keeping of such records were "stimulated to better performance" in 1558, "some registers, not only the early ones, are illegible, others defective and occasionally erroneous." [229] And yet "many are beautiful," though he does not further elaborate on whether beauty confers utility for the purpose of medical analysis. [230] Tankard also observes

that "the earliest bill of mortality is from about 1532, but they do not begin to survive in any numbers until the end of the sixteenth century," [231] too late for use in sweating sickness research.

Dobson summarizes the possible benefits of using parish records also, and tries to compare parishes in three counties in southeastern England for a period of roughly two centuries. And while "the burial registers record the final departure of the deceased from this world... they tell us little about the individual," [232] even if often including cause of death (when known) and other "intimate details." For Dobson, the utility of such records has far more to do with the history of population than with specific causes of death. Migration and mobility are instead key features of her work, as briefly mentioned by Casadevall et al. in their assessment of microbial virulence, and by Slack in his primarily economic approach to mobility in Tudor society. Dobson prizes being able to know age at time of death, and this can prove a useful source of data for some studies, though she acknowledges that prior to the first truly modern British census of 1821, "there is no precise information of the age structure of the living population." [233] Since comparing age at time of death for at-risk populations for different age groups is "the primary step for calculating age-specific mortality rates and life expectancy," this puts a major obstacle in the way of much demographic analysis. The problems of population estimates have already been mentioned, though Dobson does offer an optimistic list of the types of written records which might be able to contribute to population studies, including perhaps medical history. [234]

We can see, then, that parish records might have the potential to offer additional sources and further insights into the nature of the sweating sickness, but this does not guarantee that they will enable us to get any closer to a plausible and strong argument along the lines of a working retrospective diagnosis. Logically speaking, there are several problems with the usage of these two kinds of records (including wills, in other words). These issues do not automatically

disqualify such records for research, including even research into the sweating sickness, but since the current project is to determine the logically best candidate for that disease, it is important to keep such issues in mind. Roberts weighs in on the issues strongly, even suggesting that much recent writing about the sweating sickness amounted to "reworkings of the standard evidence with nothing really fresh to say," [235] though in fairness this was in 1971, even before Wylie and Collier really got things going with the earlier work into viruses. Roberts refers to the earlier piece by Hoskins, who "noticed the number of harvest failures and the high mortality, especially in 1557-58 (though this is too late for sweating sickness), and he (Hoskins) found that parochial registers of burials and wills a death-rate four times higher than usual." [236] Roberts goes on to suggest that later outbreaks, often blamed on influenza, may have actually been sweating sickness instead, though as we have seen, influenza is a highly problematic candidate. Also departing from influenza, as already discussed, are Carlson and Hammond, though their work yet considers details emerging from studying parish records, in that "the observation of long runs of burials that were dominated by one sex or the other suggested that groups of villagers were infected virtually simultaneously, possibly during work or social activities that involved one sex or the other." [237]

Another equally hesitant scholar dealing with this issue is John Moore, speaking to the "population crisis" in the Midlands during the later Tudor and early Stuart years. For him, "it is difficult to avoid the conclusion that around one in five of the population in midland England died from a combination of the 'sweat' in 1550-52, followed by influenza, typhus, probably also dysentery, and sometimes plague in 1556-60." [238] He tries to extrapolate some of the work done by Dyer in particular, who had estimated the average household size in England during most of the sixteenth century to have been approximately 5.1 persons, though taking such data mainly

from Norfolk might distort matters some. The real problem for Moore, however, is that most parish records "start in 1558-59 and are, therefore, useless for studying the pre-Elizabethan period." [239] Even if they could prove of more specific usage, "very few early parish registers consistently record the parentage of children christened and buried, so that the incidence of infant- or child-mortality does not assist in the identification of prevalent diseases," [240] and he again lists influenza, typhus, and plague as the likeliest killers of the very old and very young when the contemporary records are not more exacting.

Finally, with wills specifically, and leaving behind more official statements like parish records, still other problems emerge, though in some ways similar to those already mentioned. First, while wills, in the broadest sense, can give genuine insight into a person's values (a major potential prize for historians, in that we usually must disavow such since we can never literally know what someone in another time and place may have thought and believed), all the primary sources about *Sudor anglicus* refer to its alarmingly rapid onset time. Wills are legal documents, "first and foremost a reliable account of mortality," [241] even if causes of death go unmentioned, and "had been introduced into England as an instrument for the distribution of alms," [242] at least at first. While the ease of making binding changes to a will may vary in time and place, it can universally be said that it nonetheless takes minimal time: officials must be summoned, statements signed and sworn, often in the presence of other witnesses like family members, and gathering such together might be quite a drawn out effort in the context of slower communication and travel times such as those prior to at least the twentieth century. It is true that deathbed confessions and wishes have been used in many times and societies for persons to make their final wishes known, though the validity and acceptability of such have also varied widely; a will exists to be a more reliable instrument. Unless, then, a person's lawyer or other

legal official happened to be nearby when one contracted sweating sickness, this timeliness issue must remain a major factor. Indeed, even if such a witness did happen to be within a useful proximity, he or she would seem to have required a strong incentive to approach someone with an illness generally believed to be highly contagious and lethal alike, and the same could be said for loved ones. While someone's last words might be respected if spoken under duress and not otherwise recorded, the whole reason for instituting them as legal instruments is to try and avoid ambiguities and misunderstandings, and this timeliness concern with their potential very late in life corrections is not something easily overcome. Peter Ackroyd, usually sober in his historiographic assessments, seems a bit hyperbolic here, thinking that "a chance encounter in the street, a beggar knocking at the door, a kiss upon the cheek, could spell death," [243] at least with this particular illness. Lorraine Attreed sums up the more rational ideal and one's preparation for death more concisely, no small feat within Tudor England in which Catholic and Protestant objectives might clash, sometimes with the true wishes of the testator becoming blurred. As she describes matters, "wills preserve those elements of the old and new religion which made the most impact on their testators. They also record the extent to which religious elements decreased in favor of temporal concerns and the desire for society to remember the deceased." [244] She notes some 1960 wills consulted for her own research, dating from 1525 to 1588, approximately ten percent of which were left by women. [245] Many persons had their wills created from their deathbeds, and the preambles of wills took on stereotypically Catholic or Protestant tones. "The men and women of Tudor England were, by and large, pragmatists," [246] writes Eamon Duffy in his history of late medieval and early modern English religion. And while the "magical, superstitious, or semi-pagan" tended to remain unusual, "late medieval

religion was both enormously varied and extremely tightly knit," [247] an attitude that seems reinforced during the Tudor years.

Second, even assuming that alterations to wills could be made with such alacrity, there is an overlap here with one of the issues facing parish records: how, ultimately, can we ascertain that persons altering their wills allegedly from concern of having contracted a fatal disease did in fact have sweating sickness, whether they ultimately died from it or not? Attreed confirms that "some of the wills were executed at the last moment, during epidemics of plague, smallpox, and sweating sickness, which accounted for much of the pain and mortality of those whose life expectancy was already short," [248] but knowing that an epidemic has begun may differ substantially from actually being exposed to the offending disease as well as actually contracting it. Life expectancy in any age can also be a problematic measure, exacerbated by factoring in infant mortality as a way of lowering the average, though it is interesting how Attreed mentions sweating sickness here as well, so at least some persons may have benefitted from being able to offer their final testaments despite possible fears of contagion by those still healthy. And maybe they died of other afflictions, and maybe they died not of disease at all but some other causes. Perhaps the suggestion made by Christiansen can help, at least for studying the further effects of the 1529 outbreak as it made its way through the Continent, though even he concedes that only Imperial records from what is now Germany (as with his emphasis on Lübeck and its cathedral records) show much potential to lead anywhere rewarding in this sense. Too little is known presently about how much it may have invaded Poland and the various Scandinavian realms as well. And if we could gain access to more wills in these areas from that exact time then we might be able to argue for an increased reliance on them. Duffy describes the organization of the populace into parishes and the structures of wills as two complex and contentious issues, and

argues overall that those "pragmatic" Tudors tended to try and navigate as best they could through often changing religious ideals and demands. Unfortunately, though, despite how tempting both parish records and wills may seem initially, we must not only proceed very carefully with either or both, but realize that it is likely that very few such of both types of records exist in reliable enough forms to prove useful. Even if one could systematically go through all such parish records for Tudor England, a methodology would have to be created which could establish which were likeliest to prove trustworthy regarding cause of death.

The same could be said for wills, but here the issue is actually compounded since no one has yet to suggest just how many such wills might reflect concerns about sweating sickness, nor even in precisely how many modern nations they might be found. A fascinating example appears in the work of Tankard, having researched changing attitudes among those appearing at people's bedsides when the end was near. One Francis Strangman, a student at Gray's Inn studying for a legal career during the sweating sickness outbreak of 1551, had a roommate who apparently died of the disease. Strangman himself wisely fled until the contagion had passed, returning to his home in Essex, "fearing that he too would die of the disease," [249] as Tankard reports. The interesting details come next: "In anticipation of his death he wrote his own will... (and his) fears were realized: he died two days later, falling ill at ten o'clock at night and dying at four o'clock the following morning," [250] according to later testimony from surviving family members, who seem to have survived the incident with no ill effects. In this case two days proved sufficient time for an improvised will; many had far less. And this will itself included a bequeathal of Strangman's soul "to my saviar Jesu Christ desiring hym of his clemencie that I may haue the fruicion of his glorye." [251] The tone is slightly more Protestant than Catholic, and Tankard herself describes the scene as typical during the awkwardness and risk of the times,

since 1551 was during the attempt by Mary I to bring England back into the Roman fold. Religious minutiae aside, the case of Strangman differed notably from another example chosen by Tankard, of Thomas Seymour, who perished in 1535. During his "final hours he was attended at various points by about a dozen people including his servants, family, friends, an apothecary, a physician, the priest and various 'wemen and maydens' who looked after him." [252] When one was connected and no one fretted about contagion, then one might indeed be well cared for in the last days. Fear of disease alters the social variables.

Still, it is Butler who perhaps sums up the overall concern with parish records most effectively, describing that with diseases in general and including the sweating sickness, "the solution to retrospective disease analysis appears instead in the descriptions of symptoms and disease progress in medical treatises and documents such as the coroner's rolls." [253] Clearly she favors the latter types of documents, though it is important to keep in mind that every component of her conclusion here is based on analysis of primary sources; one must presume that secondary materials must be used to augment what the Tudor citizens themselves had recorded for posterity.

Table 5: Parish Records and Wills for Explaining Sweating Sickness:

	Author(s)	Potential Benefit to Research	Potential Drawback to Research
Parish Records	Dobson; Dyer	Records may list causes of death. They often account for small populations. A high volume of records exists.	Causes of death are often unreliable. The most accurate records do not begin until 1538, many not until 1558.
Wills	Attreed; Slack	Wills may list more accurate causes of death than parish records.	Wills cover earlier years, but often just as ambiguously. Alterations to wills by genuine Sweat victims may have been made too abruptly to be reliable.

Note: The key issue for the usage of parish records and/or wills for helping to assess the influence and effects of sweating sickness is that studying them is a quite large project, considering their number and geographic spread (parish records still tend to be kept at the local parish level, while wills making reference to the disease appear to be far fewer in number), and the accuracy of determining any cause of death prior to the more exacting methods beginning in 1538 remains problematic and often appear recorded as whatever an assigned coroner or religious official decided.

E. Summarizing the Major Argument.

So far we have examined quite a range of arguments, from different types of specialists who have reached different conclusions, each with its own merits. Still, the most compelling explanation for sweating sickness, keeping in mind the potential hazards of retrospective diagnoses, thus far is arbovirus, even a particular arbovirus not previously mentioned, though first we have to keep our tight logical approach and eliminate other contenders. The key reasons for acceptance of an arbovirus thesis thus far include the following considerations. First, the bacterial diseases put forth over the centuries can be systematically ruled out based on two notable principles: they were either known at the time as being separate diseases, or the reported signs and symptoms match poorly with historical accounts.

The bacteriological diseases can then be summarized as having the following more specific issues. With plague, we have a mix of the terminological issue (actual plague, "plague" as described by primary and secondary authors alike, and even "ague," which might likewise have more than one working definition: the writers herein consulted have suggested such disparate illnesses as malaria and typhus!), with the extra concern that this, too, had already afflicted England, and so much of the rest of Europe, that contemporaries recognized that true plague was quite different from sweating sickness. Even in all its terrifying forms, and knowing that their only real succour was avoidance, persons in that earlier century understood that this diaphoretic illness was something new to them, and that it only seemed to affect England, whereas plague has historically proved far less discriminatory, including back then.

With rheumatic fever, one might point out that its fever and fatigue and pains could at least lead one in the direction of sweating sickness, especially if consulting the compiled list of signs and symptoms for the Sweat by Carlson and Hammond, or considers the still useful case

offered by Hecker more than a century ago, this disease is the most difficult of all those mentioned herein to try and diagnose retrospectively, simply due to its symptomology which aligns with multiple other diseases. In other words, fever and fatigue and joint pains can arise in too many afflictions, and even now testing for rheumatic fever remains complex, even for living subjects.

That leaves anthrax, which, in truth and despite its being championed mainly by McSweegan, does have probably the closest descriptive links to sweating sickness among the bacterial diseases, its rarity in two of three forms among humans weakens it significantly. The other form, cutaneous, is also uncommon, yet even so, it presents with cutaneous eschars which, while perhaps formerly mistaken as the remnants of plague buboes or other skin disorders, comprise a feature never found among Sweat victims: they never developed external growths of any kind. The spore longevity of anthrax, its association with the wool industry, and the sloughed tissue which might be mistaken as plague buboes (though only by a poor observer or one who had never witnessed both), do show some promise, we remain left with the problems of poor matching of signs and symptoms, and that most forms of anthrax are rather difficult to contract by humans, even with repeated exposures.

For the viruses, then, the best example with which to begin is CCHF, and while a useful contender and itself an arbovirus, is problematic first since the ticks that enable its acquisition by humans are not found in Britain, nor is there any known evidence to place such ticks in England during the fifteenth or sixteenth centuries, so another member of this genus must be proposed instead. Second, as the work of Jonsson et al. reports, placing hantaviruses in Britain is historically an equally implausible exercise, which makes HPS and HFRS both more untenable as contenders than several of the recent commentators would prefer. Accordingly, the

suggestion of hantaviruses remains compelling and is otherwise largely plausible. Despite the trend begun by Wylie and Collier in 1981, however, and that HPS matches the signs and symptoms of sweating sickness more closely than either CCHF or HFRS do, HPS is a pulmonary disease, a component not found, or at least not described, by those who witnessed Sweat epidemics and their victims.

Speaking broadly of both types of viruses, if we cannot say that any type of such viruses have ever been discovered in the British Isles, and likewise have no historical reason for postulating that they might have been found previously in certain places within Britain, only to vanish later, then it clearly becomes very difficult to maintain the position that they could have any connection with sweating sickness. Dyer is worth remembering here as well, for his observation that the apparent differences among each outbreak of sweating sickness must necessarily make a combined assessment about its origins difficult if not impossible. As for the hosts and the specific diseases, HFRS (hemorrhagic fever with renal symptoms) is carried and transmitted by the striped field mouse, *Apodemus agrarius*, which can be located, even with its disjunctive distribution, only with concentrations in parts of eastern Europe and Asia, with no historical distribution in Europe west of Poland. And HPS (hantavirus pulmonary syndrome) is carried and transmitted by the deer mouse, *Peromyscus maniculatus*, indigenous strictly to North America (thus its relevance in studying the 1990s hantavirus issues in the United States Southwest). With both these small rodent species, there simply exists no known evidence to place it in the British Isles, including during the sixteenth century. And even with the disagreements about the specific path of infection for sweating sickness, these become simply mooted once there is no logical way to conclude for the existence of hantaviruses in England.

The only remaining viral candidate is influenza, and many who cite it give the impression that they just want a quick definition to work with before moving on to their next area of focus. In this group of Sweat scholars we find Purdell ("probably a form of influenza with pulmonary complications"); O'Malley ("which some (have) labeled a form of influenza"); Lawrence Conrad et al. ("the disease was most probably influenza"). What remains important to keep in mind is the main issue overall: the signs and symptoms of influenza simply have a very low correspondence with those of sweating sickness, even though there is no problem locating plenty of influenza in England during the fifteenth and subsequent centuries. And as Gottfried, and Heyman et al. remind us, sweating sickness appears in the primary accounts as lacking respiratory issues or secondary acquisitions of pneumonia; even the latter of these might have been associated at the time with true plague, in its pneumonic and most lethal variant. Also, other scholars like Hope-Simpson, McSherry, and Moore, have noted that the Tudors themselves understood flu and Sweat as discrete illnesses. Influenza was certainly devastating, and we have already encountered its alarming death toll during the late 1550s especially, but we can safely rule it out for further argumentation.

Perhaps Heyman et al. act as the most systematic eliminators of other contending diseases in this regard, as we have encountered them previously noting the major logical problems of Sweat being linked to typhus, anthrax, and even food poisoning, even if they collectively remain cautious regarding other viral illnesses. Among the promising accounts verifying even rudimentary knowledge of different diseases appears in the Johnson letters, this one from Bartholomew Warner to John Johnson during the 1551 outbreak:

> I hoped this other daye by George Graunt to have sent youe Cavalcanti's byll, which I wrott youe of in my lettres sent youe by the said Graunt, but I coulde not gett them in tyme; and wher I wrott youe the 5[0]li to be delyvered [a]t xijs [j]d,

> hit was at xijs ...d, as appereth by the byll which I send yo[ue] herin, with the lettre of advyse also.
> I wolde to God youe wer her in London, for thankes be to God, the sycknes ys nere seassed, for yesterdaye by the report from the Clarckes' Hall ther dyed in London but xj, wherof one of the sweat and one of the plage in St. Sepulchre's parishe, the rest of agues and other dyseazes, which is nothing to be counted. We rest very desyrous to her frome youe.
> The peple murmure of the calling downe of the shilling to vjd, and I feare hit (reference to letter 707). The trewth I cannot lerne, for that I may not go to the Court, and agayne my frend John Lorde ys departed, of whom I might have had some knowledge. And thus after my harty commendacions, my syster's and my wyve's to youe and youres, I byd youe well to fare in God. Frome London, th exxiijth of July, 1551.
> Youres, Barthilmew Warner.
> Add. To the worshipfull John Johnson, marchaunt of the Staple at Calleys, yeve thes at Calleys.
> End. From Barthilmewe Warner at London le 23 July; receyved at Calleis l[e] 25; aunsweryd le last August. [254]

So at this point the disease had ceased, though Warner categorized plague as something other than Sweat, in turn from various "agues" and "other dyseazes," appearing among the economic concern regarding the currency. Moving from this systematic elimination of other diseases, along with the problems already examined regarding arguing in favor of poisons, and also showing the notable research problems in using any combination of parish records and personal wills for support, we can at last arrive at the first part of our working diagnosis, one curiously mentioned initially by someone who offers little attention indeed to sweating sickness itself, and actually writes regarding diseases which affect the nervous system, a strange place already, it might seem, to find a reference useful to the purpose herein. "The only arbovirus endemic in Britain is louping-ill, a tick-borne virus, which is a very rare cause of meningitis or encephalitis," [255] as this scientist writes, though this is all he even says initially, between equally brief entries for adenoviruses and microbiological investigations. This of course is also known as "LIV," and as will be increasingly and progressively demonstrated, becomes the most promising overall contender for the etiology of English sweating sickness. First, arboviruses

make for more plausible candidates than hantaviruses, chiefly for the problem of explaining that the latter appear to have never existed within the British Isles. Second, louping-ill presents with similar signs and symptoms to what we know of the Sweat, and it is important to point out that this matching will never be precise, for two reasons. The first is that we have to rely on primary sources, which in this case can be ambiguous, incomplete, contradictory, or even use different terminology from what we might hope (curiously, these same problems tend to afflict parish records and wills, for that matter). The other is that many of these symptoms, as we have seen, remain common to potentially many diseases: it is only by accumulating a group of them that they being to become more convincing. In the case of arboviruses, there may exist the additional problem of how tick-borne illnesses may progress through various initial signs and symptoms before truly manifesting as the likes of encephalitis, dengue fever, or yellow fever, and clearly none of these three, at least, are sweating sickness (though encephalitis offers some promising overlaps of major symptoms with Sweat, while it tends to lack the hallmark diaphoresis which gives the latter its moniker).

Third, the idea of arboviruses in general is supported by numerous contributors to the ongoing "Sweat-dialogue" if such a term may be permitted, although none of them specify louping-ill in their work; that remains unique to this dissertation. Among these arbo-supporters, though, are Wylie and Collier (who make the initial proposal in this direction), Carlson and Hammond (though this project will ultimately split off from their research, as will be shown later), and Arrizabalaga. Theilmann and Cate lend their support less directly, and as they do not specify arboviruses as such, nonetheless they logically dismiss the hantavirus thesis generally. Fourth, LIV is actually the most promising explanation of the following considerations: ease of accounting for its presence in England, including during the Tudor years; relevance of its

presence among both livestock and more exotic species, also disseminated throughout England and Britain, which can help explain not just why the illness could spread but also help understand why it tended to affect well-off men more than other human groups (especially as in the work of Carlson and Hammond with their thesis about hunting, and that of McSweegan and his thesis about the Tudor wool industry); and its ongoing need for self-perpetuation via both ticks and livestock, especially sheep, but also in how perhaps it could have become dormant for periods of years between epidemics, based on highs and lows of both hunting (particularly of deer and grouse), and the wool industry (affected as it often was by enclosure practice and the rule of supply and demand, mainly on the Continent, home of most of the market for English wool). Of course no one would want "infected wool," as with the thesis proffered by McSweegan involving anthrax, and yet we also know that the Sweat only ventured to the Continent during the fourth epidemic; prior to that, the disease was not exported elsewhere, not even to Scotland or Wales or Ireland, so it proved to be self-limiting, at least with humans. If the notion about a social elite succumbing to Sweat more easily and more often, then the locations of these persons and their already lower numbers compared to the whole population, help build the case further.

There are surely numerous ways for viruses and their related diseases to travel, and there seems little reason why ticks cannot unwittingly join human hosts or non-human vectors during their travels; indeed, part of the scenario for sweating sickness getting to England the first time, in the late fifteenth century, entails its being transported in the wake of Henry Tudor's recruiting efforts. "Pathways include entry of infected vector," to borrow from one recent article specifically explaining how arboviruses might enter Britain, plus "contaminated animal products, live animals and infected humans." [256] The British even maintain a Terrestrial Animal Health

Code to assess similar risks, updated most recently in 2010, though the writers of this particular article do "not set out to assess or rank the probabilities of entry," [257] but rather to consider how such might occur. Viremic livestock might also be introduced, which can include horses, which might in turn have potentially included the mounts of mercenaries and others loyal to the up and coming Tudor cause. Once *in situ*, arboviruses might move about in other ways. As an example, and showing a connection with the work of researchers like McSweegan, Carlson and Hammond in particular (more for their historical thesis than their biological one), "movement of sheep has been associated with dispersal of LIV, a variant of TBEV (tick-borne encephalitis virus)" [258] in Britain. Fortunately, the risk of infection into Britain remains low, though in our own time this is due more to import bans in restricted zones. In the case of Tudor England, luck may have played more of a part instead. Arboviruses are also known to typically have no treatments presently, typically take three to fifteen days to manifest symptoms in a victim, and survivors may or may not acquire immunity to the related disease. So the emphasis here is not that arboviruses in general, or LIV in particular, necessarily invaded Britain at any particular time, but to show instead how it is logically plausible for them to have done so at potentially any time that livestock species or humans crossed the Channel northward.

Other research addresses the question of the actual epidemiology of LIV, including in red grouse, still a popular game species, as it would have been for the Tudors. As reported, "all stages of *Ixodes ricinus* (the tick carrying the virus) feed on large mammals but only rarely are adult females found feedings on small mammals or grouse," [259] so these ticks prefer larger creatures, perhaps benefitting from the often higher mobility of such. In our own time, sheep themselves get vaccinated against louping-ill, and must receive periodic boosters, so one must not conclude either that louping-ill is merely some strange disease from the past. Interestingly,

this same study also "examined the possible role of mountain hares and red deer as potential non-viraemic amplifiers of the virus," [260] yet found no evidence of "non-viraemic transmission" in either of these species (meaning they have to already be infected, carrying the virus in their bloodstream, to be able to infect others through tick bites). It remains interesting how these other species might fit into the late medieval and early modern hunting thesis, too. Even more tellingly, these researchers suggest "that louping-ill virus causes localised population sinks and the grouse population within these sinks are sustained by immigration." [261] The grouse, in other words, die off from this viral disease, a detail which, when added to the researchers' additional contention that winter plays a role here as well, would help justify the multi-year breaks in Sweat epidemics, and interestingly give the appeal for more Tudor climatic data a boost. While no historical data exist for tracking influx of red grouse to Britain during the relevant years, their populations peaking again could have helped the virus and the disease to reemerge. LIV in Britain also remains genetically distinct from similar viruses even found as close as parts of mainland Europe.

Yet "mortality due to louping-ill... is difficult to quantify in the field," [262] as another research team concludes, although some of the sheep, especially lambs, in their own study appeared to have acquired some immunity to LIV during the research, and up to now we have encountered nothing suggesting that one could develop immunity to sweating sickness. Perhaps the primary accounts differ in this regard (again, the only notable case of someone surviving more than one case of the Sweat while presenting with symptoms both times (thereby suggesting a lack of immunity) was Cardinal Thomas Wolsey); or perhaps the disease works differently to some extent in humans, which is true of many diseases but seems less rigorous in an academic treatise. Regardless, this is not a strong enough point to truly logically threaten the LIV thesis.

Much more specifically, the same team emphasizes that "amplification of louping-ill virus... requires ticks to acquire virus from the blood of a host." As with plague, we have here a confirmed insectoid vector, yet also one that makes humans living in close proximity able to transmit the disease to each other via tick bites. The only confirmation issue here is that ancient, medieval, and early modern accounts from around the world that discuss disease outbreaks tend to remain silent regarding small pests like insects. Unless such appeared in truly massive and exceptional numbers, they simply tend to be absent from so many primary materials, so the prevalence of ticks in relation to English sweating sickness, as with the prevalence of requisite fleas in relation to the world's plague epidemics or pandemics, remains an unknown for us.

As a final consideration for the louping-ill thesis at this time, it is worth considering controls, some of which hearken back to earlier historical periods. Interestingly, "understanding and controlling pathogens in complex multi-host systems (as with LIV, and some of the other diseases mentioned herein) is a particular challenge in epidemiology." [263] And while LIV "causes mortality in sheep and red grouse," [264] the focus of this more recent work by some of the same researchers considers a place known as Bowland Fells, a place of some 23,000 hectares (about 57,000 acres) of northern English moorland, of which 763 hectares are modern "enclosed land." One familiar with Tudor history must at once notice the relevance of a land enclosure, which in this more recent case includes areas of common grazing and a mass of protected land "used for sheep and red grouse production." Brown hares, rabbits, and roe deer are also found there. While Bowland Fells does not precisely match the enclosed areas used in Tudor times, the logic was largely the same: protecting land less for environmental reasons and much more so for economic ones, including the exploitation of sheep for wool (and meat) as well as a mix of game species for hunting, in this case, as during the medieval and early modern periods, for the rich as

part of their sport and noble custom. This case also discusses the inoculation processes, with a result that "suggests that sheep are the essential vertebrate host for this virus," but also, that "the length of exposure to ticks had an enormous influence on an animal's serological state," [265] including whether it became ill and if it lived long enough for hungry ticks to continue passing the virus onward. And even these researchers remain very cautious about whether this disease could ultimately be wholly eradicated: with sufficient numbers of hosts it could of course go on; yet with insufficient numbers it might "rest" and go into hiding. Yet it can be specifically identified, since "a genetic tripeptide marker is unique for each virus species within the genus so the louping-ill virus can be distinguished from all other flaviviruses." [266]

So, LIV remains, at least in parts of Britain. It has most likely mutated from its prior form in which it could kill human hosts into something which thankfully no longer bothers us, even though a variety of non-human animal species in Britain still suffer and often die from it, as the accounts of grouse, hares, some wild deer, and some livestock species dying from it into the present attest. One can only surmise why it may have evolved in such a manner to no longer trouble humans directly, though it clearly affects various species in which some humans maintain a vested interest. So in the process of filling in a historiographic blank space within the period of Tudor England, we can say that LIV remains the best candidate for the sweating sickness.

Notes for Part IV.

1. World Health Organization, *World Health Organization, Geneva, CH*, February, 2016, accessed February 18, 2016, www.who.int/mediacentre/factsheets/en/trypanosomiasis.
2. Arthur M. Spickett, "Merck Vet Manual," *Merck & Company*, *Kenilworth, NJ*, November, 2013, accessed February 23, 2016. www.merckvetmanual.com/.../sweating_sickness/overview_of_sweating_sickness.
3. As Bauman explains with plague, "rats, mice, and voles are the hosts... they harbor the bacteria but do not develop the disease (though many larger mammals can acquire plague, including pet and livestock species). In this cycle, fleas (*Xenopsylla cheopis*, the "rat flea") are the vectors for the spread of the bacteria among rodents (Bauman, 508)." The "human flea" is

Pulex irritans, one of six in this genus, and the only one not wholly indigenous to the Americas. Both *X. cheopis* and *P. irritans* can carry *Y. pestis* and also transmit it into the bloodstream of another animal, and even so, some scholars debate the true venue of plague (especially the Black Death of the fourteenth century), and which flea (if any) was ultimately responsible for its highly geographically successful transmission. In any case, the bacteria eventually occlude the esophagus of the fleas so that they can no longer ingest blood, and the fleas then try desperately to obtain more blood by moving to usually mammalian hosts, with the potential to infect any number of new hosts this way. As the larger host animals become infected themselves, "they act as *amplifying hosts*... (italics his) (to) support increases in the numbers of bacteria and infected fleas."
4. Centers for Disease Control, *Centers for Disease Control, Atlanta, GA*, September 14, 2015, accessed February 18, 2016, www.cdc.gov/plague. See also Mayo Clinic, *Mayo Clinic, Rochester, MN*, March 26, 2013, accessed February 22, 2016, www.mayoclinic.org/diseases/plague/basics.
5. Theilmann and Cate, 383.
6. Theilmann and Cate, 388.
7. Paul Slack. *The Impact of Plague in Tudor and Stuart England* (London: Routledge, 1985), 228.
8. Creighton, 311.
9. Hecker, I. F. C. (B. G. Babington, trans.) *The Black Death in the Fourteenth Century*. (London: Schloss, Foreign Bookseller, 1833), 158, italics his.
10. Vivian Nutton, "The Reception of Fracastoro's Theory of Contagion: the Seed that Fell Among Thorns," in Michael R. McVaugh and Nancy G. Siraisi, eds., *Medical Renaissance Learning: Evolution of a Tradition*. *Osiris*, 2, 6 (1990), 234.
11. Sloan, 475.
12. Sloan, 475.
13. R. S. Roberts, "Sweating Sickness in England: an Alternative Interpretation." *Suid-Afrikaanse Tydskrif vir Geneeskune*, 47, 1 (1973), 4.
14. Hecker, I. F. C. (B. G. Babington, trans.) *The Epidemics of the Middle Ages* (London: Trübner & Co., 1859 (1844)), 173.
15. Bauman, 540.
16. Mayo Clinic, *Mayo Clinic, Rochester, MN*, February 18, 2014, accessed February 23, 2016, www.mayoclinic.org/rheumatic-fever/basics.
17. Hecker, "Epidemics," 171.
18. Hecker, "Epidemics," 173.
19. Hecker, "Epidemics," 176.
20. Hecker, "Epidemics," 267.
21. Hecker, "Epidemics," 267.
22. Hecker, "Epidemics," 239.
23. Hecker, "Epidemics," 267.
24. Hecker, "Epidemics," 267.
25. Bauman, 54.
26. Carlson and Hammond, 24.
27. Centers for Disease Control, *Centers for Disease Control, Atlanta, GA*, September 1, 2015, accessed February 19, 2016, www.cdc.gov/anthrax. See also Mayo Clinic, *Mayo Clinic, Rochester, MN*, July 16, 2016, accessed February 20, 2016, www.mayoclinic.org/anthrax/basics.

28. Bauman, 545.
29. Centers for Disease Control, *Centers for Disease Control, Atlanta, GA*, September 1, 2015, accessed February 19, 2016, www.cdc.gov/anthrax. See also Mayo Clinic, *Mayo Clinic, Rochester, MN*, July 16, 2016, accessed February 20, 2016, www.mayoclinic.org/anthrax/basics.
30. Edward McSweegan, "Anthrax and the Etiology of the English Sweating Sickness." *Medical Hypotheses*, 62 (2004), 156.
31. Smith is very specific here, citing 516,573 acres of enclosed land, in 34 counties, accounting for 2.76% of what was available: deforested arable land, so moors, rocky hills, mountains, still extant forests, and marsh are not accounted for in her sources. And rather than focus on disease, Smith emphasizes other aspects of agriculture, the productivity of which was increased during the sixteenth century in England by "two relatively new techniques – the cultivation of 'floating' meadow land and 'up and down' farming." (Smith, 83) Floating meadows referred to lower and moister meadowlands with which to grow winter hay, "which tripled the yield of higher, drier fields and produced spring grass a month earlier," certainly a boon for farmers and their livestock alike. The animals themselves also benefitted from not getting slaughtered "during the lean months of March and April," and also evolved into larger herds of larger members, thereby producing more fertilizer with which to feed arable soils. The up and down system entailed switching between using the land to grow wheat, and using it as pasturage, which had the same effect: "more food for both human and beast." This differed from medieval crop rotation, which kept smaller fields isolated in terms of what each was growing during each year or left to lie fallow so nutrients could have a chance to replenish the soil. The early modern practice kept all the land active, without exhausting it, and this "periodic alternation improved and enriched the soil, increasing the yield and making the land easier to till."
32. McSweegan, 156.
33. McSweegan, 156.
34. Theilmann and Cate, 386.
35. Bauman, 546.
36. Bauman, 732.
37. Bauman, 732.
38. Arrizabalaga, 64.
39. F. Graham Crookshank, "Influenza and the Story of a 'New Disease'." *The Journal of Nervous and Mental Disease*, 49, 6 (1919), 560.
40. Wylie and Collier, 425.
41. Charles Creighton. *A History of Epidemics in Britain* (Cambridge: Cambridge University Press, 1894), 304.
42. Creighton, 304.
43. Theophilus Thompson. *Annals of Influenza or Epidemic Catarrhal Fever in Great Britain from 1510 to 1837* (London: Sydenham Society, 1852), 2.
44. Arturo Casadevall, Ferric C. Fang, and Liise-anne Pirofski, "Microbial Virulence as an Emergent Property: Consequences and Opportunities." *PLoS Pathogens*, 7, 7 (July, 2011), 1.
45. Casadevall, Fang, and Pirofski, 2.
46. Robert E. Hope-Simpson, "Recognition of Historic Influenza Epidemics from Parish Burial Records: a Test of Prediction from a New Hypothesis of Influenzal Epidemiology." *The Journal of Hygiene*, 91, 2 (October, 1983), 295.
47. Hope-Simpson, 297.
48. Carlson and Hammond, 32.

49. Carlson and Hammond, 33.
50. Purdell, 18.
51. Arrizabalaga, 64.
52. Articles written by Arrizabalaga, Bridson, Christiansen, Holmes, Hunter, McSherry, and also Thwaites, Taviner, and Gant, each consulted herein, all refer to the 1981 article by Wylie and Collier. Even when these other specialists disagree with the earlier piece, they acknowledge it as instrumental in provoking the most recent and still ongoing academic discussion of English sweating sickness.
53. Wylie and Collier, 437.
54. Nash, 18.
55. Nash, 20.
56. Roberts, 4.
57. Robert Gottfried, "Population, Plague, and the Sweating Sickness: Demographic Movements in Late Fifteenth-Century England." *Journal of British Studies*, 17, 1 (Fall, 1977), 17.
58. Gottfried, "Population," 22.
59. Centers for Disease Control, *Centers for Disease Control, Atlanta, GA*, February 22, 2016, accessed February 20, 2016, www.cdc.gov/flu. See also Mayo Clinic, *Mayo Clinic, Rochester, MN*, September 29, 2015, accessed February 23, 2016, www.mayoclinic.org/diseases-conditions/flu.
60. Bauman, 710.
61. Eric Bridson, "The English 'Sweate' (Sudor Anglicus) and Hantavirus Pulmonary Syndrome." *British Journal of Biomedical Science*, 58, 1 (2001), 4.
62. Centers for Disease Control, *Centers for Disease Control, Atlanta, GA*, February 29, 2016, accessed February 18, 2016, www.cdc.gov/ncidod/arboviruses.
63. Arrizabalaga, 64.
64. Arrizabalaga, 66.
65. Carlson and Hammond, 26.
66. Carlson and Hammond, 28.
67. Carlson and Hammond, 35.
68. Carlson and Hammond, 41.
69. Marion E. England, et al., "*Hyalomma* Ticks on Northward Migrating Birds in Southern Spain: Implications for the Risk of Entry of Crimean-Congo Haemorrhagic Fever Virus to Great Britain." *Journal of Vector Ecology*, 41, 1 (June, 2016), 127.
70. Lisa J. Jameson and Jolyon M. Medlock, "Tick Surveillance in Great Britain." *Vector-Borne and Zoonotic Diseases*, 11, 4 (April, 2011), 403.
71. Jameson and Medlock, 411.
72. Hughes and Larkin, 430-431
73. Carlson and Hammond, 45.
74. Bridson, 2.
75. Dyer, 376.
76. Carlson and Hammond, 30.
77. Carlson and Hammond, 31.
78. Carlson and Hammond, 36.
79. Carlson and Hammond, 38.
80. Bridson, 1. Virtually unique among authors consulted here, Bridson does an almost comprehensive job summarizing other scholars and their contributions, then moves into a general

history of hantavirus and how its study relates to the English sweating sickness. He also suggests a question which often goes ignored by commentators, in that "the mysterious disappearance of the disease after 1551 has yet to be explained (6)." As he notes, "there remains room for more work and more theories," even though we do not really know where the disease "went" after that. Part of this project, of course, is to show that it is actually still around, but simply a different version of itself.

81. Bridson, 2.
82. Bridson, 4.
83. Bridson, 4.
84. Dyer, 376.
85. Dyer, 384.
86. Dyer, 383.
87. Dyer, 382.
88. Paul Heyman, Leopold Simons, and Christel Cochez, "Were the English Sweating Sickness and the Picardy Sweat Caused by Hantaviruses?" *Viruses* 6 (2014), 159.
89. Heyman, Simons, and Cochez, 159.
90. Arrizabalaga, 66.
91. Theilmann and Cate, 391.
92. Heyman, Simons, and Cochez, 163.
93. Heyman, Simons, and Cochez, 160.
94. Bauman, 735.
95. Bauman, 735.
96. Arrizabalaga, 65.
97. Bridson, 4.
98. Bridson, 4.
99. Gert E. Olsson et al., "Hantavirus Antibody Occurrence in Bank Voles (*Clethrionomys Glareolus*) During a Vole Population Cycle." *Journal of Wildlife Diseases*, 39, 2 (2003), 303.
100. Colleen B. Jonsson, Luiz T. M. Figueiredo, and Olli Vapalahti, "A Global Perspective on Hantavirus Ecology, Epidemiology, and Disease." *Clinical Microbiology Reviews*, 23, 2 (2010), 412.
101. Bridson, 4.
102. Bridson, 4.
103. Jonsson, Figueiredo, and Vapalahti, 414.
104. Jonsson, Figueiredo, and Vapalahti, 414.
105. Jonsson, Figueiredo, and Vapalahti, 414.
106. Jonsson, Figueiredo, and Vapalahti, 416.
107. Jonsson, Figueiredo, and Vapalahti, 420.
108. Paul R. Hunter, "The English Sweating Sickness, with Particular Reference to the 1551 Outbreak in Chester." *Reviews of Infectious Diseases*, 13, 2 (March – April, 1991), 303.
109. Hunter, 304.
110. Hunter, 304.
111. Hunter, 306.
112. Frederick F. Holmes, "Anne Boleyn, the Sweating Sickness, and the Hantavirus: a Review of an Old Disease with a Modern Interpretation." *Journal of Medical Biography*, 6, 1 (February, 1998), 43.
113. Holmes 44.

114. Holmes 46.
115. Guy Thwaites, Mark Taviner, and Vanya Gant, "The English Sweating Sickness, 1485 to 1551." *The New England Journal of Medicine*, 336, 8 (February, 1997), 581.
116. Holmes, 47.
117. Hecker, "Epidemics," 170.
118. Thwaites, Taviner, and Gant, "English Sweating Sickness," 581.
119. The more modern assessment of Thomas Wolsey surviving both the 1517 and 1528 epidemics may have begun in more modern scholarship with Creighton, who himself refers mainly to early work on the Sweat by Vergil and Forrestier, but also the Marquis of Mantua, visiting England in 1517. The Marquis confirms the typically rapid onset of the disease, and how it lasts, the more dangerous component anyway, approximately a day. "It is fatal to take," he writes, "during the fit, any cold drink, or to allow a draught of air to reach the drenching skin," (Creighton, 246) and other similar warnings. Yet Hutchinson, as a biographer, is among the few to refer to the sweating sickness in 1528 as "infecting 40,000 people in London alone and killing more than 2000 of them. Wolsey and the Duke of Norfolk both contracted the disease and recovered." (Hutchinson, "Young Henry," 232) Historians Webster and Dyer accept the account of Wolsey surviving the disease twice, as does scientist McSherry, not really questioning such reports, and Wolsey himself is not known to have kept something as useful to later historians as a diary. During that same epidemic, Tankard also writes that "Henry VIII had been sufficiently alarmed to write to Wolsey commanding him to have general processions made throughout the realm to try to avert God's wrath," (Tankard, "Johnson Family," 5) though just what these processions were or how they would serve to gratify God enough to end the illness no longer appear to exist. Other than his "alarm" about the disease itself, Henry was also quite absorbed by this time with his "Great Matter" in securing a male heir, which in practice meant unloading one wife to acquire a second, and the King's relationship with his Cardinal, who was supposed to secure the royal divorce despite strong papal resistance and delays, had largely deteriorated by then. No other monarchs seemed in the habit of officially offering prayers or other religious acts to secure their realms from divine displeasure, so even if Henry made such appeals, they might not have benefitted from recording in the first place.
120. Guy Thwaites, Mark Taviner, and Vanya Gant, "The English Sweating Sickness, 1485 – 1551: A Viral Pulmonary Disease?" *Medical History*, 42 (1998), 98.
121. Thwaites, Taviner, and Gant, "Viral Pulmonary Disease," 96, italics theirs.
122. Thwaites, Taviner, and Gant, "Viral Pulmonary Disease," 98.
123. Heyman, Simons, and Cochez, 160.
124. Holmes, 46.
125. Holmes, 46.
126. Jonsson, Figueiredo, and Vapalahti, 424.
127. Jonsson, Figueiredo, and Vapalahti, 423.
128. Jonsson, Figueiredo, and Vapalahti, 430.
129. John Kelly. *The Great Mortality: an Intimate History of the Black Death, the Most Devastating Plague of All Time*. New York: Harper Collins, 2005, 7.
130. J. A. McSherry, "Sweating Sickness: a Glimpse Behind the Shroud of History." *The Practitioner*, 229, 1403 (May, 1985), 398.
131. McSherry, 398.
132. Krista J. Kesselring, "A Draft of the 1531 'Acte for Poysoning'." *The English Historical Review*, 116, 468 (September, 2001), 897.

133. Kesselring, 898.
134. Arrizabalaga, 64.
135. Heyman, Simons, and Cochez, 160.
136. Wright, 69.
137. Sally Hickey, "Fatal Feeds? Plants, Livestock Losses and Witchcraft Accusations in Tudor and Stuart England." *Folklore*, 101, 2 (Autumn, 1990), 131.
138. Hickey, 132.
139. Hickey, 135.
140. Sim, Alison. *The Tudor Housewife*. Stroud, GL: The History Press, 2014 (1996), 90.
141. Dobson, 25.
142. Dobson, 25-26.
143. Dobson, 189. Dobson's work also speaks against the common stereotype surviving into our own time about Tudor attitudes regarding hygiene. While bathing and air entering a home through open windows might both be perceived as unhealthy by Tudor subjects, there was nonetheless wide recognition of issues that affected health. As Dobson describes, "one of the most striking of the environmental images was the concept of 'bad air' and an obsession with the noxious, odorous smells and stenches that appeared to emanate from all sources of corruption and cause all sorts of fatal consequences (Dobson, 10)," a suitable summary of the old miasma explanation. And she continues, noting that "streams of effluvia and noxious vapours were believed to arise from open sewers, churchyards, slaughter houses, butchers' shops and lanes, dead flesh, burial grounds, cesspools and from every other sort of putrefaction, excrement, decay, human and animal filth, (16)" while "putrid exhalations might also arise and be contained in such closed spaces as cellars, garrets, cells, common lodging houses, tenements, alleys and alehouses." As for personal hygiene, "cleanliness of the body meant washing with soap and water, or vinegar; cleanliness of apparel required frequent washing and changes of clothes... cleanliness of the home necessitated abundant ventilation, open windows, the breathing of pure air (33)." While this sounds more encouraging and healthier, some of these notions could be prohibitively expensive for many, and Dobson herself notes that the earlier medieval notion of "corrupt airs" remained, which might in truth carry fatal illnesses. This does not indicate contradiction necessarily on her part, but rather part of the slow shift from medieval to early modern ideas, likely with doses of misinformation for contemporaries.
144. Boorde, Andrew. *The Fyrst Boke of the Introduction of Knowledge. A Compendyous Regyment; or, A Dyetary of Helth Made in Mountpyllier. Barnes in the Defence of the Berde* (London: Adamant Media, 2001 (1547)), 20.
145. Boorde, 289-290.
146. G. V. Poore, "London, Ancient and Modern, from a Sanitary Point of View." *Public Health*, 23, 236 (1888) 342.
147. Poore, 342.
148. Poore, 342.
149. John S. Lee, "Piped Water Supplies Managed by Civic Bodies in Medieval English Towns." *Urban History*, 41, 3 (February, 2014), 384.
150. Dolly Jørgensen, "Local Government Responses to Urban River Pollution in Late Medieval England." *Water History*, 2, 1 (May, 2010), 37.
151. Jørgensen, 49,
152. Describing assessments of Tudor morality and how such might fit into the study of ethics more generally is surely a worthy yet logically separate topic than the history of disease and

medicine as explored in this dissertation, though they have been considered in more detail in a previous section. By this point, when Jørgensen cites Tudor morals and whether contemporaries had certain obligations to behave (especially toward the poor, but also in terms of whether one's behavior could or should influence one's physical health), the other writers cited thus far discussing aspects of this also include Slack, Wallis, Vaughan, Hecker, and Poore. Slack notes for instance how "the obligations of the rich were often summed up under the headings of charity and hospitality. These Christian virtues were as ill-defined as poverty itself (or honor, as Chamberland describes it)... the poor were thus an integral part of a Christian commonwealth... (yet) condemnation of the poor was becoming respectable." (Slack, "Poverty and Policy," 19)
153. Lee, 386.
154. Lee, 369.
155. Louise H. Curth. "Lessons from the Past: Preventive Medicine in Early Modern England." *Medical Humanities*, 29, 1 (June, 2003) 17.
156. Curth, 17.
157. Curth, 17.
158. Poore, 342.
159. Andrew B. Appleby, "Diet in 16th Century England: Sources, Problems, Possibilities," in Charles Webster, ed. *Health, Medicine, and Mortality in the 16th Century* (Cambridge: Cambridge University Press, 1979).
160. J. Kelly, 63.
161. Poore, 342.
162. Roberts, 39-40.
163. Roberts, 40.
164. Hickey, 132.
165. Sim, 79.
166. Sim, xiv.
167. Hickey, 132.
168. Dobson, 189.
169. Creighton, 278.
170. Wylie & Collier, 442.
171. Casadevall, Fang, and Pirofski, 3.
172. Casadevall, Fang, and Pirofski, 3.
173. Slack, "Poverty and Policy," 43.
174. Slack, "Poverty and Policy," 44.
175. Smith, 81.
176. Slack, "Poverty and Policy," 48.
177. Paul Slack, "Mortality Crises and Epidemic Disease in England, 1485 – 1610," in Charles Webster, ed. *Health, Medicine, and Mortality in the 16th Century* (Cambridge: Cambridge University Press, 1979), 16.
178. Wright, 105.
179. Slack, "Mortality Crises," 21.
180. Slack, "Mortality Crises," 47.
181. Slack, "Mortality Crises," 48.
182. Carlson and Hammond, 29-30.
183. Slack, "Mortality Crises," 53.
184. William G. Hoskins, "Harvest Fluctuations and English Economic History, 1480 – 1619."

The Agricultural History Review, 12, 1 (January, 1964), 28.
185. Bridson, 4.
186. Bridson, 5.
187. Hoskins, 33.
188. Hoskins, 34.
189. Bridson, 5.
190. Hoskins, 38.
191. Carlson and Hammond, 49.
192. William Kelly, "Visitations of the Plague at Leicester." *Transactions of the Royal Historical Society*, 6 (1877), 446. After evaluating the fourteenth century in particular, Kelly moves through the fifteenth, and his descriptions of Leicester during the sixteenth prove quite fascinating, especially when considered as part of the background of sweating sickness historiography. While he admits that the corporation archives for the city contain less information about this time, Kelly writes that "there can be no doubt that the plague prevailed in Leicester during the years 1559, 1560, and 1561 (W. Kelly, 400)," and his work is also among the earlier pieces to consider the potential utility of parish records for such. The St. Martin's register, "the earliest we possess" for the area, shows marked increases in local burials for these years, dropping again after 1561. Only one hint about sweating sickness appears in his work, and even then when he quotes John Buck who had written some time earlier, in 1849: "all those great visitations which we have been considering – I mean the black-death, the sweating-sickness, and the plague – have very much in common with each other (446)." During the mid-nineteenth and later-nineteenth century, historians were already demonstrating the problems in differentiating among diseases from previous centuries.
193. Bridson, 5.
194. Caius, 15.
195. Heyman, Simons, and Cochez, 156.
196. Jonsson, Figueiredo, and Vapalahti, 417.
197. Jonsson, Figueiredo, and Vapalahti, 417.
198. Carlson and Hammond, 50-51.
199. Carlson and Hammond, 48.
200. Heyman, Simons, and Cochez, 158.
201. Nutton, "Caius, Linacre," 234.
202. Ruth H. Keogh, and D. R. Cox. *Case-Control Studies* (Cambridge: Cambridge University Press, 2014), ix.
203. The authors describe how two main potential sorts of bias exist for case-control studies. The first is selection bias, "in which both the exposure and the outcome have a common effect (Keogh and Cox, 23)," like circular reasoning or "begging the question" in logic. It occurs "if controls are sampled in such a way that they do not represent the intended underlying population." The second type is retrospective exposure ascertainment bias, which "arises if some aspect of the data collection differs systematically between cases and controls (24 – 25)." Simpler examples include recall bias and information bias, though neither of these would apply since sixteenth century sufferers of sweating sickness of course cannot be interviewed.
204. Keogh and Cox, 12.
205. Leon Gordis. *Epidemiology, Fifth Edition* (Philadelphia: Elsevier Saunders, 2014), 203.
206. Gordis, 210.

207. John Christiansen, "The English Sweat in Lübeck and North Germany, 1529." *Medical History*, 53 (2009), 419.
208. Robert S. Gottfried, "Bury St. Edmunds and the Populations of Late Medieval English Towns, 1270 – 1530." *Journal of British Studies*, 20, 1 (1980), 4.
209. Gottfried, "Bury St. Edmunds," 8.
210. Gottfried, "Bury St. Edmunds," 24.
211. Gottfried, "Population," 13.
212. Christiansen, 421.
213. Sara M. Butler. *Forensic Medicine and Death Investigation in Medieval England* (New York: Routledge, 2015), 2.
214. Steven Gunn and Tomasz Gromelski, "For Whom the Bell Tolls: Accidental Deaths in Tudor England." *The Lancet*, 380 (October, 2012), 1222.
215. The main focus of this article by Gunn and Gromelski is accidental death, and some such activities remain risky even now, including "farming, building, and mining (Gunn and Gromelski, 1222)." They also make the case that for accidental deaths, labor accounted for about half, and travel (via horses and carts) for roughly another quarter: "working practices and technologies determined the kinds of accidents to which people were susceptible." Sports injuries largely made up the rest, since archery, jousting, hunting, dueling, swimming, and football all remained popular, though the latter two were typically recreational activities of commoners alone. Wounds and injuries of all sorts might become infected, and anything of an antibiotic nature was misunderstood as such, though treating open wounds with the likes of topical administrations of wine, honey, or even spider silk might help prevent infection. More common knowledge generally recognized a need for preventive measures, and that major organs and tissues needed protecting, but armor would only typically be worn by nobles.
216. Butler, 182.
217. Butler, 227.
218. Barbara Harvey and Jim Oeppen, "Patterns of Morbidity in Late Medieval England: a Sample from Westminster Abbey." *Economic History Review*, 54, 2 (May, 2001), 215.
219. Harvey and Oeppen, 216.
220. Thwaites, Taviner, and Gant, "English Sweating Sickness," 580.
221. Dyer, 364.
222. Dyer, 364.
223. Dyer, 364.
224. Thwaites, Taviner, and Gant, "English Sweating Sickness," 580.
225. Dyer, 364.
226. While other research has often downplayed small communities, Dyer agrees that the sweating sickness was mainly rural, which means parish records, even when scanty, might be worth consulting. Dyer also offers intriguing maps considering the implications of using such records. Still, while the incubation of sweating sickness indeed seems to have been quite short, he admits we seem to have little way of confirming this, even with parish records, which do not describe details of diseases: they merely, and certainly not always, list apparent cause of death. The encouraging part here for the usage of parish records, though, is that the timing of burials tends to reflect the times of infections of diseases, including the Sweat. Dyer also confirms the preponderance of rural communities for the Sweat, though another factor emerging in his work is that roads were needed for the traveling of diseases. He estimates that perhaps a third of English parishes contain records of deaths from this particular disease. What emerges from such data are

notable increases in burials in "unmistakable rarity" which then disappear "almost as soon as (they have) begun." (Dyer, 365) He seeks patterns in these brief but sharp burial increases, and maintains that even with the ambiguities in having persons listed as having died from sweating sickness, they may yet be sufficient in both accuracy and number to offer further insights. So parish records, if they are to help at all with this research, should be used not to try and verify cause of death, but to help isolate increases in burial rates, especially in areas where it is believed or known that outbreaks occurred.
227. Slack, "Mortality Crises," 23.
228. Bridson, 2.
229. Hope-Simpson, 294.
230. Of some concern here is that Hope-Simpson seeks to identify historical trends of influenza outbreaks by comparing patterns from older parish records to known data from twentieth century outbreaks, but there exist no more recent outbreaks of sweating sickness to examine. Further, by studying parish records, "the causes of most excess mortalities, many of which were large and sustained, were not identifiable... sometimes it was possible to assign the excesses to plague, typhus or smallpox from evidence in contemporary literature or in the burial registers themselves, and it was evident that these and the other conditions mentioned previously did not produce excess mortalities that mimicked the typical influenzal curve (Hope-Simpson, 302)." One may thus feel more inclined to agree with her assessment that "the recording of epidemics in the contemporary literature of previous centuries is haphazard."
231. Tankard, "Protestantism," 14.
232. Dobson, 83.
233. Dobson, 162.
234. The list of records Dobson uses includes "churchwarden's books, quarter session rolls, hospital lists, workhouse records (these would only appear during Elizabeth's reign), medical attendance at jails (rare), medical case books and diaries, ledgers, doctor's account books, bills from practitioners, probate inventories, probate accounts, family letters, personal diaries, medical recipes, state papers, newspapers (also appearing later), advertisements, medical journals, Bills of Mortality, estate records, agricultural surveys, annals, weather journals and coroner's inquests. Together these comprise a more fruitful collection than any single source (Dobson, 231)." That would surely seem the case, and this dissertation indeed considers many of these types of records and what they may or may not help to explain regarding sweating sickness.
235. C. A. Roberts, "The Use of Literary and Documentary Evidence in the History of Medicine," in Edwin Clarke, ed. *Modern Methods in the History of Medicine* (London: The Athlone Press, 1971), 46.
236. C. A. Roberts, 49.
237. Carlson & Hammond, 34.
238. Moore, 55.
239. Moore, 45.
240. Moore, 56.
241. Gottfried, "Population," 16.
242. Lorraine C. Attreed, "Preparation for Death in Sixteenth Century Northern England." *The Sixteenth Century Journal*, 13, 3 (Fall, 1982), 37.
243. Ackroyd, 20.
244, Attreed, 38.

245. In addition to concerns over disease and its effects, Attreed informs us "that the turbulence of the (sixteenth) century dissuaded many testators from relying on the church structure, either Anglican or Catholic, to help them achieve salvation (Attreed, 56)." Creating a will is partly a statement of personal faith, that a person's survivors will indeed carry out that person's requests. Further, "at such a time testators' beliefs are probably clearer than at any other time in their lives, and they have a chance to influence society's opinion." The sixteenth century also witnessed "ostentation in funerals and burials (57)," despite financial concerns or the hygiene issues as presented by authors like Gunn and Gromelski, Bingham, Slack, and Dobson, each of whom explains hygienic concerns further.
246. Eamon Duffy. *The Stripping of the Altars: Traditional Religion in England c. 1400 – c. 1580* (New Haven: Yale University Press, 1992), 502.
247. Duffy, 6.
248. Attreed, 40.
249. Danae Tankard, "The Reformation of the Deathbed in mid-Sixteenth-Century England." *Mortality*, 8, 3 (2003), 263.
250. Tankard, "Reformation of the Deathbed," 263.
251. Tankard, "Reformation of the Deathbed," 264.
252. Tankard, "Reformation of the Deathbed," 255.
253. Butler, 231.
254. SP 46/7 f.8.
255. D. C. Shanson, "Infections of the Central Nervous System." *Microbiology in Clinical Practice*. Amsterdam: Elsevier Science, 1989, 249.
256. P. Gale, L. Kelly, and E. L. Snary, "Pathways for Entry of Livestock Arboviruses into Great Britain: Assessing the Strength of Evidence." *Transboundary and Emerging Diseases*, 62, 2 (April, 2015), 116.
257. Gale et al., 115.
258. Gale et al., 119.
259. P. Hudson, et al., "The Epidemiology of Louping-ill, a tick-borne Infection of Red Grouse (*Lagopus lagopus scoticus*)." *Parassitologia*, 39, 4 (December, 1997), 319.
260. Hudson et al., 320.
261. Hudson et al., 321. Even more interesting is how hares play a "significant role" in the "dynamics of this disease," since "without the hares, the tick population and consequently the viral infection cannot persist but when hares are moderately abundant with respect to the grouse density they can sustain both the tick and virus population." (Hudson et al., 322)
262. M. K. Laurenson, et al., "Prevalence, Spatial Distribution and the Effect of Control Measures on Louping-ill Virus in the Forest of Bowland, Lancashire." *Epidemiology and Infection*, 135, 6 (2007), 99.
263. M. K. Laurenson, et al., "The Role of Lambs in Louping-ill Virus Amplification." *Parasitology*, 120 (February, 2000), 963.
264. Laurenson, "Role of Lambs in Louping-ill Virus," 964.
265. Laurenson, "Role of Lambs in Louping-ill Virus," 970.
266. Hudson et al., 322.

V. Conclusion: Rouen, Rascals, and Run-aways.

Creighton was the earliest to make the denial: that whatever the Sweat was, and even if he himself could not fully assess its identity, it still differed from the later outbreaks of something that is recorded historically separate as the "Picardy Sweat," or sometimes "miliary fever." Fascinatingly, Creighton also noted some of the similarities, even suggesting that sufferers of each disease might have ultimately succumbed to hyperpyrexia caused by the excessive dedication of loved ones to keep victims warm, too warm in these cases, typically by covering them with too many blankets or insisting that they wear too many clothes (though in the latter instance, no one appears to have commented on the ease or, more likely, comparative difficulty, of getting someone so symptomatic to actually have enough awareness and motor control to change clothing). Holmes, meanwhile, insists that Creighton "makes a strong case for the sweating sickness being caused by a virus imported from Picardy to Milford Haven," [1] by the mercenaries in the employ of Henry Tudor. "Wet years" preceding that and each subsequent epidemic were central to how Creighton pleaded his case, so here we have not just the still more recent reference to climatic influences, we have Creighton, despite his apparent denial, specifying the Picardy-to-Wales route of the disease, with an assumption that it was viral. Yet to maintain responsibility to all known primary materials, as well as subsequent analyses based on them, the idea of the Picardy Sweat must be revisited, even if only to perhaps dismiss it as a later distraction from a different disease. In other words, the two-fold question must be raised: what was this Picardy Sweat, assuming it can be more fully known (and without delving into another retrospective diagnosis!); and, based on what has been reported about this other disease, does any basis exist for arguing that the two "sweats" may have actually been one and the same?

First, the locales need to be considered. There has never been any dispute known as to whether Henry Tudor, future King of England via conquest and victory at Bosworth, hired and relied upon a number of foreign mercenaries. Nor does anyone appear to contest from where these men came. Henry is known to have recruited initially from the region of northern France, after living in exile in Brittany with the support of Duke Francis II. What differs among various interpretations is that "the location and date of the first case of the Sweat are a source of disagreement among medical historians." [2] Some favor the date of August 7, 1485 at Milford Haven, essentially right as Henry's army arrived in Wales, while others prefer the later time after Henry and his band reached London. This comes from Devereaux, who earlier noted that the Picardy Sweat was allegedly of less concern to victims, and purportedly also presented with some type of skin eruptions, perhaps reminiscent of plague buboes, though the latter condition was recognized as specifically a telltale sign of plague instead.

Hutchinson reintroduces us to this theme while discussing the early life of Henry VIII. As for his unfortunate elder brother, Hutchinson agrees that his fate was indeed most likely traceable to tuberculosis, and yet perhaps "it is possible that he (Prince Arthur) succumbed to the *sudor Anglicus*, the so-called English sweating sickness." [3] While this is unlikely, what matters for us now is the "cruel irony that this disease (the Sweat) may have been introduced into England by the French mercenary soldiers of Henry Tudor, as the scourge made its first appearance shortly after he landed with his small army at Milford Haven, Pembrokeshire, on 7 August 1485." [4] There is no disagreement about the soon-to-be Henry VII using foreign mercenary troops, nor is there even any dispute regarding from where he hired them, indeed not far from where he had lived in exile in order to prepare for the invasion of his homeland. Also worth noting here is the similarly apparently unchallenged notion that said troops never became

ill until they crossed back over the Channel and arrived in Wales. Even modern scientists weigh in with this subject, some of them mentioning how Lord Stanley "excused himself from the battle (of Bosworth) because his army allegedly suffered from 'the sweat' although this did not prevent him (and them) from changing sides at the last moment." [5] Wylie and Collier also point out this widely accepted truth, of Thomas Lord Stanley ignoring the summons of his sovereign, Richard III, though this seems to necessitate arguing that nearby areas in northern England may have had to endure sweating sickness outbreaks, though "none of the London-based chroniclers seem to have looked far enough north for the origins" [6] of the disease, helping lead these authors to argue against London as the source.

A fine summary of this decision-making is offered in the admittedly now dated text of historian A. L. Rowse, who describes the initial invading force of Henry as numbering only two thousand or so, and Stanley distrusting "two councils" held just prior to the coronation of Richard. As for later problems with royal authority,

> "early in the year (1485) Lord Stanley had asked leave to absent himself from Court to visit his family; he had been absent from his post for months when Richard required him to attend or to send his son and heir, Lord Strange, instead. This Stanley did, just before news came of Henry's landing. Stanley's brother, Sir William, was Chamberlain of North Wales and Henry's route had passed along the borders of his jurisdiction. Suspicious and alarmed, Richard summoned Lord Stanley, head of the family, to Nottingham at once. Lord Stanley replied that he was ill of the sweating sickness... and Richard threatened those who did not come to his aid with death and confiscation." [7]

"Henry had an adventure" on the road to his fate, as Rowse further elucidates, arguing that this was later related to Vergil himself. Henry "was in great anxiety, since he could not assure himself of Lord Stanley, whose son Richard was keeping as a hostage and would certainly kill if Stanley came out openly on Henry's side." [8] King Richard was rumored to be nearby with a sizable force of his own, and Henry encamped with his smaller army, later meeting with both

Stanley brothers. But descriptions of that meeting seem noncommittal. Henry needed their help, though the timing and arrangement of forces during the battle (Stanley was actually off to the side, making his support of anyone seem an open question), enabled him to wait and decide whom to ultimately favor. Yet apparently, prior to attacking, "Richard sent a message to Lord Stanley ordering him to join in against the enemy without delay," [9] threatening still to execute his son. And "Henry was also kept in anxiety as to what Lord Stanley would do," having refused a "dubious reply" even after the meeting with the brothers the night before.

An earlier biography of the future king disagrees in turn with that notion, saying simply that the new sweating disease, "beginning in London, spread over the rest of England." [10] Yet this is also problematic, since if the illness was such a part of Stanley's initial refusal, historian Gladys Temperley in the same text describes this response merely as "an evasive answer." [11] This collection of apparent contradictory assessments of the disease does not help us isolate matters much further, and if the disease appeared first in London, then one must account for why the capitol was Henry's next stop, at least initially. As Temperley relates, the new king "was busy preparing for his coronation when the 'sweating sickness,' hitherto unknown in England, appeared in London. The disease was very virulent," [12] and she describes its rapidity and cites some early mortality reports, yet her most helpful comment in this context is political, since "the visitation (of the disease) was popularly regarded as an omen of 'a stern rule and a troubled reign'," in the sense of a judgment based on divine displeasure. Hall warned of the omen also, writing how "this cōtagious & euell plague chaunced in the first yere of kyng Henryes reigne as a token and a playne signe (if to the vaine judgemēt of the people whihce cōmonly cōmen more fantastically then wisely, any faith or credite is to be had geuē or attributed) that kyng Henry should haue a harde and sore beginning." [13] More important historically though is the timing,

which Rowse confirms: Henry "made his ceremonial entry into the City on 3 September 1485, though an outbreak of sweating sickness postponed the coronation till 30 October." [14] But this is Rowse's second and final mention of the disease, after the above passage in which Stanley claimed his own illness. It is typical of the facile attitude discussed so many times, and alluded to earlier in this piece as well. Most want an easy answer for the sweating sickness, as with earlier attempts to lazily use terms like "plague" or "ague," or to use the actual term but leave it in isolation, as Rowse does, even while otherwise helping explain the odd details and shifting loyalties just before the Tudor dynasty began at Bosworth. What matters is that a new disease began affecting England in the fall of 1485, and now we know what it most likely was.

In addition to these problems of assessing some of the details, while primary sources about sweating sickness cannot help in this capacity of comparing it to the Picardy Sweat (simply because the final Sweat epidemics all far predate the earliest Picardy outbreak), primary accounts dating to the seventeenth and even eighteenth centuries may help to illustrate the situation, in addition to helping assess the relevance to the focus of this dissertation. After all, if later epidemics of some other disease (assuming it truly was something different) turn out instead to have been the same disease, then we would clearly be another important step closer to understanding, and perhaps even more cogently retrospectively diagnosing, the disease. One commonality, to start with, concerns the likelihood of relapse, which "has led some medical historians to believe that the Sweat was one of the virulent relapsing fevers," [15] while a later commentator describes how even Vergil, at the time, "recognized relapses, often multiple, as being common," [16] and of course Wolsey stands as the best known multiple case sufferer. Holmes further refers to Forrestier and Caius as the main contemporaries to offer accounts of the disease, and how together they "suggest humours, environmental pollution, planetary influences

and bad English living habits as causes." [17] He describes Hecker, too, as criticizing Linacre for ignoring the disease at the time, and for arguing that it was a variant of Picardy Sweat, sometimes known as miliary fever. This latter term may only convolute the issue at this point, as some point to "miliary fever" as something perhaps new for the Continent during the eighteenth century, sometimes only since it appears on the death certificate of Wolfgang Mozart as the cause. Even Heyman et al. refer in passing to the death of Mozart in this context. Heyman et al. even note that the Picardy Sweat (*suette miliarie* for them) had some 196 outbreaks from the first one in 1717 and up to 1874, and suggest that, as with the prior English version, the irregularity of the outbreaks "suggest an ecological or meteorological trigger." [18]

And third, what might this brief comparison help illustrate regarding English and French interactions during not just the Tudor period but also during later times, since Picardy Sweat appears for the first time more than two centuries after the Sweating Sickness is first known to have visited England? It is Patrick, a half century ago, who weighs in with this initially, maintaining that early twentieth century speculation seems to have made the connection between the two, even if Picardy Sweat is first known to appear in historical literature in 1717, and only on the Continent. Patrick favors some toxic explanation, and voices his frustration at being unable to resolve questions about either disease from known primary accounts. Wylie and Collier are among those, interestingly, who seek to deny the existence of evidence linking the Sweat following Henry Tudor's army during 1485 during the buildup and journey of those forces from Milford Haven to Bosworth. They want to know how it could have apparently not afflicted the army in transit, especially with its otherwise described rapid onset and incubation time. Indeed, this is a critical issue for the whole idea of Henry's forces unwittingly transporting the disease at all, exacerbated by the fact that they landed in Wales, and yet there are of course no

descriptions of Welsh Sweat epidemics, nor of sickness at sea during the crossing. Accordingly, the most plausible way to conclude is to posit that since the disease was thus far unknown, in 1485, and since prior to the twentieth century the key killer of troops had always been various diseases (malaria, dysentery, typhus, and others), then contemporary accounts may simply have not left us any descriptions of Henry's troops perhaps dying off en route even as his army ultimately grew by the time of the showdown with Richard's army. High enough mortality rates do not really appear until afterward, as with Henry's coronation further south, as we have seen. As for Wales, perhaps the same can be said on a larger scale, that the disease did its work on the army as it traveled, vanishing in Wales while remaining to inflict southern England during the coronation and then perhaps lying in wait to strike again later with the four later outbreaks. Sloan and McSherry seem willing to accept some portions of Wales as suffering from the disease, though neither specifies beyond this, perhaps taking it for granted that Wales must have been hit at least by this first epidemic. And this latter point only becomes tenable if the army had very little contact with anyone in Wales, though in fairness, speed and stealth were certainly among the future king's priorities once he landed, even with a force of some two thousand. A good summary of these key points appears in the work of Guthrie Vine from a century ago, who worked with the fourteenth century "litil boke" about pestilence by Joannes Jacobi. The problem, as Vine sees it, is that "we have no evidence that any disease like the sweating sickness existed around Rouen previous to the departure of Henry Tudor for England." [19] Volcy really throws off the origin, extending to a portion of modern France which almost borders Switzerland: "la gran controversia giró en torno a la enfermedad llamada *sudor de Picardía*, *sudor picardo* o *sudor miliar*," he writes, "que después de su aparición en Montbéliard, Francia en 1712, recorrió las regiones de Normandía y Picardía." [20] Montbéliard lies indeed far

southeast, though this is the only mention of that locale in the literature. That the Picardy Sweat emerged over two centuries later suggests to Vine that it might be "not unreasonable to suppose that the seeds (Fracastoro's own term) of this later endemic disease may always have lain latent in this region," [21] so that whatever conditions the disease needed to develop, then, "must have been supplied in England." This allows for consistency, but almost reverses the apparent causality, in a sense. Something enabled the disease to develop with Henry's troops after they crossed the Channel, yet lay in wait on the Continent for later outbreaks.

So, to summarize, accounts of the sweating sickness of the sixteenth century in England and of the Picardy Sweat of later decades on the Continent have the following attributes in common. To begin with, we have occasional equivocations in the secondary literature, such as that of John Kelly, writing about the Black Death but also simplifying matters by describing "another major disease... the sweating sickness – or the Picardy sweat – appeared six times between 1485 and 1551." [22] This seems the only time the Sweat of Tudor England is called the Picardy instead, and Kelly's inaccurate count of the epidemics is rather careless. And this is the whole problem: the English and Picardy sweats are separated not just by the Channel but by time, if one uses the last Sweat epidemic of 1551 compared to the first mention of the Picardy Sweat in 1717. Many of the signs and symptoms match. Additionally, the louping-ill thesis might be applied, though this could prove problematic in that tick-borne encephalitis diseases generally are not found in this part of western Europe: Rouen and Picardy. The best comparison ultimately is offered by Heyman et al., whose comparative chart helps illustrate this further. To add to this graphic discussion, the author has added his own column, to include louping-ill virus and a similar summary of its etiology.

Table 6: Comparison of the English Sweating Sickness, Picardy Sweat, HPS, and HFRS (taken from Heyman, Simons, and Cochez, "Were the English Sweating Sickness and the Picardy Sweat Hantaviruses?" and reprinted with permission)

	English Sweat	Picardy Sweat	HPS (hantavirus pulmonary syndrome)	HFRS (hemorrhagic fever with renal syndrome	LIV* (louping-ill virus)
Vector	?	?	rodent	rodent	rodent
Pathogen	? (virus?)	? (virus?)	hantavirus	hantavirus	arbovirus
Infection Mode	? (zoonotic? human-to-human?)	? (zoonotic? human-to-human?)	aerosol	aerosol	zoonotic (aerosol possible)
Incubation Time	1 – 44 days	6 days	1 – 40 days	1 – 40 days	1 – 5 days (variable by species)
Disease Stages	headache, myalgia, abdominal pain, vomiting, delirium, cardiac palpitation, breathlessness, death	febrile phase, sweating, hemorrhages, rash, death	febrile phase, pulmonary edema, diuresis, death	febrile phase, shock, oliguric phase, diuretic phase, death	fever, chills, muscle aches, fatigue, encephalitis
Duration	24 hours (if fatal)	10 – 14 days	10 – 20 days (2 – 5 days, if fatal)	10 – 20 days	5 – 10 days (1 – 3 days, if fatal)
Mortality	30% – 50%	0% – 20%	40%	< 1%	5 – 10% (variable by species)
Seasonality	summer	summer	summer	spring, summer	spring, autumn

* LIV has been added to this table by the current author.

Even more curiously, while no further attempt will be made to equate what follows with any early modern European sweating diseases, records held within the British National Archives also include the following historical concerns. First is a report dating to as recently as June 11, 1851, part of the Home Office records. It discusses a letter, itself not included in the Archives, from Commander Lefebvre of a British ship to the Lieutenant Governor of the Isle of Guernsey, the British Crown dependency off the coast of Normandy. One may wonder, due to the geography of Guernsey and its proximity to Normandy, whether concern for the Sweat may be justified, especially if one accepts the identification of English sweating sickness with Picardy sweating sickness. Even without the original letter, the reply makes for intriguing reading:

> "Admiralty, 11th June 1851. I am commanded by my Lords Commissioners of the Admiralty to send you herewith, for the information of Secretary Sir George Grey, a copy of a letter, dated the 3 June, from Commander Lefebvre of Her Majesty's steamer Cuckoo to the Lieut. Governor of Guernsey, and of its enclosure, relative to the appearance of an epidemic (sweating sickness) at Dialette a finale place on the Coast of France. I am, Sir, Your most obedient humble servant, Wm Hamilton." [23]

No crew members of *HMS Cuckoo* are known to have encountered disease epidemics, either during this three-hundredth anniversary of the final Sweat epidemic, or afterwards. The small French port in question is misspelled and should be Diélette, and nothing in its history accords with epidemics, either.

Even more recently still comes another communiqué, number 20468 and held within the Foreign Office records of the British Archives. It dates to June 16, 1906, and refers to an incident precisely one month previously. Forwarded to officials at Bordeaux, part of "Commercial France," it concerns an "Outbreak of Sweating Sickness in Departments of Charente and Charente Inferieure," from Sir Francis Bertie, the British Ambassador to the French Republic. It reads:

> "Sir, I have the honour to transmit to you herewith from the 'Journal Officiel' of the 13th instant copies of a Decree containing regulations for the destruction of Rats on ships coming from countries infected with the plague. I have the honour to be, with great truth and respect, Sir, Your most obedient, humble servant, Francis Bertie." [24]

So what, then, are we to make of these accounts? Sometimes others have appeared sporadically, with nothing to lend them any scholarly force, as with Thompson describing the history of influenza from the early Tudor years to his own mid-nineteenth century: "during the prevalence of Influenza in Britain," he records, "Spain was afflicted with Sweating-sickness," [25] though in referring here to an 1803 account by Joaquin de Villalba, a Spanish historian and epidemiologist, this brief mention is highly problematic. In the meantime, as for Diélette, Charente reveals no other known association with sweating sickness, and is mostly inland, far south of Normandy. Its only notable history with disease epidemics occurred when phylloxera arrived in 1872 and wreaked havoc with the local wine industry. This disease only attacks grape vines, appears to be endemic to the eastern part of North America, and uses the fly *Daktulosphaira vitifoliae* as its vector. Like sweating sickness, it has no known cure, but this hardly gives the two diseases enough commonality for other comparisons. And the French mercenaries who came over with the future Henry VII in 1485 appear to have been carriers yet not sick themselves, a scenario not often considered. One observer notes that "the sweating sickness did not affect Scotsmen, Irishmen or Frenchmen," [26] though clearly affected many on the Continent in 1529. Could it be said somehow to affect those of Germanic ancestry, which applies to the English? Louping-ill remains with us, and maybe these were lesser known, more modern cases which did not reach the level of epidemics; it might just as easily be argued that both these more recent messages amounted to cases of fear surrounding a part of modern France

which might have reminded a few persons of an unusual time in early modern European history, when an unknown disease hitched a ride over the Channel with some unwitting troops.

Fussner tells us that Sir Walter "Ralegh once observed that the 'industry of an historian having so many things to weary it, may well be excused, when finding apparent cause enough of things done, it forbeareth to make further search.' This disarming remark was intended to legitimize conjecture, not laziness." [27] And Fussner refers to modern historians being unable to resolve the "climate of opinions" regarding Tudor historiography generally, and agrees with Bacon from centuries before that the Tudors themselves offered "no history of learning and the arts. This meant no history of science," [28] and also no history of philosophy, literature, politics, or economics, which may then leave a reader wondering how to possibly extrapolate something as specific as a retrospective medical diagnosis for a disease poorly understood at the time and contentiously debated into the present. Fortunately Fussner wrote this interpretation almost a half century ago, and Sweat historiography has certainly blossomed hugely since then, even with all its resulting disagreements. So, to offer a final conclusion then, after summarizing the prior arguments regarding all the offered culprits for the English sweating sickness, its link to the Picardy Sweat, and overcoming the major issues with making an academic and logical rather than laboratory and empirical retrospective medical diagnosis, we can briefly reconsider the other arguments this dissertation sought to argue. The arguments already answered include, first, that the mystery of the sweating sickness deserved additional research and consideration, since despite its having received considerable attention in specialty publications in papers written by experts in a mix of fields, no one had ever offered something truly comprehensive. Second, it has been shown that whatever the disease was, its signs and symptoms strongly suggest that it did not result from the introduction of any type of toxic agent or known poison into its victims.

Third, it has also been demonstrated how the potential promise of other types of primary source materials, in the form of both parish registers and personal wills, are sufficiently problematic (in terms of their dates of accumulation, their accuracy in recording relevant data, and the daunting task of attempting to evaluate all those which survive from the period) to discount them as useful sources other than as potential and occasional sources which may help confirm other aspects of this retrospective piece of history. And fourth, it has been shown how the remaining method would be a process of logical induction that considered both primary source reports of the disease itself, as well as other primary source materials which help put understanding of the disease into its proper context, so that to understand it and ultimately arrive at the likeliest diagnosis, Tudor society itself had to be understood from a combined perspective involving key aspects of early modern science and medicine. Ultimately, and particularly within this fourth consideration, the arguing had to proceed systematically until all the less likely candidates, bacterial and viral, could be eliminated from further consideration.

The fifth consideration brought us outside of England, even more than the fourth sweating sickness epidemic of 1528 did, and linked the study of Tudor science and medicine to another mysterious disease which has even less supporting scholarship, despite its own outbreaks having occurred more recently. Finally, then, we can ultimately say that while the English and Picardy sweating diseases have some common points, most clearly in point of geographic origin, concluding that they are one and the same is appealing from the perspectives of medicine and history, but at this stage must remain tenuous at best. The reason is straight-forward, considering that this dissertation consists in an effort to retrospectively diagnose a single disease, to add another disease for consideration, of which even less may be known based on primary accounts, necessitates a logical leap which is currently unwarranted. The English Sweat did appear in

England five times during the period of the Tudor monarchy, and this project has shown that it was most likely an ongoing and uncontrolled case of louping-ill virus, LIV, which is of far more concern to sheep and red grouse than to humans, though humans too can contract it and die from it, even now. Despite the myriad of other arguments put forth over the centuries since, the symptomology, transmission methods, and environment of Tudor England together offer the most coherent and plausible case for this. Perhaps we can never know with complete certitude what the English sweat was, but any retrospective case takes the best information and data available and reaches an inductive conclusion to fit the evidence and supporting logic, and that is what this project has done throughout.

Notes for Part V.

1. Holmes, 45.
2. Devereaux, 1193.
3. Hutchinson-2, 60
4. Hutchinson-2, 60
5. Heyman, Simons, and Cochez, 155.
6. Wylie & Collier, 428.
7. A. L. Rowse, *Bosworth Field: from Medieval to Tudor England* (Garden City, NY: Doubleday & Company, 1966), 216.
8. Rowse, 217.
9. Rowse, 218.
10. Temperley, 194.
11. Temperley, 20.
12. Temperley, 40.
13. Hall, 425.
14. Rowse, 229.
15. Devereaux, 1192.
16. Holmes, 43.
17. Holmes, 44.
18. Heyman, Simons, and Cochez, 153.
19. Vine, xv.
20. Volcy, 425.
21. Vine, xvi.
22. J. Kelly, 280.
23. HO 45/3801.

24. FO 368/19/52.
25. Thompson, 4.
26. C. J. Hackett, "Diagnosis of Disease in the Past," in Edwin Clarke, ed. *Modern Methods in the History of Medicine* (London: The Athlone Press, 1971), 100. Hackett also revisits an older insight about Sweat, that "of the sweating sickness it has been said that whereas in plague the patient becomes sick before he dies, in this disease death occurred before the patient became sick," alluding to the rapid onset.
27. Fussner, 234.
28. Fussner, 234.

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My thanks are due to the courteous and knowledgeable and especially patient staff of both the British Library and the British National Archive, who both instructed and tolerated me during visits to both facilities during early July, 2015. My dissertation research has been quite enhanced by the materials I was able to study at both facilities.

Additionally, I would like to extend my appreciation to the staff of both Valley Library and Knight Library in Oregon, particularly in the persons of Anne Bahde at the Oregon State University Special Collections and Research Center at Valley Library, and Bruce Tabb at the University of Oregon Special Collections and University Archives at Knight Library.

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