**The Influenza Problem: Paradigms, a Pandemic, and the Search for Pfeiffer’s Bacillus.**

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September 2021

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# **Abstract**

This dissertation challenges existing histories of the 1918-19 influenza pandemic which vilify the work of contemporary scientists for believing that a bacteria, *B.influenzae*, was the cause of flu. We now know influenza to be caused by a virus, but in 1918 medical thought was dictated by the bacteriological paradigm, which asserted that a bacteria was always the cause of infectious disease. However, in 1918 the bacteria which was ‘known’ to be the cause of influenza was not reliably found in sick patients. Doctors continued to search for the bacteria, even as a number of other aetiological agents, including a filter-passing virus, were proposed as alternative causes of the disease. Historians, including Mark Honigsbaum, see this preoccupation with *B.influenzae* as evidence of the scientific community being a prisoner of the bacteriological paradigm. Using Thomas S. Kuhn’s framework of scientific development, this dissertation undertakes a study of two British medical journals, *The Lancet* and the *British Medical Journal*, in order to ascertain the characteristics of the scientific community in the pandemic and post-pandemic periods, including how far they can be said to adhere to or move away from the paradigm which underpinned their work. It will conclude that rather than representing a moment of paradigm imprisonment, contributions to the two journals alongside contemporary medical literature instead demonstrate a period of re-evaluation and questioning that fits Kuhn’s model of ‘revolutionary science’. This will impact how historians conceptualise scientific development, particularly within the fields of bacteriology and virology, during the pandemic period.

# **Acknowledgments**

Writing about one global pandemic in the midst of another presented a number of challenges, and as with all large bodies of work (created in COVID conditions or otherwise) it could not have been done without help. My heartfelt thanks therefore go to the following:

To all the staff of the British Library and Wellcome Collection who have worked tirelessly to bring researchers back to their reading rooms.

To my fellow SASiety Committee Members and all the attendees of the online writing retreats. Spending our afternoons working ‘together’ over the last eight months has fostered a sense of community that I could have only wished for when my research began.

To the Office of National Statistics, for inadvertently paying for many of my academic books by admitting me as a COVID-19 Infection Survey participant.

To my family for their unwavering support and their willingness to listen patiently as I enthused, raged, and sobbed about my research in equal measure. To Mimi for our secret conversations, and to Scarlett for the companionship and laptop assistance.

And to my friends, who have cheered me up and cheered me on over a difficult year. Particular thanks to L. Beavis for the medical discussions and countryside walks, to P. Glover for keeping me sane and always sending encouragement at just the right moment, and to A. Aizenman, without whom I never would have encountered Kuhn, let alone understood his work!

My research focuses on scientific divisions during a devastating pandemic. I am grateful for every scientific advancement in the current pandemic produced by a unity towards a common goal.

# **Glossary of Medical Terms**

**aetiology.** The cause (or causes) of a disease, including the causative agent. Can also be spelt ‘etiology’.

**bacillus.** Plural Bacilli. Rod shaped bacteria, for example Pneumobacillus.

**bacteria.** Single-celled organisms.

**bacteriology.** The study of bacteria and bacterial diseases.

**blood agar.** An enriched medium used to cultivate bacteria for the purposes of study or diagnosis. It is often used to grow ‘fastidious’ organisms that do not grow well on other mediums.[[1]](#footnote-1)

**catarrh.** A build-up of mucus in your throat and nose, often caused by an infection in the mucus membranes. Flu symptoms commonly include catarrh.

**coccus**. Plural Cocci. Spherical shaped bacteria, for example Streptococcus.

**culture.** The method by which micro-organisms are grown on a suitable medium from a sample in order to facilitate identification and diagnosis.

**epidemic.** An outbreak of a disease that is widespread in an area, community, or region at the same time.

**Gram positive/Gram negative.** Refers to the outcome of a microbiological staining technique used to identify bacteria. Depending on the colour of the stain, scientists can ascertain the type of bacteria present.

**host.** An organism which harbours a smaller organism, providing it (often involuntarily) with nourishment and shelter. Viruses require living cells within a host organism in order to replicate.

**influenza.** A contagious respiratory illness caused by an RNA virus. Colloquially known as ‘flu’, or in 1918, ‘grippe’.

**medium.** A semi-solid, solid, or liquid matter used to grow microorganisms under laboratory conditions. Agar is an example of a solid medium used in microbiological culture.

**naso-pharynx**. a part of the upper-respiratory tract behind the nasal cavity and extending to just above the top of the throat. Nasopharyngeal swabs are often taken to test for influenza.

**pandemic**. an outbreak of a disease that is widespread in multiple countries or continents at the same time, impacting the majority of the population.

**pathogen**. Any infectious organism which causes disease in its host.

**pathogenicity.** The capability of an infectious organism to cause disease.

**pleomorphism.** The existence of irregular or variant forms of a specific type of microorganism. Different sizes or shapes are examples of pleomorphism.

**prophylaxis.** Treatment given to prevent infection, onset, or severity of symptoms. Vaccines are a prophylactic measure against influenza.

**pulmonary.** Of the lungs.

**sputum.** Liquid excretion from the respiratory tract. Usually coughed up, comprised of mucus and saliva.

**virology.** The study of viruses and viral diseases.

**virus.** An infective agent that can only multiply within a host’s living cells. Too small to be seen under a microscope.

**virulence.** The degree of damage caused by an infectious pathogen.

# **Introduction**

The early June editions of the 1918 *British Medical Journal* brought news of an ‘epidemic of an acute catarrhal infection in Spain.’[[2]](#footnote-2) This epidemic, mentioned first on June 1st and again on June 8th, attracted the attention of the *BMJ* as its death toll rose, with discussions of the disease’s aetiology dominating its written accounts. The symptoms described on June 1st deemed it ‘not improbable’ that the ‘acute catarrhal disease is what we are accustomed to speak of as ‘influenza’’, and by June 8th the disease was ‘clearly of the character of influenza’ in both symptoms and prognosis.[[3]](#footnote-3) This certainty, however, was juxtaposed by an inconvenient fact: in the cases studied, influenza wasn’t always found.

The June 8th report ends by stating that ‘before coming to any conclusion, it is obvious that further bacteriological information is awaited’. Specifically, the *BMJ* was awaiting confirmation thatPfeiffer’s Influenza Bacillus had been found in sampled cases, or if it had not been, that another bacteria could instead be identified as the epidemic’s cause. Pfeiffer’s Bacillus *(b. influenzae)* had been identified by German Richard Pfeiffer in 1892, and between then and 1918 it had been near-unanimously accepted as the cause of flu within medical circles. When a new, flu-like disease emerged in 1918, the search for Pfeiffer’s Bacillus therefore dominated investigative efforts into the disease’s origins. Found in some cases but not in others, sometimes as the dominant bacteria, other times in near negligible amounts, the illusive influenza bacteria puzzled doctors the world over even as they insisted on its link to the pandemic they were experiencing.

Medical historian Mark Honigsbaum sees this insistence as evidence of ‘medical researchers [becoming] prisoners of particular paradigms and theories of disease causation’, and bemoans the millions of young lives lost and ‘many research hours wasted’ from the medical profession being blinded by Pfeiffer’s bacillus as the pandemic’s cause.[[4]](#footnote-4) My dissertation will interrogate this position. Utilising Thomas. S. Kuhn’s framework of scientific development, I will explore the nature of the existing bacteriological paradigm within understandings of influenza before examining the extent to which the 1918-19 pandemic can be seen as falling victim to the paradigmatic rigidity of existing influenzal and bacteriological knowledge. I will prove that rather than representing a moment in which the scientific community was blinded by the existing paradigm, contemporary British medical journal publications demonstrate that the 1918-19 pandemic forced a re-evaluation of the existing paradigm and introduced a period of questioning and experimentation that fits Kuhn’s model of ‘revolutionary science’. By recharacterizing the nature of bacteriological work during the pandemic period, my findings will also provide new insight into post-pandemic attitudes towards influenza’s aetiology and to the eventual paradigm shift caused by the discovery of the influenza virus.

The 1918-19 Influenza Pandemic caused at least 50 million deaths and infected an estimated 500 million worldwide.[[5]](#footnote-5) In three main waves, what we now know to be an RNA virus of the genus *Orthomyxoviridae* swept around the world, causing what Howard Phillips and David Killingray termed ‘the single worst demographic disaster of the twentieth century’ which was ‘without doubt…the most devastating infection to strike the world since the Black Death’.[[6]](#footnote-6) In Britain, influenza was responsible for at least 151,000 deaths, the majority of which were young adults (aged between 20-40) rather than the more traditionally impacted elderly and very young.[[7]](#footnote-7) Of those afflicted, many experienced what we would still recognise as typical influenza symptoms (fever, runny nose, body aches and fatigue), however in serious cases doctors were faced with patients exhibiting severe pulmonary distress, most strikingly visible in a distinct ‘purple-blue tinge to the face’ known as heliotrope cyanosis.[[8]](#footnote-8) These alarming symptoms, combined with the unusual demography of patient mortality and the absence of any kind of effective treatment, brought what was known about influenza under increased scrutiny both during and in the aftermath of the pandemic. Within these discussions, no aspect of was questioned more than that of influenza’s aetiology.

Juxtaposing this, within the historiography of the pandemic influenza’s cause is the one aspect of which there is no doubt. The influenza virus was isolated first in pigs in 1931, and then as a virus able to infect humans in 1933.[[9]](#footnote-9) As a result, the majority of historical engagement with the pandemic (traced by Phillips and Killingray as having ‘a relative lull for almost 40 years’ following the pandemic, with a series of ‘spasmodic bursts of interest’ producing ‘sharp spikes’ in the 1950s and 1960s, continuing in an upwards curve from the 1970s to the present day) understood influenza solely in virologic terms.[[10]](#footnote-10) This includes Alfred W. Crosby’s seminal work on US pandemic experiences ‘*Epidemic and Peace’* (later republished as ‘*America’s Forgotten Pandemic: The Influenza of 1918*) in 1977, Phillips and Killingray’s 2003 edited volume based on papers presented at the inaugural conference on the 1918-19 Pandemic held in 1998, multiple regional histories of the pandemic (including Niall Johnson’s work on demographic impacts in Britain and Ida Milne’s response to the ‘lacuna’ of the pandemic in Irish historiography), and a growing number of popular histories (see John M. Barry’s 2004 ‘*The Great Influenza: The Story of the Deadliest Pandemic in History* and Laura Spinney’s 2017 ‘*Pale Rider: The Spanish Flu of 1918 and How It Changed the World’*).[[11]](#footnote-11)

The application of modern influenza knowledge to understandings of the 1918-18 pandemic has produced several important cross-disciplinary works on the cause and origins of the pandemic, including Jeffery K. Taubenberger’s pivotal 2006 report on the sequencing of the 1918 virus from preserved tissue, and John S. Oxford and Douglas Gill’s 2019 analysis of contemporary medical reporting of an outbreak of influenza at Aldershot in 1917 informed by modern virologic knowledge.[[12]](#footnote-12) However the application of modern knowledge has also clouded understanding of pre-virology reactions to the pandemic within historical accounts, a trend Michael Bresalier terms as the ‘ahistorical tendency to retrospectively *viralise* the pandemic’.[[13]](#footnote-13) Bresalier uses the phrase in specific reference to the lack of recognition for ‘the historicity of the relationship between influenza, the pandemic and virus research’ within understandings of influenza research in the inter-war period, however the detrimental impacts of ‘viralisation’ are also visible in histories of the 1918-19 pandemic.

The most visible impact of ‘viralisation’ is the dismissive attitude towards contemporary medical science within histories of the pandemic. Just as Honigsbaum views adherence to the Pfeiffer bacillus cause of flu as a ‘misconception’ which caused ‘many research hours [to be] wasted’ before it was eventually ‘corrected’, Mitchell Hammond judges medical understandings of influenza during the pandemic as ‘impotent’, whilst George Denher states, in a chapter of his history of influenza titled ‘Misidentifications and False Starts’, that the ‘medical establishment’s fundamental understanding of the illness was faulty’.[[14]](#footnote-14)

Alfred Crosby gives arguably the most scathing critique of medical attitudes and actions during the pandemic. Focussing on US experiences, Crosby labels ‘the physicians of 1918’ as ‘participants in the greatest failure of medical science in the twentieth century, or, if absolute numbers of dead are the measure, of all time’.[[15]](#footnote-15) Bresalier is particularly apprehensive of these types of retroactive judgements, even as he recognises it as the norm that ‘historians treat Pfeiffer’s bacillus as the wrong aetiological agent around which an entire generation organised its knowledge’.[[16]](#footnote-16) To relegate the search for Pfeiffer’s Bacillus, or any other non-virological cause of flu, to the realm of mistakes, misunderstandings or, as Tom Quinn puts it, ‘excitement and optimism’ for something which ‘proved to be a dead end’, risks ignoring the ‘significance of the bacillus and bacteriology’ within medical understandings of influenza in 1918-19.[[17]](#footnote-17)

The second impact of ‘viralisation’ also serves to distort our understanding of the bacteriological world of flu. Working with the chronological omniscience gained from knowing when the influenza was discovered (and from that when the paradigm shift in influenza knowledge occurred), historians commonly write viral histories of the influenza pandemic which place the 1918-19 pandemic as a stepping-stone on the way to later virological advancements. Certainly, there is room for debate on the extent to which the pandemic provided an impetus for increased research into viruses (known as ‘filter-passers) in the interwar period, as Ton van Helvoort’s article on post-pandemic bacteriological and virological advances and Bresalier’s multiple articles on influenza and virus research show.[[18]](#footnote-18) However for the majority of engagement with the pandemic’s link to the development of virology this connection is not made critically. Rather the present knowledge that influenza research eventually led to a virus causes historians to place often undue emphasis on the place of virus research during the pandemic year.

This is evident most overtly in Tom Quinn’s history of flu, which divides influenza’s history into two phases based upon virological advancements. Phase two starts with the ‘devastation’ of the 1918 pandemic before moving on to a period of ‘enlightenment’ once the virus was identified in the 1930s.[[19]](#footnote-19) Positioning the 1918 pandemic within the ‘enlightenment’ phase, rather than the ‘first phase of complete ignorance’ demonstrates the intrinsic link in Quinn’s mind between the 1918 pandemic and the development of virology as a discipline.[[20]](#footnote-20) His brief comment on aetiological debates during the pandemic also reflects this link, with his oversimplified insinuation that by the end of 1918 medical opinion was united in the belief that ‘an unknown and unfilterable pathogen was responsible’ for the pandemic being immediately followed by a paragraph on what we *now* know about influenza’s origin.[[21]](#footnote-21) Throughout, the emphasis in on the influenza virus, despite its discovery being over a decade after the end of the pandemic.

Quinn is by no means alone in this blinkered view of the pandemic’s aetiological history. Laura Spinney’s popular history of influenza, ‘*Pale Rider: The Spanish Flu of 1918 and How It Changed The World,’* presents a similar disparity of focus on virological versus bacteriological debates in 1918, with just two pages dedicated to bacteriological debates, compared to an entire chapter on virus research transitioning from the pandemic to the interwar period.[[22]](#footnote-22) Both Niall Johnson’s history of Britain’s experiences in the pandemic, and Michael B. A. Oldstone’s chapter on influenza in ‘Viruses, Plagues and History’compound medical investigation in the pandemic with post-pandemic viral studies, whilst Phillips and Killingray’s decision to include both a historian’s and virologist’s foreword to their edited collection exemplifies the dominance of viral knowledge within discussions of historical influenza.[[23]](#footnote-23) In the latter case, the inclusion of virologist perspectives can be ascribed to the intended interdisciplinary reach of the conference which inspired the edited collection. However, following Bresalier’s reasoning, it still needs to be asked why a bacteriologist was not also allowed to offer comment, seeing as influenza in 1918 was understood overwhelmingly in bacteriological terms.

The way in which the ‘viralisation’ of 1918-19 pandemic histories shifts the narrative of the pandemic towards one of virological enlightenment also conveniently aligns discussions of medical activity with the ‘triumphalist brand of medical history’ associated with the bacteriological advances of the 19th century and the successes of wartime medicine in reducing mortality among troops.[[24]](#footnote-24) Phillips and Killingray see the relative absence of the pandemic from historical enquiry up until the 1970s as partially caused by the pandemic’s medical ‘failures’ amounting to ‘an enormous rout in the war against disease for the medical profession’ which did not fit the general view of medical progress over disease in the period.[[25]](#footnote-25) Framing the pandemic as a key step towards a future scientific triumph resolves this issue. John M. Barry’s overtly ‘viralised’ reframing of scientific work in the pandemic is a key example of this. Barry lionises the work of American doctors and scientists during the pandemic, seeing them as ‘warriors’ who ‘concentrated on constructing the body of knowledge necessary to eventually triumph’.[[26]](#footnote-26) Laura Spinney’s choice to name Part 6 of her book, which starts with her chapter on virus research, ‘Science Redeemed’ also reflects this reframing effort.[[27]](#footnote-27) Through the redemption of the pandemic’s viral links, the contemporary ‘failures’ of modern medicine’s workers can be cleared of their black marks.

My dissertation does not intend to vilify, lionise or redeem 1918-18 scientific efforts. Instead, it will respond to Bresalier’s call for a move beyond ‘viralised’ historical study in order to draw focus to the complexities of scientific research and influenzal knowledge-building during the pandemic. By questioning the extent to which the existing paradigm influenced scientific thought, I will establish the character of the scientific community at the outset of the pandemic, before charting its development as experiences in the laboratory threw doubt on influenza’s aetiological agent. This will situate my work within a relatively niche section of pandemic histories engaged in discussions of influenza’s contemporary aetiology and the community that studied it. Alongside Bresalier’s work, my study will be influenced by van Helvoort’s paper on the bacteriological paradigm in post-pandemic influenza research, and by Eugenia Tognotti’s article on ‘scientific triumphalism’ within Italian bacteriological experiences of the pandemic.[[28]](#footnote-28). I will also be engaging with Honigsbaum and Barry’s direct references to paradigms and Kuhn’s theories of scientific development.

My dissertation is organised into three chapters. Chapter 1 outlines Thomas S. Kuhn’s framework of scientific development and establishes my reasoning for utilising it as a heuristic device by which to analyse contemporary scientific responses to the pandemic. Chapter 2 contextualises the discovery of Pfeiffer’s bacillus and establish the nature of the bacteriological paradigm in influenza research from 1889 to pre-pandemic 1918. The first four sections of Chapter 3 provide a detailed analysis of *British Medical Journal* and *The Lancet* responses to the pandemic between June 1918 and December 1919, with the final section exploring post-pandemic medical literature on influenza up until the discovery of the influenza virus in 1933, in order to provide comment on the position of the bacteriological paradigm after the tests of 1918-19. My conclusions will therefore be formed from a consideration of both pandemic and post-pandemic influenza research, producing a nuanced view of the leading aetiological debates within the period which will support my argument that the pandemic exemplifies a period of revolutionary science rather than a stubborn blindness in support of the existing paradigm.

# **Chapter I: Paradigms and Revolutions: Scientific Development and Thomas S. Kuhn.**

Thomas S. Kuhn’s work on paradigms is referenced by multiple historians exploring the impact of the 1918-19 pandemic on medical thought. This section will establish Kuhn’s philosophy of scientific development, before exploring these 1918-19 literature references and concluding on the suitability of Kuhn’s philosophical framework for an investigation into the scientific developments produced by the 1918-19 Influenza Pandemic.

## ***Section I: Kuhn and Scientific Development***

Kuhn, a physicist turned historian and philosopher of science, ‘single handedly changed the currency of the word *paradigm*’ with the publication of his essay, *The Structure of Scientific Revolutions*, in 1962.[[29]](#footnote-29) *Structure*, as it will henceforth be referred, represented the culmination of over a decade’s worth of exploration into the ways in which scientific theories develop, are maintained, and are subsumed by new concepts. Kuhn viewed the natural sciences’ lack of engagement with questions of the fundamental aspects that made up their field as a problem without comparison within the social sciences, and as such proposed a new method of interpreting the process of scientific development in which history could ‘produce a decisive transformation in the image of science by which we are now possessed’.[[30]](#footnote-30) This image had been dominated by a focus on scientific achievements, with historians of science responsible for chronicling the discoveries that constituted these achievements, adding them to the ‘ever growing stockpile that constitutes scientific technique and knowledge.’[[31]](#footnote-31) Kuhn proposed an alternate historical focus in which it is the process of scientific development, rather than the ‘finished’ result, that bears the brunt of historical scrutiny.

By focussing on the practice of science, the definition of what constituted scientific development could be extended to include not just the moments that led to a ‘stockpiled’ discovery, but also those which did not fit existing ideas of scientific ‘fact’. This included views of nature which the ‘development-by-accumulation’ theory of scientific progress labelled erroneous or outdated, but which detailed study revealed to have been comprised of ‘the same sorts of methods… that now lead to scientific knowledge’.[[32]](#footnote-32) In this way, ‘rather than seeking the permanent contributions of an older science to our present vantage’ and disregarding that which did not fulfil that objective, historians of science could ‘display the historical integrity of that science in its own time’.[[33]](#footnote-33) This was significant not just for broadening the scope of historical inquiry into the sciences, but also for bringing about a new conceptualisation of scientific development that informed the main body of *Structure*’s thesis.

If old and present science are both considered equally scientific, rather than one being ‘false’ and another ‘true’, then historians must seek to explain how opinion changed from one theory to another, whilst not losing sight of the fact that both sciences represent incommensurable but equally valid views of natural principles. *Structure* sought to offer a solution to this by outlining the method by which science, as a ‘collective enterprise pursued by a community of scientists’ rather than just the individuals responsible for singular discoveries, underwent periods of development and change as new discoveries were made and assimilated into the scientific canon.[[34]](#footnote-34) It was in this process of change and development that Kuhn saw patterns of ‘extraordinary’ and ‘normal science’, and from these observations proposed the use of the term ‘paradigm shift’ to explain the movement from one version of science to the next.

Paradigms, for Kuhn at the time of writing *Structure*, were the ‘examples of actual scientific practice…[including] law, theory, application, and instrumentation’ which ‘provide models from which spring particular coherent traditions of scientific research’.[[35]](#footnote-35) By this he refers to the foundation-setting ability of a paradigm as an exemplar discovery which provide a basis from which all scientific work within the field can be undertaken, so long as those working in the discipline are committed to ‘the same rules and standards for scientific practice’.[[36]](#footnote-36) The concept of paradigms haunted Kuhn for the rest of his career, with critical responses tothe use[s] of the term in *Structure* prompting him to defend its usage through a process of redefinition and clarification, from which paradigms were replaced by the concepts of exemplars and disciplinary matrixes.[[37]](#footnote-37) For the purposes of this study, however, I will be referring to paradigms as Kuhn originally intended within *Structure*.

*Structure* identified two distinct periods of scientific activity, defined as ‘normal’ and ‘extraordinary science. Within normal science, scientists working under a particular paradigm direct their research towards the articulation of ‘phenomena and theories that the [discipline’s] paradigm…supplies’.[[38]](#footnote-38) The paradigm binds the group of scientists together within a common understanding of the world and the methods by which further enquiry into its workings can be understood. Within normal science scientists can answer questions produced by the paradigm, apply the paradigm to new areas of study whilst still being guided by its methods or key beliefs, and work to iron out any of the wrinkles within the original paradigm now that full attention can be directed to it within the discipline formed around it.[[39]](#footnote-39) Normal science is the state of ‘business as usual’. It is not meant to produce ‘major novelties’ instead it exists to ‘add to the scope and the precision with which the paradigm can be applied’ and to solve puzzles provided by the paradigm. As Kuhn articulates, though the paradigm defines the solution to problems posed within its discipline, it does not tell scientists how to get to that point. Puzzle-solving thus allows scientists to work out how the solution is reached, with the paradigm also offering the ‘rules of the game’ to ensure that the solution[s] reachable are defined and the methods in which they are obtained fit the known standards.[[40]](#footnote-40)

In the process of this puzzle-solving, anomalies are encountered that do not fit the existing paradigm. Many of these anomalies are resolved through puzzle-solving or paradigm articulation, however on occasion they cannot be assimilated into the existing paradigm, and the discipline enters a crisis period as its acknowledgement of the anomaly changes the focus of the discipline to resolving the anomalous issue.[[41]](#footnote-41) Within this crisis period, consensus on the existing paradigm is weakened as ‘formerly standard solutions of solved problems are called into question’ alongside the questions posed by the anomaly.[[42]](#footnote-42) In this environment normal science cannot continue, and so instead of a cumulative process of puzzle-solving and information gathering in line with the current paradigm, the discipline instead switches to a period of ‘extraordinary’ or ‘revolutionary’ science in which the discipline ‘search[es] for a resolution to its paradigm problems’ by ‘push[ing] the rules of normal science harder than ever’, and then going beyond these rules to search ‘at random’ for answers for which the existing paradigm does not suggest solutions.[[43]](#footnote-43)

The culmination of this period of extraordinary science is the end of the crisis. Kuhn sees this as occurring in one of three ways:

1. The anomaly at the centre of the crisis is resolvable by normal science ‘despite the despair of those who [had] seen it as the end of the existing paradigm.’[[44]](#footnote-44)
2. The ‘crisis provoking problem’ is recognised as providing a significant challenge to the existing paradigm but scientists ‘conclude that no solution will be forthcoming in the present state of the field’.[[45]](#footnote-45) This often entails the recognition that current methods or tools are unable to provide answers to the crisis generated questions, therefore requiring more ‘developed tools’ at a future point.
3. The crisis cannot be resolved and instead ushers in ‘a new candidate for paradigm’.[[46]](#footnote-46)

The movement from crisis under one paradigm to the introduction of another is a decisive break in the scientific development of the discipline. As Kuhn stresses, the new paradigm is not formed from cumulative insights gained from the normal science of the old paradigm, rather it is a ‘reconstruction of the field from new fundamentals’.[[47]](#footnote-47) These fundamentals may have been provisionally constructed within the period of extraordinary/revolutionary science under the old paradigm, but the transition to a new paradigm will still result in the total transformation of the profession, whose ‘view of the field, its methods and…goals’ will have changed completely.[[48]](#footnote-48) This process of paradigm shift is not instant even when the new paradigm contender has been identified. The new paradigm must be accepted by the majority of the scientific community for the change to occur, and due to the magnitude of the transformation of the community as a result of paradigm shift, the new paradigm has to be proven to be worthy of the switch. Namely, the new paradigm must be seen to be the only way to resolve the crisis-causing problem, and though it will fundamentally change how the discipline operates, it must preserve a ‘a great deal of the most concrete parts of past achievement’. [[49]](#footnote-49)In this way the new paradigm does not force the community to completely start from scratch, rather it transforms the way in which previous discoveries are understood as they are viewed under the context of the new paradigm.[[50]](#footnote-50)

This transformation, for Kuhn, represented a change in world view that made the old and new paradigms incommensurable. He went as far as to state that rather than just a difference in interpretation, operating under the new paradigm was to ‘work in a different world’ to the one that had gone before.[[51]](#footnote-51) Within this, concepts, key terms, and methods take on new meanings as dictated by the new paradigm, with even data attained by the old paradigm being viewed in a new light as it is reinterpreted under the new paradigm’s ‘normal science’.[[52]](#footnote-52) To try and view the data as it was understood under the old paradigm is therefore impossible. Its meaning has changed under the new paradigm, and though a scientist can recognise its previous meaning, it does not fit into the new paradigm’s construction of the world.

To elucidate the point, I call upon the image of a snakes and ladders game. The board represents the scientific community, and the players/counters the scientists. The paradigm is the instruction manual, granting an explanation of the rules of the game, defining the roles of the counters, and describing the board so that a player can recognise the confines of the game and what should occur within it (thus describing the discipline under which the scientific community is unified). In this way the paradigm (instructions) ensure that the scientists (counters) operate within the context of their discipline (the board). If the counters were played outside of the board, or without the instructions, the game would not work. Only with the paradigm can a scientific community develop.

One day the players open the snakes and ladders box and find that a new piece has been added. The piece doesn’t immediately fit the game – *how is it played, what effect is it having on the games’ outcome, how can the counters interact with it*? Sometimes, after a bit of thinking, the extra piece is revealed to have always been in the box, having previously been unnoticed. The anomaly (extra piece) is assimilated into the existing game and Snakes and Ladders can still be played as per the original instructions. However, sometimes no amount of head-scratching can get the new piece to easily fit. The players, through the counters, may try different ways of interacting with the piece, even going outside of the instructions – *perhaps if we play the game with two boards, or add another extra piece, or include more dice rolls?* Occasionally in the process of these experiments, an extra page of the instructions is found, and the anomaly can be assimilated into the existing understanding of how the game is played. Other times, the players acknowledge that there just isn’t a way for the piece to fit the game but decide to research it later – perhaps when they can use their phone to look up what the piece could do – and in the meantime decide to play a ‘normal’ game of snakes and ladders and leave the extra piece in the box. Either the anomaly eventually fits the paradigm, or the scientists admit that though the anomaly exists they cannot yet work to assimilate it or investigate it further within the current technological constraints of their period.

Sometimes though, the players will open the box and find that not only is the new piece still there, but now the instructions have changed. The new piece’s function is now described, and it has fundamentally altered how the game can be played. Now every time the counter falls on a ladder you fall down, and on a snake, you slide up. The old paradigm (snakes down, ladders up) has been replaced, and if you want to play the game you have to play it as the new instructions suggest (this does require the consensus of the group - everyone must agree that the old game didn’t work once the extra piece was found and that the new way to play the game makes more sense. If only a minority don’t agree, maybe they don’t get invited to the next snakes and ladders games night). In this way paradigm shift occurs, and the players (scientists) can no longer view the game (the discipline) in the same way. Incommensurability now exists between the two version of snakes and ladders.

The new version of the game, though, is not built from scratch. Previous understandings of the counters, the board and the general rules of play still exist - *players still know to roll dice to decide movement* - but under the new instructions the players’ understanding of how the counters, board, dice and pieces interact is fundamentally different. Even if both versions use similar concepts, the new game cannot be played by the old rules, and if a player of the new game was to try and talk to a player of the old, they would not reach an easy understanding. They are living, by Kuhn’s principle, in different worlds, even if some elements are recognisable from the previous version.

Of course, science is far more complex than a snakes and ladders game, but the analogy is useful to demonstrate Kuhn’s theories of incommensurability and world view change without entering into what Ian Hacking described as the ‘immense philosophical dogfight’ about the nature of comparable theories and scientific development that followed the release of *Structure*.[[53]](#footnote-53) Beyond its ontological debate generating capacity, Kuhn’s theory of incommensurability between paradigms is also a key facet of his call for a new way of doing history of science. Recognizing that there exists a level of incommensurability between worldviews following a shift in paradigm requires acknowledgement that historians, as well as scientists, working under the new paradigm will not be able to fully understand the thought style existent under the previous one. To attempt to ‘display the historical integrity of [a type of] science in its own time’ rather than ‘seeking the permanent contributions of an older science to our present vantage’ is to Kuhn the ideal outcome of the historiographic revolution he saw as in its early stages in 1962. [[54]](#footnote-54) However this revolution could not occur without the recognition that even with the focus on viewing scientific developments within the context of their period, present day scientific knowledge would still create a barrier between a historian and complete understanding of the past.

This is particularly relevant to my proposed use of Kuhn’s framework within a study of scientific development in the 1918-19 pandemic, as can be demonstrated by first returning to references to Kuhn within existing 1918-19 pandemic literature.

## ***Section II: Kuhn and the 1918-19 Pandemic***

Mark Honigsbaum describes his 2019 work, *The Pandemic Century*, as ‘a book about the way that advances in the scientific knowledge of viruses and other pathogens can blind medial researchers’ to new epidemic threats.[[55]](#footnote-55) Within this he makes specific reference to the bacteriological paradigm as a barrier to developments in virologic understandings of the 1918 flu. This use of paradigms, though not directly referencing Kuhn’s theories, nevertheless applies aspects of Kuhn’s principles, namely that the dominance of the existing paradigm causes scientists (or in this case doctors) confronted with a ‘normal science’ threatening anomaly to double down on existing principles, blame their instruments or the capabilities of their colleagues for not being able to resolve the anomaly within the bounds of the existing paradigm (echoing Kuhn’s assertion that within initial efforts to resolve an anomaly ‘…failure to achieve a solution discredits only the scientist and not the theory…’), and in Honigsbaum’s words ‘become prisoners of particular paradigms’.[[56]](#footnote-56) The structure of *The Pandemic Century* highlights this process, charting ‘how…the outbreak [of the epidemic each chapter focuses upon] undermines confidence in the dominant medical and scientific paradigm, highlighting the dangers of overreliance on particular technologies’.[[57]](#footnote-57)

Just as Honigsbaum implicitly references aspects of Kuhn’s concept of paradigms, so too does his discussion of paradigm prisoners elucidate the need for a more detailed application of Kuhn’s framework to a study of the 1918-18 pandemic. Honigsbaum’s statement that the dominance of the bacteriological paradigm caused ‘many research hours [to be] wasted and millions of young people’ to die echoes sentiments expressed by Alfred Crosby (‘…the physicians of 1918 were participants in the greatest failure of medical science in the twentieth century, or if absolute numbers of dead are the measure, of all time’) which judge the activity of 1918 doctors by understandings formed by the present paradigm.[[58]](#footnote-58) With the awareness of the flu’s true virological origins informed by understandings of viruses under the current paradigm, the actions of doctors in 1918 searching for a bacteriological cause appears to be evidence of the failure Crosby suggested. However, as Bresalier notes, this form of retroactive aetiological diagnosis is not constructive when attempting to understand ‘the significance of [Pfeiffer’s] bacillus and bacteriology’ within contemporary understandings of influenza.[[59]](#footnote-59) Kuhn’s explanation of paradigm incommensurability can guide historians of science towards a non-judgemental view of past ‘failures’, making it useful for a study of the 1918 pandemic.

I propose that Kuhn’s framework of scientific development also offers a useful heuristic device for determining the extent to which the 1918-19 pandemic represented either a challenge to the existing paradigm, or as Honigsbaum suggests, an example of the existing paradigm continued to have over medical knowledge throughout the pandemic.

Within his popular history of the pandemic, John M. Barry makes explicit reference to Kuhn’s work to caution against the approach I am proposing, deeming it unsuitable for a discussion of medical progress during the pandemic period. Incorrectly summarising Kuhn’s theory as being one in which ‘the prevailing paradigm tends to freeze progress’ until the erosion of ‘the foundation of the paradigm’ causes it to collapse and scientists to ‘cast about for a new paradigm’, Barry criticises Kuhn’s concept of scientific progress for failing to reflect the fluidity of scientific development.[[60]](#footnote-60) Within Barry’s interpretation of Kuhn’s framework it is only after the abandonment of the previous paradigm that a new one is ‘cast about for’, a concept which directly opposes Kuhn’s insistence that the rejection of a paradigm without the acceptance of another is to reject science itself.[[61]](#footnote-61) This fundamental misunderstanding of *Structure’*s core concept degrades the quality of Barry’s criticism, however though also based on this misunderstanding, Barry’s issue with applying Kuhn’s concept of the scientific method to the study of medicine does bear some relevance to this study. Barry argues that progress in science is ‘more fluid than Kuhn’s concept suggests’, and that a key part of this fluidity is the ability of the scientific method to move amoeba-like with ‘soft and ill-defined edges’ rather than be restricted to only testing existing hypotheses as defined by the current paradigm.[[62]](#footnote-62) As this section has explored, normal science under Kuhn’s concept of scientific progress is not limited to just testing hypotheses set by the prevailing paradigm, rather it aims at answering a myriad of questions and clarifications left open by the paradigm upon its point of adoption. However even with this element of Barry’s criticism being based on shaking foundations, his identification of the fluidity of scientific development, especially in the context of medicine, and the issues with applying Kuhn’s framework to such a field are reflected in broader discussions of *Structure’*s place in understandings of medical scientific development.

Within his chapter on the ‘History of Science and Medicine’ in *The Oxford Handbook of the History of Medicine*, Staffan Müller-Wille states that Kuhn’s framework of scientific development ‘lends itself with difficulty to being applied to the history of medicine’.[[63]](#footnote-63) His reasoning for this echoes Barry: medicine is largely composed of ‘a pluralism of beliefs and methodologies’ which does not fit Kuhn’s idea of a singular paradigm directed science, and also relies upon ‘borrowing from and communicating with’ other disciplines which under Kuhn’s framework would not be possible due to the incommensurability between paradigm following disciplines.[[64]](#footnote-64) Even in moments where a medical paradigm can be seen, with Robert Koch’s achievements in biology being cited as an example, anomalies are instantly found and reacted to, with the end result of a ‘state of ‘permanent crisis’’ rather than clearly defined epochs of normal and then revolutionary science.[[65]](#footnote-65) Put simply, medicine according to Müller-Wille is too fluid, too composed of what Barry would term ‘soft and ill-defined edges’, to be able to be analysed through a Kuhnian lens.[[66]](#footnote-66)

As a solution to this, Müller-Wille turns to the work of Ludwik Fleck. Fleck, a Polish bacteriologist and immunologist, published his monograph *Genesis and the Development of Scientific Fact* (henceforth referred to as *Genesis*)in 1935, establishing what Wojciech Sady, in Fleck’s entry in the Stanford Encyclopaedia of Philosophy, considers ‘the first system of the historical philosophy and sociology of science’.[[67]](#footnote-67) Kuhn acknowledges the influence Fleck’s work had on his own concept of scientific development, both in *Structure* and in the forward to the English translation of *Genesis*, and though he did not consider Fleck to be a principial impact on the development of *Structure*, stating ‘I am not sure that I took anything much more concrete [than the realisation that scientific revolutions had a sociological component] from Fleck’s work’, Muller-Wille highlights the trend within the philosophy of science to compare the two accounts of scientific development.[[68]](#footnote-68)

Fleck, like Kuhn, concerned himself with defining the nature of the community in which science was developed. For Fleck, ‘science is a process carried out by groups’ rather than being based on individual acts.[[69]](#footnote-69) This process is possible as a result of scientists within a group operating under the same ‘thought style’, with the resulting ‘thought collective’ dictating scientific practices and how the group understand the world. The thought style acts in some ways like Kuhn’s paradigm, with Fleck describing it as ‘almost always exert[ing] an absolute compulsive thought upon [an individual within the thought collective’s] thinking’ and ‘with which it is not possible to be at variance’ whilst remaining within the associated thought collective.[[70]](#footnote-70) However, where Kuhn’s paradigms are identifiable by those within the disciplines defined by them (often having been introduced within textbook examples at the outset of a scientist’s practice), Fleck suggests that ‘the individual within the collective is never, or hardly ever, conscious of the prevailing thought style’.[[71]](#footnote-71) This aligns with his argument that a thought collective is not informed solely by scientific concepts (as with Kuhn’s paradigms), but rather also by external beliefs (be they religious, political or cultural) which influence what is known and how new knowledge is understood. Using understandings of syphilis as an example, Fleck highlights how existing religious ideas of the disease as a ‘carnal scourge’ resulting from the belief in ‘disease as a punishment for fornication’ combined with astrological and medical speculations on aspects of the diseases aetiology to eventually form an ‘idea of a causative agent [which] can be traced through the modern aetiological stage as far back as the collective notion of a disease demon’.[[72]](#footnote-72) Previous ideas, themselves informed by social constructs beyond the sciences, had a significant impact on current notions of syphilis as a disease, even if the old ideas were incorrect according to new evidence. The thought collective comprised of all these aspects, with individuals within the collective unknowingly being influenced by prior understandings when formulating the knowledge required for new ones.

This represents the key difference between Kuhn and Fleck’s beliefs of scientific development. As we have explored, for Kuhn the adoption of a new paradigm causes incommensurability with old understandings of the discipline. Even the most fundamental parts of the discipline’s knowledge are reshaped through paradigm shift. For Fleck, however, the process of scientific development is cumulative, with prior understandings, even if later proven ‘false’, still informing present understandings of key concepts. Scientific discovery, for Fleck, is thereby delineated as a three-way system of knowledge creation, with ‘the existing fund of knowledge’ acting as a ‘third partner’ in the creation of new knowledge alongside the ‘knowing subject and the object to be known’.[[73]](#footnote-73) In many ways this concept of cumulative scientific development is better suited to a study of medicine than Kuhn’s incommensurable paradigm theory. As Fleck demonstrates with his history of syphilis, medicine does not outright reject old ideas when faced with new facts. Fleck’s proposed cumulative model of scientific development therefore offers the fluidity in conceptualising the history of medicine as a scientific discipline that Müller-Wille sees Kuhn’s theory as precluding.

However, though the suitability of Fleck’s philosophy of science to the study of medical scientific development must be acknowledged, its utility is limited for my discussions of influenza in 1918. Unlike Kuhn’s defined structure of scientific revolution and paradigm shift, Fleck’s more nebulous process of translated concepts between thought collectives does not offer a clear answer to how thought styles are initially formed, nor how they react to moments of key discovery that Kuhn would call paradigm shifts. Within a cumulative model of development new ideas are influenced by, and so absorb or otherwise accommodate, previous understandings within the creation of new knowledge, but that does not account for moments of drastic change in understanding that fundamentally change the way the discipline perceives its previous knowledge. Rheinberger notes that ‘ Fleck does not have a very convincing response to this question [of what causes major changes in the thought style]’.[[74]](#footnote-74) Indeed, Fleck’s suggestion that ‘great transformations in thought style, that is important discoveries, often occur during periods of great social confusion’ where different points of view and their associated contradictions are seen in greater light (itself a vague statement) is relegated to an endnote within *Genesis* rather than engaged with in the main body of the work.[[75]](#footnote-75) The cumulative nature of scientific development is well suited to Fleck’s syphilis `case study, but it does not allow for an investigation of the transformative impact of singular discoveries within a discipline, as with the identification of the flu bacillus. Furthermore, when answering this thesis’ questions on the impact of the 1918-19 pandemic on bacteriological understandings of flu, Fleck’s philosophy would suggest only a cumulative outcome, which does not correspond to the historiography of the pandemic which considers it a key moment of shift. For this reason, Kuhn’s framework of scientific development will continue to be invoked as a heuristic device for the purposes of this study, with its structural rigidity, rather than hindering a discussion of medical development, instead assisting in establishing the extent to which the events of 1918-19 can be considered as fitting the definition of ‘revolutionary science’. From this, it will be possible to extrapolate how far the 1918-19 pandemic represented a moment of paradigm shift within medical understandings of influenza, and to make clear the power of the existing bacteriological paradigm within bacteriological practices associated with ascertaining the flu’s aetiology. Before entering this discussion, Chapter II will first provide the context of 1918-19 influenza knowledge, with an exploration of the 1892 discovery of the influenza bacillus and the way in which the influence of the bacteriological paradigm produced a ‘new way of knowing’ flu.

# **Chapter II: Germ Theory, Germ Facts: 19th Century Influenza and the Bacteriological Paradigm**

This chapter contextualises the paradigm to which Honigsbaum views 1918-19 scientists as being held prisoner. The Pfeiffer bacillus dominated discussions of influenza in the early part of the pandemic, and even once other potential aetiological agents were introduced it remained relevant within both pandemic and post-pandemic debates on the disease. The discovery of the bacillus also aptly demonstrated the influence of the bacteriological paradigm on medical understandings of disease during the late 19th century and shows how the paradigm was still establishing itself by the time of the influenza bacillus’ discovery. Throughout the chapter, the process of influenza knowledge creation will be considered, providing a clarifying framework for the bacteriological developments which were produced by 1918-19 pandemic experiences.

## ***Section 1: The Influenza Bacillus***

The *BMJ* ran a special feature in the journal’s January 16th, 1892 edition. In a segment titled ‘The Influenza Bacillus’, Dr R. Pfeiffer’s ‘preliminary communication on the exciting causes of influenza’ was published alongside supporting articles on the bacillus’ cultivation and appearance within ‘the blood of influenza patients’.[[76]](#footnote-76) Pfeiffer, a German scientist working at the Berlin Institute of Infectious Diseases, detailed his initial findings of ‘a bacillus of a definite species…found in absolutely pure cultures, and mostly in immense quantities…exclusively in cases of influenza’.[[77]](#footnote-77) The bacilli, described as ‘tiny rodlets about the thickness of the bacilli of mouse septicaemia, but only half of the length’ were found in 31 cases of influenza by Pfeiffer, with the identification of a distinct influenza bacillus endorsed by both Pfeiffer’s colleague - esteemed Japanese bacteriologist and student of Robert Koch’s school of bacteriological thought - Dr S Kitasato, and by another German physician, Dr P Canon.[[78]](#footnote-78) Following descriptions of how the bacilli are obtained – specific details referring to the percentage of sugar agar needed for colony growth, the temperature required for multiplication, the process of staining samples to identify the bacilli in the blood – all three articles identify the bacilli as standing ‘in direct relation to influenza’, with Pfeiffer concluding that he considered himself ‘justified in pronouncing the bacilli just described to be the exciting causes of influenza.’[[79]](#footnote-79) These findings were corroborated, first by an article on January 23rd responding to Pfeiffer’s report with observations of the bacillus within sputum and agar cultures of influenza patients in London, and then a week later (30th January) by a further response to the findings that (albeit tentatively) asserted that ‘the problem as to the micro-organism of influenza has at last been solved’.[[80]](#footnote-80)

The positive reception did however come with a caveat, expressed in the closing statement of the 30th January report wherein the author stressed that despite the potential of the discovery for future understandings of influenza, ‘it cannot be expected swiftly to arrest an epidemic’.[[81]](#footnote-81) This sentence is indicative not just of the immediate context of Pfeiffer’s findings, explored below, but also the broader picture of how his discovery fit into, and indeed acted as an integral part of, what Michael Bresalier termed the ‘new way of knowing influenza’ that was produced by experiences of epidemic influenza in the final decade of the 1800s.[[82]](#footnote-82)

Pfeiffer’s flu samples, as well as those of his supporting authors and of the author of the comment made on the 23rd, were taken from those afflicted with ‘Russian Flu’, the name given to an influenza pandemic that swept the globe between 1889 and 1891. Initially reported in Russia in early Autumn, cases were noted in England and Wales in December 1889, from which point fatalities rose across three waves to total an approximate 110,000 deaths by the end of 1892.[[83]](#footnote-83) Finding the microbial cause of the pandemic was a natural goal of the medical community from the pandemic’s onset, however, as the 30th of January 1892 article warns, even isolating the bacteria responsible would not provide an immediate solution to the pandemic problem. As Nancy Tomes identifies in her work on the acceptance of germ theory in the US, ‘for the first fifty years…the germ theory provided its greatest utility as a guide to the *prevention* of disease through modification of individual and collective behaviour’.[[84]](#footnote-84) The identification of the flu bacillus could potentially offer insight into prophylactic measures able to prevent future pandemics, but it would do little to assist in the pandemic at hand.

It was therefore in the pandemic’s aftermath that the significance of Pfeiffer’s discovery could truly be realised. Reflecting on the pandemic once it was over, it became clear that existing theories of contagion, specifically in relation to influenza, no longer supported the witnessed disease progression. These theories, broadly described, were that influenza occurred cyclically due to disease reintroduction or environmental conditions; that it was as a result of weather patterns or astral conditions; or that it was caused by an airborne pathogen and so transmitted miasmically. A germ theory of influenza had to contend with these existing explanations of disease, and medical experiences of the Russian pandemic created the perfect conditions for this challenge.

The Russian Flu pandemic marked the first British experience of epidemic level influenza since 1847, indeed for over 40 years prior to 1899 influenza deaths had been declining, with under 100 deaths from influenza reported in 6 of the 9 years preceding the outbreak.[[85]](#footnote-85) Prior to 1847, influenza had reached epidemic levels at roughly twenty-year intervals. However, between 1847 and 1889 influenza cases had dropped drastically, leading the 1920 report on the 1918-19 Pandemic to state that by 1889 ‘the country had been free from pandemic influenza for more years than in any previous epoch since the middle of the 17th century’.[[86]](#footnote-86) The lack of a flu pandemic in the 1870s, and the emergence of one in 1889, therefore discredited the idea of pandemic influenza as a cyclically explainable disease. This was further reinforced by the continued presence of influenza epidemics in the 20 years that followed. As Bresalier notes ‘in no year between 1890 and 1915 did fewer than 496 Londoners’ die’ from influenza, with epidemics occurring in 1895, 1899-1900 and 1908-9 and an average of over 11,000 deaths per year from 1908-1918.[[87]](#footnote-87) Flu, it appeared, had entered a new age. If it were ever cyclical in nature – or for that matter a result of weather or astral conditions – from 1889 it had undergone a drastic change. This ‘new’ flu was endemic in its epidemics, prompting what Bresalier terms ‘new ways of knowing influenza’ that reflected its constant threat and offered new suggestions on how to understand its spread.[[88]](#footnote-88)

This ‘new way of knowing’ influenza also served to challenge existing theories of its transmission. As late as the 1880s, the germ theory of disease was an outlier to more popular miasmic or zymotic theories of disease. These theories, broadly defined as considering disease stemming from ‘bad air’ which carried infection (miasmic) or spontaneously generating poison creating chemical ferments (zymotic), were dominant within medical opinion due to the strength of the ‘sanitary science’ movement in offering solutions to prevent or control outbreaks.[[89]](#footnote-89) If disease was caused by bad air or the rotting fermentation of ‘decaying filth’, then measures which proposed ways to sanitise disease ridden areas or keep your domicile clean from disease carrying vapours offered legitimate solutions to the problem of disease spread.[[90]](#footnote-90) Accepting germ theory as the cause of disease required a complete re-evaluation of what diseases ‘were’, and also did not offer easy solutions to deal with their occurrence. Further to this, the science of germ theory required a move away from the traditional clinical and epidemiological methods of disease observation. Rather than observe the patient’s symptoms or examine trends in reported cases, germ theory required the adoption of a laboratory view of disease. This required access and understanding of new scientific methods, and for those of the existing school of thought the ‘experimentalism’ of a theory based on outlier notions of microbial life was not worth the rejection of ‘insights derived from decades of clinical and epidemiological observation’.[[91]](#footnote-91)

Nancy Tomes describes this division of thought in the late 1800s as a ‘civil war over the truth of germ theory’.[[92]](#footnote-92) By the flu pandemic of 1889-91, this war was falling firmly in the germ theorists’ favour, with the work of German physician Robert Koch being instrumental in the legitimisation of both germ theory and the laboratory method in the wider scientific medical imagination. In working to prove a causal link between anthrax and the *Bacillus anthracis*, Koch pioneered new experimental techniques used to identify microorganisms, including new methods of growing and staining samples which made isolating a specific bacterium from a culture far easier.[[93]](#footnote-93) This facilitated Koch’s isolation of the tubercle bacillus in 1882, a feat which Tomes views as ‘probably the single most dramatic discovery of the golden age of bacteriology’.[[94]](#footnote-94) As well as proving that tuberculosis was a contagious disease caused by a specific bacteria, Koch’s process of isolating the bacillus also paved the way for decades of future disease discoveries.

In order to prove that a specific bacillus causes a disease, it must be established that the bacillus alone is responsible, rather than its existence in a sample being coincidental or evidence of a secondary, unrelated infection. Recognising this issue from his earlier work on anthrax, for his studies into tuberculosis Koch established a methodological approach which could prove without doubt if a specific microbe was the causal agent of a disease. First, a pure culture must be obtained from a sick patient (by means of growing a sample on a suitable solid medium) and once isolated this culture is then used to inoculate an animal host. If the animal becomes sick and cultures taken from it match those found in the original sample it could be proven that the bacillus was the cause of the disease, establishing ‘the necessary role the specific bacterium in the disease process’.[[95]](#footnote-95) This process provides step-by-step conditions under which any potential microbial agent could be isolated as a diseases’ causative agent. Koch’s Postulates, as they became known, allowed Koch to isolate the cholera bacillus in 1884, and enabled other researchers over the next thirty years to identify the causal organisms of ‘diphtheria, typhoid, scarlet fever, erysipelas, pneumonia, leprosy, gangrene, tetanus, gonorrhoea…bubonic plague, dysentery, whooping cough, syphilis’ and, of course, influenza.[[96]](#footnote-96)

The influence of Koch’s Postulates on microbiological discoveries is evident in the reporting of Pfeiffer’s influenza discovery in 1892. All three articles accompanying the discovery’s announcement in the *BMJ* detail the process by which pure cultures were obtained through the use of a nutrient medium (with the method remarked on by Dr Kitasato being one devised by Koch himself).[[97]](#footnote-97) Two reference the results of animal inoculations, and though inoculation experiments were only minimally successful (Pfeiffer stating that ‘only in apes and rabbits could positive results be obtained’ out of a potential pool of animals which also included ‘guinea-pigs, rats, pigeons and mice’, and Canon admitting that ‘experiments on animals always yielded a negative result’) the fact that the bacillus could only be found in sick patients and was reproducible on agar and to some extent in animals meant it fulfilled Koch’s Postulates, and thus could be deemed a legitimate discovery.[[98]](#footnote-98)

Though granted legitimacy by Koch’s Postulates, Pfeiffer’s discovery did not immediately change mainstream opinion on the cause of influenza. In fact, Bresalier’s ‘new way of knowing influenza’ was a product of the post-pandemic years, with experiences of the pandemic shifting opinion towards the general acceptance of Pfeiffer’s bacillus as the bacteriological cause of flu.

## ***Section II: A ‘New Way of Knowing’ Influenza***

The 1899-91 pandemic had challenged multiple ways of knowing influenza. The pandemic’s timing disproved the idea of cyclical outbreaks, as well as the concept of reimported infection at measurable durations.[[99]](#footnote-99) So too did pandemic experiences highlight a key issue with relying on a clinical model of influenza diagnosis – namely that there was no clear consensus on what influenza looked like. As we have explored, for 40 years prior to the 1889 outbreak flu had been a relatively uncommon affliction, leading to an unfamiliarity within the medical sphere with influenza’s symptoms. Efforts were taken to remedy this, first by consulting older medical texts, and then by attempting to build a new clinical picture of the disease through contemporary observations. As Bresalier notes, neither provided a clear answer. Older medical texts appeared to be describing an entirely different disease, with the catarrhal influenza of the 1850s bearing little resemblance to the respiratory symptom dominant influenza of 1889. Contemporary observations had almost the opposite issue. In reported findings of 1889 influenza patients, there appeared no organ or system not impacted by the disease. Rather than just one clinical description not fitting, doctors were now faced with multiple different symptom accounts, leading to their being ‘numerous clinical interpretations’ of the disease within the medical establishment at the outset of the pandemic.[[100]](#footnote-100) The identification of a bacterial cause did not completely solve this problem, but in cases where influenza was suspected it offered a method which could identify true cases, so long as Pfeiffer’s bacillus could be found within patient samples.

In the aftermath of the pandemic this method was subject to continued scrutiny as medical opinion began to more readily accept a bacteriologically dominant way of practicing medicine. Bresalier views this shift in accepted thought style, at least within British medical opinion, as being intrinsically linked to investigations into the flu bacillus, citing Edward Klein’s inquiries into influenza and the bacillus’ role as key to establishing the ‘medical relevance’ of bacteriological thinking.[[101]](#footnote-101) This disregards the strides made towards the acceptance of bacteriological principles following the numerous other bacterial discoveries in the last years of the 1800s, including Tomes’ ‘single most dramatic discovery of the golden age of bacteriology’ (the isolation of the tubercule bacteria), however Bresalier’s assertion does indicate the significance of post-pandemic bacteriological investigations as legitimacy granting endeavours for bacteriology as it related to flu treatment.[[102]](#footnote-102)

This process did not result in a complete acceptance the ‘bacteriological paradigm’.[[103]](#footnote-103) Pfeiffer’s bacillus proved very difficult to cultivate in post-pandemic tests, leading to results that often failed to fulfil all of Koch’s Postulates. Influenza’s broad reported symptoms also continued to be a roadblock to a unanimous agreement on Pfeiffer’s bacillus’ link to flu. Within the understood bacteriological paradigm, in line with Koch’s Postulates, a bacteria should be found aetiologically responsible for a specific disease’s presentation. Influenza did not have one sole form; indeed, influenza could present with as much variety as to appear as near separate diseases in different patients. The question was therefore raised: was the influenza bacillus responsible for just one variant of the disease known as ‘influenza’, and for other types, as Bresalier puts it, ‘were other agents at work?’[[104]](#footnote-104)

The responses offered to these questions, while not demonstrating complete acceptance, do indicate the strength of the Pfeiffer’s Bacillus paradigm for understandings of flu by the early 20th century. Pfeiffer initially suggested that the influenza bacillus excreted a toxin which could spread to different organs and produce the various observed symptoms.[[105]](#footnote-105) In the wake of increasing scepticism caused by issues finding the bacillus in 1899 flu cases (as well as it being found cases of other diseases and in healthy people), Pfeiffer then suggested that instances where the bacillus was not found in otherwise symptomatic cases was a result of ‘pseudo-influenza’ being confused with the ‘true’ bacteriological form. Other bacteriologists, offering an alternative explanation, suggested an expansion of influenza’s recognised aetiology to include a group of diseases caused by a variety of bacteria rather than a single disease with just one aetiological cause. This went against all existing thought on singular disease aetiology, and thus for those that did not adopt either the ‘pseudo-influenza’ or mixed aetiology theories, suspicion remained that the true cause had not been found.[[106]](#footnote-106)

What both theories and the insistence in another cause show though is that in all instances the ‘new way of knowing influenza’ was bacteriological. Those that did not believe Pfeiffer’s bacillus to be the true cause of influenza were nevertheless looking for an alternative bacteriological explanation. Meanwhile bacteriologists entertained the notion of deviating from the existing norm of singular aetiological explanations simply to account for Pfeiffer’s bacillus remaining a key part of influenza understanding. If other bacteria were found to play a part in influenza cases, this was in addition to, not instead of *B.influenzae*. Influenza’s definition should be expanded, be it through Pfeiffer’s explanation or alternate suggestions, so that Pfeiffer’s bacillus could continue to play a significant role.

Mark Honigsbaum’s discussion of the ‘bacteriological paradigm’ does not account for the nuance of Bresalier’s late 19th century ‘new way of knowing influenza’. To Honigsbaum, as to many other historians of the 1918-19 pandemic, the insistence on a bacteriological cause of flu was an all-consuming medical belief by 1918, with the pandemic either challenging this idea or falling victim to the strength of the paradigm caused by Pfeiffer’s Bacillus. As this section has indicated, though the discovery of Pfeiffer’s’ Bacillus did represent a significant moment in changing attitudes towards influenza, it was not instantly nor universally accepted. Its longevity within understanding of influenza is testament to the growing acceptance of germ theory and the need for theories of influenza to fit the bacteriological model, but equally important to consider is the context of the bacillus’ discovery. Pfeiffer’s bacillus as a (tenuously) paradigmatic model of influenza aetiology was a product of the rejection of existing theories of disease and clinical models of diagnosis enforced by experiences of the 1899 pandemic. The 1918-19 pandemic, as the first pandemic experience of flu since Pfeiffer’s discovery in 1892, thus acted as the first true challenge to ‘Pfeiffer’s paradigm’, and from that the bacteriological paradigm writ large. Chapter III will explore this challenge.

# **Chapter III: ‘Further Bacteriological Information Awaited’:**

## ***Section I: Testing the Paradigm – A Discussion of Methods***

In order to ascertain the impact of the 1918-19 pandemic on the medical community’s adherence to the bacteriological paradigm, one must establish a suitable methodology for analysing the community’s knowledge creation outputs. I identified two prominent British medical journals for this process – *The Lancet* and the British Medical Journal – and utilised a chronological approach in order to chart changing attitudes towards Pfeiffer’s bacillus and the bacteriological paradigm writ large throughout the duration of the pandemic. A chronological approach was required due to the variety of terms associated with influenza in use throughout the pandemic. Both *The Lancet* and the British Medical Journal have searchable digitised archives, however Tom Jefferson and Eliana Feronni’s 2009 study of the pandemic using the *BMJ’s* digitised archive highlights the flaws with using a search-term based approach to studies of 1918-18 medical thought. In order to ‘look at the pandemic through the eyes of contemporary *BMJ* contributors and readers and give them their voice back’, Jefferson and Feronni used a series of search terms (“influenza,” “flu,” “pandemic,” and “Spanish influenza”) which unintentionally produced the ‘perilous’ interpretation of prior events from a modern perspective that their article sought to avoid.[[107]](#footnote-107) Though ‘influenza’ was a recognised diagnostic term it was not the sole one used to describe the pandemic, with ‘epidemic catarrh’, and ‘purulent bronchitis’ also used within medical discussions during the 1918-19 period.[[108]](#footnote-108) Similarly, though the outbreak was referred to as a ‘pandemic’ in some instances, the use of ‘epidemic’ as a descriptor was more common (with the influenza referred to as an ‘epidemic’ 13 times in 1918 *BMJ* articles compared to only 5 references to it as a ‘pandemic’) , yet did not feature as a chosen term within the Jefferson and Ferroni article.

The authors identify ‘55 articles of varying nature—including studies, reports, and letters—published between July 1918 and October 1920’ which included ‘unusual or forgotten observations and still unresolved questions’ about the pandemic.[[109]](#footnote-109) Though they acknowledge that this does not represent a ‘systematic review’ and encourage future engagement with the search function of the *BMJ* archive, the limits of utilising specific search terms when studying the nebulously diagnosed disease of influenza prompted me to instead employ a more comprehensive study of each journal by manually checking every weekly publication from first mention of the pandemic in June (*BMJ*) or July (*Lancet)* 1918 until the final issue of each journal in December 1919. In doing so I gained an insight into not just the changing attitudes towards the flu’s aetiology over the duration of the pandemic and post-pandemic period, but also of the medical communities into which new influenza knowledge was being shared and collaboratively produced.

In total I consulted 293 articles, correspondences, and parliamentary notices across both journals. 129 of these were from the *BMJ* and 164 from *The Lancet*, with the latter also accounting for the largest spike in influenza mentions in the November 1918 issues (38 relevant mentions, compared to a smaller but still significant rise to 11 mentions by the *BMJ*). Figure 1 demonstrates this rise, as well as showing general trends in article increases and decreases which broadly align with case instances in the pandemic’s three main waves. These waves are illustrated for comparison in the graph reproduced from Pearce et al.’s article ‘Understanding mortality in the 1918–1919 influenza pandemic in England and Wales’, with the second wave’s peak in late October and early November 1918 correlating with the increase in articles on influenza in both journals.[[110]](#footnote-110)

Figure 1: A graph Illustrating the Incidence of Mentions of Influenza in BMJ and Lancet Articles, June 1918-December 1919.

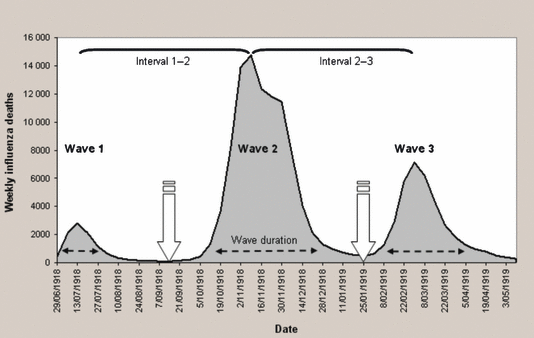


Figure 2: A time-series plot graph showing the three main waves of influenza deaths in Britain, from June 1918-May 1919. Source: Dora C. Pearce, Paul K. Pallaghy, James M. McCaw, Jodie McVernon, John D. Mathews, ‘Understanding mortality in the 1918-1919 influenza pandemic in England and Wales’, Influenza and Other Respiratory Viruses, 5.2,(2011) 89–9, (p.90).

Of these articles, 60 within *The Lancet* expressly mentioned Pfeiffer’s bacillus or discussed the aetiology of influenza (representing 47% of all influenza mentions), with 29% of *BMJ* articles doing the same. These statistics, almost one half of all mentions in *The Lancet* and almost a third in the *BMJ,* make clear the significance of the debates surrounding influenza’s aetiology within the medical community’s influenza knowledge output. This section will investigate the nature of these debates, examining how each journal’s communities developed their positions on influenza throughout the pandemic and analysing the extent to which these developments demonstrate either Honigsbaum’s insinuated ‘blindness’ caused by the bacteriological paradigm, or instead offer evidence of a transition to revolutionary science and paradigm shift caused by bacteriological findings attained during the pandemic.

As part of this analysis, the nature of the paradigms in question must first be made clear. Though Honigsbaum views the continued insistence on the role of the influenza bacillus as evidence of the strength of the bacteriological paradigm, I argue that Pfeiffer’s discovery of the influenza bacillus represents its own influenzal paradigm, which was then challenged in 1918. As such, it is two paradigms that this section will examine in accordance with Kuhn’s theories of scientific revolutions: the influenzal paradigm that saw Pfeiffer’s bacillus described as influenza’s primary aetiological agent, and the wider bacteriological paradigm which underpinned all knowledge on diseases and pathogenic organisms. Differentiating between these paradigms allows for a more nuanced understanding of influenza knowledge within the period and is particularly useful when accounting for distinctions in approaches to vaccines and filter-passing viruses within both journals.

## ***Section II: Early Signs: June – September 1918***

The *BMJ* was the first of the two journals to report on a potential pandemic in two June 1918 articles responding to newspaper reports of ‘the epidemic in Spain’.[[111]](#footnote-111) The epidemic is posited as influenzal in the initial article on June 1st based upon symptoms reported by the popular press, with links also being drawn to ‘a severe outbreak of influenza in a certain district of this country’ during May and an ‘outbreak of influenza among German troops’ which had delayed their recent offensive.[[112]](#footnote-112) The lack of reported bacteriological evidence is here used to allay ‘alarmist suggestions’ of plague and cholera rather than be used as a diagnostic tool for influenza, but by June 22nd, with the epidemic in Spain ‘now reported to have caused 700 deaths in ten days’, attention turns to initial bacteriological findings, and the first noted absence of Pfeiffer’s bacillus.[[113]](#footnote-113) It is acknowledged that ‘although the disease is clearly of the character of influenza, bacteriological examination has not resulted in the discovery of the influenza bacillus’.[[114]](#footnote-114) This is rationalised in the article by explaining that ‘it is well known that the bacillus influenzae is quite commonly absent in cases clinically characteristic of influenza’, whilst the other organism found in the examinations (a ‘parameningococcus’) is explained as potentially ‘Micrococcus catarrhalis, which has some superficial resemblance to the parameningocccus’ and is often found in influenza-type cases.[[115]](#footnote-115) The article concludes by stating that ‘it is obvious that further bacteriological information is awaited’, namely the identification of the influenza bacillus or ample evidence of an alternate bacteria, such as a parameningococcus, which would change the epidemic from one of flu to another disease.[[116]](#footnote-116)

The awaited bacteriological evidence came in the next article on the epidemic – now noted as ‘the Influenzal *Pandemic*’ [emphasis own] on July 13th. The article first reports on the spread of ‘the influenza that we read so much about in the daily papers’ across ‘almost all the countries of Europe’, before going on to discuss the symptoms and prognosis of those afflicted. The disease’s character, and specifically the low mortality in comparison to previous influenza epidemics, is noted as having caused speculation that ‘we are not now dealing with an epidemic or a pandemic of influenza at all’, but the article notes that ‘bacteriological evidence is accumulating to prove that the influenza bacillus is responsible for at least a considerable proportion of cases’, thereby proving that influenza is the responsible agent.[[117]](#footnote-117) The absence of the influenza bacillus in pharyngeal swabs, and the finding of ‘pneumococci and what at first sight appear to be pneumobacilli predominating, and Micrococcus catarrhalis, streptococci, staphylococci, and other microbes’, does not hinder this judgement, for the pneumobacilli are explained as evidence of the influenza bacillus’ known tendency to appear polymorphic in culture (‘these pneumonobacilli are in many cases influenza bacilli of aberrant type’).[[118]](#footnote-118) The predominant presence of pneumococci is further dismissed by the article adding that ‘the pneumococci are in many instances of the saprophytic type, and not pathogenic to guinea-pigs’, thereby failing Koch’s 3rd postulate and disproving all causative link between the pneumococci and the onset of disease.[[119]](#footnote-119) The article is clear: even if another organism is more prevalent than the known cause of flu, it is the influenza bacillus that dominates any discussion of aetiology.

In its first reports of influenza the *BMJ* therefore establishes its position as one in favour of the influenza bacillus. This is striking when compared with *The Lancet*’s first article mentioning the ‘present widely spread epidemic’, also on July 13th, which concludes that an alternative organism is responsible as a result of the ‘absence of the bacillus influenzae’ from upper air-passage samples.[[120]](#footnote-120) Taking an altogether more critical tone, authors Little, Garofalo and Williams cited the difference in clinical course, the absence of some gastro-intestinal symptoms commonly associated with ‘the form of influenza due to the *Bacillus Influenzae’,* and a characteristic variance in leucocytosis as reasons why in their opinion the present epidemic ‘cannot properly be considered’ influenza.[[121]](#footnote-121) Most significant to their conclusions though is the complete absence of the ‘organism of influenza -viz, *Bacillus influenzae*’ in either smears or cultures, and the presence instead of a diplococcus found ‘present with no exception’.[[122]](#footnote-122) This diplococcus was found in outstanding predominance in ‘direct smears from the naso-pharynx, throat and sputum’, and was exceedingly abundant in sputum cultures, prompting the authors to conclude that with it ‘long [having] been recognised that attacks of so-called influenza are often due to organisms other than *Bacillus influenzae*…we consider [the causative organism of the present epidemic] to be the…diplococcus.’[[123]](#footnote-123)

The authors display a confidence in their claim of an alternative causative organism, both in the publishing of their findings and in the defence of their methods. They acknowledge that the diplococcus, when inoculated intraperitoneally’ is non-pathogenic to mice and therefore unable to fulfil Koch’s third postulate, but see the fulfilment of the first two postulates, and the promise of positive results from ‘further animal inoculation’ as proof enough that their claim has merit.[[124]](#footnote-124) This, along with their concluding note that ‘it has long been recognised that attacks of so-called influenza are often due to organisms other than *Bacillus influenzae*…many have been indicated’ demonstrates a significant doubt in the existing influenzal paradigm.[[125]](#footnote-125) This doubt, however, was not unanimous within the medical community, as proven by the authors’ pre-emptive defence of their laboratory methods. ‘It may be contended’ they state, ‘that the absence of the influenza bacillus in their results ‘is due to the fact that we did not use media containing whole blood’.[[126]](#footnote-126) However, they quickly dismiss this potential critique by stating that ‘it has been our experience that *Bacillus influenzae* grows abundantly on legumin serum agar’ and that ‘we frequently obtain it when making routine bacteriological examinations from the naso-pharynx’.[[127]](#footnote-127) Their methods, they argue, are sound, and the mention of finding the bacillus in ‘routine bacteriological examinations’ could also hint at their further scepticism that the bacillus is the direct cause of epidemic influenza.

That this article is the first engagement within *The Lancet* on the subject of the influenza epidemic suggests a more critical position to the cause of influenza than that held by the *BMJ*. However, these positions are far from fixed, with the *BMJ* going on to publish a series of articles which are sceptical of influenza’s involvement in the pandemic in July and August of 1918, and *The Lancet* printing a well-received correspondence which comes to the defence of Pfeiffer’s bacillus.

The *BMJ* published seven articles discussing influenza between July 20th and the end of September 1918, six of which discuss influenza’s aetiology and the absence of the influenza bacillus. Though this absence is acknowledged, it is not enough to prompt a rejection of the bacillus as the cause of influenza. Rather, the fact that influenza’s ‘causal agent is known’ causes Julius Burnford to argue that ‘there is no precise [by which he means bacteriological] evidence that we are dealing with influenza’ in his July 20th note on epidemics, whilst a *BMJ* article on ‘*The Pandemic of Influenza’* on July 27th explained the presence of non-*B.influenzae* microorganisms (including pneumococci, micrococcus and pneumobacilli) as evidence of a symbiosis between Pfeiffer’s bacillus and other secondary organisms, rather than evidence that the bacillus was not the pandemic’s causative agent.[[128]](#footnote-128) The overall continued acceptance of the influenzal paradigm is made clear by the latter article’s statement that ‘the general consensus of opinion seems to indicate Pfeiffer’s Bacillus influenzae as the infecting agent. But by no means all the medical men and bacteriologists who have investigated the epidemic will agree with so summary a generalization’.[[129]](#footnote-129) It is the generalisation of the role of the bacillus that is framed as the point of contention (e.g. not in detail stipulating the symbiotic or conjunctive role of the bacillus with a secondary organism), rather than the bacillus’ overall role as influenza’s causative agent.

In just one instance the relative absence of the influenza bacillus is not accompanied by a statement that insists upon its causative link. The August 10th article in the *BMJ* on *‘The Bacteriology of the Influenza Pandemic’* reports on German experiences of the pandemic, and notably the bacteriological findings from a number of German laboratories, including Pfeiffer’s laboratory in Breslau. The article notes that ‘Pfeiffer’s bacillus has been found only exceptionally’ in Munich, found only in some instances in Breslau and Strasburg and was absent entirely from investigations in Frankfort.[[130]](#footnote-130) Pfeiffer himself is noted as having ‘reserved his final opinion’ on whether the pandemic is the same as that in which he identified the influenza bacillus, pending further investigation into the ‘cause of [the] discrepancy’ in finding the bacillus in samples, whilst Kolle, writing from Frankfort, posited that the identification of a diplococcus, which he regarded ‘as the agent of the secondary infection in the “Spanish Disease”, could mean that the current pandemic ‘may not be identical with the pandemic influenza of 1880-93.[[131]](#footnote-131) Where previous articles had assuaged doubts in the role of Pfeiffer’s bacillus with explanations of symbiosis or pleomorphism, this article states the findings without such caveats. Indeed, the German findings of the diplococcus are linked to similar findings from the US, and reference is made to the *BMJ* articles published on July 13th and 20th as supporting evidence that the diplococcus could be the true agent of infection in the pandemic, despite those articles concluding at the time that the influenza bacillus was ultimately still to blame.

In what is likely a nationalistically motivated attack (given the existing conflict, even with the article representing a moment of truce in the pursuit of shared knowledge), the article goes further to question German laboratory methods, stating that ‘Pfeiffer’s bacillus was not found until 1892, although it should have been impossible to overlook it in 1889’ in response to the Munich Medical Union’s statement that the limited findings of the bacillus were ‘in “keeping with the precedent” of findings from 1889.[[132]](#footnote-132) They also print the acknowledgement by Ulrich Friedemann, writing from Berlin, that the ‘influenza bacillus…might be evading capture on account of faulty methods’.[[133]](#footnote-133) Though this can be read simply as a form of nationalistic smugness, the reference to ‘faulty methods’ gains significance when compared both to the 13th July Lancet article which pre-emptively dismissed accusations of poor methodology, and to the condition of Kuhn’s revolutionary science in which the community accept that ‘the crisis provoking problem’ is not solvable within the constraints of current methods or tools.[[134]](#footnote-134) Though faulty methods are not identical to technologically limited ones, this initial evidence of methods being questioned by the scientific community when faced with the ‘crisis provoking problem’ of the influenza bacillus’ absence is significant when considering the extent to which the pandemic can be said to place the medical field into a period of revolutionary science. It is particularly significant when considering the role questions of methodology have in *The Lancet*’s discussions of influenza from July 13th to September 28th.

In the week following Little, Grafallo and Williams’ article on a potential diplococcal cause of the pandemic, *The Lancet* published a correspondence by Dr John Mathews which responded to the article’s findings. Though admitting that the paper is ‘interesting’, Matthews is quick to state that ‘the conclusions that the epidemic is not caused by Pfeifer’s bacillus are at least open to question.’[[135]](#footnote-135) What follows is a critique of Little et-al.’s choice of medium ‘hitherto it has never been suggested, as far as I am aware, that Pfeiffer’s bacillus will grow on a medium devoid of blood’, their choice of interpretative method ‘[the authors]…rely on not recognising Pfeiffer’s bacillus in direct smears, but in the case of…[the] influenza bacillus the evidence derived from direct smears is apt to be fallacious’, and the validity of their conclusions.[[136]](#footnote-136) These conclusions are insinuated as ignoring the basic, textbook principles of influenza knowledge (‘in most textbooks influenza is usually coupled with typhoid fever as a cause of leucopenia’ rather than being identified through leucocytosis as Little et.al claimed), and are also criticised for having ‘neglected the elementary precaution of demonstrating that the diplococcus…is not to be found in normal conditions as their “influenza” cases’ – thus failing Koch’s first postulate despite the authors’ emphasis that they had fulfilled the first two.[[137]](#footnote-137)

Matthews’ criticism ends by stating that in his experience ‘I have recovered Pfeiffer’s bacillus in every case, a result partly attributable perhaps to the use of an improved medium, an account of which I hope to publish in the immediate future.’[[138]](#footnote-138) In this statement, and in the promised article ‘*On a Method for the Culture of Pfeiffer’s Influenza Bacillus That Gives Profuse Growth and is o a Marked Degree Selection for This Organism’* published one week later, Matthews therefore dismisses the potential for a diplococcal cause of the pandemic and reinforces the position of Pfeiffer’s bacillus. Where the absence of the bacillus had caused an anomaly which threatened the influenzal paradigm, Matthews’ demonstration of a successful method/tool (in the form of a ‘trypsinised blood’ medium) to isolate the bacillus provided evidence that the existing scientific community had the tools to solve the crisis period and explain the anomaly within the boundaries of the existing paradigm.[[139]](#footnote-139)

This support for the influenza bacillus is visible within other Lancet articles published within the July-September 1918 period. The August 10th issue joins the *BMJ* in commenting upon German bacteriological findings, though *The Lancet*’s reporting is altogether less critical than that of the *BMJ*. Where the *BMJ* is succinct in their criticism that ‘it should have been impossible to overlook [the bacillus] in 1889’, *The Lancet* article is more lenient in their statement that ‘although it should have been impossible to overlook it in 1889, thus it may be that it will yet turn up in due course’.[[140]](#footnote-140) Similarly, though *The Lancet* mirrors the *BMJ* in acknowledging that ‘the findings of [the] pleomorph Gram-positive diplococcus [reported by Kolle in Frankfort] is very interesting in view of the observations of Rosenow and his pupils in the United States’, unlike the *BMJ*’s mention of earlier articles they do not relate it to the earlier diplococcus findings by Little et-al. – potentially due to the dismissal of their findings by Matthews’ contributions.[[141]](#footnote-141)

Though there are still expressed doubts on the differing presentation of the present epidemic of influenza versus that of previous years (as with the article on the ‘symptomatology and morbid anatomy of so-called “Spanish Influenza”’ by E. Rivax Hunt on September 28th in which the present epidemic is differentiated from ‘true influenza’ due to its differing presentation) the role of the bacillus, unlike in the *BMJ*, is not questioned within *Lancet* articles published in the early pandemic period.[[142]](#footnote-142) This is indicative of a wider trend within the two journals, with the *BMJ* expressing more scepticism of the influenzal paradigm than *The Lancet* as the pandemic’s second wave spread through Britain in the autumn of 1918.

## ***Section III: Vaccines and Viruses: October – December 1918***

On October 14th 1918, the War Office hosted ‘an important conference…on the utilisation of bacterial vaccines for the prevention and treatment of influenza.’[[143]](#footnote-143) Reported in both the *BMJ* and *The Lancet* on October 26th, the conference consulted several prominent pathologists and military officials, and served to ‘formulate definite recommendations as to the employment of vaccines in view of the probability of an extensions of the epidemic of influenza’.[[144]](#footnote-144) Central to these discussions was the bacteriological composition of a potential vaccine, thereby forcing both the doubts and defences of Pfeiffer’s bacillus to the forefront of discussion within the two journals over the autumn period.

The chairman’s notes of the conference (identically published in both journals) drew attention to the increasing divisions over the role of the bacillus, stating that ‘…After discussing the available evidence on the bacteriology of the present epidemic the majority of those present were agreed that there was considerable doubt as to the primary etiological significance of the *Bacillus influenzae* of Pfeiffer’.[[145]](#footnote-145) Significantly, the chairman does not note a similar consensus being found for an alternate primary aetiological candidate, thereby reflecting the differing opinions on a potential pneumococcal or diplococcal agent seen in the early reporting of the pandemic. Only in the case of secondary pulmonary complications are ‘pneumococci and streptococci’ mentioned as significant associated organisms.[[146]](#footnote-146) The chairman’s notes do, however, posit that ‘the existence of some as yet undiscovered virus must be regarded as possible’ as the main cause of the pandemic.[[147]](#footnote-147) Though ‘virus’ in this context does not refer to a virus in the modern context, suggestion of an ‘as yet undiscovered’ cause of influenza further demonstrates the doubts held by some over the role Pfeiffer’s bacillus played in the pandemic’s (and by extension, influenza’s) aetiology. Doubts amongst the conference attendees extended beyond the existing organisms associated with flu to also consider a solution outside the boundaries of existing knowledge.

Despite this expression of doubts, however, the conference’s overall recommendations were still in support of Pfeiffer’s bacillus as a significant, if not *the* significant, aetiological agent. The chairman notes that there were ‘no doubts as to the very frequent presence of Pfeiffer’s organism in this epidemic, nor as to the great importance of the part which it has played in the production of symptoms and complications of the disease’.[[148]](#footnote-148) Accordingly, the *Bacillus influenzae*, alongside ‘the pneumococcus and the streptococcus’ are agreed as the only organisms to be included in the vaccine, with a brief mention of the logistical challenges in identifying required *B.influenzae* strains the only clarifying note on the committee’s decision.[[149]](#footnote-149)

The publication of the War Office conference’s recommendations prompted responses in the correspondence section of both the *BMJ* and *The Lancet*, however it is the latter that sees most objection to the report. Two letters to the editor were published in the week following the War Office Conference report, with a further three reiterating the initial criticisms the week after. This consensus of critical opinion is explainable not just by its conformation to *The Lancet*’s typically ‘pro-Pfeiffer’s bacillus’ standpoint during the pandemic, but also due to the author of one of the initial correspondences being Dr John Matthews. Matthews found himself ‘wholly at variance with the suggestions of the Committee’ regarding the dosage of the influenza bacillus in the proposed vaccine, and so ‘venture[d] to criticise’ the published notes from the conference.[[150]](#footnote-150) His criticism trades upon the reputation he gained from the positive reception of his culture medium a month prior, with references to his ‘more than two years’ experience’ of his medium’s success in isolating the bacillus and his experience of influenza infections being ‘extensive’.[[151]](#footnote-151) In reasserting his professional standing, Matthews also suggests that the ‘imperilling’ dosage suggestions are a result of ‘scarcity of practitioners’ able to advise on vaccine dosage, which he views ‘not a question of pure science so much as a question of practice.’[[152]](#footnote-152) In this way, Matthews belief in the influenzal paradigm – supported by the success of his culture medium and the esteem it granted him – allowed him the confidence to critique the Committee’s ‘gifted and able bacteriologists’.[[153]](#footnote-153) The critique’s framing as one purely of dosage concerns also allowed him to express the belief that though the majority of the committee may have agreed to the dosage terms (and by extension the expressed doubts about *B.influenzae*’s role in the pandemic), it was in fact informed by a dependence ‘to a very great extent on the experience of at least a minority of its members’ which meant the Committee’s ‘opinion loses its weight’.[[154]](#footnote-154)

Matthew’s letter is referenced in two out of the three correspondences on the subject of ‘*Influenza and Preventive Inoculation’* in the following week’s journal, with W.H. Wynn agreeing ‘with Dr John Matthews that the doses recommended at the War Office Conference are inadequate and likely to imperil the value of the vaccine’ and Thomas Horder offering a scathing critique independent of Matthews’ argument which nonetheless ‘[endorses] emphatically the criticism appearing in Dr John Matthews letter in *The Lancet* of Nov 2nd as to the suggested dose of Pfeiffer’s bacillus’.[[155]](#footnote-155) Horder’s letter also recommends the use of Matthew’s medium for the cultivation of the bacillus for the vaccine, viewing it as an ‘essential’ part of vaccine logistics which ‘the committee does not expressly say’ anything about.[[156]](#footnote-156)

Horder’s thinly veiled critique of the Committee’s competence regarding vaccine production logistics is mild in comparison to his letter’s opening remarks. Horder lambasts the ‘lack of cogency in the conclusions arrived at by the committee’, asking whether ‘it would not have been wise if the committee had made a more frank confession of our present state of ignorance’ on influenza’s aetiology rather than giving ‘dogmatic advice’ in ‘the absence of accurate data’.[[157]](#footnote-157) He points out the inconsistency in the Committee’s statement of doubts about the role of Pfeiffer’s bacillus then being followed by an insistence on its inclusion within a vaccine, and eludes to the wider discontent shown in the correspondence section of the journal following the conference notes’ publication by confidently stating that ‘many of your readers must have been struck’ by the apparent disparities in the Committee’s conclusions.[[158]](#footnote-158)

Horder’s reference to the consensus of the community on his viewpoint can also be seen as contributing to his confidence in questioning whether ‘there is not present a slight savour of “intensive cultivation” about the whole argument, as though military necessity led to a forcing of the pace?’.[[159]](#footnote-159) Horder frames this expediting of vaccine decisions as a negative, thereby placing blame on the War Office for what he deems a foolhardy attempt to make bricks out of straw. Though this is not echoed in the other correspondences responding to the conference’s decisions, the dominance of the military over medical influenzal decisions is a core theme of Michael Bresalier’s 2011 article ‘*Fighting Flu: Military Pathology, Vaccines, and the Conflicted Identity of the 1918-19 Pandemic in Britain*’. Bresalier sought to demonstrate ‘how the wartime organisation of British medicine shaped definitions and knowledge of influenza’, specifically how ‘medical officials mobilised military pathology’ in order to offer solutions both to ‘the bacteriological problems thrown up by the pandemic’, and to the demands for prophylactic or therapeutic vaccines, largely for the purposes of reducing sickness in troops.[[160]](#footnote-160)

By 1918 pathological laboratories were ‘at the core’ of a militarised medical system which ‘bound the organisation, production and application of medical knowledge to the war’.[[161]](#footnote-161) Initially acting in a supportive role to assist in the ‘diagnosis and treatment of infectious diseases’, laboratories soon found their role transition to ‘the production of preventative and therapeutic techniques’, of which the antityphoid vaccine provided a model success.[[162]](#footnote-162) Bresalier underscores the significance of vaccine successes in charting not just the use of ‘vaccines for cholera, plague and dysentery’ alongside typhoid, but also by stressing that ‘the military relevance of laboratory medicine’ was further bolstered by the efficacy of treatments for diseases such as tetanus and diphtheria despite the serum therapies in question being pre-war discoveries.[[163]](#footnote-163) ‘For every disease’ Bresalier concludes, ‘the AMS [Army Medical Service] turned to pathology for solutions.’[[164]](#footnote-164)

The influenza pandemic proved no different. Concerns over an autumn second wave were already being voiced in August 1918, and with them came calls for the consolidation of laboratory knowledge on influenza in order for a central authority to manage the development of treatments and vaccines.[[165]](#footnote-165) This focus was military in nature, with Bresalier noting that it was ‘the threat of yet another epidemic jeopardising military operations’ that prompted the creation of the Committee which presented their findings at the War Office Conference on 14th.[[166]](#footnote-166)Framed in this light, the decision of the Committee to include Pfeiffer’s bacillus in the proposed vaccine despite an acknowledgment of the uncertainty of its role in the epidemic can be viewed as another aspect of the militarisation of influenza knowledge production during the period.

The decision to produce a mixed vaccine, made up of multiple organisms viewed as potential primary aetiological agents as well as the now doubted Pfeiffer’s bacillus, appears a pragmatic, rather than purely scientific, decision when the influence of the military is considered. Though mixed vaccines had proven largely ineffective in peacetime (as had pure Pfeiffer’s bacillus vaccines), the prospect of a militarily crippling second wave required *something*, and in the absence of any existing preventive or therapeutic drug a vaccine that *could* help (‘by reducing the incidence of disease and mitigating more severe complications’) was better than delaying production until an unspecified date at which a consensus could be reached on what definitely *would*.[[167]](#footnote-167) Bresalier does acknowledge that the push for vaccine development did have wider ramifications for the development of influenza knowledge beyond its military applications, arguing that pathologists saw the development of a successful vaccine as ‘a way to resolve influenza’s etiology’ by identifying the causative agent based on if a vaccine was effective’.[[168]](#footnote-168) However, his overall framing of British influenza knowledge production as a near-total militarised process places the onus of scientific community development on an entity – the War Office– outside of the community’s sphere. Though his article does demonstrate the merit of considering the impact of medical militarisation for understanding British pandemic control and prevention measures in 1918, for my study of changes in the thought style of the medical community, including adherence or divergence from the community’s founding paradigms, it is important to view debates around influenza vaccine composition not just as arguments on a military logistical issue, but rather articulations of paradigm challenges and changes in the thought style that affected the community’s fundamental understandings of influenza during the pandemic.

When this framing is taken into account, reactions to the October 14th War Office Conference’s recommendations demonstrate the medical community’s challenges to both the influenzal and bacteriological paradigms. Of the two, the influenzal paradigm sees the majority of the challenges, with the Conference Committee’s doubts on the bacillus’ role echoed in H.A. Des Vieux’s statement that the Committee’s conclusion that ‘influenza is caused by an unknown microbe’ represents ‘quite a tenable theory’ which ‘remains sub judice’.[[169]](#footnote-169) Though *The Lancet* correspondence’s endorsement of Matthews’ medium suggest a general support of the influenza bacillus, and thus the influenzal paradigm, Des Vieux’s letter proposing that influenza actually be understood as a ‘fever syndrome’ caused by any of the organisms found frequently, alongside Henry T Gillet’s correspondence insisting that ‘the primary infection in this epidemic is a streptococcus’ because ‘Pfeiffer’s bacillus is by no means always present, whereas streptococcus is’ demonstrate that there was growing scrutiny surrounding the aetiological role of the influenza bacillus during the autumn of 1918.[[170]](#footnote-170) The lack of consensus on what the *true* influenzal aetiological agent could be, however, prevented the autumn of 1918 from being a moment of paradigm shift. Under Kuhn’s framework of paradigm shift there is no persuasive paradigm candidate to which the community could switch, and from a pragmatic military perspective the abandonment of the existing aetiological agent would leave the medical community without a base for their vaccine formulas.

That the influenzal paradigm was only in the early stages of being challenged under the framework of ‘revolutionary science’ is evident in responses to a letter published in both the *BMJ* and *The Lancet* in the late autumn of 1918. Responding to articles in each journal which discuss influenza’s aetiology, Dr Robert Donaldson asserted that he had found ‘a certain organism’ in every case he has examined which has more compelling an argument for the role of primary aetiological agent than the ‘two infection theory’ (a primary infectious agent opens the way for a secondary infection, the latter of which results in deaths and is more visible in post-mortem cultures), and of the possibility of a filter-passer as the cause.[[171]](#footnote-171) He rejected entirely the role of the influenza bacillus (‘in view of the rarity with which Pfeiffer's bacillus has been found, the latter organism has nothing to do with the epidemic’) and instead argued for his organism, defending its previous lack of identification on its ‘extremely pleomorphic’ character when cultured.[[172]](#footnote-172) Donaldson claimed his organism fulfilled Koch’s postulates, including pathogenicity in animals which the influenza bacillus could not fully claim. However despite his convincing argument, Donaldson did not receive any immediate response to his article in either journal, with his call for ‘others who are working on the problem of the epidemic’ to ‘corroborate or otherwise [disprove] my findings’ going initially unanswered.[[173]](#footnote-173) Despite voiced doubts on the influenza bacillus, the community was not at the point within Kuhn’s framework in which consensus was reached on the inability of the old paradigm to support scientific development, or on a new contender for a paradigm for which all the community could agree to support. Donaldson did not receive the same level of response that Matthews’ defence of the bacillus had received a few months earlier, demonstrating the continued strength of the influenzal paradigm even with growing recognition of the anomaly the bacillus at its centre was causing in pathological reports.

As part of Donaldson’s letter in the *BMJ*, he dismisses the prospect of influenza’s primary agent being a filter-passer in favour of pushing his organism as the primary contender. His dismissal is largely technical, demonstrating a near textbook example of Kuhn’s end of crisis scenario in which technological limitations means ‘that no solution will be forthcoming in the present state of the field’ in his statement that ‘if there be such an agent it is unlikely that any vaccine, prophylactic or otherwise, now in use will be of any value, and the substantiation of this view would probably condemn us to wait for some considerable time before an efficient and scientific method of prevention or cure is found’.[[174]](#footnote-174) However, Donaldson’s mention in his concluding paragraph that his theory of dual-phase organisms (in which his organism’s pleomorphic nature could be explained as part of a longer bacterial life-cycle) ‘is a view not inconsistent…with a filter-passer theory’ shows that even with the acceptance of technological limitations, the prospect of a filter-passer as a factor in influenza’s aetiology was credible enough a concept to bear positive mention in a discussion of the disease.[[175]](#footnote-175) This follows a wider trend within *BMJ* autumn reporting which published multiple articles on filter-passers and their link to the present pandemic, thereby providing a challenge to the bacteriological as well as influenzal paradigm.

Filter-passers were first identified in tobacco plants in the 1890s. Sap from diseased plants was found to be infectious to healthy plants, even when the sap had been passed through a Pasteur-Chamberland filter. Bacteria did not easily pass through these filters, and unsuccessful attempts to culture the infectious sap further supported the conclusion that the infectious material causing the tobacco plant disease was not a bacteria, nor could it be a fungus. Thus the causative agent, though it clearly existed, remained unknown. Also in 1898, a similar unknown, invisible filter-passing organism was found to cause foot and mouth disease in animals, and in 1901 studies of yellow fever found that filtered serum from a sick patient caused the disease in a volunteer despite no bacteria or ‘visible microorganisms’ being observed.[[176]](#footnote-176) Unidentifiable pathogenic organisms were therefore known by 1918 to cause disease in plants, animals and humans, a fact which Melvin Santer argues ‘upset the prevailing model of the bacterial or fungal cause of contagious disease’, and prompted existing bacteriological (and fungal) paradigm to appear ‘too restrictive to explain the nature of all contagious disease agents’.[[177]](#footnote-177) Santer acknowledges that ‘the paradigm was not discarded’ solely as a result of these late 19th and early 20th century discoveries, however the existing pressures on the paradigm caused by these discoveries are important to consider when analysing the reception of filter-passer findings in relation to the 1918 pandemic.[[178]](#footnote-178)

*The Lancet* saw just two direct mentions of filter-passers in 1918 discussions on flu, the first being within Donaldson’s November 23rd letter, and the second in a report from a meeting held on November 15th by the Royal Academy of Medicine in Ireland which suggested that the effectiveness of vaccines both with and without the influenza bacillus ‘suggested that a filter passer was at fault’.[[179]](#footnote-179) This differs greatly from *BMJ*’s coverage of filter-passer theory developments, with six mentions of filter-passers in articles from November 2nd, including a detailed report published on December 14th 1918 of experiments undertaken by Major H Graeme Gibson, Major FB Bowman and Captain J I Connor in order to corroborate findings by French bacteriologists Charles Nicole and Charles Lebailly. These experiments involved injecting monkeys (and in the original study, two men) with filtered and unfiltered bronchial secretions from influenza patients and observing if the injected subject developed influenza symptoms. In both the original study and replicated study by Gibson et al., as well as in a further self-inoculation experiment by Dujarric de la Riviere, bronchial secretions which had been passed through a Chamberland filter were found to cause a ‘marked attack of influenza’, proving that ‘the virus of influenza can pass through a Chamberland filter’ and so must be a filterable organism.[[180]](#footnote-180) These findings did not represent a moment of complete paradigm shift, in fact the *BMJ* article concluded that ‘those who accept [a hypothesis associated with filter-passers causing disease] may still maintain their belief in Pfeiffer’s organism’ by assuming ‘that it has a filtrable stage’.[[181]](#footnote-181) However, the report summary’s concluding sentence, which admits that ‘the virus may be totally distinct, and the majority of the members of the War Office Conference last October were of opinion that the existence of some as yet undiscovered virus must be regarded as possible’ does indicate the growing doubts towards the existing paradigm as the filter-passer theory gained more credibility.[[182]](#footnote-182)

Not all opinions on the filter-passer theory were as positive, with an earlier discussion of influenza’s aetiology though itself responding favourably to Nicole and Lebailly’s findings, noting the sentiment within the wider medical community that ‘the suggestion that the true virus is a filter-passer as it has been said…is only a cloak to our real ignorance as to its nature.’ [[183]](#footnote-183) Similarly, a Lancet article published on December 28th 1918 dismissed the filter-passer theory as being only a ‘remote possibility’, arguing that an ‘underlying ultra-microscopic agent’ was inconceivable as a singular cause of a disease which affected ‘its victims in such diverse ways’.[[184]](#footnote-184) These doubts reflect the broader crisis point which the pandemic produced, with not just influenza’s aetiology, but also its presentation and the techniques used to isolate it, under question. As the pandemic’s second wave began to wane in the early months of 1919, these questions were amplified as both journals’ contributors reflected on their pandemic experiences.

## ***Section IV: Ends and Beginnings: January – December 1919***

In the first issue of the new year, the *BMJ* noted that, judged on a comparison of mortality statistics, ‘it appears that the subsidence of the existing wave of influenza is definitive’.[[185]](#footnote-185) It would prove to be the only thing definitive about the influenza pandemic within the minds of Britain’s medical men. The pandemic had tested what was known about influenza’s symptomology, epidemiology and aetiology. Whilst vaccine debates and discussions over culture mediums had gone some way to unify, or at least direct, scientific thought towards the most prominent bacillus-based conundrums (*Is the unreliability of the bacillus found in culture a result of culture medium or method? What composition of bacilli should a prophylactic or therapeutic vaccine use? Are the high instances of pulmonary complications a result of infection by secondary agents, and if so, which ones*?), the second wave’s end, as well as the armistice of the war, reduced the impetus on prophylactic vaccine research and afforded many contributors time to analyse results gained at the pandemic’s height and from them attempt conclusions on a far wider variety of questions.

Of these questions, it was the role of the filter-passer in influenza, rather than that of Pfeiffer’s bacillus, that dominated early 1919 discussions in both journals. *The Lancet’s* first issue of the year featured comment on a ‘series of observations from various sources...appearing to establish a “filter passer” as the primary agent and essential cause’ of influenza, whilst Major General Sir John Rose Bradford, Captain E. F. Bashford and Captain J.A. Wilson’s ‘Preliminary Report on the Presence of a “Filter Passing” Virus in Certain Diseases’, published in both journals on February 1st 1919, drew wide support for the filter-passer theory.[[186]](#footnote-186)

The concept of a filter-passer was enticing, however, for different reasons than in 1918. Whereas the initial discovery of a filter-passer acted as an outlier challenge to the Pfeiffer bacillus led influenzal paradigm, by 1919 its existence offered the potential to ‘harmonise’ contradictory knowledge gained during the pandemic, thus working to strengthen rather than challenge the paradigm.[[187]](#footnote-187) Though initially found sparingly in cultures, the adoption of Matthews’ medium (and later similar blood agar mediums proposed by Levinthal and Gordan) had increased the identification of Pfeiffer’s bacillus in samples, to the point that by January 1919 ‘trained bacteriologists of the first class…[can] announce that Pfeiffer’s’ bacillus is obtainable in abundance from the sputum of practically every case of the disease’.[[188]](#footnote-188) As much as its role was doubted, these findings meant that it could not be completely excluded from consideration as it perhaps could have been when it was found in only limited instances in the first few months of the pandemic. That this increase in successful culture coincided with the increasing severity of the epidemic further insinuated the continued significance of the bacillus, with the timeline of identification also fitting that of the last influenza pandemic, wherein ‘the bacillus was only found towards the end of the epidemic in 1892 by Pfeiffer’.[[189]](#footnote-189) Influenza knowledge therefore needed to account for both the initial absence and later prevalence of the bacillus, whilst also explaining the existence of numerous other cocci/bacilli found in cultures. The presence of a filter-passer – in itself another anomaly to be added into the influenzal canon - was thus co-opted to fulfil this harmonising role.

Robert Donaldson, as we have seen, had hypothesised on a link between his pleomorphic organism and the filter-passers in his December 21st 1918 correspondence to the *BMJ* (‘It [the prospect of the organism being pleomorphic and having multiple identifiable stages] is a view not inconsistent, as I have said, with a filter-passer theory’), and his 1919 letters to *The Lancet* advance both his organism and the role of the filter-passer in this light.[[190]](#footnote-190) On February 15th he responded favourably to Bradford et al.’s ‘preliminary report…on a filter-passing virus found in certain diseases’, linking their findings of the filter-passer in influenza to those of Nicolle and Lebailly before stating that, in light of this and American bacteriologist Edward Carl Rosenow’s filter-passer discoveries ‘it seems necessary to emphasise’ that the he ‘did not necessarily exclude the possibility of a filter-passer phase’ of the organism he described (Organism “D”).[[191]](#footnote-191) By this, he stressed, he does not mean that the filter-passer and Organism “D” were consecutive infections (with one or other being a primary invader and the other secondary), but instead argues for a pleomorphic understanding of the causative agent of influenza that can account for both findings in culture.

Under a pleomorphic understanding of influenza’s aetiological agent, the lack of consensus within culture findings can be explained by viewing the different isolated organisms (a filter-passer, Pfeiffer’s bacillus, varying strepto-/pneumo-/micro-cocci) as phases within a singular organism’s overall life. Bacteriologists testing samples from both sick and post-mortem patients, and at different times during the pandemic’s waves, could therefore expect to see different presentations of the organism in culture. *The Lancet’s* January 4th 1919 article on the pathology of influenza described this as potentially having two phases, ‘a minute filterable form which becomes haemal and a bacillary form mainly developed in the air passages’, the second of which could resemble Pfeiffer’s bacillus or any other of the pathogens found regularly.[[192]](#footnote-192) This line of reasoning technically disqualified Pfeiffer’s bacillus from consideration as flu’s aetiological agent (‘it follows that Pfeiffer’s bacillus, as that is usually understood and described, can no longer be looked upon in the light of a responsible etiological factor’), however Donaldson’s clause that Pfeiffer’s bacillus is excluded, as it ‘is usually understood and described’ suggests an effort to reshape its involvement in the influenzal paradigm, rather than an outright rejection.[[193]](#footnote-193) Nevertheless, the presence of a filterable virus, or indeed a pleomorphic unidentified organism, did serve to challenge the influenzal paradigm and bring into motion a period of true revolutionary science according to Kuhn’s framework. This is represented cogently in a response to Donaldson’s February 15th letter.

On February 15th, Donaldson reasoned that ‘until it is proved beyond any questions of doubt that the filterable virus can exist in no other form’, it is ‘obviously fallacious’ to argue that the presence of both a filter-passer and other bacilli/cocci indicates multiple infective agents rather than one agent with multiple forms.[[194]](#footnote-194) This line of reasoning was echoed by a supportive response to Donaldson’s letter on February 22nd, in which F. G. Crookshank emphasised that ‘there is no a priori reason to assume that filter-passing organisms have no other stage or form of existence’.[[195]](#footnote-195) Crookshank viewed the what Donaldson termed as ‘fallacious’ arguments against this theory of pleomorphism as a demonstration of ‘excessive reliance on positivist forms of thought and analytical methods of examination’ that served to limit scientific development and ‘will not carry us much further’. In the conclusion of his letter he therefore called for more emphasis on ‘synthetic and imaginative faculties, without which’ he felt – echoing Darwin – ‘there will be no useful observation.’[[196]](#footnote-196) In other words, he called for a rejection of the known – the paradigm – in favour of a ‘synthetic (meaning dependent ‘upon the facts about the world’ not just those of the internal discipline) approach to scientific research.[[197]](#footnote-197) This bears marked similarity to Kuhn’s definition of extraordinary/revolutionary science, in which the rules of normal science (the paradigm) are pushed to their limits and then explored beyond in the search for solutions to the anomaly.[[198]](#footnote-198) Crookshank, to use the Snakes and Ladders Analogy, is calling for the players to shake the box and try out new ways to play with the extra piece.

Crookshank’s letter did not receive specific published responses in the weeks following its publication. However, his call for a period of revolutionary science to resolve the influenza aetiology anomaly was answered in the general trends, or rather lack thereof, in aetiological discussions across both journals in the first six months of 1919.

The strongest contender to the filter passer theory was that of symbiosis. Within the symbiotic theory of influenza’s aetiology, the Pfeiffer Bacillus (or indeed in some instances a filter-passer) is theorised as the initial invader, which cooperates symbiotically with other organisms which cause the fatal symptoms and appear most commonly on post-mortem cultures. *The Lancet*’*s* January 4th article on influenza’s pathology, though supporting the filter-passer theory in the first instance, concluded by stating that should the filter-passer be found not to be pleomorphic with bacterial forms, then ‘the symbiotic theory will have to be accepted’, a version of which, they confessed ‘appeal[ed] more’ to them when viewed in the context of their recent pandemic experiences.[[199]](#footnote-199) *The Lancet* article suggested that ‘one organism, the filter-passer, [prepares] the ground for another, the influenza bacillus, which for a time flourishes and had the upper hand, and in its turn prepares the way for, and is replaced by, a member of the streptococcus group, or by the pneumococcus…’, a pattern of infection echoed (minus the filter-passer stage) by Captain William T Munroe’s article ‘*Some Bacteriological Findings in Epidemic Influenza*’ published in the *BMJ* on the 22nd March (‘the influenza bacillus was probably present in all cases at the beginning of the illness and that the pneumococcus and the streptococcus were mainly responsible for the pulmonary complications’) and W. James Wilson and P. Steer’s ‘*Bacteriological and Pathological Observations on Influenza as Seen in France During 1918*’ (we consider that in influenza Pfeiffer’s bacillus acts as a pioneer and prepares the way for pneumococci, staphylococci and streptococci’).[[200]](#footnote-200)

The symbiosis theory accounted for the presence of both the influenza bacillus and other bacilli/cocci in cultures, thereby allowing for an expansion of the influenzal paradigm rather than an outright rejection. The influenza bacillus could be said still to play an important role in the disease, without its irregularities in culture being used as proof to the contrary. Symbiosis was also in support of the broader bacteriological paradigm, with an April 12th *Lancet* article pointing to questions of symbiosis as the natural progression in bacteriological research from ‘the early years of…obtaining pure cultures…studying morphological differences for the purposes of classification…working out the problem of identifying disease’ to the more advanced present investigations into ‘the biochemical properties of our cultures’.[[201]](#footnote-201) Other solutions put forward to resolve the crisis of influenza’s aetiology did, in some instances, reflect this continued adherence to the bacteriological, if not the influenzal, paradigm, however the period of revolutionary science also produced ‘solutions’ to the crisis inducing anomaly that did not align with either paradigm.

Conforming with the bacteriological paradigm, several articles proposed alternate bacteriological agents as the cause of the influenza pandemic. These included the ‘usual suspects’ of 1918 (streptococcus, pneumococcus, M. Catarrhalis etc), but also more left field suggestions including a diphtheria-Pfeiffer’s bacillus hybrid or a previously misidentified bacillus of plague type.[[202]](#footnote-202) Others had, from late 1918, challenged the knowledge underpinning the influenzal paradigm alongside its presumed bacteriological cause, questioning whether “influenza” (as it continued to be scare-quoted) was instead ‘the name given to a well-recognised fever syndrome’, or a disease ‘affecting the cerebro-spinal nervous system’ in order to explain the pandemic’s symptom presentation and, in the latter case, the absence of the bacillus from lung lesion cultures despite pulmonary symptoms.[[203]](#footnote-203)

The biggest diversion from both paradigms was the suggestion of a mycotic aetiological agent. Mycosis refers to infection of tissue by fungi, suspicions of which were generated in influenza research by reports of ‘spores’ resembling Donaldson’s ‘Organism “D”’ found in culture, alongside his suggestions of pleomorphism and organism life-stages.[[204]](#footnote-204) The possibility of mycotic infection was initially posited by H.J.B. Fry in the July 12th edition of *The Lancet*, who concluded that ‘influenza may be of mycotic origin’ based upon his findings and their similarities to Donaldson’s.[[205]](#footnote-205) Increased legitimacy was granted to the idea, however, by G.E. Beaumont’s August 9th article ‘*The Aetiology of Influenza*’ which represented a detailed summary of the influenzal field. Following a comprehensive overview of doubts surrounding Pfeiffer’s bacillus and a discussion of the potential role of filter-passers in the disease, Beaumont, working alongside Fry, concludes that his ‘examination of material obtained from patients suffering from influenza has shown the presence of a mycotic organism’ and that it was therefore ‘possible that the disease is a mycosis’.[[206]](#footnote-206) The potential for these findings to act as an anomaly resolving force is recognised in Beaumont’s concluding statement that the demonstration of a mycotic organism in influenza cases ‘afford[s] a means of unifying the seemingly discordant results that have been obtained by other workers’.[[207]](#footnote-207) This same hope of unification is echoed by the report on his paper in the same issue which predicts that ‘if [Beaumont’s] results are confirmed… then the relation of the various forms of the parasite to one another and to the different organisms found in this disease by other observers will afford interesting matters for investigation’ and could ‘clear up the confusion at present existing by explaining the discrepancies and coordinating the divergent views’.[[208]](#footnote-208)

Despite the demonstrated popularity of the mycosis theory as a way to resolve the influenza anomaly, it remained just one of multiple ‘out of the box’ suggestions put forward by the scientific community within the period of revolutionary science. A reason for this, and for why revolutionary science did not lead to a paradigm shift in 1919, is alluded to within J. Ramsbottom’s 6th September 1919 comment upon Beaumont’s article, in which he expresses reluctance in accepting the mycosis theory without ‘rigid proof’ that Beaumont’s findings, and the methods that produced them, were correct.[[209]](#footnote-209)

For scientific revolution to occur, Kuhn stresses the importance of community consensus on the adoption of a new paradigm. For consensus to be attained, the new paradigm must be persuasively presented as a suitable alternative, with its viability also convincing adherents to the old paradigm that it would no longer be able to ‘solve all of [the community’s] problems’ as it had before.[[210]](#footnote-210) This can be achieved by demonstrating that the new theory ‘can solve the problems that have led the old to a crisis’, by offering a new theory which ‘appeal[s] to the individual’s sense of the appropriate or aesthetic – that the new theory is said to be “neater”, “more suitable”, or “simpler” than the old’, or by being so different from the existing paradigm that the theory’s ‘shock value’ proves ‘especially persuasive’.[[211]](#footnote-211) Ramsbottom’s request for more laboratory proof of the mycotic theory therefore represents an attempt to test whether it quantitatively fit any of these criteria.[[212]](#footnote-212)

For Kuhn the act of conversion to a new paradigm is neither instantaneous nor unanimous. Rather, the gradual increase in supporters for a new paradigm as it is refined, and further proof of its suitability is confirmed, eventually results in a shifting of professional allegiances from the old paradigm to the new.[[213]](#footnote-213) Though the first half of 1919 saw an increase in support for new aetiological theories (with the filter-passer and symbiosis theories undergoing the most refinement and so gaining the largest number of shifted allegiances), in the second half of the year contributors to the *BMJ* were more vocal in their criticisms of the filter-passer theory, whilst *Lancet* authors largely reneged their support of any specific theory on the aetiology of flu.

J. A. Arkwright published his ‘Criticism Of Certain Recent Claims To Have Discovered And Cultivated The Filter-Passing Virus Of Trench Fever And Of Influenza’ in the *BMJ* on August 23rd. In it, he notes the growing body of work on the subject of filter-passers and offers his own findings as a means of critiquing the claims of influenza’s filter-passer cause published by Bradford, Bashford and Wilson in February and May of 1919. Despite following the same methods as Wilson, and being ‘familiar practically’ with the methods and media used, Arkwright was ‘quite unable to distinguish macroscopically or microscopically between tubes inoculated with…influenza virus…and control tubes’.[[214]](#footnote-214) More alarmingly, he found that the culture tubes were ‘always’ contaminated by bacteria, including those shared by Wilson for Arkwright to re-examine.[[215]](#footnote-215) Rather than proving the existence of a filter-passer, Arkwright concludes that ‘Captain Wilson has been misled by the appearance in films of bacteria…and in the case of cultures from which bacteria has been absent he has been deceived by the diplococcus-like or dumb-bell shaped forms…and he has regarded these…as organisms’.[[216]](#footnote-216) From this he also questions the veracity of Bradford et-al.’s inoculation results, arguing that ‘if the cultures used [to inoculate animals and fulfil Koch’s third postulate] were contaminated with bacteria to the same extent as those from which the films have been shown to the writer’ then it is those bacteria, not a filter-passer, that are responsible for observed symptoms.[[217]](#footnote-217) For these mistakes, Arkwright blames Bradford et-al.’s isolation from other bacteriologists, ‘a tendency to place undue reliance on the efficacy of filters’, and inadequate laboratory techniques and methods.[[218]](#footnote-218) In an attempt to refine the filter-passer contender for paradigm, Arkwright’s results instead cast doubt on the theory’s suitability, both with his own findings and in calling into question the capabilities of the theory’s strongest proponents.[[219]](#footnote-219)

Further evidence of the filter-passer theory’s limited persuasibility is demonstrated in Ciauri’s dismissal of the filtrable virus theory in the *BMJ’*s 22nd November 1919 ‘Epitome of Medical Literature’ section. Ciauri compares those that discard ‘obvious bacilli like Pfeiffer’s bacillus’ in favour of a filter-passer to ‘Plato’s cave dwellers, who never saw anything but the shadow – the shadow in this case being the filterable virus.’ He sees the adoption of a filter-passer theory as a rejection of the bacteriological paradigm, and warns against ‘the wholesale relegation of bacteria to a comparatively unimportant position’ as a secondary agent to an initial filterable invader.[[220]](#footnote-220) He is more willing to accept symbiotic or pleomorphic explanations (‘the invisible virus is something which mobilizes the latent pathogenicity of the invading germs, or it may be that certain pathogenic micro-organisms take on a filtrable stage in which they spread contagion’) which ‘saves us from a wholesale repudiation of bacteria as etiological factors in disease’, thereby demonstrating both the continued significance of the bacteriological paradigm within understandings of disease, and the lack of a singularly persuasive contender for a new influenzal paradigm.[[221]](#footnote-221)

Ciarui’s mention of ‘the obvious bacilli like Pfeiffer’s bacillus’ is a significant departure from the general trend towards a rejection of the Pfeiffer bacillus within 1919 journal publications. Certainly it did not return to become the dominant theory – articles in the *BMJ* and *The Lancet* between September and December 1919 continue to propose anthrax-like bacilli, diplococci, pneumococci and streptococci as potential causes – but it did regain some credibility in the latter months of the year.[[222]](#footnote-222) W. Camac Wilkinson, writing in the November 1st 1919 edition of the *BMJ*, proposed a symbiosis of ‘Pfeiffer’s bacillus and Staphylococci’, whilst a note on research undertaken by Coronini and Priesel on ‘The Pathology and Bacteriology of the Influenza of 1918’ in the November 29th 1919 ‘Epitome of Current Medical Literature’ showed a reluctance to dismiss the possibility of Pfeiffer’s Bacillus being found in their cultures (‘In sections of lungs from many cases they saw organisms which they believed to be [a] coccus, but…were *possibly* Pfeiffer’s bacillus’ [emphasis own]).[[223]](#footnote-223) The most striking endorsement came on December 13th, where R. Kraus is reported as being ‘convinced that the causal agent of the epidemic of 1918-19 is Pfeiffer’s’ bacillus.’[[224]](#footnote-224)

Contributors to *The Lancet* remain more sceptical of Pfeiffer’s bacillus, with the increase in references in the final months of 1919 largely discrediting the bacillus, and in doing so further demonstrating the ‘revolutionary science’ period the journal’s community was experiencing. Subba Rae, in a correspondence on November 29th 1919, reflected on their own findings in light of Beaumont’s 9th August article on influenza’s aetiology, stating that ‘my mind was so much mesmerised by the so-called Pfeiffer’s bacillus that I paid no attention’ to other organisms found in culture, dismissing them as ‘accident or artefact’.[[225]](#footnote-225) Rae’s reflection on being ‘mesmerised’ by the bacillus bears striking similarity to Honigsbaum’s suggestion of ‘paradigm blindness’. However, the fact that Rae is able to reflect on this mesmerisation demonstrates that they are no longer a ‘prisoner’ of the old paradigm and are instead operating within a revolutionary period in which new theories can be considered.

This move beyond ‘prisoner’ status is further elucidated by Sheldon F. Dudley’s ‘Note on B. Influenzae’ which alongside recommendations for further studies on the bacillus’ symbiotic connections to filter-passers also suggested that there would be merit to studying the number of people who ‘in normal times harbour *B. Influenzae* in their naso-pharynx.[[226]](#footnote-226) If the bacillus was found in swabs from the healthy population it would fail Koch’s first postulate (‘the microorganism must be found in diseased but not healthy individuals’).[[227]](#footnote-227) The fact that Dudley considers there to have been ‘enough evidence…collected to make it worthwhile’ to investigate a theory which would disqualify the bacillus from aetiological contention shows how far from blindness the medical community had come by the end of 1919, even if the period of revolutionary science was yet to produce a viable new paradigm.[[228]](#footnote-228)

The state of influenza knowledge at the end of 1919 is best summarised by W. Hughes opening statement in his article on ‘The Value of Antistreptococcus Serum in Influenza’: ‘That the bacteriological reports of the recent epidemic of influenza have been conflicting is not to be wondered at when we have to deal with several organisms, each in itself capable of producing the pathological result’.[[229]](#footnote-229) The period of revolutionary science triggered by the pandemic produced multiple theories of influenza’s causation in response to doubts around the Pfeiffer bacillus. None, however, were persuasive enough to be accepted as the new paradigm during the pandemic and immediate post-pandemic period. As the final section of this chapter will explore, acceptance of a new paradigm required technological advancements, theory refinement, and over a decade of heated medical debate.

## ***Section V: The Influenza Problem: January 1920 – July 1933***

By the end of 1919, the only real consensus on the aetiology of influenza was that there was no consensus. The crisis caused by the anomalous appearance of Pfeiffer’s bacillus in influenza cultures had prompted a period of revolutionary science, with the existing influenzal paradigm being challenged by a series of alternate theories about influenza’s aetiology. One of these theories, that of a filter-passer being the primary aetiological agent, also served to challenge the bacteriological paradigm which had dictated scientific inquiry and understandings of disease causation since the 19th century.

This section will offer a snapshot into the prolonged period of revolutionary science between January 1920 and the discovery of the influenza virus in July 1933. Michael Bresalier considers this a significant period of ‘knowledge making’ in which the 1918-19 pandemic became ‘inextricably linked’ with virus research undertaken by the UK’s Medical Research Council (henceforth MRC). [[230]](#footnote-230) This suggestion eludes to the dominance of the filter-passer theory of influenza’s causation within medical thought, if not the acceptance of it as a new influenzal paradigm before the eventual discovery of the influenza virus in the 1930s. Ton van Helvoort, however, argues that the bacteriological paradigm’s influence remained strong not just in the decade following the pandemic, but also in the way research was undertaken following the discovery of the influenza virus.[[231]](#footnote-231)

By examining a selection of medical literature alongside official British reports and memorandums on influenza produced during the 1920-1933 period, I will chart the impact of the revolutionary science period on attitudes towards the Pfeiffer bacillus and the popularity of the filter-passer theory of influenza’s aetiology. This will highlight how both the influenzal and bacteriological paradigms were challenged by the refinement of the filter-passer theory, with technological advancements producing increasingly persuasive evidence of its suitability as a new paradigm. However, a demonstrated resurgence in popularity of the Pfeiffer bacillus, alongside continued expression of doubt on filter-passers late into the 1920s, will also work to conclude that though Bresalier’s work on the MRC’s attitudes towards virus research suggests a pre-discovery acceptance of a new influenzal paradigm, in reality wider medical literature is more closely aligned with van Helvoort’s insistence on continued bacteriological paradigmatic influence.

The introduction to the 1920 official ‘*Report on the Pandemic of Influenza 1918-19’* demonstrates the division of opinion on the cause of influenza in the immediate aftermath of the pandemic. In his summary of the report’s chapters and their contributor’s, Chief Medical Officer George Newman notes that ‘the problem of the cause of influenza is not yet solved’ with ‘clinicians, bacteriologists and epidemiologists’, including two contributor’s to the Report (Dr French and Dr Frederick W. Andrewes’) unable to agree on the role of Pfeiffer’s bacillus in the disease.[[232]](#footnote-232) That two contributors to the same report hold opposing views of the pandemic’s aetiological agent - French believing the bacillus ‘has not yet been deposed from its place as the causal organism’ and Andrewes remaining more sceptical - exemplifies the transitionary period of theory testing which Kuhn deemed vital for the adoption of a new paradigm. As an adherent to the old paradigm, French was unwilling to accept that a new theory could resolve the crisis-causing anomaly (or indeed may view the anomaly as having been resolved by the existing paradigm). Andrewes, though far from a proponent of the new theories which arose from the early revolutionary science period of 1919, was closer to being persuaded, and, crucially, recognised that the existing paradigm was unable to fully explain the anomaly of the bacillus’ absence in culture for the first months of the pandemic.[[233]](#footnote-233)

Andrewes, a respected pathologist who had trained under Klein (the British doctor who had corroborated Pfeiffer’s findings of the influenza bacillus in 1892 ), was not willing to conclude equivocally that the Pfeiffer’s bacillus was not the cause of influenza, though did acknowledge that ‘the position of Pfeiffer’s bacillus as the primary cause of the disease has been in no way strengthened’ by work undertaken during the pandemic.[[234]](#footnote-234) ‘The crucial tests to which it has been submitted’ he noted, ‘seem to indicate it rather as a secondary infection’ but all the same ‘it cannot be asserted that, as a primary cause, it is wholly out of court’.[[235]](#footnote-235)Alongside his reluctance to disavow the role of Pfeiffer’s bacillus, he was unconvinced of the filter-passer theory, stating in spite of several pages detailing the varying research undertaken by the likes of Nicolle and Lebailly, and Gibson et.al, that ‘while…some of the observations summarised above are suggestive of a filter-passer…it cannot be said that any of them offer concrete proof of such a proposition.[[236]](#footnote-236) Andrewes cited Arkwright’s criticism of filter-passer research methodology as a reason to be suspicious of results suggesting a filter-passing cause (‘results so far recorded…must be received with caution’) and overall remarks that ‘far more data are required before we can fully accept a filter-passing virus as the primary cause of influenza’.[[237]](#footnote-237) This demonstrates that despite refinement during the latter part of 1919, the filter-passer theory was still far from convincing all echelons of the influenzal paradigm’s old guard.

That Andrewes did not show more support for the prospect of a filter-passing cause of flu is interesting when his remarks on the wider state of bacteriology are considered. Before engaging in a discussion of the pandemic’s aetiological findings, Andrewes noted ‘certain factors which have contributed to widen the outlook of the pathologist’, including advances in ‘the technique of bacteriology’ and ‘the interpretation of the data which it reveals’.[[238]](#footnote-238) Specifically, Andrewes drew attention to the ‘horizon of the bacteriologist’ being widened by the fact that ‘he is no longer restricted to the bacteria proper in his search for a pathogenic microbe’: microparasites and the ‘so-called “filter-passing” group of microbes’ are both mentioned as credible new avenues of investigation.[[239]](#footnote-239) This he saw as important, for it could work to counter the restrictive adherence to germ theory which he believed ‘is now so firmly established that we are in danger of too readily accepting a microbe as the causal agent of the disease’.[[240]](#footnote-240) Viewed in isolation, Andrewes’ comments suggest growing doubts in the ability of the bacteriological paradigm to support disease research, and suggest a rejection of the influenzal paradigm given that it was formed upon ‘germ theory’ principles. Yet his chapter’s conclusions on the role of Pfeiffer’s bacillus within the 1918-18 pandemic instead show a continued adherence to the influenzal paradigm, with his brief reference to the potential of a symbiotic element to the pandemic’s mortality figures supporting, rather than challenging, a bacteriological concept of disease.[[241]](#footnote-241) This contradiction is indicative of the personal cognitive as well as professional divisions caused by the influenzal paradigm’s movement into revolutionary science, with those unconvinced by a potential new paradigm theory (namely the filter-passer) still faced with the crisis-causing anomaly that prompted the revolutionary period and which remains unsolvable within the constraints of the existing paradigm.

Andrewes’ chapter also indicates that while pandemic experiences, especially concerning the role of filter-passers, had begun to sow doubts surrounding the strength of the bacteriological paradigm, it was far from being challenged in the same way as the influenzal paradigm in the immediate post-pandemic period. This supports van Helvoort’s argument in the continued strength of the bacteriological paradigm as it related to influenza in the interwar years, though the fact that the official report singles out only the Pfeiffer bacillus and the filter-passer as potential causative organisms, rather than draw attention to the numerous other bacteriological contenders posited in 1918-19, does suggest that the scientific community had moved towards what Kuhn identified as the testing period ‘as part of the competition between two rival paradigms for the allegiance of the scientific community’.[[242]](#footnote-242) Rather than the early revolutionary science period which aims to identify theories which could resolve the crisis-causing anomaly, the later stages of revolutionary science are comprised of just one theory being tested against the existing paradigm. The filter-passer represents this new theory, with continued evocation of Pfeiffer’s bacillus by medical literature acting as the existing paradigm which the new theory is tested against. The existing paradigm in question is the influenzal paradigm, however the fact that the new paradigm contender represents a non-bacteriological agent also demonstrates that despite evidence of the continued strength of the bacteriological paradigm in influenza research, it was still being challenged by the revolutionary science period in the early 1920s.

1922 saw the return of epidemic influenza in Britain. In response, the Ministry of Health formed an ‘Influenza Committee’ made up of many of the voices considered influential in 1918-19 discussions of the disease.[[243]](#footnote-243) The notes of this committee’s frequent meetings provide insight into the research being undertaken in response to continuing questions about influenza’s aetiology. As with the 1920 Report, it is discussion of Pfeiffer’s Bacillus and the filter-passer which dominates. Refinement of the filter-passer theory is demonstrated in reported investigations by Dr Mervyn Gordon (who is ‘continuing his search for the presence of a filter-passer and endeavouring to confirm the work’ of other filter-passer proponents) as well as Professors McIntosh and Dean.[[244]](#footnote-244) The Pfeiffer Bacillus, meanwhile, was reported as present in some cases (Dr Perdrau of Lambeth Infirmary reported the bacillus being isolated ‘in all the cases’ and Dr McIntosh finding it ‘in the majority’ from Middlesex Hospital) but not in others, thus recreating the same anomalous conditions that caused the revolutionary science period in 1918-19.[[245]](#footnote-245) Indeed Dr Fildes, from the London Hospital, demonstrated a similar change of opinion as experienced by doctors in late 1918, in which he ‘was at first of the opinion that Pfeiffer’s bacillus played no part in the epidemic…but his later experience [of finding the bacillus in culture] led him to modify his conclusion’.[[246]](#footnote-246)

Some of the investigations into the 1922 influenza were working to justify its varied presence, e.g. the work done ‘at Sheffield…where they are attempting to correlate the occurrence of Pfeiffer’s bacillus with various stages of the disease’.[[247]](#footnote-247) However other investigations reported by the Committee demonstrate the lack of faith in the existing influenza paradigm, including Professor Dean’s ‘inquiry into the Pfeiffer’s’ bacillus in both sick and healthy persons’ which echoed Sheldon F. Dudley’s 1919 Lancet Article in suggesting that the bacillus was found only coincidentally in influenza cases and did not fulfil Koch’s first postulate in only being found in cases of disease.[[248]](#footnote-248) This opinion was reiterated by the Committee’s report on ‘Influenza Bacilli in Respiratory Inflammations’ which concluded that ‘One may safely say that during epidemic and the earlier post-epidemic periods influenza bacilli are so commonly found in the respiratory mucosa of the general population that no certain deduction in favour of their pathogenic activity may be drawn from their presence even in abnormal number in the discharges or local lesions of respiratory disease’.[[249]](#footnote-249)

Though the Influenza Committee reports show a high level of scepticism in Pfeiffer’s bacillus, despite it in some instances being found in 1922 cultures, this scepticism was not unanimous within the wider medical community. Also published in 1922, though made up of research gathered in 1918-18, James McIntosh’s ‘Studies in the Aetiology of Epidemic Influenza’ concluded that: ‘The almost constant presence of B influenzae in the secretions and lesions of influenza during the late epidemic is indicative of an aetiological relationship’.[[250]](#footnote-250) McIntosh considered the bacillus to have fulfilled all of Koch’s postulates, and conversely saw there to be ‘not the slightest evidence in favour of the presence of a filter-passing virus of the disease’, with reports to the contrary being lambasted as ‘trivial’ and produced by investigators which McIntosh insinuated lacked experience and used the filter-passing virus to avoid ‘producing a description of the morphology’ of their supposed pathogenic microbe.[[251]](#footnote-251) McIntosh’s strong criticism of the filter-passer theory demonstrates the gradual process of conversion to a new paradigm, especially when the new contender for paradigm remains in his mind crude and not persuasively refined.[[252]](#footnote-252) That his report is prefaced by an introduction by the MRC that clarifies that McIntosh’s view is one ‘which, as is well known, was not held by all the writers in the previous Report published by the Council on the subject’, and that the MRC ‘does not hold itself responsible for the scientific conclusions reached by the authors of reports which they may publish’ only further highlights the divisions within the community caused by the revolutionary science period.[[253]](#footnote-253)

F. G. Crookshank warned of a similar disparity of views within his edited collection on the subject of influenza, also published in 1922. ‘The problems involved in any general discussion of influenza’ Crooshank wrote ‘are so many, so various, so complex, so antithetical, and so profound, that…any attempted systematic exposition by a group of workers would almost of necessity lack…unity of presentation…so diverse are the views that may be maintained with apparently equal right in respect of this or that relevant issue’.[[254]](#footnote-254) The collection of essays within the volume are therefore evidence of ‘a certain community of opinion’, but Crookshank is clear to stipulate that ‘no one is to be supposed necessary in either agreement or disagreement with the notions of any other’.[[255]](#footnote-255)

The collection’s chapter on bacteriology represents a third of the total volume (175/525 pages). Written by Robert Donaldson (whose work Crookshank had supported in *The Lancet* in 1919), the chapter provides a lengthy overview of contemporary thought on influenza’s aetiology, with Donaldson astutely surmising the revolutionary scientific character of the field in his statement that ‘bacteriologists…came to belong to one or other of three groups: (i) those who believed Pfeiffer’s bacillus to be the cause of influenza; (ii.) those who did not; and (iii.) those who could not make up their mind’.[[256]](#footnote-256) Donaldson himself was a member of the second group and was scornful of the acceptance of Pfeiffer’s bacillus, stating that ‘no organism…has had such wide acceptance on grounds so inefficient as Pfeifer’s so-called *B.influenzae*’.[[257]](#footnote-257) He took issue with the excuse that ‘failure to find the organism is due to the employment of unsuitable media or of faulty technique’, seeing it as a ‘gross and wholly unwarranted aspersion of the skill of a large number of bacteriologists’.[[258]](#footnote-258) Indeed his concern for the perceived competency of the medical community is the impetus behind his insistence that ‘if…it is not the cause, the sooner we dismiss it from our calculations the better, since for twenty-eight years it has stood in the way of any real advance towards discovery of the actual cause’.[[259]](#footnote-259) Progress cannot be made, according to Donaldson, until the bacillus has been ‘rendered unassailable or absolutely discredited’.[[260]](#footnote-260)

Even with his strong assertions in the ‘fallacious’ and ‘illegitimate’ claim of aetiology held by Pfeiffer’s bacillus and its adherents; Donaldson still appears on the fence in his call for a dismissal of the bacillus in the name of scientific progress.[[261]](#footnote-261) His repeated use of ‘if’ alludes to a continued element of doubt in his dismissal of the bacillus (‘if, however, it is not the cause’) and indeed in the notion of a singular causative agent at all (‘Pfeiffer’s bacillus is either the bacteriological cause of influenza as I have defined it (if such cause there be) or it is not’).[[262]](#footnote-262) Also interesting is the fact that Donaldson was equally dismissive of the filter-passer theory as he was that of Pfeiffer’s bacillus. He considered ‘the whole subject of filter-passers [as] one bristling with pitfalls and difficulties’ and concluded that ‘there is not the slightest shred of evidence that [influenza] is due to a so-called filter-passing virus’.[[263]](#footnote-263) Donaldson, echoing his articles from 1918-1919, was in favour of a pleomorphic explanation of influenza, and briefly mentions his own mycotic organism as a contender for aetiological agent.[[264]](#footnote-264) However where in 1918 he deemed the filter-passer theory credible enough to warrant a comment suggesting that his view is ‘not inconsistent, as I have said, with a filter-passer theory’, by 1922 he has rescinded all support for the concept of filter-passers.[[265]](#footnote-265) This is interesting, for it suggests that in Donaldson’s case the refinement of the filter-passer theory between 1919 and 1922 had reduced, rather than bolstered, his desire to convert to the new paradigm. It also serves to demonstrate the continued power of the bacteriological paradigm, wherein even with the influenzal paradigm in doubt, the prospect of a bacteriological cause allowed Donaldson an alternate aetiological solution which did not require aligning himself with what he considered an unpersuasive filter-passing paradigm contender.

Donaldson’s support for the filter-passer theory was only ever secondary to his own bacteriological and mycotic ideas, however even some of the filter-passer’s strongest 1918-19 proponents indicated a continued doubt in the possibility of a filter-passer, rather than Pfeiffer’s bacillus, cause of flu. R. Dujarric de la Rivière was a French microbiologist who had published a report in 1918 detailing his findings of a filterable virus in influenza samples.[[266]](#footnote-266) His work on filter-passers was referenced in the *BMJ* in that year, as well as in the 1920 Report on the 1918-19 Pandemic, therefore making his voice significant within filter-passer research in the post-pandemic period.[[267]](#footnote-267) However where one would expect his published work to forefront his adherence to the filter-passer theory, in reality his 1929 book ‘*Etiologie et prophylaxie de la grippe: Bacille de Pfeiffer, virus filtrant grippal*’ provided an account of both the Pfeiffer bacillus and the ‘filterable influenza virus’ which had dominated his pandemic era research. The bacillus and the virus receive roughly equal comment in the book, and though the Pfeiffer’s bacillus chapter is broadly critical (mentioning again the presence of the bacillus in ‘healthy people’), in the case of both the bacillus and the filter-passer Dujarric de la Riviere was unable to make specific conclusions based upon the technological limitations of his period.[[268]](#footnote-268) For the bacillus, the suggestion among some (including Pfeiffer) that *B.influenzae* remained dormant in a healthy population before increasing in pathogenicity was ‘an opinion which is very difficult to support or deny’, whilst for the filter-passer ‘the morphology…escape[s] our current optical means’ [all translations own].[[269]](#footnote-269)

The mention of technological limitations even after a decade of research and theory refinement makes Dujarric de la Riviere’s text significant to consider, despite his nationality separating him from the British scientific community on which this dissertation has focussed. Bresalier’s framing of the interwar period as one of significant advancements in the proto-field of virology suggests an increase in technological capabilities which would support the isolation of the influenza virus from animals in the early 1930s. Dujarric de la Riviere’s 1929 comment on the inability of current ocular technology to isolate microscopic viral matter does not support this assertion. I am by no means attempting to argue that virus research was not developed during the early interwar period, indeed Bresalier’s detailed review of the MRC’s study of canine distemper shows that ‘virus research was becoming an established medical scientific field’ by 1931.[[270]](#footnote-270) This move towards the formation of a new discipline – virology – aligns with Kuhn’s framework which gives the generation of new disciplines as one outcome of paradigm shift. [[271]](#footnote-271) However the fact that these developments are not reflected in the wider literature on influenza emphasises that for the medical community writ large influenza knowledge was still firmly within a period of revolutionary science.

This is clearly indicated within the 1927 *Revised Memorandum on Influenza*, published two years before Dujarric de la Riviere’s book. The revised memorandum, unlike McIntosh’s study of influenza published five years earlier, is unwilling to assert that the Pfeiffer Bacillus, is influenza’s causal agent. Instead the memorandum admits that ‘as yet we do not know the nature of the living virus to which influenza is due’ and that ‘no conspicuous advance has been made recently in our knowledge of the bacteriology of influenza’.[[272]](#footnote-272) The memorandum identifies the ongoing debate between the ‘adherents of Pfeiffer’s theory and those who believe that the true causal agent is some other organism – probably a filter-passer’, but is pessimistic of any resolution being forthcoming.[[273]](#footnote-273) It argues that because both sides of the argument lack the ability to offer concrete proof on their theory (‘…reports on the isolation of a filter-passer, while not lacking conviction on the part of the authors, recognise that the work has not yet advanced to the stage which would entitle this theory to general acceptance’ whilst ‘the claims for Pfeiffer’s bacillus are now put forward in a somewhat temperate fashion, with the admission that they are not fully sustained’), those in favour of the bacillus will ‘continue for an indefinite period to point to the absence of a better claim’ and assert their own “victory” in the meantime, while proponents of the filter-passer theory will face an uphill battle to acquire enough evidence to ‘carry conviction’.[[274]](#footnote-274) The filter-passer theory, to use Kuhn’s framework, continues to be unable to convince the community to reject the existing influenzal paradigm. Paradigm shift can therefore not occur, and the 1927 memorandum must conclude that more work (in Kuhn’s model, theory refinement) must be done ‘to solve the influenza problem’.[[275]](#footnote-275)

In the first weeks of July, 193, the *BMJ* and *The Lancet* reported on ‘A Virus Obtained From Influenza Patients’ which was found to be pathogenically transmissible to ferrets.[[276]](#footnote-276) From their experimental results, authors Wilson Smith, C. H. Andrewes and P. P. Laidlaw reported that a filter-passing virus obtained from throat washings from influenza patients was transmittable to ferrets who then developed contagious influenza symptoms akin to human infection. Samples from healthy individuals did not cause the same reaction, and antibodies taken from convalescing influenza patients were ‘capable of neutralising the virus of the ferret disease’.[[277]](#footnote-277) The filter-passer thus met all of Koch’s postulates. It was influenza’s causative organism.

The discovery was positively received, with the editorial response to Smith et-al.’s *Lancet* paper on July 8th celebrating that ‘although the authors show great modesty in drawing conclusions from their work, there seems little doubt that…they have offered almost conclusive evidence that the primary cause of human influenza is a filter-passing virus’.[[278]](#footnote-278) The *BMJ* too praised Wilson et-al.’s ‘brilliant piece of work’, and both journals are hopeful for the ‘next advances’ in influenza research under the new understanding of the disease’s aetiology.[[279]](#footnote-279) Included in both editorial articles, though not in Wilson et-al.’s paper itself, are reflections on the state of the field prior to the new discovery. *The Lancet*, in their praise for the findings, reference the ‘long controversy between the protagonists of Pfeiffer’s bacillus and those of a filtrable virus’, whilst the *BMJ* preface their discussion of the discovery by describing the community into which it was presented as one where ‘the possibility of any further advance seemed rather remote’.[[280]](#footnote-280) As this section has shown, this period of debate is characteristic of Kuhn’s revolutionary science, however so too is the in some ways sudden shift to a new paradigm prompted by Wilson et-al.’s findings.

Just as Kuhn reminds us that conversion to a new paradigm is a gradual process of professional realignment, so too does his admit that in some cases ‘the new paradigm…emerges all at once, sometimes in the middle of the night, in the mind of a man deeply immersed in crisis’.[[281]](#footnote-281)The publication of Wilson et-al.’s findings represent that moment. Existing understandings of influenza, directed by an influenzal paradigm that labelled Pfeiffer’s *B.influenzae* as the cause of the disease, were overturned by the proof of a filter-passing aetiological agent. The world of influenza research after July 1933 was incommensurable with that which came before, with what was known about the disease fundamentally altered by the discovery of the filter-passing virus. It is this shift that allowed a F. M. Burnet and Ellen Clark’s 1946 ‘*Influenza: A Survey of the Last 50 Years in Light of Modern Work on the Virus of Epidemic Influenza*’ to confidently open with the statement that: ‘Influenza in its serious form as exemplified by the pandemic of 1918-19 still remains the biggest unsolved problem of theoretical epidemiology and public health practice. It is, however, no longer a disease of unknown etiology’.[[282]](#footnote-282)

The 1933 Wilson et-al. paper represents the moment of influenzal paradigm shift, however for the bacteriological paradigm things are not as a clear cut. Although Wilson et-al.’s findings of a pathogenic filter-passing virus did challenge bacteriological understandings of disease causation, it did not force a complete conversion to a new way of understanding disease that was incommensurable with the old. Ton van Hoort argues that the bacteriological paradigm continued to dictate how laboratory findings were understood even past the point of influenzal paradigm shift, with ‘research [into viruses] undertaken on the basis of the germ theory of infectious disease’ which crucially saw ‘ the heuristics of ‘Koch’s postulates’…thought valid whether the infectious agent was filterable or not’ (as demonstrated in Wilson et-al.’s report).[[283]](#footnote-283) Bresalier refutes this claim, arguing instead that ‘viruses…were construed as complex research problems’ with those researching them identifying as ‘experimental pathologists rather than bacteriologists’.[[284]](#footnote-284) This dissertation recommends further study of both these claims, for a conclusion on the moment of shift – if indeed there was one – from the bacteriological to a more inclusive pathological paradigm (or alternatively the moment in which a clear virological subdiscipline diverted from the bacteriological main) requires investigation not just into post-1933 influenza research, but also into how other diseases, both viral and bacteriological, were researched and understood in the second half of the twentieth century.

# **Conclusions**

The introduction of the London County Council 1919 ‘Influenza Report of Chief Medical Officer of Health’ described 1918 as ‘a great influenza year’.[[285]](#footnote-285) My dissertation has shown that the pandemic which swept the world in 1918 produced not just an influenza year, but instead over a decade of debate and developments caused by the anomalous culture results of Pfeiffer’s bacillus. My analysis of contemporary medical journals and texts has demonstrated that theses anomalous results produced a crisis period which, rather than prompting the paradigm blindness described by Honigsbaum, instead brought about a period of questioning and debate which is emblematic of Thomas S. Kuhn’s ‘revolutionary science’. These findings serve to recharacterize historical understandings of scientific work during the pandemic period, and in doing so answer Bresalier’s call for a non-viralised analysis of 1918-19 bacteriological investigations.

By identifying an influenzal paradigm which dictated knowledge as a subset of the bacteriological paradigm during the period, I have opened the field both for further study into influenzal knowledge generation in the 19th and 20th century, and for potential investigations into the ways in which other disease-specific paradigms interact with broader paradigms which dictate the type of the disease’s causative organism (e.g. bacterial, viral, fungal). I have demonstrated the utility of applying Kuhn’s framework of scientific development to discussion of medical knowledge creation and paradigm challenges, and so recommend its use in further studies of this nature.

Though I have succeeded in fulfilling this dissertation’s aims, the impact of the present COVID-19 pandemic placed some limitations on my work for which further study is merited. My focus on two British journals provided a detailed picture of the British medical community’s response to the challenges of the influenza pandemic, however unimpeded access to archival material would allow for a consultation of additional British journals, or alternatively facilitate the geographical widening of the communities studied to also include North American, European or Non-Western perspectives. Crosby and Barry’s histories of the pandemic highlight the rich scientific debate within contemporary US medical discussions on the pandemic, whilst contemporary summaries of the state of the influenzal field also pay heed to Japanese bacteriological investigations.[[286]](#footnote-286) Broadening a study of the pandemic’s challenges to the influenzal and bacteriological paradigms in this way is therefore a meritorious task.

My research could also be expanded upon by applying my research methodology to a broader period. A systematic review of all *BMJ* and *Lancet* publications between 1920 and 1933 could build upon my review of medical literature to establish a wider picture of the interwar influenza community’s reactions to the ‘revolutionary science’ period. Similarly, an analysis of post-1933 viral research using my methodological approach could provide further insight into the development of virology as a discipline, and the extent to which the discovery of the influenza virus can be seen as a new anomaly that would serve to eventually challenge the bacteriological paradigm.

In July 1918, the British medical community heard the first reports of a new pandemic for which they awaited further bacteriological evidence. My dissertation has proven that once it became clear that this evidence was not forthcoming, Britain’s medical men embraced the uncertainty of an anomaly, crisis and revolutionary science period which would culminate in influenzal paradigm shift. The pathologists of 1918-19 were not prisoners of their paradigm. It is time for historians of the pandemic’s scientific impact to shake off our own shackles and recognise this fact.

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# **Appendix I: Figure List**

[Figure 1: A graph Illustrating the Incidence of Mentions of Influenza in BMJ and Lancet Articles, June 1918-December 1919. 51](#_Toc81352302)

[Figure 2: A time-series plot graph showing the three main waves of influenza deaths in Britain, from June 1918-May 1919. Source: Dora C. Pearce, Paul K. Pallaghy, James M. McCaw, Jodie McVernon, John D. Mathews, ‘Understanding mortality in the 1918-1919 influenza pandemic in England and Wales’, Influenza and Other Respiratory Viruses, 5.2,(2011) 89–9, (p.90). 52](#_Toc81352303)

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35. Kuhn, *Structure*, p.11. [↑](#footnote-ref-35)
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37. It is beyond the scope of this project to enter into a detailed discussion of Kuhn’s changing philosophical approach to his use of paradigms. For an articulation of Kuhn’s later thought on paradigms, see 'Second Thoughts on Paradigms', in Thomas S. Kuhn, *The Essential Tension: Selected Studies in Scientific Tradition and Change* (USA: The University of Chicago Press, 1977), pp. 293-319. For a well contextualised discussion of the development of Kuhn’s thought post-*Structure,* see James A. Marcum, 'From Paradigm to Disciplinary Matrix and Exemplar', in *Kuhn's The Structure of Scientific Revolutions Revisited*, ed. by Vasso Kindi and Theodore Arabatzis (USA: Routledge, 2012), p. 41-63 , and for further exploration of Kuhn’s conceptualisations of paradigms see James A. Marcum, *Thomas Kuhn's Revolution: An Historical Philosophy of Science* (Great Britain: Continuum, 2005) and 'Chapter 3: Paradigms', in Alexander Bird, *Thomas Kuhn*(USA: Routledge, 2014), p. 65-96. [↑](#footnote-ref-37)
38. Kuhn, *Structure*, p.24. [↑](#footnote-ref-38)
39. Ibid, pp. 25-7. [↑](#footnote-ref-39)
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45. Ibid. [↑](#footnote-ref-45)
46. Ibid. [↑](#footnote-ref-46)
47. Ibid, p.85. [↑](#footnote-ref-47)
48. Ibid, pp.85-89. [↑](#footnote-ref-48)
49. Ibid, p.168. [↑](#footnote-ref-49)
50. It should also be noted that, under Kuhn’s framework, a paradigm cannot ever be rejected without a viable replacement being adopted in its stead. To do so would be to reject science itself. Only the adoption of a new worldview can facilitate the continuation of scientific research (Kuhn, *Structure*, p.79). [↑](#footnote-ref-50)
51. Hacking, 'Introductory Essay', in Kuhn, *Structure*, p. xxviii. [↑](#footnote-ref-51)
52. Marcum, *Kuhn’s Revolution*, p.72. [↑](#footnote-ref-52)
53. Hacking, 'Introductory Essay', in Kuhn, *Structure*, p. xxx. [↑](#footnote-ref-53)
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55. Honigsbaum*, Pandemic Century*, p.xiii. [↑](#footnote-ref-55)
56. Kuhn*, Structure*, p.80; Honigsbaum*, Pandemic Century*, p.xiii [↑](#footnote-ref-56)
57. Honigsbaum, *Pandemic Century* p. xxvii. [↑](#footnote-ref-57)
58. Honigsbaum, *Pandemic Century* p.xiv, Crosby, *America’s Forgotten Pandemic,* p.10. [↑](#footnote-ref-58)
59. Bresalier*,* ‘A Most Protean Disease’, p.484. [↑](#footnote-ref-59)
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64. Ibid, pp.473-4. [↑](#footnote-ref-64)
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66. The inflexibility of Kuhn’s framework is also commented upon by Ernst Mayr within his article on ‘*The Nature of the Darwinian Revolution*’, with Mayr concluding that biological discoveries require a more nuanced model of scientific revolution than is facilitated by Kuhn’s framework. (Ernst Mayr, 'The Nature of the Darwinian Revolution', Science, 176.4038, (1972), 981-989 (p. 989)). [↑](#footnote-ref-66)
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212. Despite Beaumont’s report indicating that the mycotic theory would be ‘simpler’ than the existing paradigm (with influenza’s apparent pleomorphism in culture explainable by mycotic life-phases rather than a complex web of symbiotic relations), enough of a version from the existing bacteriological norm to warrant some ‘shock value’, and of course capable of solving the aetiological anomaly, the lack of evidentiary support in response to Ramsbottom’s comment meant that the mycotic theory was not persuasive enough to gain the support required to present a true challenge to the influenzal paradigm. [↑](#footnote-ref-212)
213. Ibid, pp.157-158. [↑](#footnote-ref-213)
214. J. A. Arkwright, ‘Criticism Of Certain Recent Claims To Have Discovered And Cultivated The Filter-Passing Virus Of Trench Fever And Of Influenza’, *British Medical Journal*, 2.3060 (23rd August 1919), 233-236, (p.234,236). [↑](#footnote-ref-214)
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219. Wilson and Bradford’s responses to Arkwright’s article were printed in the same issue. Though defending their methods, both acknowledged that ‘at the present moment it cannot be claimed that [their] work has proved that filter-passing organisms in the diseases in question have been grown in pure culture’ in ‘any of the diseases for which the claim was made’. Doubts around the filter-passer theory were thus acknowledged even by those that had ardently supported it. (Sir John Rose Bradford and Captain J. A. Wilson, ‘Notes on Dr Arkwright’s Article’, *British Medical Journal*, 2.3060 (23rd August 1919) 236-237 (pp.236-237).) [↑](#footnote-ref-219)
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226. Sheldon F. Dudley ‘A Note on B. Influenzae’, *The Lancet*, 194.5011 (13th September 1919), 476-477, (p.477). [↑](#footnote-ref-226)
227. Julia A. Segre, 'What does it take to satisfy Koch’s postulates two centuries later? Microbial genomics and Propionibacteria acnes', *J Invest Dermatol*, 133.9, (2014), 2141-2142 (p. 2141). [↑](#footnote-ref-227)
228. Dudley, ‘A Note on B. Influenzae’ p.477; Alexander Fleming and Francis J. Cleminger had also published findings discrediting the influenza bacillus in 1919, concluding that it was ‘commonly present in catarrhal conditions of the respiratory tract for some years before the recent epidemic’ and so was unlikely to be the organism responsible for suddenly causing a world-wide pandemic. (Alexander Fleming and Francis J. Cleminger, ‘An Experimental Research Into The Specificity of the Agglutinins Produced by Pfeiffer’s Bacillus’, *The Lancet*, 194.5020 (15th November 1919), 869-871, (p.870).) [↑](#footnote-ref-228)
229. W. Hughes, ‘The Value of Antistreptococcus Serum in Influenza’, *The Lancet*, 194.5018 (1st November 1919), 782-784, (p.782). [↑](#footnote-ref-229)
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231. Van Helvoort, ‘A Bacteriological Paradigm in Influenza Research’, p.5. [↑](#footnote-ref-231)
232. George Newman, ‘Chief Medical Officer’s Introduction’ *in* *Report on Public Health – Medical Subjects No.4: Report on the Pandemic of Influenza 1918-19*, (London: HMSO, 1920), p. ix. [↑](#footnote-ref-232)
233. Ibid; Frederick W. Andrewes, ‘Chapter IV: The Bacteriology of Influenza’ in *Report on Public Health – Medical Subjects No.4: Report on the Pandemic of Influenza 1918-19*, (London: HMSO, 1920), p.116. [↑](#footnote-ref-233)
234. Andrewes, ‘Chapter IV: The Bacteriology of Influenza’, p.125. [↑](#footnote-ref-234)
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243. These included Chief Medical Office Sir George Newman, founding member of the Medical Research Council Sir William Leishman, and 1920 Report contributor Dr French among others. (Wellcome Library, PP/JRH/D/36, Influenza Committee 1922 - papers etc: Influenza Committee Ministry of Health 27th January 1922.) [↑](#footnote-ref-243)
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