Fighting Flu: Military Pathology, Vaccines, and the Conflicted Identity of the 1918–19 Pandemic in Britain

MICHAEL BRESALIER
Centre for the History of Science, Technology, and Medicine, University of Manchester, Second Floor, Simon Building, Brunswick Street, Manchester M13 9PL, UK.
Email: michael.bresalier@manchester.ac.uk

ABSTRACT. This article explores the decisive role of British military medicine in shaping official approaches to the 1918 influenza pandemic. It contends that British approaches were defined through a system of military pathology, which had been established by the War Office as part of the mobilization of medicine for the First World War. Relying on the bacteriological laboratory for the identification and control of pathogenic agents, military pathology delivered therapeutic and preventive measures against a range of battlefield diseases, and military and civilian authorities trusted that it could do the same with influenza. This article traces how it shaped efforts to establish the etiology of the pandemic and to produce a general influenza vaccine. It highlights the challenges involved in both strategies. Understanding the central role of military pathology helps make sense of the nature, direction, scale, and limitations of medical mobilization against the pandemic in Britain and the authority accorded to specific medical bodies for elaborating and coordinating strategies. Crucially, it demands that we rethink the relationship between the war and pandemic as one about the social organization of medical knowledge and institutions. **Keywords:** 1918 influenza pandemic, British military medicine, influenza vaccines, pathology and war.
On 13 November, two days after armistice, Sir Arthur Newsholme, Chief Medical Officer to Britain’s public health body, the Local Government Board (LGB), organized an emergency meeting of medical authorities at the Royal Society of Medicine. Like every major city in Europe, and most inhabited places around the world, London was in the grip of a devastating influenza epidemic. Over one million, nearly a quarter of the population, had been sick in the preceding month. Thousands of workers, from bankers to dockers, were laid up in bed or in hospital. Public services suffered massive staff shortages and the transport system had ground to a near standstill. Hospitals, dispensaries, infirmaries, and doctor’s offices were overrun with the ill. Physicians and nurses, their numbers already depleted by the war, struggled to cope. Many medical men and women were themselves stricken and many had already died in their own wards. All around London, military camps constructed to house troops on their way to front were filled with returning soldiers who had survived the war but now battled a disease so virulent and with such perplexing symptoms that many medical observers first doubted that it was influenza. When Newsholme opened the “Discussion on Influenza,” the pandemic’s identity was at the forefront of his agenda: “Is it one disease, or a group of diseases? And is the disease now prevailing the disease which prevailed in the spring, and still more in July, of this year?”

In hindsight, historical epidemiologists have divided the 1918–19 pandemic into three consecutive waves. A relatively mild epidemic beginning in summer 1918 was quickly followed by a lethal epidemic in autumn after which developed a somewhat less virulent epidemic in spring 1919. While each wave presented its own clinical and epidemiological characteristics, retrospective accounts have nonetheless linked them together into a single cataclysmic pandemic. Newsholme and his contemporaries had no such luxury.

1. For contemporary accounts of the epidemic in London, see reports in The Times between October and December 1918. See also, Andrea Tanner, “The Spanish Lady Comes to London: The Influenza Pandemic 1918–1919,” London J., 2002, 27, 51–76.
2. Arthur Newsholme, “Discussion on Influenza,” Proc. Roy. Soc. Med., 1918–19, 12, 1.
3. See, for example, Niall P. A. S. Johnson, Britain and the 1918–19 Influenza Pandemic: A Dark Epilogue (London: Routledge, 2006); David K. Patterson and Gerald F. Pyle, “The Geography and Mortality of the 1918 Influenza Pandemic,” Bull. Hist. Med., 1991, 65, 4–21; Erwin O. Jordan, Epidemic Influenza: A Survey (Chicago: American Medical Association, 1927).
At the time, the relationship between the summer and autumn epidemics baffled medical experts. The summer epidemic shared characteristics with previous epidemics, particularly that of 1889–90, with doctors describing extreme body aches, prostration, fever, sore throat, dry cough, nausea, and general lassitude in most patients they saw. Yet important aspects did not fit the established picture. Experts and officials were unable to agree that it was “influenza” until August 1918. By then, early signs of a second epidemic had already started to be reported in military garrisons in France and Flanders; but little about it correlated with what had been seen in the spring. Indeed, it was so stunningly virulent that many medical experts thought they were encountering an entirely new disease. Not only did the epidemic display a new kind of virulence, who it killed also changed. A generation of textbooks taught that influenza-related deaths occurred among the very young, aged, and infirm. Epidemiologists who had plotted the age-distribution of influenza’s mortality since the 1890s typically traced a “U” mortality curve. Yet in autumn 1918, the U turned into a “W”: the disease now killed a disproportionate number of men and women in the prime of life.4

The disjuncture between the summer and autumn epidemics made determining influenza’s identity crucial. For Newsholme, to approach prevention rationally, medical professionals had to know what they were fighting. “The first difficulty,” he insisted, “is to define influenza.” Before influenza could be “brought within the scope of prevention,” he argued, doctors needed “further knowledge—epidemiological, pathological, and bacteriological.”5 But from where was such knowledge to come, and from which authorities were official strategies to be elaborated? The source for answers was clear enough to Newsholme. They had to come from the system of military medicine that had been organized for the war effort and, which by 1918, determined the direction and imperatives of Britain’s corresponding war against disease.

This paper explores the decisive role of British military medicine in shaping official strategies against the pandemic. Through the spring

4. Major Greenwood, “The Epidemiology of Influenza,” Br. Med. J., 1918, 2, 563–66; Christopher Langford, “The Age Pattern of Mortality in the 1918–19 Influenza Pandemic: An Attempted Explanation Based on Data for England and Wales,” Med. Hist., 2002, 46, 1–20.

5. Arthur Newsholme, “Discussion,” 1; Newsholme, “Epidemic Catarrhs and Influenza,” Lancet, 1918, 192, 599.
and autumn 1918, the War Office, the Army Medical Services (AMS), and the Medical Research Committee (MRC), which had coordinated mobilization of British medical science, jointly produced official knowledge and epidemic strategies. Prior to the pandemic, these authorities had worked together to create a system of military pathology, which linked pathological laboratories to base and field hospitals in France and Flanders. This system was organized to collect, isolate, and identify pathogens from the battlefield, and to facilitate production of vaccines and antisera. Military pathology delivered therapeutic and preventive measures against a range of battlefield diseases, and its planners trusted that it could do the same with influenza.

British approaches to the pandemic were governed by a military logic in which the pathological laboratory provided key solutions to understanding and managing the disease. This logic dictated two interrelated strategies: the identification of the primary causative agent and the rapid production of preventive vaccines against it. During the war, these strategies were central to the military management of infectious disease. While not adhered to blindly, during the pandemic they acted as important reference points from which medical practices were developed and organized. This argument draws special attention to the war as the over-arching context in which British responses to the pandemic were defined. One aim is to counter the tendency in historical work to trace “responses to” the pandemic without examining the contemporary medical knowledge on which they were based. In doing so, I want to provide a new way of thinking about the relationship between the war and the pandemic. Much interest has focused on how war conditions created novel ecological conditions for the rapid spread and increased virulence of influenza. I take a different approach. I show how the wartime organization of British medicine shaped

6. Cay R. Prüll, “Pathology and Surgery in London and Berlin 1800–1930: Pathological Theory and Clinical Practice,” in Pathology in the 19th and 20th Centuries: The Relationship between Theory and Practice, ed. Cay R. Prüll (Sheffield: European Association for the History of Medicine and Health Publications, 1998), 71–100.
7. William B. Leishman, “Organization of the Pathological Service,” in History of the Great War Based on Official Documents. Medical Services. Pathology, ed. William G. MacPherson, William B. Leishman, and S. L. Cummins (London: Her Majesty’s Stationery Office [H.M.S.O.], 1923), 1–31.
8. For example, Robert J. Brown, “The Great War and the Great Flu Pandemic of 1918,” Wellcome Hist., 2003, 24, 5–7.
definitions and knowledge of influenza. Understanding this helps
make sense of the nature, direction, and scale of medical mobiliza-
tion against the pandemic and the authority accorded to specific
medical bodies for elaborating and coordinating strategies.

The relationship between the militarization of medicine and the
pandemic has started to receive some historical attention. Carol
Byerly has examined how the organization of military medicine
within the U.S. Army affected its approaches to the pandemic.⁹
John Barry has elucidated the efforts (and failures) of laboratory
pathologists enlisted in the American war machine to identify and
control the influenza germ.¹⁰ But these studies take it as self-evident
that responses were made for a disease caused by a virus that no
medical expert at the time knew existed or how to study or control.
By privileging a modern disease concept, they tend to exclude
from analysis concepts developed and used by historical actors.¹¹ As
a result, they explain little of how certain kinds of medical knowl-
edge were produced and used, and how they were challenged and
changed in the process.¹² More promising to this end is the work
by Anne Rasmussen, who has convincingly argued that in war-torn
France “the management of influenza took place within the mili-
tary health organization” and has examined how what counted as
useful knowledge was dictated by military interests and impera-
tives.¹³ Rasmussen stresses the central role of military medicine and
bacteriological knowledge in shaping approaches to the pandemic.

Contextualizing the military organization of medical knowledge
and strategies helps to focus attention on the ways in which certain

⁹. Carol R. Byerly, Fever of War: The Influenza Epidemic in the U.S. Army during World
War I (New York: New York University Press, 2005).
¹⁰. John M. Barry, The Great Influenza: The Epic Story of the Deadliest Plague in History
(London: Penguin Books, 2004).
¹¹. Adrian Wilson, “On the History of Disease-Concepts: The Case of Pleurisy,” Hist.
Soc., 2000, 38, 271–319; Andrew Cunningham, “Identifying Disease in the Past: Cutting
the Gordian Knot,” Asclepio, 2002, 54, 13–34.
¹². Byerly, Fever of War; Barry, Great Influenza. A recent exception is Mark
Honigsbaum, Living with Enza: The Forgotten Story of Britain and the Great Flu Pandemic of
1918 (London: Macmillan, 2009). Honigsbaum highlights debates among British bacteriol-
ogists over the nature of the etiological agent. He does not, however, set these debates in
the wider context of British military pathology.
¹³. Anne Rasmussen, “Prevent or Heal, Laissez-Faire or Coerce? The Public Health
Politics of Influenza in France, 1918–1919,” in Influenza and Public Health: Learning from
Past Pandemics, ed. Tamara Giles-Vernick and Susan Craddock (London: Earthscan,
2010), 73.
approaches were legitimized and shaped the meanings of the pandemic. In what follows, I trace how in Britain pathology was organized for war and the concomitant fight against influenza. While historians are familiar with the bacteriological problems thrown up by the pandemic, I highlight the ways in which medical officials mobilized military pathology to generate ways of understanding and controlling them that fit the demands of a nation in total war.  

WAR PATHOLOGY

That the pandemic and official responses to it in Britain should be defined through military medicine is understandable when one considers the extent to which medicine had been organized on military lines. By 1918 over 50 percent of the profession had been recruited or conscripted into the Royal Army Medical Corps (RAMC). The problems they worked on were primarily military and required ways of seeing and managing diseases acquired on battlefields and in military hospitals. Military and civilian medicine converged around the maintenance and management of military manpower, including civilians who were the reserve labor pool for the military and the war industries. The joint medicalization of the military and militarization of medicine bound the organization, production, and application of medical knowledge to the war.

The wartime medical system combined medical institutions for the treatment, care, and management of personnel at the front with

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14. Alfred W. Crosby, America’s Forgotten Pandemic: The Influenza of 1918 (Cambridge: Cambridge University Press, 1989); Martha L. Hildreth, “The Influenza Epidemic of 1918–1919 in France: Contemporary Concepts of Aetiology, Therapy, and Prevention,” *Soc. Hist. Med.*, 1991, 4, 277–94; Eugenia Tognotti, “Scientific Triumphalism and Learning from Facts: Bacteriology and the ‘Spanish Flu’ Challenge of 1918,” *Soc. Hist. Med.*, 2003, 16, 97–110.
15. Jay M. Winter, The Great War and the British People (London: Macmillan, 1985), 186.
16. Ian R. Whitehead, “The British Medical Officer on the Western Front: The Training of Doctors for War,” in Medicine and Modern Warfare, ed. Roger Cooter, Mark Harrison, and Steve Sturdy (Amsterdam: Rodopi, 1999), 161–84.
17. Roger Cooter, “War and Modern Medicine,” in Companion Encyclopedia of the History of Medicine, ed. William F Bynum and Roy Porter (London: Routledge, 1993), 1536–73; Roger Cooter and Steve Sturdy, “Introduction,” in War, Medicine and Modernity, ed. Roger Cooter, Mark Harrison, and Steve Sturdy (London: Sutton, 1998), 1–21; Mark Harrison, “Medicine and the Management of Modern Warfare,” *Hist. Sci.*, 1996, 34, 379–410.
medical institutions at home.\textsuperscript{18} Highly rationalized, it depended on triage, teamwork, specialization, and large-scale communication, linking Casualty Clearing Stations (CCS) at the front to field and base hospitals attached to each army division.\textsuperscript{19} Field hospitals were supported by “territorial” hospitals in mainland England, which were connected to major London and provincial teaching hospitals and run by consultants who were given temporary ranks in the RAMC and paid part-time salaries.\textsuperscript{20}

At the core of this system was a network of pathological laboratories that supported every level of medical practice and institution. Pathology laboratories were an organizational innovation in military medicine spurred by the growth and successes of bacteriology in civilian medicine and public health. Training in pathology and bacteriology was established as part of the curriculum of the RAMC in 1903.\textsuperscript{21} But the pathological laboratory was made a fundamental unit of military medicine only during the war.\textsuperscript{22}

The organization of pathological laboratories involved close collaboration between the War Office (WO) and the MRC. Created in 1913, the MRC established its authority in the coordination of medical science for the war effort.\textsuperscript{23} In his first annual report, MRC Secretary, Walter Morley Fletcher, justified its role in terms of the value of medical research for the “efficiency of the fighting forces” and for its future applications in peacetime. The “conditions of war,” he argued, offered “special opportunities . . . for disciplined study and for repeated observation.”\textsuperscript{24} Every aspect of the war demanded “the application of the scientific method”—from the

\begin{footnotesize}
\begin{enumerate}
\item Mark Harrison, \textit{The Medical War: British Military Medicine in the First World War} (Oxford: Oxford University Press, 2010).
\item Ian R. Whitehead, \textit{Doctors in the Great War} (London: Leo Cooper, 1999), 210.
\item Roger Cooter, \textit{Surgery and Society in Peace and War: Orthopaedics and the Organisation of Modern Medicine, 1880–1948} (London: Macmillan, 1993), 111.
\item Cay R. Prüll, “Pathology at War 1914–1918: Germany and Britain in Comparison,” in \textit{Medicine and Modern Warfare}, ed. Cooter, Harrison, and Sturdy, 131–62.
\item Leishman, “Organization,” 1–31.
\item Joan Austoker, “Walter Morley Fletcher and the Origins of a Basic Biomedical Research Policy,” in \textit{Historical Perspectives on the Role of the MRC: Essays in the History of the MRC of the United Kingdom and Its Predecessor, the Medical Research Committee, 1913–1953}, ed. Joan Austoker and Linda Bryder (Oxford: Oxford University Press, 1989), 22–33; Robert E. Kohler, “Walter Fletcher, Frederick G. Hopkins, and the Dunn Institute of Biochemistry: A Case Study in the Patronage of Science,” \textit{Isis}, 1978, 69, 331–55.
\item First Report of the Medical Research Committee, 1914–15 (London: H.M.S.O., 1916), 1–2.
\end{enumerate}
\end{footnotesize}
management of manpower and hospitals to medical diagnoses and
treatments. Coordinated laboratory work in pathology was crucial
to this goal.\textsuperscript{25}

Sir William Boog Leishman, a career military pathologist, spe-
cialist in tropical medicine, and founding member of the MRC,
spearheaded the integration of pathology into the medical service.
A former student of the renowned pathologist and bacteriologist,
Sir Almroth Wright, Leishman had been instrumental in the devel-
opment of the military’s antityphoid vaccine at the Royal Army
Medical College at Millbank and the large-scale inoculation of
troops.\textsuperscript{26} In October 1914, he became Advisor in Pathology to the
Director-General of the AMS (DGAMS) and was detailed to estab-
lish pathological laboratories in France and Flanders.

Leishman and a small cadre of advisors defined the role of path-
ology laboratories along lines that fostered opportunities for “better
cooperation between clinicians and pathologists.”\textsuperscript{27} He promoted
laboratory knowledge and practices as necessary for “the mainte-
nance of the health of the troops and for the effective treatment of
the sick and the wounded.”\textsuperscript{28} British military pathological laborato-
ries were initially organized to provide doctors with bacteriological
services for the routine diagnosis and treatment of infectious dis-
eases.\textsuperscript{29} The high incidence of tetanus, gas gangrene, venereal dis-
eases, and blood poisoning in the early months of the war, along
with the emergence of new infectious diseases, such as trench foot,
sand-fly fever, and cerebrospinal fever, served as an important
rationale for expanding the role of pathological laboratories.

Beginning in 1914, Leishman oversaw the deployment of nearly
one hundred pathologists at eighty-five hospital laboratories in
France and Flanders, the development of a fleet of twenty-five
mobile laboratories to provide bacteriological services at the front,
and the creation of a central research laboratory at Boulogne.\textsuperscript{30} The

\textsuperscript{25} Austoker, “Walter Morley Fletcher,” 23–24.
\textsuperscript{26} William Leishman was one of “Wright’s Men”; see Michael Worboys, “Almroth
Wright at Netley: Modern Medicine and the Military in Britain, 1892–1902,” in Medicine
and Modern Warfare, ed. Cooter, Harrison, and Sturdy, 77–97.
\textsuperscript{27} Prüll, “Pathology at Wär,” 144.
\textsuperscript{28} Leishman, “Organization,” 5–6.
\textsuperscript{29} Cay Prüll argues that this was a defining feature of British clinical pathology. See,
“Pathology at Wär.”
\textsuperscript{30} Leishman, “Organization,” 6.
routine work of the laboratories involved testing and monitoring patients, performing autopsies, analyzing specimens, preparing and administering vaccines and serum therapies, and wiring important or unusual findings to appropriate agencies back home. The pathological department at the Royal Army Medical College was the system’s institutional hub. It trained pathologists and analyzed pathological material, and aided in the confirmation of difficult-to-diagnose diseases. College staff maintained close relations with pathologists at various London research institutions and medical schools, with their well-equipped laboratories and trained staff.\textsuperscript{31}

The College’s most important role was the production of preventive and therapeutic techniques. The large-scale manufacture and inoculation of antityphoid vaccine at its Vaccine Department was a model for the successful application of laboratory methods to pathological problems, and shaped military medicine’s parallel war on disease.\textsuperscript{32} Antityphoid vaccination dramatically reduced the disease’s incidence and spurred the production and widespread use of vaccines for cholera, plague, and dysentery. Although serum therapies against tetanus and diphtheria had been proven in peacetime, their efficacy during the war, along with new serum therapies against gas gangrene, trench fever, and other diseases, further underscored the military relevance of laboratory medicine. For every disease that cropped up during the war, the AMS turned to pathology for solutions.

The MRC played a key role in the mobilization of pathology. Although it maintained relations with the Royal Navy and the Royal Air Force, it mostly assisted the AMS, and mostly in pathology.\textsuperscript{33} Crucially, it linked together civilian and military medical expertise. It encouraged pathologists working in hospitals, universities, and research institutions to take on war-related responsibilities. Most needed little convincing, and many were commissioned by the RAMC. The MRC’s leading pathologist, Almroth Wright, headed the AMS’s central pathology laboratory at Boulogne, where

\textsuperscript{31} Ibid., 23–24.
\textsuperscript{32} Paul J. Weindling, “Between Bacteriology and Virology: The Development of Typhus Vaccines between the First and Second World Wars,” \textit{Hist. Phil. Life Sci.}, 1995, 17, 81–90.
\textsuperscript{33} Leishman, “Organization,” 20.
he worked with young assistants, Alexander Fleming and Leonard Colebrook, on wound infections and antiseptics. The RAMC also commissioned (Captain) S. R. Douglas, who oversaw the production of antityphoid vaccine at Wright’s Inoculation Department at St. Mary’s Hospital. Civilian pathologists at St. Mary’s played an important role in military pathology, particularly in efforts to type bacteria and manufacture therapeutic agents.

More generally, through a system of grants, the MRC ensured that military laboratories were supplied with trained staff, materials, and equipment. Leading pathologists, including Paul Fildes, were seconded on temporary commissions. Pathological research at universities and hospitals was coordinated with research in military institutions. It organized special research committees to combat specific illnesses. These committees fostered the organizational notion of “team work” between military and civilian researchers and between clinicians and pathologists. The circulation of medical information was vital to making the system work. The MRC’s statistician, John Brownlee, joined the War Office to compile statistics on the sick and wounded in the forces, developing a novel system of card indices for rapidly transferring information from hospital records to a central database. It published leaflets and pamphlets on special areas of research, and the Medical Supplement, a compendium and analysis of foreign medical publications.

34. Joan Austoker and Linda Bryder, “The National Institute for Medical Research and Related Activities of the MRC,” in Historical Perspectives on the Role of the MRC, ed. Austoker and Bryder, 35–57, 39.
35. Ibid., 39. See, Wai Chen, “The Laboratory as Business: Sir Almroth Wright’s Vaccine Programme and the Construction of Penicillin,” in The Laboratory Revolution in Medicine, ed. Andrew Cunningham and Perry Williams (Cambridge: Cambridge University Press, 1992), 245–92.
36. For the MRC’s organizational activities, see Annual Report of the Medical Research Committee, 1916–17 (London: H.M.S.O., 1918); Annual Report of the Medical Research Committee, 1917–18 (London: H.M.S.O., 1920).
37. Steve Sturdy, “War as Experiment: Physiology, Innovation and Administration in Britain, 1914–1918: The Case of Chemical Warfare,” in War, Medicine and Modernity, ed. Cooter, Harrison, and Sturdy, 65–84.
38. For the concept of “team work,” see Roger Cooter, “Keywords in the History of Medicine: ‘Teamwork,’” Lancet, 2004, 363, 1245; Steve Sturdy and Roger Cooter, “Science, Scientific Management, and the Transformation of Medicine in Britain c. 1870–1950,” Hist. Sci., 1998, 36, 421–66; Arthur L. Thomson, Half a Century of Medical Research. Origins and Policy of the Medical Research Council (UK), 2 vols. (London: H.M.S.O., 1973), I: 95–99.
39. Medical Research Committee, Interim Report on the Work in Connection with the War at Present Undertaken by the Medical Research Committee (London: H.M.S.O., 1915), 3.
The MRC also drove efforts to standardize methods, equipment, and materials. Most notable was the large-scale preparation of uniform culture media for pathology units, to ensure accuracy in routine diagnostic work. When this was institutionalized, the MRC turned its attention to supporting research on war-related problems. Small teams of physicians and pathologists did most wartime pathological research. Rather than draw a line between specialist and routine pathology, Fletcher recognized that “routine work in proper hands may be expected to suggest and give an opportunity for research . . . [and] might provide . . . valuable gains in knowledge and in methods of treatment.”

Military pathology made it possible to mobilize laboratory research on an unprecedented scale. Moreover, it appeared to work. Fewer soldiers died of infections than in any previous war. Antityphoid vaccination symbolized this success. Leishman had organized efforts to redress problems in the preparation and testing of an antityphoid vaccine developed by Wright before the war, and by 1916 technical improvements and the imperatives of manpower economy transformed it into a model for military approaches to infectious disease prevention. So when an epidemic that appeared to be influenza broke out among troops in spring 1918, the War Office, AMS, and MRC trusted that if the suspected influenza germ could be identified, a preventive vaccine could be developed to protect soldiers and military interests.

Influenza was hardly new to the British military in 1918. The disease had broken out for three consecutive years between 1915 and 1917. On AMS estimates, over thirty-six thousand cases were admitted to its hospitals in France in 1916 and nearly thirty

40. Ibid., 3–6; *First Report of the Medical Research Committee, 1914–15* (London: H.M.S.O., 1916), 31–48.
41. Austoker and Bryder, “The National Institute for Medical Research,” 53.
42. For the relationship between the war and the professionalization of pathology, see William D. Foster, *Pathology as Profession in Great Britain and the Early History of the Royal College of Pathologists* (London: E&S Livingstone, 1965), 19–20.
43. *Interim Report on the Work. . . by the Medical Research Committee*, 32.
44. Harrison, *Medical War*, 292–95.
45. Anne Hardy, “Straight Back to Barbarism: Antityphoid Inoculation and the Great War, 1914,” *Bull. Hist. Med.*, 2000, 74, 265–90.
46. Leishman, “Organization.”
thousand in 1917. Medical officials considered the numbers “normal,” but a drag on manpower.

Although treatment was, as it had been in peacetime, largely symptomatic, with antipyretics, hydration, and, wartime conditions permitting, bed-rest, this depended on a reasonably accurate diagnosis. Diagnosing influenza during nonepidemic periods remained notoriously difficult, since it lacked a pathognomonic sign and its symptoms were easily confused with other diseases. By the outbreak of the war, however, some progress had been made as diagnosis increasingly was confirmed by the bacteriological identification of a specific germ, *Bacillus influenzae*, first identified in 1892 by the Berlin bacteriologists, Richard Pfeiffer and Shibashuro Kitasato. Pfeiffer had developed special methods for its cultivation, which, he found, required a substrate of blood—in particular, hemaglobulin—as a growth medium. Through the 1890s, he promoted the bacillus as the primary cause of influenza and his blood-agar culture technique as necessary for establishing its identity. Identification of the germ became widely accepted as the best way to distinguish “true influenza” from other influenza-like conditions. In Britain, Pfeiffer’s claims were first substantiated in 1893 by E. E. Klein, a founder of British bacteriology, in a series of investigations for the Medical Department of the LGB. Pfeiffer’s techniques were slowly incorporated into bacteriological practice and “Pfeiffer’s bacillus” gained standing as the “germ of influenza.” Both became part of the general organization of pathology for war.

47. Herbert French, “Influenza,” in *History of the Great War. Diseases of the War*, 2 vols. ed. William G. Macpherson, Wilmot P. Herringham, Thomas R. Elliot, and Andrew Balfour (London: H.M.S.O., 1920), I: 174–211, 174.
48. Ibid., 174.
49. Richard Pfeiffer and M. Beck, “Weitere Mittheilungen über den Influenza-Erreger,” *Dtsh. Med. Wochenschr.*, 1892, 18, 465; Richard Pfeiffer, “Preliminary Communication on the Exciting Causes of Influenza,” *Br. Med. J.*, 1892, 1, 128; Shibashuro Kitasato, “On the Influenza Bacillus and the Mode of Cultivating It,” *Br. Med. J.*, 1892, 1, 128.
50. Richard Pfeiffer, “Die Aetiologie der Influenza,” *Zeitschrift. Hyg.*, 1893, 12, 357–86.
51. E. Klein, “Some Remarks on the Influenza Bacillus,” *Br. Med. J.*, 23 1892, I, 171; E. Klein, “Report on Influenza in Its Clinical and Pathological Aspects,” Local Government Board, *Further Report and Papers on Epidemic Influenza*, 1889–92 (London: H.M.S.O., 1893), 85–153.
52. For example, Alfred C. Coles, *Clinical Diagnostic Bacteriology* (London: J&A Churchill, 1904), 144–49.
Most military pathologists were familiar with preparing media for the bacillus and with how to identify it in films and cultures (Figure 1). From as early as 1915, its isolation was used to distinguish the various atypical respiratory conditions encountered on the battlefield and local outbreaks of influenza in France. The bacillus first came to general military attention in late December 1916 when it was isolated from an epidemic of “purulent bronchitis” at Etaples. A team of investigators reported that during February and March 1917 in more than 45 percent of all pulmonary autopsies performed in the hospital, purulent bronchitis was the “primary condition.” Most soldiers died of “lung block,” resulting from the accumulation of fluid and pus in the lungs. The bronchi and lungs of soldiers filled with pus, causing emphysema and cyanosis and some observers estimated a mortality rate of 50 percent.

Fig. 1. Standard culture preparations of Pfeiffer’s bacillus. Left: \textit{B. influenzae} in blood agar, indicated by small pocks. Right: photomicrograph of \textit{B. influenzae} in pure culture. \textit{Source:} Richard Pfeiffer, “Influenza und die Gruppe der hämoglobinphilen Bakterien,” in \textit{Lehrbuch der Mikrobiologie}, ed. E. Friedberger and R. Pfeiffer (Jena: Verlag Von Gustav Fischer, 1919), 743. Photograph by Wellcome Library, London; used with permission.

53. Adolphe Abrahams, Norman F. Hollows, John W. H. Eyre, and Herbert French, “Purulent Bronchitis: Its Influenza and Pneumococcal Bacteriology,” \textit{Lancet}, 1917, 191, 377–80; Adolphe Abrahams, “Epidemic at Aldershot: Discussion,” \textit{Trans. Roy. Soc. Med.}, 1917, 12, 97.
54. J. A. B. Hammond, William Rolland, and T. H. G. Shore, “Purulent Bronchitis: A Study of Cases Occurring among the British troops at a Base in France,” \textit{Lancet}, 1917, 190, 41–47.
55. Ibid., 43.
56. John Adami, “Influenza,” in \textit{History of the Great War}, ed. MacPherson, Leishman, and Cummins, 417.
struck investigators was that in smears and cultures of sputa and lung samples from twenty cases they tested, *B. influenzae* appeared to be the primary agent, even though the disease bore little clinical or histological resemblance to influenza. Observations by a team of medical experts at Connaught Hospital at Aldershot Command in September 1917 supported this observation.57 The Aldershot team identified *B. influenzae* as the primary agent in the eight cases they tested and other well-known respiratory germs—particularly pneumococci, *Micrococcus catarrhalis*, and streptococci—as secondary infections. Like their counterparts at Etaples, the Aldershot group concluded that the isolation of *B. influenzae* from the majority of cases of purulent bronchitis indicated a “serious form of influenzal infection.”58 Pathologists and physicians working at the No. 3 Canadian General Hospital at Boulogne, who carried out a “full clinical, pathological and bacteriological study” of purulent bronchitis, supported this conclusion. From all but one of the nine cases, they were able to grow *B. influenzae* in pure culture, which they interpreted as a key indicator that the bacillus caused the disease.59

Pathologists used the identification of *B. influenzae* to frame purulent bronchitis as a type of “influenzal infection.” But the outbreaks were so local that they were treated as one of the many anomalous respiratory infections encountered in the war. It was only when an almost identical condition appeared in large numbers of soldiers in August 1918, and in civilians in October, that the same pathologists started to connect purulent bronchitis to the autumn influenza epidemic. When the official military history of the 1918–19 pandemic was written in the early 1920s, the severe outbreaks of purulent bronchitis that began in December 1916, and the milder outbreaks of influenza in the intervening years, were characterized as the “first phase.”60 The isolation of the *B. influenzae* was essential to drawing this link. “It was evident, in light of these careful studies, that the frequent presence of the *B. influenzae*, noted by the earliest of observers was of no little significance, and further,
that influenza existed among the troops prior to the onset of the pandemic, sporadically...and not of sufficient frequency to cause alarm, but there, nevertheless.”

Studies on purulent bronchitis underscored the extent to which pathological perceptions of influenza were directed by its presumed etiological link with *B. influenzae*. Bacteriological diagnosis rested on the assumption that “true influenza” was present in only those cases from which *B. influenzae* alone was consistently isolated. Its presence, even in obscure or borderline conditions, was enough to establish a disease’s influenza identity, while its absence cast doubt on it. Up until 1918, military pathology thus worked on the assumption that influenza was a bacterial disease caused by Pfeiffer’s bacillus. Trust vested in the bacillus accordingly shaped official strategies when a widespread epidemic broke out among troops in spring 1918. But important anomalies in the bacteriology also challenged this consensus.

Signs of what was later identified as the spring wave in Britain were first encountered in the British Expeditionary Force in France and Flanders in March 1918. Physicians and pathologists attached to the First and Second Armies reported local outbreaks of a mild but rampant fever in troops in Rouen and Wimereux in the ill-famed Ypres salient, where “disease of all sorts seemed to flourish.” The fever raised concern because the British and American armies were bracing for a major German assault on the Western Front. By early May, it had affected thousands of soldiers in the Second Army and had started to appear in other parts of the military. Colonel A. B. Soltau of the AMS claimed that it had “important military bearings” on the supply of manpower and the fighting fitness of the army: “Whole units were sometimes put out of action. One army brigade of artillery...had at one time two-thirds of its strength laid up, and was unable to go into action, though badly needed, for three weeks.” By June, it had turned into an epidemic and

61. Ibid., 420.
62. A. B. Soltau, “Discussion on Influenza,” *Proc. Roy. Soc. Med.*, 1918–19, 12, 27.
63. Ibid., 28. See also Michael Oldstone, *Viruses, Plagues and History* (Oxford: Oxford University Press, 1998), 172–73.
64. Influenza Committee of the Advisory Board to the D.G.A.M.S., France, “The Influenza Epidemic in British Armies in France, 1918,” *Br. Med. J.*, 1918, 2, 505–9, 505.
65. Soltau, “Discussion,” 28.
admissions to military hospitals sky-rocketed.\textsuperscript{66} The Second Army’s CCS admitted over 1,900 cases in the first week of June and nearly 3,900 in the second.\textsuperscript{67} The Grand Fleet reported that an estimated 10 percent of men had been struck.\textsuperscript{68} The Army reported a total of 226,615 cases with a further 93,670 soldiers incapacitated.\textsuperscript{69} By the end of the month, the epidemic reached Britain’s civilian population, most likely transmitted by military personnel.\textsuperscript{70}

The identity of the epidemic perplexed army doctors and pathologists. While it shared characteristics with influenza, important aspects did not fit the established picture. It occurred in summer instead of autumn. Rather than the usual susceptible groups—the very young, aged, and infirm—it affected young, healthy soldiers.\textsuperscript{71} Few of the typical symptoms associated with influenza’s complications or sequelae were evident in cases admitted to hospitals; morbid anatomists in the First and Second armies noted that in the relatively small number of fatal cases, death appeared to be caused by a strange pneumonic and hemorrhagic condition that produced a suffocating cyanosis.\textsuperscript{72} But most of the confusion stemmed from uncertainties about its etiology.

Suspecting the epidemic was influenza, through spring and summer 1918, AMS pathologists tried to isolate \textit{B. influenzae} from sick soldiers’ sputum, nasal passages and blood, and from the lesions of the few cases that ended up on the autopsy table. Yet so seldom were their efforts successful that many concluded that it was at best associated with, but not essential to, the epidemic. Its absence led some to question whether the epidemic was influenza.

Names reflected these uncertainties.\textsuperscript{73} The spring fever was first classified as “Pyrexia of Unknown Origin” (P.U.O.), a category that had been introduced in 1915 for infectious diseases, like “trench fever,” for which neither specific causal agents nor pathognomonic

\textsuperscript{66}. French, “Influenza,” 174.
\textsuperscript{67}. Influenza Committee of D.G.A.M.S., France, “Influenza Epidemic,” 505.
\textsuperscript{68}. D. Thomson and R. Thomson, “Influenza (Part I),” \textit{Annals of the Pickett-Thomson Research Laboratory} (London: Bailliere, Tindall and Cox, 1933), 8.
\textsuperscript{69}. James McIntosh, \textit{Studies in the Aetiology of Epidemic Influenza}, Medical Research Council Special Report Series, no. 63 (London: H.M.S.O., 1922), 6–7.
\textsuperscript{70}. Tanner, “Spanish Lady,” 54.
\textsuperscript{71}. Ibid.
\textsuperscript{72}. Herbert French, “Influenza,” \textit{Guy’s Hospital Gazette}, 1919, 33, 118–27.
\textsuperscript{73}. Tanner, “Spanish Lady,” 54.
signs could be determined.\textsuperscript{74} When the fever became epidemic in troops in June 1918, P.U.O. was replaced by a new term, “three-day fever,” which reflected its typical clinical course: “three days’ incubation, three days’ fever, and three days’ convalescence.”\textsuperscript{75} It was only when the epidemic reached the civilian population in late June 1918 that it started to be called “influenza,” first in the lay press, where it was erroneously labeled “Spanish Influenza,” and then in the medical press, where, beginning in mid-July, it was used cautiously and the notion of the epidemic’s Spanish origins immediately challenged. The \textit{Lancet} was hesitant to describe it as “influenza” because of the term’s use by the media, public, and many practitioners to denote all sorts of mild respiratory conditions. The editors often put the term in scare quotes.

These uncertainties affected official responses. Notably, the DGAMS did not officially recognize the disease as “influenza” until the first week of August, when the summer epidemic had all but smoldered out. Historians have noted that British military officials wanted to conceal the disease and that censorship not only delayed reports of the epidemic but also contributed to the popular notion that it was of Spanish origin.\textsuperscript{76} However, the key reason for the delay was that military experts and officials were genuinely confused about its identity. Arthur Newsholme, who, on similar grounds, decided not to issue an official LGB memorandum to civil authorities in summer 1918, shared the DGAMS’s confusion.\textsuperscript{77} No one was certain about the disease. The key constraint was that the bacteriological evidence did not sufficiently support such a classification. Rather, it generated controversy. A contradictory bacteriological picture emerged in summer 1918. The medical and general press printed accounts from German bacteriologists who claimed they too had failed to find the bacillus. Pfeiffer himself was reported as remaining silent.\textsuperscript{78} At the
same time, anecdotal reports from British pathologists in France suggested that the bacillus was occasionally found and in some cases was being characterized as the predominant organism in the epidemic. The *British Medical Journal (BMJ)* argued that “the general consensus of opinion seems to indicate Pfeiffer’s *Bacillus Influenzæ* as the infecting agent.” This contradicted not only the editorial views of the *Lancet*, which doubted whether the epidemic was influenza, but also reports in the BMJ’s own pages.

Through summer 1918, two loosely defined camps of pathologists clashed over the causal agent and identity of the epidemic: the “Pfeiffer school” argued that the epidemic was influenza, and attributed the inability to find *B. influenzae* to technical failures; the “anti-Pfeiffer school” argued that its absence indicated either that the epidemic was not influenza or that influenza was caused by another organism. Questions about the causative agent were inevitably questions about professional competence, skills, and interests. They also had profound implications for understanding influenza’s pathology and for its prevention with vaccines. It is worth looking more closely at work on influenza’s bacteriology during the first wave, as uncertainties and disputes about its etiology had important bearing on the elaboration of official strategies during the second wave.

**DISPUTED GERMS**

Contrary to the BMJ, in summer 1918, bacteriological evidence and opinion weighed against Pfeiffer’s bacillus. Its absence was explained in various ways. Many pathologists followed the assumption that without *B. influenzae* there could be no influenza. In the first published bacteriological report on the pandemic in Britain, which appeared in the *Lancet* on 13 July 1918, three Canadian

79. Edward B. Krumbhaar, “The Bacteriology of the Prevailing Epidemic,” *Lancet*, 1918, 192, 123.
80. “The Pandemic of Influenza,” *Br. Med. J.* 1918, 2, 91–92.
81. Ludwik Rajchman, editor of the MRC’s Medical Supplement, used the term “Pfeiffer school” to describe those who believed that “true influenza epidemics” were caused only by *B. influenzæ*, “Influenza,” *Medical Supplement*, 1 October 1918, 354. Paul Fildes and James McIntosh used the term “anti-Pfeiffer school” to describe its critics. Paul Fildes and James McIntosh, “The Aetiology of Influenza,” *Br. J. Exp. Path.*, 1920, 2, 159.
82. See Fildes and McIntosh, “Aetiology of Influenza”, McIntosh, *Studies in the Aetiology of Epidemic Influenza*. 
Army Medical Corps pathologists took this reasoning to its logical conclusion. Working from their mobile laboratory on the Western Front, they encountered hundreds of cases of the spring fever. Suspecting influenza, they ran bacteriological tests on nasal and sputa samples from twenty soldiers. But they were unable to isolate \textit{B. influenza}. They instead found a small diplococcus in great abundance and often in pure cultures. Based on this evidence, they concluded that the epidemic was not influenza and that their diplococcus was likely “the causative organism.”

While the Canadians would be hard pressed to find supporters for their diplococcus, many pathologists employed similar reasoning. Paul Fildes and James McIntosh, former colleagues at the London Hospital’s Department of Bacteriology, typified those who stood by Pfeiffer’s bacillus. Both men were experienced and respected researchers, and worked together throughout the war. McIntosh remained in civilian service at the London, while Fildes served on a temporary commission as pathologist to the Royal Naval Hospital at Haslar in Plymouth. Here, with MRC support, he ran a pathology laboratory. His position gave him unobstructed access to sick servicemen at Haslar, including those who started flooding into the wards in August with the so-called Spanish influenza.

As in other naval bases, the disease had been endemic in Portsmouth since spring 1918, and Fildes and his colleagues had made it the “subject of considerable inquiry and study.” At first Fildes was not convinced that it was Influenza: “The question of influenza as a factor in the disease was discussed and studied,” he noted, “but owing to the complete absence of bacteriological support the causative importance of this bacillus was discredited.” He attributed the epidemic to one of a number of “respiratory”

83. T. R. Little, C. J. Garofalo, and P. A. Williams, “Absence of the \textit{Bacillus Influenzae} in the Exudate from the Upper Air-Passages in the Present Epidemic,” \textit{Lancet}, 1918, 192, 34.
84. Ibid.
85. G. P. Gladstone, C. J. G. Knight, and Graham Wilson, “Paul Gordon Fildes, 1882–1971,” \textit{Bio. Mem. Fell. Roy. Soc.}, 1973, 19, 317–74.
86. MRC, Influenza Research by Dr. Fildes, FD1/530, National Archives, Kew, Richmond, Surrey (hereafter NA).
87. Paul Fildes, S. L. Baker, and W. R. Thompson, “Provisional Notes on the Pathology of the Present Epidemic,” \textit{Lancet}, 1918, 192, 697.
88. Ibid.
organisms—hemolytic streptococci, pneumococci, or *streptococcus pyogenes*—which, he thought, “had taken on increased virulence, owing to the somewhat unsatisfactory weather and presence of large numbers of young ‘new entries’ in the barracks,” among whom the epidemic was largely confined.89

McIntosh shared Fildes’ skepticism. A taciturn Scotsman who came to the London Hospital in 1908 after spending two years in Paris on Elie Metchnikoff’s bacteriological course at the Pasteur Institute, McIntosh was well established in London pathological circles.90 Like many civilian pathologists, he had started investigating the influenza in summer 1918. With clinical material taken from a few cases and postmortem examinations, he tested for *B. influenzae* using ordinary blood agar and found no trace. As he later admitted, he concluded, “perhaps rather rashly,” that the bacillus “did not play an important part in the causation of the epidemic” and ceased to pursue this line of investigation.91 While Fildes was able to suggest other possible candidates, McIntosh failed to find “a single type of bacterium . . . with any constancy.”92 Despite this difference, both men’s approaches to the epidemic reflected the grip that Pfeiffer’s bacillus had on some pathologists. When they failed to find the bacillus, the natural conclusion was that the epidemic was not influenza.

But what was the epidemic and what was its cause? A host of prospective diseases and agents emerged from pathological laboratories and medical minds. Researchers probing the respiratory tracts of the sick were supplied with an abundance of well-known microorganisms, many of which were pathogenic and could even be epidemic under the right circumstances. Yet no one could agree on the main culprit.

A week after the Canadian pathologists’ publication, two pathologists at the Central Royal Air Force Hospital in Hampstead, Oliver Gotch and Harold Whittingham, identified *M. catarrhalis* as the “predominating organism” in fifty cases, and suggested that it was “probably the specific organism” of the epidemic, acting alone or,
more likely, as a mixed infection in conjunction with \textit{B. influenzae} (which they had isolated from a small number of patients).\textsuperscript{93} Still other candidates were proposed from the various types of familiar respiratory germs, including meningococci, pneumococci, pneumo-bacilli, streptococci, and staphylococci. A brief consensus emerged shortly after the summer epidemic subsided when reviews of laboratory work in Britain, Germany, France, and the United States noted that one type of respiratory germ, the “diplostreptococci,” was almost “universally recorded.”\textsuperscript{94} Yet, no sooner had this agent been identified as a possible candidate than pathologists questioned its status. While some suggested that it was the specific cause, others argued that it was part of a unique compound infection involving various agents, and still others insisted that it was nothing more than a secondary infection, with the primary agent still unknown.\textsuperscript{95}

These skirmishes illuminated the difficulty pathologists faced when abandoning or challenging a familiar disease category. Those skeptical about whether the epidemic was influenza faced the daunting prospect of not just characterizing the agents and identity of yet another disease, but also of forging a consensus about them.

Proponents of Pfeiffer’s bacillus did not need to traverse this minefield. Accepting that the summer epidemic was influenza, they argued that the problem was to deploy the right techniques in the right manner to isolate the bacillus. Many defended the role of bacillus, but in Britain none was more vocal or more assiduous than John Matthews. A member of Wright’s Inoculation Department, Matthews worked on various aspects of vaccine production, including making stock “Anti-catarrh” vaccines from Pfeiffer’s bacillus, which were marketed and sold through Parke, Davis and Company, and supplied to the Army.\textsuperscript{96} During an epidemic in London in March 1915, he successfully isolated and made what he claimed to be an effective vaccine from it. At the time he insisted that only the rigorous use of established culture technique could produce

\textsuperscript{93} Oliver H. Gotch and Harold E. Whittingham, “A Report on the ‘Influenza’ Epidemic of 1918,” \textit{Br. Med. J.}, 1918, 2, 82.

\textsuperscript{94} “Influenza,” \textit{Medical Supplement}, 1 October 1918, 358.

\textsuperscript{95} Ibid., 358.

\textsuperscript{96} For the Inoculation Department’s vaccines, see Chen, “The Laboratory as Business.”
successful results: “nothing short of the employment of an agar plate, rich in fresh blood, gives one any confidence in giving an opinion. This, I imagine, is the experience of every competent bacteriologist.”

In summer 1918, he wielded this view against those who questioned the bacillus. He took particular aim at the Canadian pathologists. Within a week of their report he accused them of a fundamental error. Their culture medium was “devoid of blood,” a remarkable oversight given the known growth requirements of the bacillus, and one that undermined their experiments. While Matthews accused the Canadians—and other pathologists—of technical incompetence, he could not level the same criticism against peers, like Fildes and McIntosh, who had followed standard procedures. Their experiences raised a different kind of problem, which had to do with the existing standard of established media and culture techniques. Blood-agar medium was finicky and, as most pathologists knew, getting its composition right was especially important to making the bacillus visible. Variations in media also produced variations in results. Matthews argued that, more than laboratory skill, lack of good quality media was the primary reason pathologists had failed to find the bacillus.

Between June and October 1918, he devoted much effort to drawing attention to and resolving these issues. In July 1918, Matthews reported that he had devised a new medium on which pathologists could more accurately and consistently visualize Pfeiffer’s bacillus. Based on a technique developed for making diagnostic blood cultures by his colleagues at the Inoculation Department, S. R. Douglas and Leonard Colebrook, Matthews’ method involved mixing a small portion of blood with the commercially manufactured pancreatic secretion, trypsin, and some broth, before combining it with agar. The process created a product that, when mixed with agar, produced “profuse growth”; and moreover it was highly “selective for this organism.”

Trypsinized blood appeared to inhibit the growth of other organisms in plate culture—a notorious problem in cultivating B. influenzae—that enabled the free growth of the bacillus. The medium gave

97. John Matthews, “Influenza or Epidemic Catarrh,” Lancet, 1915, 185, 727.
98. John Matthews, “On a Method for Preparing Medium for the Culture of Pfeiffer’s Influenza Bacillus,” Lancet, 1918, 192, 104.
Matthews' exceptional results during the summer epidemic. He recovered the bacillus from a dozen cases at St. Mary's Hospital, "frequently in profuse and practically pure cultures." Confident that he had found a solution to the culture problem, he claimed that, "if this medium be used, Pfeiffer's bacillus will be found universally associated with the epidemic." He started campaigning to get the medium into the hands of other pathologists and to standardize its manufacture.

Matthews' medium appeared to live up to its billing. When a far more deadly epidemic appeared in troops in August, pathologists who had at first failed to isolate Pfeiffer's bacillus reported regularly finding it, and many attributed their success to the medium. Fildes and McIntosh were spurred to re-investigate the role of Pfeiffer's bacillus. Fildes used Matthew's medium to isolate the bacillus from new cases in August and devised his own medium, "K," which employed the peptic digest of sheep's blood. McIntosh reported "successful results with a version of Matthews' trypsinised blood medium." Recanting his earlier doubts, he now testified that, "the Bacillus influenza belongs to the delicate group of hemophilic bacteria which require special media for their isolation. Up to the present it was generally supposed that ordinary laboratory media to which some blood had been added were sufficient." Matthews had apparently proved this assumption wrong. Using variants of his medium, pathologists who had initially questioned the summer epidemic's identity became convinced that, provided sufficient care was given to isolation methods, Pfeiffer's bacillus could be found in every case.

Innovations of media were not new and the support for Matthews' media did not assuage doubts about the bacillus' role. The most damning criticism came in the M.R.C.'s Medical Supplement. Its editor, Ludwik Rajchman, a Polish émigré bacteriologist who had come to King's College in 1910 as a laboratory assistant, worked with Fildes at the London Hospital, and was made head of London's Central Laboratory on Dysentery in 1914.
highlighted the contradictory nature of laboratory evidence. In an editorial printed on 1 October 1918, he concluded that there was “sufficient material to shake the orthodox conception out of its high altar.” Rajchman argued that the mere presence of *B. influenzae* did not prove its causal identity with influenza. Any one of the abundant organisms found during the epidemic could meet this criterion. Moreover, reports on the morbid anatomy of the summer epidemic failed to directly connect it to the pathological lesions of the disease—particularly lobar pneumonia and hemorrhages in the respiratory tract. He insisted that: “The cold logic of the *post-mortem* room in the dispassionate home surroundings does not leave any doubt that [*B. influenzae*] when present did not play any more important part [in the epidemic] than the ubiquitous diplostreptococci.”

The *Medical Supplement*’s intervention affected the debate on the epidemic’s identity and the subsequent direction of official pathological work. Powerful as an arm of the MRC, its role as a conduit and mediator of militarily relevant medical information shaped professional opinions and agendas. Its reviews were regularly summarized in the medical and general press, and taken as definitive. With this backing, Rajchman’s report made an impact. The *Lancet* commented that it could “recall no more masterly review in our language of this or, indeed, any other ‘war disease’ during the last four years... that we should take it as a basis” for understanding the epidemic’s bacteriology. Rajchman’s review further sanctioned the exploration of alternatives to Pfeiffer’s bacillus. If this was the culprit, proof of its identity needed to be better established. If the agent was some other entity, then pathologists had better find it fast. Rajchman not only illuminated existing divisions among pathologists, he also raised the key question of what laboratory knowledge should form the basis of official pandemic strategies.

What might have appeared as little more than intraprofessional rivalry in summer 1918 was, by the time of Rajchman’s review, a matter of grave medical and military importance. Divisions among

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103. Marta A. Balinska, “Ludwik Rajchman: Pioneer of International Health,” *Int. Health Hist. Newsletter*, 1995, 1, 8–9.
104. Anon., “Influenza,” *Medical Supplement*, 1 October 1918, 359.
105. Anon., “Lessons of a Great Epidemic: The Pathology of Influenza,” *Lancet*, 1919, 193, 25.
pathologists had consequences for approaches to the far more catastrophic autumn epidemic. Ideally, wartime medical officials wanted a uniform strategy based on the rapid identification of the disease and its pathogen, followed by the equally rapid production and distribution of a vaccine. In reality, official strategies in autumn 1918 reflected uncertainties among laboratory and medical experts.

Although the summer epidemic was mild, and received only passing attention from civilian medical and public health authorities, some military pathologists and officials worried that it would be followed by a more virulent recrudescence. Records of the 1889–92 pandemic supported this expectation: they showed how two deadly epidemics succeeded a mild one. The MRC put credence in this precedent and became the first government body to draw attention to possible “secondary waves of the infection.”

Assuming that the summer epidemic was influenza, but undecided about its specific cause, Fletcher and his colleagues believed that it was not a matter of whether there would be a recrudescence, but when. No one could predict how quickly it would come or how severe it would be. But planning was imperative.

On 5 August 1918, Fletcher sent a memorandum to the BMJ and the Lancet calling on pathologists and practitioners to make preparations for a second epidemic. He specifically asked for the results on influenza’s bacteriology to be sent directly to the Committee. The memorandum was the first part of an evolving plan to coordinate influenza research. The MRC wanted to ensure that laboratory and clinical work was well organized and, as far as possible, centrally administered. Changes in influenza’s virulence provided the rationale. For when the MRC issued its memorandum, evidence was already emerging that the anticipated second wave had been identified in troops in France and at bases at home.

106. Richard Reece (War Office) to Walter Fletcher, 3 November 1918, and Walter Fletcher to Richard Reece, 6 November 1918, FD1/535, NA; Memorandum on a scheme of inquiry concerning influenza, FD5/186, NA.
107. Anon., “Bacteriology of the Influenza Pandemic,” Br. Med. J., 1918, 2, 140.
108. Influenza Committee: correspondence with LGB and War Office, FD1/535, NA.
109. Medical Research Committee, “Memorandum,” Lancet, 1918, 192, 717–18. Walter Fletcher had circulated the letter to the medical press on 5 August 1918. See Walter Fletcher, “The Influenza Epidemic,” Lancet, 1918, 192, 643.
110. General Scheme of Influenza Investigations, 11 November 1918, FD1/533, NA.
As the summer epidemic abated among civilians in late July, a new form of the disease appeared in among troops in late August. For thousands of soldiers, a dreadful array of secondary infections, rarely seen in previous epidemics, led to severe and often deadly pneumatic complications.\textsuperscript{111} By November, it had engulfed the nation. The numbers of dead in London alone crept toward sixteen thousand.\textsuperscript{112} Estimates put the minimum total death toll for Britain at two hundred thousand, a large number of whom were soldiers.\textsuperscript{113} Most died between October and December 1918.

The second wave likely gained a foothold in Britain at naval ports in Portsmouth, Southampton, and Liverpool.\textsuperscript{114} Reports indicated that the early outbreaks were highly virulent and far more deadly than anything seen during the summer. While resembling influenza, what was singularly outstanding, according to observers, was how rapidly it turned into a lethal respiratory condition.\textsuperscript{115}

At Portsmouth, Fildes and his colleagues were struck by the “acute alteration” in the disease’s characteristics. Young naval recruits were the most severely affected. By the end of August, cases among them became “more and more numerous and the clinical aspect more and more acute.”\textsuperscript{116} Fildes later reckoned that Portsmouth was an epicenter from which the epidemic radiated out to the rest of the nation.\textsuperscript{117} What he and his colleagues observed on their wards and in their morgues foreshadowed what would happen once the disease spread into civilians. Large numbers of young men with acute

\textsuperscript{111} Mark Harrison notes that “influenza was rife throughout Mesopotamia and it was the main cause of admissions to hospital among British troops in the last months of the war and after armistice,” \textit{Medical War}, 284.

\textsuperscript{112} William Hamer, Chief Medical Officer of the London County Council estimated the number of deaths between October and December 1918 to be close to sixteen thousand. William H. Hamer, \textit{Report on Influenza by the County Medical Officer of Health} (London: County Hall, 1919). Current estimates support this figure. Niall P. A. S. Johnson and Juergen Mueller, “Updating the Accounts: Global Mortality of the 1918–1920 ‘Spanish’ Influenza Epidemic,” \textit{Bull. Hist. Med.}, 2002, 76, 105–15; Langförd, “Age Pattern,” 1–20.

\textsuperscript{113} Johnson and Mueller, “Global Mortality.” 113. For difficulties in estimating mortality among soldiers, see Johnson, \textit{Influenza Pandemic}, 69–93.

\textsuperscript{114} For the spread of the epidemic in Britain, see Johnson, \textit{Influenza Pandemic}, 37ff.

\textsuperscript{115} Adolphe Abrahams, Norman E Hollows, and Herbert French, “A Further Investigation into Influenzo-Pneumococcal and Influenzo-Streptococcal Septicaemia: Epidemic Influenza ‘Pneumonia’ of Highly Fatal Type and Its Relation to ‘Purulent Bronchitis’,” \textit{Lancet}, 1919, 193, 1–11.

\textsuperscript{116} Fildes, Baker, and Thompson, “Provisional Notes,” 698.

\textsuperscript{117} Ibid., 697.
pneumonia were admitted to hospital, and among these the death rate was very high. Postmortems showed that rather than dying from what seemed to be a general infection, “patients appeared to be drowned” by a massive accumulation of pus, fluid, and blood in their lungs, bronchial tree, and upper respiratory tract. In a typical case, significant parts of the lungs of a “well developed muscular young man, aged eighteen” were “airless” and full of blood and mucous, a condition that induced marked cyanosis. As he slowly suffocated, his fingers and lips turned blue and his complexion a pallid gray.\textsuperscript{118}

This shocking pathological picture soon became the norm. Herbert French, Aldophe Abrahams, N. F. Nallows, and J. W. H. Eyre, consulting pathologists at Aldershot hospital, who had described cases of purulent bronchitis in 1916, produced the standard descriptions of soldiers dying from the strange heliotrope cyanosis. In the weeks that followed, physicians and pathologists describing thousands of similar cases would recognize cyanosis as a sign of imminent death and the cyanotic patient, suffering acute respiratory damage, became the iconic symbol of the epidemic.

The early signs of a new wave prompted the MRC to expand its plans for “an organized scheme” of pathological and medical work. In a series of “emergency arrangements” in September, Fletcher asked MRC pathologists at various universities and institutes to put aside their current research to work on influenza.\textsuperscript{119} He hoped their work would be coordinated with research pathologists in the army and navy, who were also asked to concentrate their efforts.\textsuperscript{120} While military officials were hesitant to prioritize studies of the epidemic, Fletcher argued that such “research was necessary from a ‘service’ point of view.”\textsuperscript{121} By early September, the MRC started working with the War Office and AMS to organize investigations into the primary cause and to determine possible methods of prevention and treatment.\textsuperscript{122}

Through the summer, official military policy had been to “carry on” in the face of the epidemic.\textsuperscript{123} But with forewarnings from the
MRC, the threat of yet another epidemic jeopardizing military operations, and new laboratory evidence from military pathologists that the disease appeared to be caused by Pfeiffer’s bacillus, on 6 August 1918 the DGAMS issued orders to return the epidemic as “influenza.” Soon after, its Advisory Board created a special “Influenza Committee” to oversee the work of its medical and pathological services on the pandemic. Along with Leishman and Fletcher, the Committee included representatives from the Army, Admiralty, and LGB. For direction, it relied on Fletcher and Leishman, who had taken up his new post as Advisor on Pathology to the War Office. From August to early October, the Committee mapped out a rudimentary response plan.

The plan involved two interrelated strategies. The first concentrated on the development of a vaccine; the second concentrated on improving bacteriological methods for vaccine manufacture. Influenza’s bacteriology posed a host of practical problems for vaccine production. With the primary cause still unknown, the microorganisms to make up a vaccine had to be selected, typed, and cultivated. While Leishman and Fletcher were under no illusion about the challenge of manufacturing an effective vaccine, they also understood its importance for consolidating the role of pathology in controlling the pandemic. A successful vaccine was seen as a way to resolve influenza’s etiology. If it prevented cases, then this could be taken as evidence of the etiological role of the agent(s) used. Much was thus at stake in producing an effective vaccine.

MIXED VACCINES

Influenza vaccines had been developed in the decades before the war, but remained unproven. Most were either so-called anticatarrhal vaccines produced using different microorganisms associated with catarrhal infections or streptococcus and pneumococcus vaccines targeted at secondary infections. Because of its identified role in the disease, Pfeiffer’s bacillus was regularly included in these mixtures. Various anticatarrhal vaccines were bought and marketed by pharmaceutical companies. Parke, Davis and Company’s “Anti-Catarrhal Vaccine,” made at Wright’s Inoculation Department, was advertised as a
Laboratories typically produced two kinds of vaccine: one prophylactic, to prevent infection or reduce complications from it, and the other therapeutic, administered after infection as a form of treatment aimed at boosting the patient’s immunity by inducing the production of immune substances called “opsonins.” This widely followed two-pronged approach was Wright’s creation; he had characterized opsonins and developed an opsonin index in 1902, and established this to produce therapeutic and prophylactic vaccines. On this model, the efficacy of a vaccine was judged in terms of either curative or preventive qualities. The therapeutic use of catarrhal vaccines had gained popularity in civilian practice. While some were reportedly highly effective, in the decade before the war there was little to suggest that they much reduced the incidence of influenza. Some evidence indicated that these, as well as pneumococcus vaccines, prevented respiratory complications, and some practitioners reported good results when vaccines were used as treatments. But nothing approaching a systematic evaluation of the kind applied to typhoid vaccines had been carried out earlier in the decade.

During the summer epidemic, military pathologists tested the efficacy of “mixed” catarrhal vaccines. Targeting secondary complications, their composition varied considerably. J. W. H. Eyre worked with the pathologists at the New Zealand Expeditionary Force’s General Hospital at Walton-on-Thames on the first large-scale investigation of a prophylactic “mixed catarrhal vaccine” (M.C.V.). Developed at Eyre’s Bacteriology Department at Guy’s, the vaccine was made from seven different heat-treated organisms isolated from soldiers and prepared for use in two dosage strengths, delivered ten days apart. Between March and August 1918, Eyre and his NZEF colleagues inoculated 16,104 new recruits and used another 5,700 as

126. John Matthews, “Influenza and Preventive Inoculation,” Lancet, 1918, 192, 602.
127. Chen, “Laboratory as Business”; Worboys, “Almroth Wright.”
128. John Eyre and John Lowe, “Prophylactic Vaccinations against Catarrhal Affections of the Respiratory Tract,” Lancet, 1918, 192, 484–87; W. H. Wynn, “The Use of Vaccines in Acute Influenza,” Lancet, 1918, 192, 874–76.
129. For American efforts to manufacture a flu vaccine, see John Eyler, “The Fog of Research: Influenza Vaccine Trials during the 1918–19 Pandemic,” J. Hist. Med. Allied Sci., 2009, 64, 401–28; John Eyler, “The State of Science, Microbiology, and Vaccines Circa 1918,” Pub. Health Rep., 2010, Suppl. 3, 125, 27–36.
130. Eyre and Lowe, “Prophylactic Vaccinations,” 484–87.
uninoculated controls. Comparing the average of all respiratory complications among the inoculated and uninoculated, the study showed that M.C.V. reduced complications. Promising as this was, it did not prevent infection. Some pathologists argued that while targeting secondary complications was necessary, the primary goal should be prevention. “[I]f the influenza can be prevented,” argued Fildes, “complications will not arise.” This goal raised the contentious problem of what organism or organisms should form the basis of a specific vaccine. So-called Pfeiffer vaccines, made from pure cultures of *B. influenzae*, had been tested in the United States and proven poor prophylactics. While variation in the composition of anticatarrhal vaccines was acceptable, introducing such variation into the composition of influenza vaccine relegated Pfeiffer’s bacillus to the status of a secondary invader, which its proponents were loath to do.

Despite their uneven record, using vaccines to combat the autumn epidemic appealed to medical officials as the most efficient approach. There were few other options, since “[n]o drugs,” declared the Royal College of Physicians, “have yet been proved to have any specific influence as a preventive of influenza.” For medical officials, a vaccine could potentially overcome this problem by reducing the incidence of the disease and mitigating more severe complications. It could also be used therapeutically on the worst cases. Since maintaining manpower was the most crucial concern of military planners as they lurched toward victory in autumn 1918, a vaccine that kept or got soldiers out of hospital beds had obvious attractions over nursing and bedside care. This logic also appealed to the LGB and other civilian medical authorities, whose ranks and thus capacity to respond to a pandemic had been seriously diminished by military demands.

131. Ibid., 487.
132. Fildes, Baker, and Thompson, “Provisional Notes,” 700.
133. Eyler, “Fog of Research.”
134. Fildes, Baker, and Thompson, “Provisional Notes,” 700.
135. On the variety treatments, see Lori Loeb, “Beating the Flu: Orthodox and Commercial Responses to Influenza in Britain, 1889–1919,” *Soc. Hist. Med.*, 2005, 18, 203–24.
136. “Memorandum by the Royal College of Physicians,” *Br. Med. J.*, 16 November 1918, 546.
137. John Eyler, *Sir Arthur Newsholme* (New York: Cambridge University Press, 1997), 272–73.
On 14 October 1918, Leishman organized a conference at the War Office with the MRC to decide on an official approach to “preventive vaccination.” Along with Eyre, Leishman invited fellow MRC member, and Professor of Pathology at St. Bartholomew’s Hospital, F. W. Andrewes, who had worked on first isolating Pfeiffer’s bacillus in Britain in 1892, and S. R. Douglas, who had been developing methods for typing “races” of bacteria, essential to vaccine development. They were joined by two military officials who would be responsible for vaccine production: Lieutenant-Colonel D. Harvey, officer in charge of the Royal Army Medical College’s Vaccine Department, and the Deputy-Surgeon General, P. W. Bassett-Smith, who oversaw the vaccine department at the Royal Naval College. The Committee aimed to establish a vaccine formula to be used throughout the military services. Vaccines developed with the known causative agents of a disease were regarded as most promising. Yet when the Committee reviewed the bacteriology of the summer and early autumn epidemics they “agreed that there was considerable doubt as to the primary etiological significance of the Bacillus Influenzae.”

Rather than risk developing a “Pfeiffer” vaccine, they advocated a “mixed” vaccine. Determining its composition involved selecting which organisms to include and in what quantities. Both decisions would be controversial. Concentrating on those agents known to play the most significant role in influenza’s complications, they decided that B. influenzae should form its basis, with streptococcus and pneumococcus selected because of their role in grave secondary infections.

Other organisms isolated from the epidemic were excluded. Two weeks after the conference, the War Office published its guidelines for manufacture and administration in the *Lancet.* Different strains of each organism were to be used; each had to be isolated from cases during the epidemic and submitted to strict tests for their “race and type.” The Committee made no recommendations for the best methods of cultivating the organisms, but did define the “relative proportions” of organisms used and the dosage

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138. Anon., “The Utilisation of Vaccine for the Prevention and Treatment of Influenza,” *Lancet, 1918,* 192, 565.
139. Ibid.
140. Ibid.
141. Ibid.
size. The vaccine would be administered in two doses, spread ten days apart, with the second dose doubling the dosage of the first. Since its primary role was preventive, inoculations were to be given before exposure to infection. But because it also targeted secondary infections, the Committee saw no reason to withhold its use on influenza cases. Finally, following the approach established for testing antityphoid vaccine, the War Office asked that steps be taken to secure exact statistical records on reactions and the incidence and complications of the disease following inoculations in all clinical settings.\footnote{142}

Large-scale manufacture for the Army and Navy was centralized at their respective vaccine departments, where production began soon after the War Office conference. The organisms used were first screened, typed, and selected by Douglas, who was assisted by the MRC in classifying the types of \textit{B. influenzae} isolated at different centers.\footnote{143} Other laboratories were left to produce the vaccine voluntarily, and no coordinated effort was made to mass-produce it for civilians.

The formula embodied the limitations of existing pathological knowledge, and immediately became the focus of debate. Many experts and observers noted that it was little more than a mixed “Pfeiffer” vaccine.\footnote{144} Pathologists with experience in making such vaccines questioned the composition, dosages, and practicalities of mass production. Many wondered why important pathogens, such as \textit{M. catarrhalis}, were excluded and criticized the quantity of organisms in the vaccine itself. Compared with other mixed vaccines, the dosages of the War Office’s formula were significantly smaller. Whereas a “mixed catarrhal vaccine” made at St. Mary’s contained three hundred million influenza bacilli, the War Office’s formula recommended only thirty and sixty million influenza bacilli.\footnote{145} W. H. Wynn, an expert on prophylactic and therapeutic vaccines at Birmingham General Hospital, argued that the dosage recommendations were “inadequate and likely to imperil the value of the vaccine.”\footnote{146}
Successful vaccines, including those for typhoid and pneumonia, contained huge numbers of organisms (a minimum of one thousand million) because, claimed Wynn, their makers understood that “the period of immunity [was] probably proportionate to the size of dose used.”\footnote{Wynn, “Preventive Inoculation,” 643.} The War Office’s dosage cast doubt on the Committee’s expertise.

Setting the tone of criticism, John Matthews argued that in the absence of experimental work on influenza vaccines, the question of dosages—and any other aspect of its manufacture—could not be “a question of pure science” but was rather “a question of practice” and reliable expertise.\footnote{Matthews, “Preventive Inoculation,” 602.} He challenged the recommendations of a Committee that did not include any practitioners and relied on the “experience of . . . a minority” of pathologists. Not surprisingly, he believed that the limitations of this approach were most glaring with respect to \emph{B. influenzae}. By not outlining a suitable culture method the Committee had demonstrated its ignorance of expert work, and in particular the relevance of his own medium. Older culture methods could not be relied upon to make the quantity and quality of bacillus needed for mass vaccination. “[I]t is almost inconceivable,” he argued, “that under old conditions a vaccine . . . could have been provided in a reasonable time for large bodies of troops. This aspect has been entirely changed by my method.”\footnote{Ibid.} Matthews insisted that his method was indispensable. It had been used for two years at St. Mary’s for mixed catarrhal vaccines, which were supplied to military authorities.\footnote{Ibid.}

While Matthews wanted a wider representation of expert opinion in formulating the vaccine, others challenged the assumptions on which the formula was based. In a scathing analysis, the eminent physician, Thomas Horder, argued that the Committee had failed to acknowledge two fundamental problems. First, they ignored the constraints posed by the lack of knowledge and consensus on the etiological agent. “Bricks cannot be made without straw,” he argued, “and dogmatic advice on the prevention and cure of a disease cannot be given in the absence of accurate data on its

\begin{thebibliography}{1}
\bibitem{Wynn} Wynn, “Preventive Inoculation,” 643.
\bibitem{Matthews} Matthews, “Preventive Inoculation,” 602.
\bibitem{Ibid} Ibid.
\bibitem{Ibid} Ibid.
\end{thebibliography}
causation.” The Committee had dismissed the primary role of *B. influenzae* and then based a vaccine upon it. The claim that such a vaccine could control influenza was without foundation. Second, even if the formula was workable, Horder argued that the Committee had failed to acknowledge the time it would take to produce. While every Committee member knew that typing strains was a “highly expert and lengthy technique,” they had proceeded “as though all this work...had already been done!” These oversights suggested to Horder that the War Office had “very little faith in its nostrum, at least as a preventive for the present epidemic.”

Not about to abandon its plans, the War Office addressed some criticisms, but ignored others. With the help of the MRC, it quickly established the best culture methods for the selection and mass production of *B. influenzae*. Matthews’ medium was recognized as a prime candidate, but so too was Walter Levinthal’s medium, a German formulation that Fildes and other pathologists had successfully employed. In early November, the MRC asked pathologists to compare the two methods. Most found that Levinthal’s medium produced better growth, while Matthews’ was better suited for isolation and strain selection. While the War Office incorporated advice on the medium it resisted changing its formula and made the vaccine as originally planned. Between November 1918 and July 1919, the Army Vaccine Department alone made 1,806,325 doses, while the Royal Navy Vaccine Department produced 144,000, enough to inoculate the Grand Fleet. Mass inoculations in the Army began on 1 November 1918, along with voluntary trials at numerous bases in England and France.

Leishman hoped the formula would be adopted across the armed forces. But, pathologists unconvinced by it continued to work

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151. Horder, “Preventive Inoculation,” 642.
152. Ibid.
153. MRC, “Circular Sent to District Laboratories,” 1 November 1918, FD1/533, NA.
154. See Walter Fletcher’s correspondence with pathologists, Medical Research Committee, FD1/534, NA.
155. Leishman, “Organization,” 30; Anon., “The Vaccines and Serums Supplied to the Royal Navy,” *Lancet*, 1919, 193, 545. One dose = 1 cc.
156. William Leishman, “The Results of Protective Inoculation against Influenza in the Army at Home, 1918–1919,” *Lancet*, 1920, 195, 366–68.
Numerous unofficial preventive vaccines were made. Commonly, these included organisms excluded from the official vaccine, in different proportions, and, most tellingly, with significantly increased dosage sizes. A number of pathologists, including Wynn, also employed aggressive therapeutic vaccines for cases with acute or chronic respiratory complications. Unofficial vaccines challenged the War Office formula and made it difficult to assess accurately its own vaccine.

Trials of the War Office vaccine involved limited organization. As with other forms of vaccination, influenza inoculation was voluntary, which reduced the number of available experimental subjects. The War Office relied on the judgments of physicians and pathologists to determine its effectiveness. The MRC helped the Advisor in Pathology to the DGAMS in France, S. L. Cummins, coordinate trials at Boulogne in November and December 1918, in which over two thousand soldiers were inoculated. The most thorough evaluation came from inoculation records supplied to Leishman from camps in the home commands, where over eleven thousand soldiers received one or two doses in November 1918, and another five thousand were inoculated between December 1918 and April 1919. While Leishman admitted the results were not conclusive or even free from “large fallacies,” he believed that they demonstrated an “encouraging” general trend. Inoculation appeared to provide moderate protection against infection and had “decidedly beneficial effects” in reducing the frequency and severity of complications. For Leishman, the results vindicated the vaccine: “they confirm and even strengthen our original anticipations.”

Yet Leishman’s assessment appeased only those pathologists who trusted the vaccine. For others, it was a vaccine built on false premises. There were questions about the identity of the disease against which it was to protect; the primary agent against which the vaccine was targeted was still unknown; and, once vaccination

157. For different vaccine preparations, see Fildes, Baker, and Thompson, “Provisional Notes,” 695–700; H. E. Whittingham and C. Sims, “Some Observations on the Bacteriology and Pathology of Influenza,” Lancet, 1918, 192, 865–71.
158. W. H. Wynn, “Use of Vaccines,” 874–76.
159. MRC, “Prophylactic Anti-influenza Vaccination,” 21 December 1918, FD1/529, NA.
160. Leishman, “Results of Protective Inoculation,” 367.
161. Ibid.
proceeded, many doubted that it was possible to collect accurate statistical evidence of its effectiveness.

Pathological investigations during the second wave illuminated the first two problems. Describing a stunningly complex disease entity, investigators struggled to produce consistent pictures of the processes of infection they encountered postmortem and were divided over which organisms contributed to which pathological changes.\textsuperscript{162} Herbert French argued that a primary infection, which he believed was \textit{B. influenza}, set-up the most severe complications.\textsuperscript{163} This view gained renewed support in autumn 1918, as pathologists began to find Pfeiffer’s bacillus with increasing frequency. Fildes and McIntosh defended its primary role and emerged as staunch proponents of the “Pfeiffer School.” The decisive evidence for them was the remarkable increase in the incidence of the bacillus. They attributed this change to improvements in culture media and methods, and not to an epidemiological factor. They claimed that the bacillus had been present throughout, but had eluded bacteriologists who lacked adequate techniques or expertise to make it visible. “[T]he epidemic can be divided into two stages,” argued McIntosh, “a first in which \textit{B. influenzae} was seldom demonstrated, and a second, in which this bacillus was demonstrated with great regularity. This fact is not attributable to any alteration in the epidemic itself, but to the application of new methods for the demonstration of the bacillus of influenza.”\textsuperscript{164}

For Fildes and McIntosh, the innovation of so-called selective media, which improved the cultivation of \textit{B. influenzae} and controlled the overgrowth of cultures by other microorganisms, put bacteriologists in a position to establish its primary role. The technique enabled researchers to do three things necessary to meet Koch’s postulates. First, they could regularly identify the bacillus from a large number of cases; second, they could identify or isolate the bacillus in broncho-pneumonia lesions clinically associated with the disease; and finally, they could use pure cultures for inoculation

\textsuperscript{162} Adami, “Influenza,” 413–66.  
\textsuperscript{163} Herbert French, “The Clinical Features of the Influenza Epidemic of 1918–1919,” in Ministry of Health, \textit{Report on the Pandemic of Influenza, 1918–1919} (London: H.M.S.O., 1920), 66–78.  
\textsuperscript{164} McIntosh, \textit{Studies in the Aetiology of Epidemic Influenza}, 33.
experiments aimed at reproducing the disease in experimental animals and humans. Fildes and McIntosh declared:

We have a characteristic living organism which is present in the lesions of influenza and has a pathogenic action for man. Pure cultures of this bacillus are capable of producing in animals a condition which has important points in common with influenza in man, and it is only the indefinite nature of the essential lesions of influenza which causes hesitation in accepting the experimental disease in animals as the same as the natural disease in man. If this relationship is accepted, then the criteria by which a bacterium should be judged the cause of a disease are fulfilled.\(^\text{165}\)

For Fildes and McIntosh, the evidence generated with selective media was enough to counter the “great revolt against the view... that B. influenzae... was the cause of the disease.”\(^\text{166}\)

Many bacteriologists refused to accept the causal relationship and criteria they proposed. Two British pathologists, H. B. Maitland and Gordon Cameron, who scrutinized their claims, argued that the body of evidence from experiments on Pfeiffer’s bacillus did not support Fildes and McIntosh’s conclusions. They noted that rather than demonstrate a causal relationship, inoculation experiments with \(B.\ influenzae\) had generally failed to produce a characteristic lesion in laboratory animals; and while the bacillus was frequently associated with lesions found in postmortem studies of humans, the failure of animal experiments favored “the opinion that \(B.\ influenzae\) is a secondary invader.”\(^\text{167}\)

Critics of the War Office vaccine made much of these discrepancies. For many, the problems surrounding prevention during the pandemic had changed little since influenza’s bacteriology first started to be explored in the 1890s. In a review on the prospects of prevention in January 1919, W. D’este Emery felt no need to change the views he had expressed twelve years earlier. Evidence from the pandemic had only confirmed that pathologists “do not know the cause of influenza. It is hardly necessary to point out,” he noted, “that if the influenza bacillus is not the cause of the disease,

\(^{165}\) Fildes and McIntosh, “The Aetiology of Influenza,” 172.
\(^{166}\) Ibid., 119.
\(^{167}\) H. B. Maitland and Gordon Cameron, “The Aetiology of Epidemic Influenza: A Critical Review,” Can. Med. Assoc. J., 1920, 6, 492.
we can scarcely hope to get good results from the use of a vaccine made from this organism as a prophylactic measure.”

Emery’s observations set the tone for debates among bacteriologists over the prospects of an effective influenza vaccine. The primary issue remained the status of the etiological agent. Supporters of Pfeiffer’s bacillus hoped that an effective vaccine would indirectly secure its etiological link to influenza. But the War Office decision to produce a mixed vaccine rather than the specific Pfeiffer vaccine only added to the doubts about its etiological status. When Leishman and his colleagues formulated the vaccine they were aware of the mounting evidence against *B. influenzae*, and understood that the mixed vaccine was an unsatisfactory solution. But they had limited options. One was to organize research into other possible causative agents. At the time, Fletcher argued that, “on the hypothesis that *B. influenzae*, no less than pneumococci and streptococci, are secondary [infections],” new research was needed on “some as yet undiscovered virus,” and, in November 1918, set in train the first investigations into the possible role of a so-called filter-passing virus. But this was a long-term solution to an immediate problem.

In the short term, a key issue concerned the evidence available to support using the official vaccine. As part of his efforts to rehabilitate the antityphoid vaccine, Leishman had made control trials and systematically collected clinical information necessary to the evaluation process. But implementing similar procedures to test influenza vaccine proved challenging. Leishman noted that, while “clear statistical evidence...should have been easy to collect through the workings of official machinery,” the strains of the epidemic on medical personnel, combined with demobilization, made it very difficult to organize field trials and to accurately collect and record results. In the data he did receive, nearly half of those inoculated had been given only one of two proposed doses; and there was limited information on the interval between inoculation and subsequent immunity or attacks of the disease. Medical statisticians

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168. W. D’este Emery, “Influenza and the Use of Vaccines,” *Practitioner*, 1919, 74, 73.
169. Walter Fletcher to Paul Fildes 22, 28 October 1918, FD1/530, NA; MRC Influenza General Research, 1918, 1 November 1918, FD1/533, NA.
170. Walter Fletcher to Paul Fildes, 28 October 1918, FD1/533, NA.
171. Leishman, “Results of Protective Inoculation,” 367.
who had scrutinized the antityphoid vaccine studies and had used them to develop new criteria for designing valid vaccine trials, were hardly impressed. Major Greenwood, a pioneer of biometrical approaches in medicine, and general editor of the British Ministry of Health’s Report on the Pandemic of Influenza, 1918–19, challenged Leishman’s claims. In the Report, Greenwood noted that the War Office trials were organized under extreme conditions, which “combined to diminish any hopes of a dramatic success in the use of anti-influenza vaccines such as crowned the anti-typhoid campaign.” Lessons about effective trial design were not applied in the War Office trials: vaccinated and control groups were not matched; and since the vaccine was administered during the epidemic, the exposure periods of the vaccinated and unvaccinated groups were not identical. But the greatest shortcoming was the poverty of organized clinical records: “the returns were so incomplete,” noted Greenwood, “evidence of true comparability in the few instances in which a large measure of protection seemed to have been conferred upon the inoculated, so slight and untrustworthy, that we are unable to say that the arguments in favour of prophylactic inoculation as a general measure have been at all strengthened by our experience of the 1918–19 pandemic.”

Questions about the composition of the War Office vaccine and results from field trials took on new meaning as official priorities slowly shifted from military to civilian needs after the end of war. Despite doubts about the efficacy of the vaccine, the LGB wanted it made available to civilian medical services. As early as October 1918, Newsholme discussed with Fletcher how to provide “a large supply of vaccine” for the general population. At the time, however, the LGB’s own laboratory lacked manpower and facilities for large-scale production, and the War Office’s efforts were devoted to the troops. Small amounts were commercially manufactured by Burroughs, Wellcome & Co. and for Parke, Davis & Co. at St. Mary’s, but could not meet the LGB’s objective of supplying

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172. For these criteria, see Eyler, “Fog of Research,” 24–26.
173. Major Greenwood, “The Prophylaxis of Influenza,” in Ministry of Health, Report on the Pandemic of Influenza, 1918–19 (London: H.M.S.O., 1920), 175.
174. Ibid., 175–76.
175. Walter Fletcher to Arthur Newsholme, 23 October 1918, FD1/535, NA.
vaccine to practitioners on demand. Through autumn 1918, an emergency “Influenza Committee,” set-up by the MRC, explored ways to shift military vaccine production to meet civilian needs once demobilization started. Fletcher was especially keen to facilitate this transfer, and worked to expedite the release of skilled pathologists. By the end of December, with assistance from the War Office, the LGB was able to distribute “considerable amounts of prophylactic vaccine” and the MRC coordinated the first official trials in the civilian population. The LGB’s Medical Department also demanded that the proportions of B. influenzae in the official formula be increased, in line with suggestions made by critics. The War Office finally relented, and in April 1919 a vaccine, with significantly more B. influenzae, was prepared for the LGB by the RAMC and supplied to all practitioners, free-of-charge, by the Government Lymph Establishment in London. But it was too late: the pandemic had almost subsided.

CONCLUSION

While the transfer of the War Office vaccine into civilian medicine was not straightforward, it underscores the crucial role of military pathology in shaping general strategies against the pandemic. As I have demonstrated, in Britain, the pandemic was defined and disputed through the specific contexts of pathology organized for war. Historical epidemiologists and modern virologists have demonstrated how war conditions produced an ideal ecology for the pandemic. I have argued that British medical institutions necessary to the war effort shaped understandings and approaches in key ways.

Official strategies were dictated by the imperatives of a system of military medicine that placed pathology at its core. Military pathology was governed by an approach focused on the identification and control of the specific causes of infectious disease. Its importance

176. Walter Fletcher to Arthur Newsholme, 23 October 1918, FD1/535, NA; Anon., “Influenza Prophylaxis Vaccines,” Lancet, 1919, 194, 616.
177. MRC, Walter Fletcher to Paul Fildes, 22 October 1918, FD1/530, NA; Walter Fletcher to George Buchanan, 4 March 1919, FD1/535, NA; Schools Reports on Cases and Treatment of Influenza, 1919, FD1/537, NA.
178. Arthur Newsholme to Walter Fletcher, 3 January 1919, FD1/535, NA.
179. Anon., “Prevention of Influenza,” Br. Med. J., 1919, 1, 19.
180. Anon., “The Prevention of Influenza,” Lancet, 1920, 195, 41; Anon., “Preparations for an Influenza Epidemic,” Lancet, 1920, 195, 271.
and consequences for British responses to the pandemic should not be underestimated. For the first time, large numbers of trained pathologists were able to investigate influenza’s etiology and pathogenesis, and to test methods, criteria, and established concepts of the disease. Because medical officials vested considerable authority in laboratory pathology, the knowledge it yielded underpinned official responses. Bacteriological studies played a key role in framing the summer epidemic and shaping strategies against the deadly autumn wave.

While military pathology provided the context in which official responses were generated in Britain, it did not go unchallenged. The laboratory system on which the War Office, MRC, and RAMC depended produced an avalanche of conflicting evidence and claims about the primary cause of the pandemic. This magnified rather than resolved fundamental problems in existing knowledge and approaches. Difficulties in identifying and agreeing on the specific cause taxed the War Office’s efforts to produce a general vaccine, and its value as a preventative instrument would remain in question. Some critics of bacteriology took these conflicts as indication of the failure of reductive approaches to disease etiology, and called for reappraisals of prevention strategies. Echoing these sentiments, one historian has described British strategies as a “failure of expertise.”

This judgment misses a key point. The War Office and MRC were widely lauded for their efforts. While disputes and uncertainties over influenza’s etiology posed challenges to producing a vaccine, the basic strategy was generally accepted. It is important to recall the exigencies under which pandemic plans were elaborated: the summer epidemic was novel in epidemiological terms and bacteriological evidence seemed to confirm this; officials had only weeks to organize their responses; and their plans were based on contested knowledge and methods.

Retrospective judgments of the failure of laboratory medicine to control influenza not only ignore the state of contemporary scientific knowledge and practices, but also how experts responded to the challenges posed by pandemic. My point here is that, if the

181. Tomkins, “Failure of Expertise.” For contemporary criticisms, see, William Hamer, *The Relationship between Influenza, Cerebrospinal Fever, and Poliomyelitis. Appendix to the Report of the County Medical Officer of Health and School Medical Officer for the Year 1918* (London: County Hall, 1919); William Hamer, “The Influenzal Constitution,” *Proc. Roy. Soc. Med.,* 1927, 20, 1349–68.
pandemic challenged the authority of military pathology, it did not undermine it. Rather, for those directly involved in its organization, the challenges of controlling the pandemic created new opportunities. The MRC, in particular, was especially keen to frame the experience in this way. After the pandemic, Fletcher argued that the rapid organization of coordinated strategies in 1918–19 was testimony to the merits of military pathology. He attributed the inability to manage the pandemic to the general lack of resources for basic pathological research during wartime. With better funds and institutional supports, this form of research organization could be improved and extended into civilian medicine. Fletcher’s vision had considerable support. Within a few years, the MRC was able to generate a consensus in government that modernizing medicine and science, with pathology a focal point of this project, could best solve the problems encountered in controlling influenza. Historians have shown how the MRC built credibility for its aims and authority during the war.\footnote{Sturdy, “War as Experiment.”} The pandemic was also enrolled in this mission, and was used to justify the reorganization of medical science in Britain. Most importantly, the system of military pathology that was mobilized against the pandemic became a key resource for developing new approaches to influenza and other troublesome infectious diseases in peacetime.\footnote{Michael Bresalier, “Uses of a Pandemic: Forging the Identities of Influenza and Virus Research in Interwar Britain,” \textit{Soc. Hist. Med.} (forthcoming); Michael Bresalier, “Neutralizing Flu: ‘Immunological Devices’ and the Making of a Virus Disease,” in \textit{Crafting Immunity: Working History of Clinical Immunology}, ed. Pauline Mazumdar, Kenton Kroker, and Jenniifer Keelan (London: Ashgate, 2008), 107–44.} Forged in the context of total war, and challenged by a devastating pandemic, military pathology emerged in the interwar years as a model for medical scientific organization.

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