

Chapter 2

The Scientific Challenge

2.1 Introduction

In the last chapter, I described how a number of argument forms that had traditionally been characterized by philosophers as weak or fallacious modes of reasoning could be shown to facilitate scientific inquiry into BSE when little was known about this new brain disease in cattle. The point was made that these argument forms have relevance to the epidemiologists and public health scientists whose task it was to identify and respond to this emerging infectious disease. However, this point requires some explanatory work if it is to have more than a very general application to the work of these public health professionals. For these professionals might ask with some justification why they should treat seriously argument forms that have been deemed to be logically inadequate by generations of philosophers. They might also wonder if philosophical discussion of reasoning has anything but the most abstract lessons for scientists who are charged with containing infectious diseases. In this chapter, I undertake this explanatory work by arguing that philosophical contributions on reasoning and argument are not only relevant to epidemiology, but that they also represent the very best prospect for investigators of addressing some of the criticisms of epidemiology that have been raised in recent years. These criticisms have been expressed most clearly by Christakos et al. (2005), although other theorists have also added their voices to the exchange. I believe, and will subsequently argue, that it is only when epidemiology and philosophy come together on questions relating to reasoning that true progress will be made on devising models of reasoning that are adapted to conditions of uncertainty. Certainly, this belief is the principal motivation for the model of reasoning that will be defended throughout this book.

If the model of scientific reasoning that I am proposing is to have epidemiological value, it must be able to demonstrate its worth to the early investigations of epidemiologists in the BSE crisis. Accordingly, this chapter will also be used to extend the examination of those investigations which was commenced in [Chapter 1](#). The aim will be to show how this model is very directly motivated by the deliberations that were undertaken as part of those investigations. Specifically, the conclusions of those early epidemiological studies set in motion an argumentative strategy that was

to shape all subsequent inquiry into BSE. That strategy was a form of analogical reasoning, the central premise of which sought to relate BSE in cattle to scrapie in sheep. The warrant that attended that premise is of critical significance in assessing the rational merits of this argument form and of the many decisions and risk assessments that were based upon analogical reasoning throughout the BSE crisis. In order to assess the strength of that warrant, we must first know something about the epidemiological context in which it was developed. A thoroughgoing examination of that context will be the second main task of this chapter.

2.2 The Current Paradigm in Epidemiology

In a recent book, Christakos et al. (2005) undertake a much needed critical examination of the modern science of epidemiology. These authors present a strong case for their claim that the current paradigm in epidemiology is fundamentally flawed. Although I do not wish to rehearse the full details of their argument, its essence can be captured by the following points: (1) the focus of epidemiology is on technical practices rather than on theoretical developments¹; (2) there has been little recognition in epidemiology of the essentially interdisciplinary nature of much public health research²; (3) the current paradigm has neglected to give any emphasis to epistemic cognition notions such as reasoning³; (4) there has been little or no attention given to the spatiotemporal characteristics of epidemics under conditions of uncertainty.⁴ To respond to these weaknesses of the current paradigm, Christakos et al. (2005: 5) propose the development of a synthetic epidemic paradigm (SEP) that will be able to account for the points listed above ‘in a mathematically tractable and epidemiologically thoughtful fashion’. The extent to which SEP succeeds in addressing the shortcomings of the current paradigm is the topic of another discussion. In the present context, I want to consider how the model of scientific reasoning that will be presented throughout this book can respond to these features and how a much greater role for philosophy within epidemiology is envisioned as a result.

2.2.1 Theoretical Development in Epidemiology

A chief concern of Christakos et al. is the lack of theoretical development within epidemiology. This theoretical void has been filled by a proliferation of studies whose main concern is the use of techniques.⁵ Kaplan (2004: 127) states that:

[T]here has been considerable lament regarding the ascendancy of technique over theory in epidemiology and its separation from basic foci of public health. Indeed, development of epidemiologic theory per se, separate from techniques for analyzing causal effects and partitioning sources of noise in data may be required.

In the absence of theoretical development, epidemiology has become preoccupied with conducting studies of associations. Many of these associations are weak and never replicated. Their combined effect has been to bring about an erosion of

public trust in the state of the science (Taubes 1995). Certainly, the findings of these various studies get us nowhere in the absence of an underlying theory that is able to offer some explanation of these associations. In relation to his own field of social epidemiology,⁶ Kaplan (2004: 128) remarks that:

[T]he spatial scope over which determinants of specific outcomes such as all-cause mortality, infant mortality, or cardiovascular disease, or other outcomes, operate is less clear. Without some theory to suggest the ways in which the determinants of these outcomes are spatially embedded, one is left with a plethora of analyses of “area” effects that seem more often driven by the level of spatial data available, or other arbitrary factors, than by reasoned etiologic considerations.

The paucity of theoretical labour in epidemiology has adverse implications for the knowledge base of the discipline. When that knowledge base is impoverished, our ability to engage in scientific explanations and predictions is considerably diminished. Yet, these are the very activities that scientists must undertake in order to mount an effective response to a newly emerging infectious disease. To predict the behaviour of an epidemic across space-time, for example, scientists must draw upon theoretical frameworks in a range of disciplines, including mathematics, systems engineering, molecular biology, toxicology, climate change and demography (Christakos et al. 2005). Where these frameworks are lacking or are otherwise poorly developed, the epidemiologist must approach the task of prediction with the burden of uncertainty. It is this burden that the epidemiologist aims to minimize through the development of ever more powerful models of reasoning.⁷

The model of scientific reasoning that I will be proposing in this book is simultaneously realistic and optimistic about the place of uncertainty in inquiry. On the one hand, it recognizes that uncertainty is an unavoidable feature of scientific inquiry.⁸ On the other hand, it believes that uncertainty can be brought within manageable limits. Each of the argument forms that will be demonstrated throughout this book serves to contain uncertainty by bridging specific gaps in our knowledge. These gaps may ultimately come to be ‘filled in’ as results from experimental studies begin to emerge. Until such time as these results are forthcoming, however, reasoning strategies that include circular arguments, arguments based on analogy and arguments from ignorance perform a useful facilitative function, that of advancing inquiry on a tentative basis. To appreciate how this is possible in the case of one of these arguments, the argument from ignorance, one need only consider one of the many ways in which this argument was used during scientific inquiry into BSE. The lack of evidence that scrapie had transmitted to humans in the 250 years it had been present in British sheep populations was taken to support the proposition that scrapie was not transmissible to humans. This proposition was something very much worth knowing, as it was used in subsequent assessments by scientists of the risk that BSE posed to human health. A renewed emphasis on theoretical developments within epidemiology is essential if the knowledge base of this scientific discipline is to be expanded. But an equally important part of this emphasis must be the development of innovative ways of reasoning from the incomplete knowledge bases of epidemiology and related disciplines.

2.2.2 *Epidemiology and Other Disciplines*

As the above example of epidemic prediction demonstrates, the theoretical frameworks that inform the scientific activities of explanation and prediction come from a range of disciplines. Interdisciplinarity is at the heart of the SEP that Christakos et al. wish to see replace the current paradigm in epidemiology.⁹ According to these authors, ‘important sources of knowledge available in the physical and life disciplines are ignored at the cost of profoundly inadequate epidemic and human exposure studies’ (2005: 4). One discipline above all others that influences how Christakos et al. approach epidemiology is philosophy. These authors reflect very deeply on questions that are essentially epistemological in nature. They argue, for example, that ‘regardless of how technical or formal [public health scientists’] research may be, they will always need to gain intellectual access to issues such as the nature and reliability of knowledge, the conception of reality, the reasoning mode, and the underlying methodological assumptions’ (2005: 66). To this extent, public health scientists may find themselves operating as ‘applied philosophers’.

Nor are Christakos et al. alone in envisioning a role for philosophy in epidemiology. While epidemiological studies have little difficulty demonstrating associations between risk factors and disease, only some of these associations are genuinely causal relationships. The strong tendency of epidemiologists to interpret even weak associations as causal relationships suggests that something may be wrong with the way these scientists conceive of the notion of causality.¹⁰ Rothman (2002: 15–16) states that how this notion is applied within science in general, and epidemiology in particular, is a question of interest to philosophers of science:

[H]ow do we go about determining whether a given relation is causal? Some scientists refer to checklists for causal inference, and others focus on complicated statistical approaches, but the answer to this question is not to be found either in checklists or in statistical methods. The question itself is tantamount to asking how we apply the scientific method to epidemiologic research. This question leads directly to the philosophy of science.

Philosophy has a further intellectual contribution to make to discussion within epidemiology. As the critical discipline *par excellence*, philosophy has a key role to play in addressing scientists’ concerns that epidemiology has for many years experienced a grievous lack of critical analysis. For example, Phillips (2008: 62) remarks that ‘epidemiology is characterized by the cranking out of thousands of new research reports per month, with little attempt at critical analysis’.¹¹ In an earlier article, Phillips et al. (2004: 2) state that:

It is troubling that we plow ahead with billions of dollars worth of research every year while making minimal effort to answer fundamental questions about what the research is really telling us. Epidemiology is far too important to our society to be treated as an exercise in uncritically following existing formulae.

Clearly, epidemiologists must engage in a critical way with the very standards and methods that are integral to their own discipline. If critical analysis can reverse the largely uncritical bent of epidemiology to date, then philosophy is the obvious

place to seek out such analysis. Trainee epidemiologists would do well, for example, to engage with the critical thinking courses of philosophy students. For it is in such courses that significant questions concerning types of arguments and standards of evidence are routinely examined and debated.¹² Christakos et al. (2005: 64) appear to concur with this emphasis on critical thinking when they state that ‘critical thinking systems based on the close interaction between concepts and experimental fact may be more valuable in public health research and development than belief-based systems’.

It emerges that philosophy has much to offer the study of epidemiology. From epistemology to philosophy of science and critical thinking, philosophy undoubtedly has a strong contribution to make to this scientific discipline. Christakos and others are to be congratulated for recognizing the significance of that contribution. In the chapters to follow, however, I want to go much further than these investigators by extending philosophy’s contribution to epidemiology to that sub-discipline of logic that is concerned with the study of informal fallacies. In navigating the logical terrain between rationally acceptable and fallacious forms of argument, informal logicians are uniquely placed to address issues of uncertainty in reasoning. Some consideration of the task of argument evaluation reveals why this is the case (we will return to argument evaluation in later chapters). A central tenet of the presumptive and pragmatic frameworks that are at the heart of the model of reasoning presented in this book is that the boundary between rational acceptability and fallaciousness in the study of argument is not pre-ordained and absolute but is subject to variation on the basis of features of context. Uncertainty is a significant feature of epistemic context and, as such, must enter into the task of argument evaluation for the informal logician. To the extent that epidemiologists must also address issues of uncertainty, most notably during scientific inquiry, there is much that these scientists can glean, I will argue, from the critical insights of informal logic. Certainly, this particular parallel between the disciplines of informal logic on the one hand and epidemiology on the other hand will be very much in evidence in the arguments presented in this book.

2.2.3 Reasoning and Epidemiology

Beyond challenging the dearth of theoretical research and lack of genuine interdisciplinarity in epidemiology, Christakos et al. mount another revealing attack on the current paradigm. This paradigm, they argue, effectively neglects the contribution of epistemic cognition notions to the study of epidemiology:

The solution of mathematical models of epidemics has been viewed as a purely *ontologic* affair that focuses on abstract and dry formulas, whereas crucial factors – such as modes of perception and reasoning, and their integration – are neglected. Thus, what the current perspective is missing is that these models are imperfect constructs of the human mind, often they do not account for essential site-specific knowledge, and they constitute an uncertain representation of reality (2005: 4; italics in original).

The emphasis given to our cognitive procedures in Christakos et al.'s epistemic cognition framework¹³ is at once refreshing and instructive. It is refreshing because for too long epidemiology has viewed our cognitive procedures with more than a little distrust. This is evident in the proliferation of formal and statistical techniques which, it is argued, deliver nature to us objectively – something that the current paradigm in epidemiology appears to have assumed our own cognitive resources are powerless to do.¹⁴ It is interesting to note that the epidemiologist is acting on the same impulse as the knowledge sceptic, who positively urges us not to accept what our cognitive procedures (e.g. perception) tell us about the world. That impulse leads both the epidemiologist and the knowledge sceptic to view our cognitive procedures as being irretrievably flawed for no other reason than they are prone to error. And a cognitive procedure that commits errors (so the argument goes) is a cognitive procedure that cannot be trusted to deliver knowledge to us. However, to dispense with our cognitive procedures on the ground that they are prone to error is to overlook the facts that these procedures can and do serve us well and that they are the basis of our many epistemic successes.¹⁵ The emphasis on cognitive procedures within Christakos et al.'s epistemic cognition framework is also instructive. For whatever epidemic models and solutions we set about developing, they are ultimately judged to be acceptable and plausible by standards that are implicit in our cognitive procedures:

[T]he solution of [an epidemic] system should follow certain rules of reasoning. But these rules are, in the final analysis, propositions about one's epistemic cognition process. The reasoning rules leading to a solution in uncertain space-time domains do not constitute an independent ontologic entity, but they are rather implicit in the epistemic cognition process that enforces them (Christakos et al. 2005: 22).

As well as urging the epidemiologist to relocate the focus of study on the cognitive procedures that generate mathematical solutions to epidemics, Christakos et al. believe those solutions should be judged by cognitively attainable standards. A similar cognitive reorientation is envisaged in the model of reasoning based on informal fallacies that will be advanced in this book. The philosophical study of fallacies has displayed the same neglect of the cognitive domain that Christakos et al. believe characterizes the current paradigm in epidemiology.¹⁶ This neglect has had rather unfortunate consequences. Despite fallacies being committed by arguers and detected (or not, as the case may be) by other arguers, the presence of a thinking agent has been largely subordinated to other issues in discussion of the fallacies. Moreover, fallacies have been evaluated according to standards that are set apart from our cognitive procedures.¹⁷ In aiming to locate fallacies firmly within a wider cognitive framework, I propose to take seriously a claim by John Woods (2004: xxvi) that 'an account of fallacies needs to be set in a more general theory of cognitive agency'. When recast in cognitive terms, many informal fallacies lose the appearance of fallaciousness that has engendered so many centuries of philosophical rejection.¹⁸ Quite apart from being 'improprieties of rational performance' or 'failures of one or another of our basic rational survival skills' (Woods 2004: 10–11), the fallacies can be shown to have positive survival value for the cognitive

agents who possess them. Of course, these arguments can still misfire and cause agents to pursue unproductive and (occasionally) catastrophic courses of action. Nevertheless, they confer sufficient epistemic advantage on any agent that possesses them to warrant their continued inclusion in what Woods calls our ‘rational survival kit’.

2.2.4 Spatiotemporal Factors and Epidemiology

Spatial and temporal factors play a significant role in the spread of any infectious disease. The widespread geographical distribution (space) of early BSE cases and the distribution of cases by month and year of onset (time) led epidemiologists to conclude that the epidemic had an extended common source (i.e. contaminated meat and bone meal). Spatial factors were of vital importance in the SARS outbreak of 2003. We now know that the outbreak in many locations began with ill travellers coming from SARS-affected areas and that healthcare settings played an important role in amplifying outbreaks (Lingappa et al. 2004). With the ongoing threat to human health from avian and swine influenzas,¹⁹ spatial and temporal considerations are integral to the epidemiological models that scientists are using to predict the behaviour of pandemic human influenza. These models predict, for example, that for effective control of this disease to be achieved, antiviral prophylaxis would have to be started within approximately 3 weeks of the first human-to-human transmissions and within two days of the onset of a new case once an outbreak is underway. This control strategy, Smith (2006) argues, would be extremely difficult to achieve in rural Southeast Asia.

Beyond models of infectious diseases, spatial and temporal factors are equally important to disease modelling in other branches of epidemiology. Christakos et al. (2005: 7) state that ‘in the case of environmental epidemiology, the adequate description of the geographical and temporal distribution of the exposures (toxic chemicals, radioactive materials, etc.) to which the population has been subjected is a crucial component of any modelling study’. Krieger (2001: 694) places similar emphasis on spatiotemporal factors in social epidemiology when she states that ‘[a]dequate epidemiological explanations . . . must account for both persisting and changing distributions of disease, including social inequalities in health, across time and space’.

Notwithstanding the centrality of spatiotemporal factors to the study of disease processes, it is clear that epidemiological investigations have not always acknowledged these factors. Christakos et al. (2005: 4) believe that the neglect of spatial and temporal factors represents one of the ‘significant limitations of the current epidemic paradigm’:

Mathematically rigorous and epidemically meaningful stochastic tools (e.g., spatiotemporal random field theory) have been ignored in favor of deterministic methods and classical statistics techniques that neglect vital cross-correlations and laws of change on space-time manifolds.

Kaplan (2004: 126) is equally critical of diagrammatic models in social epidemiology in which ‘[i]nformation is seldom available at the multitude of levels portrayed in such models and, where available, is often measured cross-sectionally, making the *temporal influences* that we consider so important in the assessment of causality opaque’ (italics added). Similarly, Holmes (1997: 111) bemoans the neglect of spatial considerations in epidemiological theory when she states that:

[M]ost classical epidemiological theory glosses over the spatial dimension of disease transmission and instead assumes that every individual is equally likely to contact every other. A key question is to what extent do we lose insight or are quantitatively misled by modelling the intrinsically spatial process of disease spread with nonspatial theory.

Krieger (2003: 384) attempts to explain the loss of ‘place’ in epidemiology and proposes a possible remedy in the form of geographic information system technology:

[D]espite epidemiology’s longstanding concern with “time, place, and person” (or, perhaps more accurately, “time, place, and population”), “place” had receded into the background by the mid-20th century, conceptually unmoored from increasingly influential etiologic frameworks based on characteristics of the individual. Fortunately, geographic information system technology has contributed in recent years to a reviving awareness that any epidemiologic explanation worth its salt must encompass geographic – and temporal – variations in population health.

The model of reasoning that will be presented in this book attempts to address this neglect of spatiotemporal factors by directly integrating these factors within the rational evaluation of the argument forms that were used by scientists during the BSE inquiry. The temporal dimension is particularly significant to such evaluation. It is to be expected, for example, that the epistemic context in which scientists conducted inquiry changed over time as more became known about BSE and as the results of experimental studies began to emerge. This context was thus dynamic and evolving rather than static and immutable as the epidemic raged on in the years during the late 1980s and early 1990s. Accordingly, the reasoning strategies that served scientists well at one time point in inquiry might be expected to become redundant at a later time point. To address this essential temporal dimension of the BSE problem, a chronological approach will be adopted when examining the reasoning of scientists. It is of interest to this study, for example, that a particular argumentative strategy was used intensively by scientists in the early weeks and months of the BSE epidemic when little was known about this new brain disease in cattle. But it is equally interesting to establish if this strategy persisted across the time course of the epidemic and, if not, how it was revised or discharged. The temporal dimension is integral to epidemic modelling in epidemiology and public health science. It will become evident to the reader that temporal considerations had an equally significant role to play in the reasoning of scientists during the BSE epidemic.

2.3 Early Epidemiological Investigations

It was described above how reasoning based on an analogy between BSE in cattle and scrapie in sheep was an integral part of the argumentative strategy that shaped scientific inquiry into BSE. This analogy had its origin in the early epidemiological investigations that were conducted into BSE and specifically a study that was undertaken by Wilesmith et al. (1988). This study sought to relate this new brain disease in cattle to scrapie in sheep and it is to it that we must turn in order to establish the strength or otherwise of this emergent analogy. Our task will be the reconstruction of the different arguments that these investigators used to support the claim that BSE and scrapie were essentially related diseases. These arguments are inter-linked in revealing and complex ways (see Diagram 2.1). One of these arguments has the status of a main or primary argument. The conclusion of this argument is the analogical thesis that motivated much subsequent reasoning during the BSE crisis. Other arguments are subarguments in that their conclusions serve as premises within the main or primary argument. These subarguments developed a number of independent lines of evidence that converged on the same primary argument. Having reconstructed these arguments, our next task will be their rational evaluation.²⁰ We cannot proceed to assess the rational merits of the different analogical arguments that were in evidence during the BSE inquiry if we don't first establish the rational standing of the analogical thesis that is the main premise of these arguments. This latter, evaluative task will reveal the tentative, presumptive nature of the early reasoning of BSE scientists.

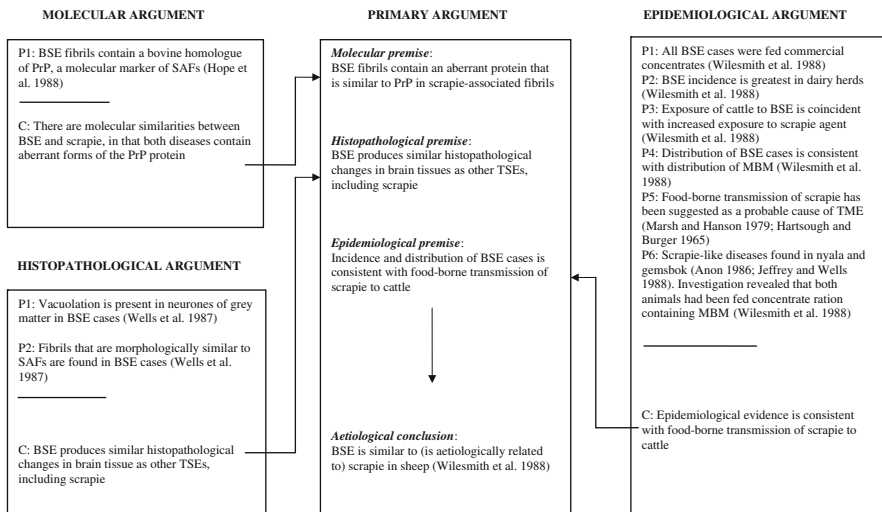


Diagram 2.1 Reconstruction of the arguments linking BSE to scrapie

The epidemiological study conducted by Wilesmith et al. (1988) was initiated in June 1987. This study undertook to obtain descriptive epidemiological, including genetic information, about early BSE cases; to monitor the incidence of BSE within herds and nationally; to collect data on the duration of the disease and the frequency of clinical signs, and to develop hypotheses about the aetiology of the disease. It is this last aspect of the study that is of particular significance to us as we seek to reconstruct the reasoning that led John Wilesmith and his colleagues to conclude that there were ‘aetiological similarities’ between BSE and scrapie in sheep. It is important to be clear from the outset about what this claim of aetiological similarity meant to these investigators. Wilesmith et al. were not merely stating that BSE was similar to scrapie in the sense that both diseases belonged to the group of TSEs (although this statement is clearly a corollary of their claim of aetiological similarity). Rather, these investigators were making the more specific claim that the infectious agent that causes scrapie in sheep had transmitted via a feed source to cattle and was now causing BSE. This specific claim of aetiological similarity, I will argue subsequently, was ‘strengthened’ during the BSE crisis to become a claim of aetiological identity (BSE *is* scrapie in cattle). This ‘bovine scrapie’ thesis and its weaker ‘similarity’ counterpart (BSE *is similar to* scrapie in sheep) assumed the status of explicit and implicit premises²¹ in the reasoning of BSE scientists. Certainly, these theses formed the cornerstone of much analogical reasoning during the BSE inquiry.

In 1988, when Wilesmith et al. were advancing their claim of aetiological similarity, it could not be directly validated. Specifically, strain-typing studies, which were to reveal that the BSE ‘signature’ was distinct not only from scrapie but also from other naturally occurring TSEs, had yet to be completed.²² Consequently, Wilesmith et al. were forced to fall back on a number of less direct sources of evidence. One such source was histopathological evidence presented in a paper by Wells et al. (1987).²³ This evidence consisted of two findings based on the investigation of



Fig. 2.1 BSE positive cows. The vacant stare, low head carriage and a wide-based hind limb stance are typical of the disease. Behavioural changes in temperament (e.g. nervousness or aggression), abnormal posture, incoordination and difficulty in rising, decreased milk production, and/or loss of weight despite continued appetite are followed by death in cattle affected by BSE (Published with the permission of the Controller of Her Majesty’s Stationery Office and the US Department of Agriculture, Animal and Plant Health Inspection Service (APHIS), respectively)

pathological material obtained from four herds with BSE cases: (1) the presence in the grey matter of neuronal degeneration that most often involved intracytoplasmic vacuoles and (2) the electron microscopic identification of fibrils that morphologically closely resembled scrapie-associated fibrils (SAFs).²⁴ (Figs. 2.2 and 2.3). It is clear that Wells et al. took these histopathological features to suggest a ‘similarity’ or ‘resemblances’ between BSE and encephalopathies caused by unconventional infectious agents in other species:

There are strong *resemblances* between this disorder of cattle and the unconventional viral agent encephalopathies recorded in several species (1987: 420; italics added).

[T]he authors regard this sporadic, slowly progressive, neurological disorder of adult cattle, characterised by grey matter vacuolation and scrapie associated fibrils, as compelling provisional evidence of *similarity* with diseases caused by unconventional infectious agents (1987: 420; italics added).

Yet, there are at least two significant ways in which these ‘similarity’ claims differ from those that were later advanced by Wilesmith et al. (1988). Firstly, Wells et al. did not attempt to draw any aetiological connection with scrapie in particular – the similarity in question was with TSEs in general. Secondly, although these resemblances were described as ‘strong’ and evidence of similarity was ‘compelling’, Wells et al. were also keen to stress the largely tentative nature of these early conclusions about the aetiology of BSE. Their caution is signalled through the emphasis of the ‘provisional’ nature of the evidence. Also, they remarked that ‘[a]dditional evidence is required. . .to establish the true nature of this entity’ (Wells et al. 1987: 420). Finally, these investigators stated that ‘[i]t should be emphasised that at present the aetiological basis of bovine spongiform encephalopathy remains

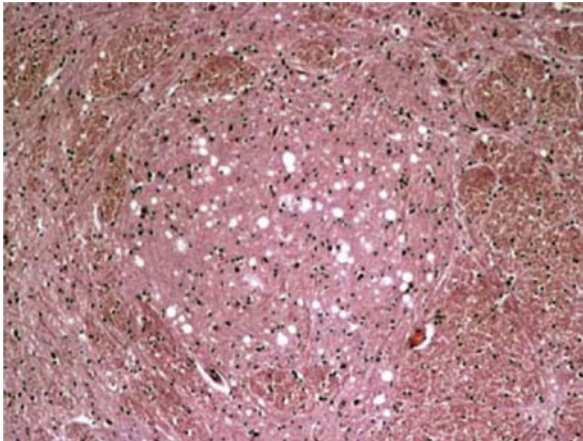


Fig. 2.2 BSE positive-neuropil vacuolation in the nucleus of the solitary tract (bovine brainstem at the level of the obex). © Crown copyright 2011. Published with the permission of the Controller of Her Majesty’s Stationery Office. The views expressed are those of the author and do not necessarily reflect those of Her Majesty’s Stationery Office or the Veterinary Laboratories Agency or any other government department



Fig. 2.3 Scrapie-associated fibril, magnification X 39,000. © Crown copyright 2011. Published with the permission of the Controller of Her Majesty's Stationery Office. The views expressed are those of the author and do not necessarily reflect those of Her Majesty's Stationery Office or the Veterinary Laboratories Agency or any other government department

unknown and no connection with encephalopathies in other species has been established' (1987: 420). The tentativeness and generality of Wells et al.'s 'similarity' claims are indicative of their rather low epistemic standing. Certainly, these claims had an altogether lower epistemic standing than the 'similarity' claims that were subsequently endorsed by Wilesmith and his colleagues. Histopathological evidence provided Wilesmith et al. with a rational basis upon which to begin developing an analogy between this new brain disease in cattle and scrapie in sheep. Yet, this evidence was still some way off establishing the more specific aetiological link with scrapie that emerged from Wilesmith et al.'s early epidemiological investigation. Additional evidence was needed in order to move from Wells et al.'s claim of similarity of BSE with TSEs in general to Wilesmith et al.'s claim of similarity with scrapie in particular.

Such evidence was to emerge from a number of other sources. One of these was a molecular study conducted by Hope et al. (1988). These investigators demonstrated that the fibrils, which had appeared morphologically similar to SAFs in Wells et al.'s study, did indeed contain a protein that is the bovine homologue of PrP, a molecular marker of SAFs.²⁵ This finding helped to strengthen the developing aetiological connection of BSE to scrapie: 'Our protein data...confirm the homology of this novel cattle disorder and scrapie' (Hope et al. 1988: 392). Further confirmatory evidence was obtained from Wilesmith et al.'s own epidemiological study. In order to establish the claim of aetiological similarity between BSE and scrapie, Wilesmith et al. needed to make a case for the claim that scrapie had transmitted to cattle. If scrapie had transmitted to cattle to cause BSE, then there were only two main routes by means of which transmission could have occurred. They included (1) direct or

indirect contact between scrapie-affected sheep and cattle and (2) the consumption of scrapie-affected sheep tissues by cattle. Research into TSEs prior to 1986 had demonstrated that these diseases could be spread through direct and indirect contact between diseased and healthy animals both within and across species. In this way, scrapie could be transmitted between sheep through contaminated pastures and enclosures (see [Section 1.2.4](#)). Also, elk had been found to develop chronic wasting disease following sporadic fence-line contact with clinically affected deer and the use of pens in which such deer were kept (see [Section 1.2.6](#)). Yet, to the extent that 20% of farms with BSE did not have sheep (Wilesmith et al. 1988), route (1) appeared an unlikely means of transmission and was rejected by these investigators.

A food-borne route of transmission did, however, receive support from several findings. Firstly, all BSE cases for which accurate records existed were at some time fed commercial concentrates, either as protein supplements used in home mixed rations or as finished rations such as pelleted calf feed and dairy cow cake. Secondly, Wilesmith et al. found a greater incidence of BSE cases in dairy herds (311 of 44,767) compared to beef suckler herds (11 of 54,166). The animals in the former herds received more commercial concentrates than the animals in the latter herds. Thirdly, a computer-based simulation model indicated that the exposure of cattle began in the winter of 1981/1982. This date is coincident with a number of developments which increased the exposure of cattle to the scrapie agent via feedstuffs.²⁶ Fourthly, the widespread geographical distribution of BSE cases is consistent with the distribution of meat and bone meal in commercial feedstuffs. The geographical variation in BSE incidence was not, however, consistent with the distribution and use of tallow in these feedstuffs. Scrapie-like agents were known to be associated with cell membranes and were expected during the rendering process to partition with the cellular residues of the meat and bone meal fraction rather than the lipids of tallow. Each of these findings served to increase the plausibility of the food-borne hypothesis of the transmission of scrapie to cattle.

Additional supportive warrant for this hypothesis was provided by the appearance many years earlier of a spongiform encephalopathy in ranch reared mink. Hartsough and Burger (1965) described several outbreaks of the disease between 1947 and 1963. They remarked that '[w]e feel that in the 1961 and 1963 episodes there is reasonable evidence of a food-borne exposure. . .we may even assume that tissues of bovine or perhaps ovine origin are possible sources for the as yet hypothetical contagion' (Hartsough and Burger 1965: 392). Later, Marsh and Hanson (1979: 460) confirmed that TME had resulted from the oral transmission of scrapie to mink: '[T]here appear to be no major obstacles remaining to the probable fact that TME originates from feeding mink scrapie-infected tissues'. The development of a spongiform encephalopathy in mink provided Wilesmith et al. with an important precedent for the food-borne transmission of the scrapie agent. Certainly, this finding constituted a further, plausibility-raising thesis in support of the claim that scrapie had transmitted to cattle via a food source. The same could be said of the discovery in a wildlife park in England of a scrapie-like disease in a nyala and a gemsbok (MAFF/ADAS 1986; Jeffrey and Wells 1988), both of which had been fed

a concentrate ration containing meat and bone meal (Wilesmith et al. 1988). Like the finding before it, Wilesmith et al. could contend that if scrapie had transmitted via a food source to infect these species,²⁷ it could do so again to cause BSE in cattle.

These additional findings complete the task of the reconstruction of the argumentation that was used by Wilesmith and his colleagues to support their claim of aetiological similarity between BSE in cattle and scrapie in sheep. As can be seen from Diagram 2.1, this claim relied on multiple sources of evidence from studies that separately examined the histopathology, molecular biology and epidemiology of this new disease. Individually, these sources carried little probative weight. Collectively, I will argue that they conferred sufficient rational warrant on Wilesmith et al.'s aetiological claim to give that claim legitimacy as a presumptive truth in subsequent scientific inquiry into BSE. This latter of our two tasks is an evaluative one. Its outcome will determine the rational credentials of the analogical thesis that was integral to much scientific reasoning about BSE.

Not every source of evidence brought forward by Wilesmith et al. to support their claim of aetiological similarity conferred the same degree of warrant on this claim. We have already seen how Wells et al.'s histopathological findings only went as far as supporting a claim of similarity to TSEs in general, rather than scrapie in particular. By themselves, these findings could not support the more specific aetiological connection of BSE to scrapie that Wilesmith et al. were attempting to draw in their early epidemiological study. Accordingly, these investigators had to bring forward other forms of evidence that would raise the plausibility of their aetiological claim by suggesting a specific aetiological link between BSE and scrapie in sheep. This other evidence included the results of a molecular study by Hope et al. as well as Wilesmith et al.'s own epidemiological findings. These latter findings included the following four claims:

- (1) All BSE cases were fed commercial concentrates.
- (2) BSE incidence is greatest in dairy herds.
- (3) Exposure of cattle to BSE is coincident with increased exposure to the scrapie agent.
- (4) Distribution of BSE cases is consistent with distribution of meat and bone meal.

Of these claims, only the first two could be established with any certainty. Claim (3) was based upon a number of factors which Wilesmith et al. (1988: 643) described as being 'undoubtedly significant in the occurrence of this epidemiological phenomenon'. Yet, upon close examination, the significance of at least two of these factors may have been somewhat overstated. The two factors in question were (i) the introduction of continuous rendering processes during the 1970s and 1980s (the effect of which was to reduce the temperature and length of time at which animal material was rendered) and (ii) the decline in the practice of using hydrocarbon solvents and terminal heat treatment for fat extraction since the mid 1970s. Both factors relate to the rendering of animal material for commercial products, including cattle feedstuffs. However, it is clear from what was known about rendering

practices on the one hand and the extraordinary resistance of the scrapie agent on the other hand that no rendering process was probably ever capable of inactivating TSE agents. During the BSE Inquiry, Lord Phillips and his team established that up until the 1970s, temperatures during rendering were not recorded or monitored systematically, that they could be very variable and that the absence of proper managerial control meant it was possible to discharge material before it was completely cooked (BSE Inquiry Report, Volume 13: 90). These various lapses in practice almost certainly resulted in a failure to deactivate the scrapie agent. Also, considerable evidence existed by 1988 to show that the scrapie agent failed to be deactivated by temperatures of 100°C or greater (Brown et al. 1982; Kimberlin et al. 1983; Rohwer 1984). Both considerations served to weaken Wilesmith et al.'s claim that changes in rendering practice played a significant role in increasing the exposure of cattle to the scrapie agent.²⁸ Claim (3), it emerges, was not as strongly warranted as it first appeared.

Claim (4) was also largely tentative in nature. When Wilesmith et al. were advancing their aetiological hypothesis, little was known about the exposure of cattle to meat and bone meal, the ingredient in commercial concentrates that was believed to be the source of infection. In April 1988, in response to a request from Mr Meldrum (Director of the Veterinary Field Service) to produce a report on BSE, Mr Wilesmith responded: 'In brief we have no conclusions to explain why BSE should have occurred from the findings of the various investigations on the feed-stuffs industry thus far' (BSE Inquiry Report, Volume 3: 70). Even at the point of publication of their epidemiological findings in December 1988, Wilesmith et al. (1988: 643) indicated that their investigations of the feed industry, and the production and use of MBM in commercial feedstuffs in particular, were still some way off completion. These investigators stated that '[s]tudies are in progress to determine more precisely the exposure of affected and unaffected animals to meat and bone meal in commercial concentrates'. It is a presupposition of this statement that the exposure of animals to meat and bone meal had *not* been precisely determined by the stage at which these investigators were advancing their claim of aetiological similarity between BSE and scrapie. Lord Phillips and his inquiry team identified some reluctance on the part of investigators to make an early approach to the feed industry to enquire about its practices in general and MBM in particular. Such an approach, it was argued, could lead to a concealment of information.²⁹ For this reason, confidential enquiries were made on an informal basis to individuals with industry experience, not all of it recent.³⁰ These 'off the record' meetings meant that Wilesmith and his colleagues were unable to confirm their food-borne hypothesis for some months after this hypothesis began to be seriously entertained by investigators.³¹ Claim (4), it can be seen, was still subject to empirical validation even as it was being advanced by Wilesmith et al. in support of their aetiological link between BSE and scrapie.

As well as epidemiological findings, Wilesmith et al. used the results of Hope et al.'s molecular study of bovine fibrils to support their claim of aetiological similarity between BSE and scrapie. Certainly, this study reveals similarities between the major protein of BSE fibrils and the PrP protein that is a molecular marker of

scrapie-associated fibrils. In this way, BSE fibril proteins were similar in protease sensitivity and size to PrP in SAFs that were purified from the brain of a Cheviot sheep with natural scrapie. Also, the N-terminal 12 amino acids in bovine PrP were identical to those found in sheep SAF protein and differed by one amino acid from mouse, hamster and human PrP. The aetiological significance of these molecular similarities is somewhat doubtful, however. By 1988, studies demonstrating molecular similarities between the proteins in different transmissible spongiform encephalopathies were commonplace. In this way, Bendheim et al. (1985) found that CJD proteins had molecular weights similar to those observed for scrapie prion proteins. These investigators also remarked that '[p]urification of the two infectious pathogens by virtually identical procedures provided further evidence for similarities in their molecular structures' (1985: 997). Kitamoto et al. (1986) found that antisera raised against hamster scrapie PrP stained amyloid plaques in the brains of humans and rodents with CJD and a human subject with Gerstmann-Sträussler syndrome. Bockman et al. (1985) reported a similar reaction to antibodies raised against the scrapie prion protein in proteins from the brains of two patients with CJD. These investigators stated '[o]ur findings suggest that the amyloid plaques found in the brains of patients with Creutzfeldt-Jakob disease may be composed of paracrystalline arrays of prions similar to those in prion diseases in laboratory animals' (1985: 73).³²

Molecular similarities between scrapie prions and prions in CJD and GSS certainly provide sufficient warrant for the claim that these diseases all belong to the group of transmissible spongiform encephalopathies caused by unconventional agents: 'We conclude that the molecular and biologic properties of the CJD agent are sufficiently similar to those of the scrapie prion protein that CJD should be classified as a prion disease' (Bendheim et al. 1985: 997). But this nosological relationship between these prion diseases is something quite different from the aetiological relationship between BSE and scrapie that Wilesmith et al. are seeking to support through Hope et al.'s molecular findings. The latter relationship does not merely seek to relate one prion disease (BSE) to another prion disease (scrapie). Rather, it goes one step further in asserting that the agent that causes scrapie in sheep is also causing BSE in cattle. It is this more specific aetiological relationship that is not supported by findings of molecular similarities between the prion proteins that cause BSE and scrapie. To see this, one need only consider studies such as that of Bendheim et al. which established molecular similarities between prion proteins in CJD and scrapie prions. These molecular similarities did not lead these various investigators to suggest that the scrapie agent had transmitted to humans to cause CJD. Indeed, by 1988 there was a developing scientific consensus that scrapie had not transmitted to humans to cause CJD despite extensive opportunity for it to do so.³³ In this case, molecular similarities between CJD prion proteins and scrapie prions led to the (warranted) conclusion that these diseases were nosologically related in the absence of any wider claim to the effect that scrapie had transmitted to humans to cause CJD. A similar situation, I am arguing, pertains in the case of Hope et al.'s findings of molecular similarities between prion protein in BSE fibrils and prions in SAFs. These molecular similarities certainly support a nosological relationship between BSE and scrapie. However, they are much less able to support the type of

aetiological relationship between these diseases that Wilesmith et al. are attempting to advance.

We have now evaluated the histopathological, molecular and epidemiological evidence that was used by Wilesmith et al. to support their claim of an aetiological relationship between BSE and scrapie. Each of these strands of evidence, we have seen, is problematic in one or more respects. In some cases, essential knowledge was lacking (e.g. knowledge of the exposure of cattle to meat and bone meal). In other cases, greater significance was attributed to certain facts than a thoroughgoing assessment of these facts would have permitted (e.g. the overstated significance of changes in rendering practices). These various problems serve to weaken the supportive warrant that particular strands can confer on Wilesmith et al.'s aetiological conclusion. Yet, these individual strands and their attendant problems are less important to determining the supportive warrant of this aetiological conclusion than is the plausibility-raising capacity of these strands considered collectively.³⁴ With each additional source of evidence, Wilesmith et al.'s claim of aetiological similarity between BSE and scrapie grew in epistemic stature. The epistemic evolution of this aetiological claim was at all possible because this claim had the status of a presumption, an epistemic category which, although tentative in nature, can improve its standing during inquiry. We will examine presumption in detail in the next chapter, where it will be seen that this notion is particularly well equipped to deal with the adverse epistemic conditions that characterize scientific inquiry in its early, emergent stage. The focus of this chapter, however, has been on an account of the evidential processes that were used by Wilesmith and his colleagues to forge an aetiological connection between BSE and scrapie, a connection that was to have a profound influence on all subsequent reasoning during the BSE crisis.

Notes

1. 'In many cases of public health research, the emphasis is solely on *data gathering* (experimental, observational, surveillance, etc.) and the black-box operation of the *techniques/instruments* employed for this purpose, without any appreciation of the kind of substantive *theoretical* modelling that underlies these techniques/instruments and gives voice to the data' (Christakos et al. 2005: 5; italics in original).
2. 'Little attention has been given to the *interdisciplinary* nature of epidemic research and development. In this manner, important sources of knowledge available in the physical and life disciplines are ignored at the cost of profoundly inadequate epidemic and human exposure studies' (Christakos et al. 2005: 4; italics in original). Clearly, Christakos et al. subscribe to the view that epidemic modelling requires the integration of knowledge bases from several disciplines. Savitz et al. (1999: 1159) make a similar point in relation to public health work in general when they remark that '[t]he phrase "the basic science of public health work" implies that the entire knowledge base underlying public health comes from epidemiology. . . it should be acknowledged that many other sciences serve public health as well. Among the basic public health sciences are clinical medicine, sociology, toxicology, molecular biology, anthropology, nutrition, sanitary engineering, policy analysis, risk assessment, industrial hygiene, economics, and political science'.
3. 'The solution of mathematical models of epidemics has been viewed as a purely *ontologic* affair that focuses on abstract and dry formulas, whereas crucial factors – such as modes of perception and reasoning, and their integration – are neglected. . .' (Christakos et al. 2005: 4; italics in original).

4. 'The fundamental *spatiotemporal* character of an epidemic under conditions of uncertainty has been mostly neglected. Intrinsically spatiotemporal phenomena, like disease propagation, are often modelled with "aspatial" and "aspatiotemporal" theories. . . This neglect has resulted in unsatisfactory analyses of major issues such as space-time prediction of disease distribution, epidemic explanation, and causation' (Christakos et al. 2005: 4; italics in original).
5. In a 2003 interview with the eminent epidemiologist Mervyn Susser for the journal *Epidemiology*, Susser is asked for his opinion of the current state of health of epidemiology. His response conveys his concern that the field has become preoccupied with a focus on methods: 'I have been writing a fair amount about this over the past dozen years, starting with "Epidemiology Today: 'A Thought-Tormented World.'" I felt that we were at cross-purposes in formulating what epidemiology was. The academic center of the field was diverging from its concern with substance into the minutiae of methods. Not to say that methods are not essential, but to make methods a principal activity, to the neglect of the object of the activity, seems to me a travesty' (Paneth 2003: 750).
6. The field of social epidemiology involves the 'examination of the role of a broad array of social factors in the development and progression of many important health problems, and in the natural history of the risk factors for those diseases and conditions' (Kaplan 2004: 124).
7. For Christakos et al. (2005: 68), stochastic modelling is the method of choice for dealing with multiple sources of uncertainty: '... in our view stochastic modelling is the primary conceptual and operational apparatus for studying fundamental uncertainties of the type happening in public health research, in general, and epidemics, in particular'.
8. That uncertainty is unavoidable in scientific inquiry is recognized by both scientists and philosophers. Christakos et al. (2005: 43) remark that '[u]ncertainty, in its various forms, is at the center of many scientific investigations and debates. Indeed, in the last few decades scientists have moved from seeing nature as inherently stable and deterministic to viewing it as uncertain, subject to unexpected shifts and changes. . .'. In this extract, Christakos et al. attribute uncertainty to nature. In Rescher's philosophical account, uncertainty, in the form of incompleteness of knowledge, stems from the process of inquiry itself: 'Certain fundamental features inherent in the very structure of man's inquiry into the ways of the world thus conspire to indicate the incompleteness of the knowledge we can attain in this sphere' (1980: 237). In Chapter 3, we will consider different sources of uncertainty during inquiry.
9. 'If the development of SEP is going to produce rigorous rules for the integrated modelling of knowledge from different disciplines and levels of organization, it must rely on an adequate understanding of scientific intradisciplinarity and interdisciplinarity in an epidemic assessment context' (Christakos et al. 2005: 14).
10. Of a paper by Adams et al. (2003) and responses to it by economists, epidemiologists and others in the same publication, Kaplan (2004: 126–127) remarks that 'one is left with the impression that a number of the authors believe that the analysis of causal relations in observational data is so flawed as to potentially threaten even the conclusion that smoking causes lung cancer'.
11. Phillips et al. (2004) include the 'critical analysis' of epidemiology as one of the areas in which the new online journal *Epidemiologic Perspectives & Innovations* welcomes submissions.
12. Weed (1995: 916) sees the development of critical thinking skills among epidemiologists as one of the key benefits to emerge from a greater alignment of epidemiology with the humanities: 'The ability to think critically is important to scientists. It is a broader ability than the concept of criticism alone, encompassing explanation, logic, creativity, and inquisitiveness. Critical thinking is useful for identifying bias, for finding hidden assumptions in causal criteria, and for developing new theories of disease causation that explain joint exposure effects. . . Critical thinking can be developed directly by studying the works of philosophers promoting it'. It is clear that an educational effort of the sort described in the main text is already underway in some public health circles. In a Technical Report prepared by the American Public Health Association (APHA), it is stated that 'APHA should continue efforts

- to strengthen training in schools of public health in the analysis of scientific evidence on public health problems' (APHA 1990: 749).
13. 'In the epistemic cognition framework, the contribution of cognition is to identify basic knowledge-assimilation, belief-forming and problem-solving processes, which are then examined by means of the evaluative standards of epistemology' (Christakos et al. 2005: 5).
 14. '[A]nother potentially significant departure of SEP from the traditional epidemic paradigm is the thesis that an epistemic cognition solution (which assumes that the relevant models describe incomplete knowledge about the epidemic and focuses on cognitive mechanisms) can lead to more adequate results than the conventional ontologic solution (which assumes that the models describe nature *as is* and focuses on *form manipulations*)' (Christakos et al. 2005: 19–20; italics added).
 15. In developing a logic of error, Gabbay and Woods (to appear) subscribe to a form of fallibilism. They state that '[f]allibilism would be a harsh and pessimistic doctrine, and a stupid one, if it didn't embed a further pair of assumptions. . . The one assumption says that the frequency with which we commit errors has not shut us down as would-be knowers; far from it. The other says that, notwithstanding the errors in which they land us, it is to the procedures in question that we owe our epistemic successes. They are not only the best that we can do, but they serve us rather well'.
 16. This neglect is a consequence of the concern not to conflate psychology with philosophy in the study of fallacies. In this way, Kahane (1980: 38) remarks that: 'Well, then, do we overstep the bounds of logic and philosophy when we theorize about fallacious reasoning. Not, it seems to me, when we attempt to specify what fallacious reasoning consists in, nor when we specify the logical factors which make fallacious reasoning fallacious. These are questions of methodology, and thus of logic and philosophy. But we do overstep when we attempt to specify psychological mechanisms that lead to fallacious reasoning, and when we devise psychological categories useful in avoiding fallacious reasoning'. A theorist who explicitly acknowledges a role for cognition in the study of fallacies is Dale Hample (1982, 1985, 1988). Hample (1982: 59) states that 'a message can only stimulate a fallacy; the actual fallacy is a *cognitive event*' (italics in original). For further discussion of Hample's views, see Cummings (2004b).
 17. Fallacy theorists have often advanced standards which actively eschew any role for the thinking agent (here, the user of argument) in argument evaluation. This can be seen in the following views of Biro and Johnson (the latter summarized by Walton):

Both these approaches [Perelman's rhetorical approach and Hamblin's dialectical approach], in spite of their great interest, share the flaw we have seen to be fatal in Sanford's treatment: relying, instead of on the necessary argument-relativity of the notion of knowability, on the essentially user-relative notions of assent and acceptance (Biro 1977: 270).

Johnson's claim is that mere "acceptance" or "effectiveness" in causing a listener to accept something she did not accept before is too weak a standard to do the job of providing a normative model of argument to help with analyzing fallacies (Walton 1993: 308).

- At the root of such criticisms lies the concern that by assigning any significance to the user or recipient of an argument in judgements of argument acceptability, we are losing all sense of the objective from argumentative discourse.
18. Hansen and Pinto (1995) bring together the work of some of the key thinkers down the centuries who have engaged with the fallacies. Starting with Aristotle (384–322 B.C.), they include the authors of the Port-Royal Logic, Antoine Arnauld (1611–1694) and Pierre Nicole (1625–1695), John Locke (1632–1704), Isaac Watts (1674–1748), Richard Whately (1787–1863) and John Stuart Mill (1803–1874). A further historical overview of the fallacies can be found in Charles Hamblin's 1970 book *Fallacies*.

19. See Lipsitch et al. (2009) for discussion of the uncertainty confronting scientists during the recent emergence of the influenza A (H1N1) virus that is responsible for swine flu.
20. As presented here, it seems that the tasks of argument reconstruction and evaluation are conceptually and temporally distinct activities with evaluation only occurring after the process of reconstruction has taken place. In reality, however, reconstruction and evaluation are interrelated stages in argument analysis. The task of argument reconstruction is already dependent on evaluative analysis, for example, when decisions concerning missing premises are made. We only accept as missing premises in an argument those claims which contribute to the *deductive validity, plausibility or rational acceptability* of an argument. And such notions are inherently normative and thus evaluative in nature.
21. An 'implicit' premise is also known by the terms 'missing' premise, 'tacit' premise, 'unstated' premise and 'hidden' premise. The standard role of these premises, according to Govier (1987: 82), is to 'fill inference gaps'.
22. Bruce et al. (1997: 499) state that '[t]he BSE "signature", based on both incubation periods and pathology, has only ever been seen in transmissions from animals suspected or known to have been infected with BSE. It has never been seen throughout an extensive series of transmissions, set up in Edinburgh between 1963 and 1994, of other naturally occurring TSEs (35 sheep and two goats with scrapie, two mink with transmissible mink encephalopathy, and a mule deer with chronic wasting disease)'.
23. Wells et al. (1987: 420) were the first investigators to call this new brain disease in cattle 'bovine spongiform encephalopathy': 'Pathological and epidemiological studies, analyses of genetic data and experimental transmission studies have been initiated but until further characterisation is achieved the authors suggest for this disorder the provisional appellation: bovine spongiform encephalopathy'.
24. Patricia Merz was the first scientist to identify SAFs (Merz et al. 1981). Using electron microscopy, Merz demonstrated the presence of SAF 'in all combinations of strain of scrapie agent and strain or species of host examined, regardless of their histopathology' (1981: 63).
25. 'In this report, we show the major protein of BSE fibrils is the bovine homologue of PrP as judged by its size, protease resistance, immunoreactivity, lectin binding and partial N-terminal protein sequence' (Hope et al. 1988: 390).
26. 'A number of factors have been identified which when combined are undoubtedly significant in the occurrence of this epidemiological phenomenon. These include: a dramatic increase in the sheep population in Great Britain which commenced in 1980 and has continued; a probable increase in the prevalence of scrapie infected flocks; the greater inclusion of sheep heads in material for rendering; the greater inclusion of casualty and condemned sheep in material for rendering as a result of the reduction in the number of knackers' yards; the introduction of continuous rendering processes during the 1970s and 1980s which may have resulted in the rendering of animal material at a lower temperature and, or, a shorter time than previously and the decline in the practice of using hydrocarbon solvents and terminal heat treatment for fat extraction since the mid 1970s' (Wilesmith et al. 1988: 643).
27. It should be emphasized that food-borne transmission of scrapie to these species is not suggested in the paper by Jeffrey and Wells (1988) or by the Report of the Chief Veterinary Officer (MAFF/ADAS 1986). In fact, Jeffrey and Wells (1988: 399) only appear to consider early feeding practice and direct and indirect (via paddocks) contact between this nyala and sheep (as well as mink and deer) as possible routes for the transmission of scrapie: 'This nyala had no direct contact with sheep or goats, and its paddock had not previously been used by those species. It was bottle-reared on domestic cow's milk by an attendant with responsibility for a small number of sheep grazed at a different location, but scrapie has not been reported in these sheep. Neither mink nor North American species of deer are kept at the wildlife park'.
28. This same point was made by Lord Phillips and his team when they remarked that '[t]he theory that BSE resulted from changes in rendering methods has no validity. Rendering methods have never been capable of completely inactivating TSEs' (BSE Inquiry Report, Volume 1: xix).

29. The Head of the Pathology Department and BSE research coordinator at the CVL, Mr Bradley, felt that an early approach to the feed industry would result in concealment of information. When asked on 27 November 1987 if an approach should be made, he responded '[n]ot yet. We must first identify the right questions to ask and we have more data to collect yet. We also need time to think. If we approach too early concealment of information is likely' (BSE Inquiry Report, Volume 3: 61).
30. In a written statement to Lord Phillips and his team, Mr Rees (Chief Veterinary Officer, 1980–1988) remarked that 'in these early stages we did manage to have some very informal discussions with a few advisers who we knew on a more personal basis, to determine the extent of the use of MBM in commercial feedingstuffs, and the distribution of MBM. These were really very informal chats as no one wanted to meet formally with MAFF at this stage' (BSE Inquiry Report, Volume 3: 63). Mr Rees also told the inquiry team 'of a single conversation that he had, on a personal and confidential basis, with an adviser to a feed manufacturer. While willing to assist, the adviser was not able to give very sound information as to the content of animal feed 4 or 5 years in the past' (BSE Inquiry Report, Volume 3: 63).
31. From evidence given to Lord Phillips and his team, it is clear that Mr Wilesmith was more interested in contaminated vaccine than feed as a possible source of infection in June 1987 (BSE Inquiry Report, Volume 3: 60). However, by December 1987 Mr Wilesmith was 'sufficiently confident' that a feed-borne source was responsible for BSE (Volume 3: 62). Yet, it was 3 months later before feed industry representatives even became aware of a suspected link between feed and BSE. In this way, the UK Agricultural Supply Trade Association told the inquiry team that they did not learn of such a link until March 1988 (Volume 3: 61). Similarly, Mr Paul Foxcroft of Prosper De Mulder (the leading renderer in England and Wales) told Lord Phillips and his team 'that he was absolutely certain that he was not aware of any suggestion of a link between BSE and animal feed until March 1988' (Volume 3: 63). The time lag between Mr Wilesmith's suspicion of a link and an opportunity to validate this suspicion through intensive investigation of the feed industry is responsible, I am arguing, for the lack of empirical validation of Wilesmith et al.'s food-borne hypothesis even by December 1988, when these investigators' epidemiological findings were published.
32. Other studies in the same vein include Baron et al. (1988) who demonstrated immunoreactivity to rabbit antiserum raised against SAF protein in scrapie-infected mice and two familial cases of transmissible dementia. Manuelidis et al. (1985) tested protein blots of human, guinea pig and hamster CJD fractions with an antibody raised against a 29-kDa band from mouse scrapie. In all CJD and scrapie fractions, 29-kDa proteins were labeled. Bode et al. (1985) used antisera raised in rabbits and mice against SAF protein from hamster brain to test SAF proteins from hamster and mouse and from CJD. The antisera detected five bands in a Western blot analysis with molecular weights of 26, 24, 20, 18 and 16 K. By gel electrophoresis, these antigens seem to be identical in hamster, mouse and man.
33. A study by Brown et al. (1987), which concluded a 15-year investigation of CJD in France and reviewed the world literature, found no evidence whatsoever of a link between scrapie in sheep and CJD in humans. These investigators stated that although numerous cases of CJD had been observed in rural sheep-raising areas in Italy, Czechoslovakia and Chile, scrapie had not been recognized in sheep for at least a century in the latter two countries while there was no evidence that the Italian cases had been exposed to infected animals. In France, no increased risk of CJD was observed in people who were most exposed to sheep or sheep products, nor was there any relationship between CJD frequency and the distribution routes of sheep products from scrapie-endemic areas. CJD cases had occurred in France in people who had never eaten lamb or other sheep tissues and a life-long vegetarian in England also developed the disease. Furthermore, scrapie had been assiduously barred from Australia, where CJD occurs with the usual frequency, and Japan also experienced CJD cases even though scrapie had not been known until 1981.
34. This account differs from Rescher's (1976) analysis of plausible reasoning in two significant respects. Firstly, Rescher (1976: 60–61) adopts a deductive approach to plausibility: 'The

presently envisaged approach to plausible inference thus proposes to assess the plausibility of a “merely plausible” piece of reasoning in terms of the plausibility of the added enthymematic premisses needed to transform it into a valid deductive argument’. As will become clear in [Chapter 3](#), the model of reasoning that I am advancing in this book is essentially non-deductive in nature. Secondly, Rescher (1976: 61) states that the plausibility of an argument’s conclusion consists in the maximum plausibility among the various minimum plausibilities of the supplemental premisses that are needed to turn the argument into a valid deduction: ‘the plausibility of the argument is to be the maximum value among the minima of the plausibilities of the enthymematic supplementations that enable a deductive derivation of the conclusion from the premisses’. My own model of reasoning adopts a cumulative approach to plausibility wherein no single premise determines the plausibility of the conclusion of an argument. Rather, all premisses contribute to the raising of the plausibility of a conclusion, with high plausibility premisses making a greater contribution to the plausibility-raising of the conclusion than low plausibility premisses.