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Abstract

The origin site for the 1918 influenza pandemic which killed more than 50 million people worldwide has been hotly debated. While the mid-western United States, France, and China have all been identified as potential candidates by medical researchers, the military context for the pandemic has been all but ignored. Conversely, military historians have paid little attention to a deadly disease which underlines the reciprocal relationship between battlefield and home front. This paper re-examines the debate about the origins and diffusion of the 1918 flu within the context of global war, bridging gaps between social, medical, and military history in the process. A multidisciplinary perspective combined with new research in British and Canadian archives reveals that the 1918 flu most likely emerged first in China in the winter of 1917–18, diffusing across the world as previously isolated populations came into contact with one another on the battlefields of Europe. Ethnocentric fears – both official and popular – facilitated its spread along military pathways that had been carved out across the globe to sustain the war effort on the Western Front.

Keywords

Spanish flu, 1918 influenza pandemic, China, First World War, epidemiology, origins of influenza pandemics

Epidemics typically travel along a society's most important and active lines of communication. The deadliest epidemics in history have been those which followed new paths carved out when previously isolated populations came into sustained contact with one another. Sometimes these paths were essentially economic, with new diseases following

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emergent patterns of trade. In the sixth century the Byzantine Empire was struck by the plague of Justinian, probably transmitted with shipments of grain from North Africa that were being imported to feed the empire's expanding cities.¹ Likewise, the Black Death of the mid-fourteenth century moved across the Mediterranean as Italian traders began plying the waters between the Black Sea and Sicily with greater frequency.² Severe epidemics have also followed military conquest or imperial expansion, moving along the sinews of war and empire. In the sixteenth century the Spanish invasion of the Aztec and Incan empires devastated Aboriginal populations of the 'New World' in several epidemics of smallpox and influenza.³ Asiatic cholera had long been endemic in India before it was exported to the Middle East and then Europe by a British military expedition from the subcontinent to Oman in 1821.⁴

Given the historical link between war and epidemic disease, it is not surprising that at the outbreak of the Great War in 1914 public health officials feared that a global war might bring new diseases home to civilian populations. 'The trail of infected armies leaves a sad tale of sickness amongst the women and children and non-combatants. Laws and regulations may govern the conduct of war, but disease and infections recognise no such laws and refuse to signal [*sic*] out the combatant only,' wrote future Canadian surgeon general Guy Carleton Jones in August 1914. 'Thus we see that war forces itself on the civilian, on the innocent child, on the non-combatant who stays at home ... for who can tell, or count up, or even recognise the victims of war when it once places its hand on a country?'⁵ Four years later, his worst fears were realized.

The 1918 influenza pandemic circled the globe in three waves: the first in the spring of 1918, the second in the autumn, and the third in the winter of 1918–19, extending in some places into 1920. The first wave is said to have caused few deaths but much sickness; it would probably have gone unnoticed in history were it not for the second and more deadly autumn wave between August and December 1918 that caused the majority of deaths. In the first wave it was the armies that suffered most severely. In the autumn and winter waves, soldiers and civilians alike died from secondary pneumonia infections

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- 1 William H. McNeill, *Plagues and Peoples*, rev. edn (Toronto: Anchor, 1998), p. 137.
 - 2 Philip Ziegler, *The Black Death* (New York: Penguin, 1998), pp. 13–17. See also McNeill, *Plagues and Peoples*, pp. 173–9.
 - 3 Alfred W. Crosby, 'Conquistador y Pestilencia: The First New World Pandemic and the Fall of the Great Indian Empires', *Hispanic American Historical Review* XLVII (1967), pp. 321–37. For a more recent synthetic treatment see Jared Diamond, *Guns, Germs, and Steel: The Fates of Human Societies* (New York: W.W. Norton, 1999).
 - 4 R. Pollitzer, *Cholera* (Geneva: World Health Organization, 1959), pp. 19–21. See also Roderick McGrew, *Russia and the Cholera, 1823–1932* (Madison: University of Wisconsin Press, 1965), pp. 17–25.
 - 5 Colonel Guy Carleton Jones, 'The Importance of the Balkan Wars to the Medical Profession of Canada', *Canadian Medical Association Journal* IV (1914), pp. 801–2. On Jones's role in the Canadian Army Medical Corps during the Great War, see J. George Adami, *War Story of the Canadian Army Medical Corps* (Ottawa: Canadian War Records Office, 1918).

which caused people to turn blue from lack of oxygen and cough up purulent, bloody sputum. It was a strange and terrifying epidemic.⁶

Yet researchers have been strangely reluctant to link the dual global catastrophes of war and disease in their analyses. Those studying the social and military history of the Great War have largely ignored the effects of disease on the battlefield or the ways in which mobilizing massive international armies may have facilitated the development of the pandemic.⁷ Those few historians who have chosen to look at the link between war and influenza have concentrated almost exclusively on writing national histories which examine how military-medical infrastructures coped with a major epidemic crisis.⁸ Social historians of disease and medicine have typically chosen to concentrate on national and often local studies which examine flu's effects on specific communities and social groups.⁹ Medical researchers, on the other hand, have been most concerned with establishing the pandemic's

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- 6 The best overview of the pandemic remains Alfred W. Crosby, *America's Forgotten Pandemic: The Influenza of 1918* (Cambridge: Cambridge University Press, 1989). See the following on the flu's virology: Jeffery K. Taubenberger, Ann H. Reid et al., 'Characterization of the 1918 Influenza Virus Hemagglutinin and Neuraminidase Genes', *International Congress Series MCCXIX* (2001), pp. 545–9; Ann H. Reid, Thomas G. Fanning, Johan V. Hultin and Jeffery K. Taubenberger, 'Origins and Evolution of the 1918 "Spanish" Influenza Virus Hemagglutinin Gene', *Proceedings of the National Academy of Science of the United States of America* XCVI (1999), pp. 1651–6; Ann H. Reid et al., '1918 Influenza Pandemic Caused by Highly Conserved Viruses with Two Receptor-Binding Variants', *Emerging Infectious Diseases* IX (2003), pp. 1249–53.
 - 7 The literature on the pandemic is large and growing. Historiographical overviews are provided in Guy Beiner, 'Out in the Cold and Back: New-Found Interest in the Great Flu', *Cultural and Social History* III (2006), pp. 496–505, and Howard Phillips, 'The Re-appearing Shadow of 1918: Trends in the Historiography of the 1918–19 Influenza Pandemic', *Canadian Bulletin of Medical History* XXI (2004), pp. 121–34. See also Howard Phillips and David Killingray, *The Spanish Influenza Pandemic of 1918–1919: New Perspectives* (London: Routledge, 2003).
 - 8 Carol Byerly, *Fever of War: The Influenza Epidemic in the U.S. Army during World War I* (New York: New York University Press, 2005). See also Mark Osborne Humphries, 'The Horror at Home: The Canadian Military and the Great Influenza Pandemic of 1918', *Journal of the Canadian Historical Association* XVI (2005), pp. 235–60, and *The Last Plague: Spanish Influenza and the Politics of Public Health in Canada* (University of Toronto Press, 2013); Eckard Michels, 'Die "Spanische Grippe" 1918/19: Verlauf, Folgen, und Deutungen in Deutschland im Kontext des Ersten Weltkriegs', *Vierteljahrshefte für Zeitgeschichte* I (2010), pp. 1–33; Manfred Vasold, *Die Spanische Grippe: Die Seuche und der Erste Weltkrieg* (Darmstadt: Primus, 2009).
 - 9 For an overview of the literature, see Beiner, 'Out in the Cold', and Phillips, 'Re-appearing Shadow'. Examples include Niall Johnson, *Britain and the 1918–19 Influenza Pandemic: A Dark Epilogue* (London: Praeger, 2005); Esyllt Jones, *Influenza, 1918: Disease, Death, and Struggle in Winnipeg* (Toronto: University of Toronto Press, 2007); Eileen Pettigrew, *Silent Enemy: Canada and the Deadly Flu of 1918* (Regina: Western Producer Prairie, 1983); Dorothy A. Pettit and Janice Bailie, *A Cruel Wind: Pandemic Flu in America, 1918–1920* (Murfreesboro, TN: Timberlane, 2008); Howard Phillips, 'Black October': *The Impact of the Spanish Influenza Epidemic of 1918 on South Africa* (Pretoria: Government Printer, 1990); Geoffrey W. Rice, *Black November: The 1918 Influenza Pandemic in New Zealand*, 2nd edn (Christchurch: Canterbury University Press, 2005).

demographic impact, sequencing the virus's genes, or using the pandemic experience to model future public health responses.¹⁰ The result is a fragmented field in which researchers rarely talk to one another. It also means that histories of the pandemic – a term which refers to a global outbreak of disease – have ironically been localized. The opportunity to examine the pandemic's relationship to a very global war has been missed.

This paper re-examines the 1918 flu's origins and its pattern of diffusion as military events. It seeks to situate the disease within the larger social and military context of the First World War and the massive movements of population which it entailed. In re-evaluating the epidemiological and historical evidence, and building on the work of previous scholars in a variety of fields, it posits that the most likely origin site for this flu was China where, during the winter of 1917–18, a new and deadly virus first appeared and then diffused around the globe with the mobilization of the Chinese Labour Corps (CLC). It criss-crossed the planet in a matter of months, following the sinews of war, moving from China, to North America, to Europe, to Africa, and then back again, killing millions in the process. The flu was thus a global disaster precipitated and best understood as a consequence of the transnational and novel nature of the First World War. Between 1914 and 1918 the centuries-old pattern of the outward movement of populations from Europe to the rest of the world was reversed as millions of colonizers, colonized, and independent non-European actors converged on battlefields and troopships from across the globe. The result was the most deadly epidemic in history.

We now know that influenza is caused by a virus which finds its natural home in wild ducks and only infects humans when it crosses the species barrier, sometimes directly passing from birds to people, sometimes moving through an intermediary species such as pigs or horses.¹¹ When an animal virus becomes a human disease, it has the potential to cause a pandemic because people have no prior immunity; there have been five such episodes in the last 125 years – 1889, 1918, 1957, 1968, and 2009.¹² The 1918 virus was by far the most severe, and researchers are still not sure why. What is clear is that it had a highly unusual combination

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- 10 Kirsty Duncan, *Hunting the 1918 Flu: One Scientist's Search for a Killer Virus* (Toronto: University of Toronto Press, 2003); Gina Kolata, *Flu: The Story of the Great Influenza Pandemic of 1918 and the Search for the Virus that Caused It* (New York: Farrar, Straus and Giroux, 1999); Heather MacDougall, 'Toronto's Health Department in Action: Influenza in 1918 and SARS in 2003', *Journal of the History of Medicine and Allied Sciences* LXII (2007), pp. 56–89; Howard Markel et al., 'Non-Pharmaceutical Interventions Implemented by US Cities during the 1918–1919 Influenza Pandemic', *Journal of the American Medical Association* CCXCVIII (2007), pp. 644–54; S. Zhang, P. Yan, B. Winchester, and J. Wang, 'Transmissibility of the 1918 Pandemic Influenza in Montreal and Winnipeg of Canada', *Influenza and Other Respiratory Viruses* IV (2010), pp. 27–31.
 - 11 For a brief scientific overview, see World Health Organization, 'Fact Sheet: Influenza', www.who.int/mediacentre/factsheets/2003/fs211/en/ [accessed 25 September 2011], and Center for Disease Control (CDC), 'Seasonal Influenza: Flu Basics', www.cdc.gov/flu/about/disease/ [accessed 23 September 2011]. Pettit and Bailie, *Cruel Wind*, provides a good overview of the science behind pandemics in ch. 1, pp. 1–37.
 - 12 W. Graeme Laver, Norbert Bischofberger and Robert G. Webster, 'The Origin and Control of Pandemic Influenza', *Perspectives in Biology and Medicine* XLIII (2000), pp. 173–91.

of genetic characteristics which made it both deadly and easily transmissible.¹³ Most unusually, it also killed those members of society who normally survive the flu with few complications: young, otherwise healthy adults between the ages of 18 and 40. Researchers think this may be because the 1918 virus turned the body's immune system against itself, making it most deadly for those with particularly robust internal defences.¹⁴

While we are learning much about the genetic nature of the virus from samples obtained from frozen corpses and archived medical slides, we know less about where it came from.¹⁵ The search for the genesis of the 1918 flu began soon after the pandemic's second wave subsided in the late autumn of 1918. As the eminent epidemiologist Edwin Oakes Jordan noted in his 1927 study – which remains the most comprehensive epidemiological work on the pandemic – 'its origin is largely shrouded in obscurity'.¹⁶ Although he believed it must have radiated outwards from either a single 'endemic focus' or from several foci, the data and knowledge available in 1927 made it difficult if not impossible to settle upon a definite epicentre.¹⁷ Jordan decided to focus his research on reports of unusually virulent respiratory diseases with similar symptomologies to the influenza seen in the deadly autumn wave but published prior to the pandemic's recognized first wave in April and May 1918. From his readings of the periodical literature, he identified three possible sites: British military camps in Great Britain and France (1916–17), Haskell, Kansas (March 1918), or China (winter 1917–18). In all three cases Jordan concluded that there was evidence that an unusually virulent and deadly respiratory disease of unknown origins was present well in advance of the first recognized wave of flu, but ultimately he decided that the evidence available in 1927 did not allow him to reach a firm conclusion about which site was the primary place of infection. Researchers still debate a European, American, or Asian origin for the virus today.

Beginning in the late 1990s, virologist John Oxford authored a series of articles which built a new case for a European origin site.¹⁸ Like Jordan, his studies point to two specific

13 Pettit and Bailie, *Cruel Wind*, pp. 22–37.

14 Kerri Smith, 'Concern as Revived 1918 Flu Virus Kills Monkeys', *Nature* CDXXXXV, 237 (2007), www.nature.com/nature/journal/v445/n7125/full/445237a.html [accessed 25 September 2011], and Darwyn Kobasa et al., 'Aberrant Innate Immune Response in Lethal Infection of Macaques with the 1918 Influenza Virus', *Nature* CDXXXXV, 319–23 (18 January 2007), www.nature.com/nature/journal/v445/n7125/abs/nature05495.html [accessed 10 September 2011].

15 On the search for the virus, see Kolata, *Flu*.

16 Edwin Oakes Jordan, *Epidemic Influenza: A Survey* (New York: American Medical Association, 1927), p. 60.

17 Ibid.

18 J.S. Oxford et al., 'Early Herald Wave Outbreaks of Influenza in 1916 prior to the Pandemic of 1918', *International Congress Series* MCCXIX (2001), pp. 155–61; J.S. Oxford, 'The So-Called Great Spanish Influenza Pandemic of 1918 May Have Originated in France in 1916', *Philosophical Transactions of the Royal Society B* CCCLVI, 1416 (29 December 2001), pp. 1857–9; J.S. Oxford et al., 'World War I May Have Allowed the Emergence of "Spanish" Influenza', *Lancet: Infectious Diseases* II, 2 (2002), pp. 111–14; J.S. Oxford et al., 'A Hypothesis: The Conjunction of Soldiers, Gas, Pigs, Ducks, Geese, and Horses in Northern France during the Great War Provided the Conditions for the Emergence of the "Spanish" Influenza Pandemic of 1918–1919', *Vaccine* XXIII (2005), pp. 940–5. For a summary of Oxford's earlier work, see Kolata, *Flu*, pp. 289–97.

outbreaks of a respiratory disease elusively called ‘purulent bronchitis’ by the British army’s doctors at two army bases in France and England.¹⁹ The first began in late December 1916, peaking in January and February 1917, at Étaples in northern France. The second occurred in March and April 1917 at Aldershot barracks, south-west of London. Both epidemics were written up in the *Lancet* by the doctors who witnessed them in the spring and autumn of 1917, well before the actual pandemic began. The articles describe soldiers dying with symptoms that resembled those seen in the deadly autumn wave of the 1918 flu: bloody sputum production, tachycardia, a high temperature, and a dusky heliotrope cyanosis which often preceded an unusually frequent pattern of deaths.²⁰

In the view of Oxford and his colleagues the peculiar conditions of trench warfare allowed these local outbreaks to emerge as a new pandemic virus, incubated by a lethal combination of gas, filth, overcrowding, and human cohabitation with livestock, specifically pigs and fowl.²¹ This suggestion is based on the assumption that pandemic viruses might emerge in fits and starts as a new strain begins to slowly cross the species barrier from birds or pigs to human beings.²² If this happened, it would be expected that the virus would initially be extremely lethal, but would also spread inefficiently while it adapted to its human hosts, which would result in short-lived and highly localized outbreaks of disease.²³ Oxford and his co-authors suggest that this is what happened during the 1916–17 outbreaks at Aldershot and Étaples, arguing that these constituted ‘herald waves’, or early instances of a new animal virus crossing the species barrier for the first time,²⁴ part of a seeding process in which a new and virulent form of influenza was planted around the world in the years before the pandemic and exploded from the metaphorical soil into a global pandemic during the autumn of 1918.²⁵

Oxford and colleagues assume that the pandemic’s explosion in the summer and autumn of 1918 can be explained by the massive movements of demobilized armies. They write: ‘demobilization in the autumn of 1918 would have provided an ideal set of circumstances for intimate person-to-person spread and wide dispersion as young soldiers returned home by sea and rail to countries around the entire globe’.²⁶ This, they

19 Oxford et al., ‘Early Herald Wave Outbreaks’, pp. 156–7.

20 Ibid. For the original articles, see J.A.R. Hammond et al., ‘Purulent Bronchitis: A Study of Cases Occurring amongst the British Troops at a Base in France’, *Lancet* II (1917), pp. 41–5, and A. Abrahams et al., ‘Purulent Bronchitis: Its Influenza and Pneumococcal Bacteriology’, *Lancet* II (1917), pp. 377–80.

21 Oxford et al., ‘Hypothesis’, pp. 940–5.

22 See R.B. Belshe, ‘The Origins of Pandemic Influenza: Lessons from the 1918 Virus’, *New England Journal of Medicine* CCCLIII, 21 (24 November 2005), p. 2209, and Ann H. Reid et al., ‘The 1918 Spanish Influenza: Integrating History and Biology’, *Microbes and Infection* III (2001), p. 86.

23 Belshe, ‘The Origins of Pandemic Influenza’, p. 2209; Reid et al., ‘1918 Spanish Influenza’, p. 86.

24 Oxford et al., ‘Early Herald Wave Outbreaks’, pp. 155–61.

25 Ibid., p. 160.

26 Ibid., p. 160.

argue, explains the long interval between the earlier cases and those in the autumn of 1918. While it is an intriguing hypothesis, it also underlines the necessity of situating flu research within a wider historical context. Demobilization cannot account for the diffusion of a fatal pandemic wave which began in late August and peaked in late October and early November, as none of the belligerents demobilized until well after the crisis passed. The German army, for example, was the first to disengage from the Western Front and return within its home borders after the armistice of 11 November, but even its march did not begin until 21 November.²⁷ On the other side, the first British troops were not ordered home until mid-December, while the majority of Canadian soldiers – the largest of the imperial contingents – did not begin to return to the Dominion until the late winter and spring of 1919.²⁸ Likewise, the Americans did not repatriate their army until well into 1919; as late as August of that year, there were still 156,000 US soldiers in Europe – or about 20 per cent of wartime strength.²⁹ The majority of French soldiers remained along the banks of the Rhine until after the Treaty of Versailles was signed on 28 June 1919; indeed, at the end of May, the French were still making preparations for a full-scale invasion of Germany in the event the Germans refused to sign the treaty.³⁰ In sum, the armies of the Great War did not demobilize en masse until well after the pandemic's *third* wave had ended in most locations, meaning that demobilization alone cannot account for the explosion of flu in the autumn of 1918. In the absence of this trigger, it is difficult to establish an unbroken epidemic chain linking the earlier European outbreaks of purulent bronchitis with the 1918 pandemic, despite the extensive and detailed medical records available for most armies throughout the period.

There are viable alternatives to the European hypothesis. In 1927, after discarding the outbreaks at Aldershot and Étaples, Jordan turned to an unusual public health report from Haskell, Kansas, dated March 1918 that linked an outbreak of influenza to deaths from pneumonia. Because the first wave of flu was known to have been rampant in American military camps in April and May 1918, it seemed possible to Jordan that the disease might have first appeared in Haskell and was then transmitted to the army camps as civilian recruits were called to the colours. Once in the ranks of the army, it would have spread quickly from west to east across the United States and then across the Atlantic to

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- 27 James E. Edmonds, *History of the Great War Based on Official Documents: Military Operations, France and Belgium, 1918*, vol. 5: *26th September–11 November 1918: The Advance to Victory* (London: HMSO, 1947), p. 615. See also Holger H. Herwig, *The First World War: Germany and Austria-Hungary, 1914–1918* (New York: St. Martin's, 1997), pp. 446–7.
- 28 Stephen Graubard, 'Military Demobilization in Great Britain Following the First World War', *Journal of Modern History* XIX (1947), p. 301. The majority of Canadian troops did not return home until late in the winter of 1919. See G.W.L. Nicholson, *Canadian Expeditionary Force, 1918–19* (Ottawa: Department of National Defence, 1962), pp. 530–3.
- 29 See E. Jay Howenstine, Jr, 'Demobilization after the First World War', *Quarterly Journal of Economics* LVIII (1943), pp. 95–105, but especially p. 103.
- 30 Margaret MacMillan, *Paris 1919: Six Months That Changed the World* (New York: Random House, 2001), p. 471.

Europe throughout the summer of 1918, perhaps mutating somewhere overseas, before radiating outwards from the continent's west coast in the early autumn with renewed force.³¹

In recent years John Barry has become the most vocal proponent of the American origins theory. In his book *The Great Influenza* and in other articles, Barry builds on Jordan's findings to argue that flu overwhelmed a local doctor in Haskell County, Loring Miner, forcing him to sleep in his buggy between night calls until he eventually became so perplexed by the disease that he filed a report with the United States Public Health Service in late March.³² But this note comprises only single sentence in the 5 April 1918 issue of *Public Health Reports*. It reads: 'On March 30, 1918, the occurrence of 18 cases of influenza of severe type, from which 3 deaths resulted, was reported at Haskell, Kansas.'³³ Using the local gossip columns of the *Santa Fe Monitor*, Barry was able to further elaborate on the significance of this otherwise obscure reference to flu activity, suggesting that these cases had actually occurred in February but were only reported at the end of March before finally being published in April.³⁴ He argues that from this localized outbreak the flu spread to nearby Camp Funston, Kansas, at the beginning of March, then to other army camps across the United States, and later around the world after American troops arrived in Europe.³⁵

At first glance the American hypothesis appears convincing. Like Oxford's theory, it presents an early case of a similar type of disease; the American theory also better fits the general epidemiological pattern while remaining consistent with the historical record and other scientific research. But in order to accept Haskell as the origin site for the pandemic, we must agree that it is the earliest outbreak of the disease evident in the record. At the same time, while Miner's report suggests anecdotally that morbidity and mortality were high, we must also ask whether they were unusual. It is possible to examine both questions using the medical records kept by the US Army which provide the most complete picture of respiratory illness activity in the United States for the immediate pre-pandemic period in 1917 and 1918.³⁶ An examination of the admission rates for the winter and spring of 1917–18 for flu-like illnesses and pneumonia suggests that morbidity and mortality spiked in two waves in December–January 1917–18 and March–April 1918 (see

31 Jordan, *Epidemic Influenza*, pp. 62–76.

32 John M. Barry, *The Great Influenza: The Epic Story of the Deadliest Plague in History* (New York: Penguin, 2005), pp. 92–6. See also John M. Barry, 'The Site of Origin of the 1918 Influenza Pandemic and Its Public Health Implications', *Journal of Translational Medicine* II (2004), www.translational-medicine.com/content/2/1/3 [accessed 13 September 2013].

33 Anon., 'Influenza: Haskell, Kansas', *Public Health Reports* XXXIII (1918), p. 502.

34 Barry, 'Site of Origin'.

35 Ibid.

36 Maj. Gen. M.W. Ireland, *The Medical Department of the United States Army in the World War*, vol. 9: *Communicable and Other Diseases* (Washington: U.S. Government Printing Office, 1928), p. 78. These statistics include admissions for influenza, bronchitis, and pneumonia.

37 Ibid., pp. 83–4. See also George A. Soper, 'Health of the Troops in the United States during 1918', *Military Surgeon* (October 1919), pp. 11–12. This source simply refers to outbreaks of flu throughout the winter and spring but cites many early outbreaks at military camps in March 1918.

Table 1. Respiratory diseases in US armies (influenza, bronchitis, and broncho- and lobular pneumonia), April 1917 – December 1918.

Month	Annual admission rate per 1,000 soldiers		Annual mortality rate per 1,000 soldiers		Case-fatality rate (%)	
	White	African American	White	African American	White	African American
Apr 1917	141.97	96.07	2.16	4.92	1.52	5.12
May 1917	113.07	125.53	1.91	—	1.68	—
Jun 1917	59.53	27.84	0.7	—	1.18	—
Jul 1917	32.74	39.57	0.26	1.8	0.79	4.55
Aug 1917	45.6	40.85	0.17	1.41	0.37	3.45
Sep 1917	58.23	54.85	0.23	2.55	0.40	4.65
Oct 1917	107.58	75.99	0.98	2.75	0.91	3.62
Nov 1917	203.84	167.64	3.81	10.4	1.87	6.20
Dec 1917	312.94	312.28	4.68	22.14	1.50	7.09
Jan 1918	344.58	308.41	5.83	16.1	1.69	5.22
Feb 1918	200.08	226.54	2.48	10.81	1.24	4.77
Mar 1918	363.91	582.16	5.22	26.71	1.43	4.59
Apr 1918	493.2	649.87	4.63	34.16	0.94	5.26
May 1918	167.51	368.7	1.54	16.57	0.92	4.49
Jun 1918	85.94	112.62	0.84	4.98	0.98	4.42
Jul 1918	73.13	145.67	0.93	5.95	1.27	4.08
Aug 1918	83.05	197.79	1.27	10.13	1.53	5.12
Sep 1918	974.36	896.71	54.06	53.86	5.55	6.01
Oct 1918	1978.15	1584.95	95.69	11.66	4.84	0.74
Nov 1918	296.27	172.86	9.74	6.37	3.29	3.69
Dec 1918	266.65	191.51	9.08	6.92	3.41	3.61

Source: Maj. Gen. M.W. Ireland, *The Medical Department of the United States Army in the World War*, vol. 9: *Communicable and Other Diseases* (Washington: U.S. Government Printing Office, 1928), p. 78.
Note that the statistics are broken down by ‘race’ in the original.

Table 1).³⁷ The annual admission rate (which measures morbidity) in December and January was 312.94 and 344.58 per 1,000 white soldiers and 312.28 and 308.41 per 1,000 African American soldiers. Morbidity dropped off significantly in February before moving higher in March and April to 363.91 and 493.2 per 1,000 white soldiers and 582.16 and 649.87 for African American doughboys. The number of admissions to hospital per 1,000 soldiers was also almost three times higher than it had been the previous year, indicating that both the December–January and March–April waves were unusually severe and thus abnormal. But the earlier wave, which occurred almost three months before the Haskell outbreak, was by far the more deadly of the two. While annualized mortality was higher in the spring wave, the case-fatality rate (which measures the virulence of the disease) for white soldiers in December was 1.5 per cent and in January, 1.69 per cent; for

38 Ireland, *Medical Department*, p. 78.

African Americans it was 7.09 and 5.22 per cent respectively.³⁸ In contrast, case-fatality rates were much lower in the March and April wave; in fact, December was an even more deadly month for African American flu victims than the period between August and November 1918, when the majority of recognized pandemic deaths took place.³⁹ This suggests that the Haskell outbreak was not the primary site of the 1918 flu's emergence; perhaps, though, it may have been among the first civilian encounters with the disease.

Although Barry was correct to conclude that flu was first reported in the civilian population at Haskell, it is unlikely that it was the origin site for the 1918 virus given the clear existence of another severe wave three months earlier which was reported across US Army hospitals. Edwin Oakes Jordan identified only one other possible site for the origins of the 1918 flu: China. South East Asia had been linked to the origins of previous pandemics – at least in the minds of their chroniclers, and we now know that new strains of influenza have often originated in Asia.⁴⁰ In 1927 Jordan observed that several contemporary English-speaking writers suggested that the flu had been brought to Europe by the CLC, raised by the French and British in northern China for service on the Western Front. One author described 'a curious, mild, febrile disease reported among Chinese labor troops on the coast of France early in the spring of 1918, about which [he had] never been able to obtain definite clinical or epidemiological information'.⁴¹ Given the limitations of the available sources, language difficulties, and a lack of detailed epidemiological records from the Chinese interior, Jordan concluded that although he could not dismiss the possibility, he was unable to fully test the hypothesis.⁴²

In recent years the Chinese origins theory has gained new support from researchers such as Christopher Langford, Dorothy A. Pettit, and Janice Bailie, who have uncovered evidence of a severe form of respiratory illness, initially diagnosed as pneumonic plague, circulating in the interior of China during the winter of 1917–18.⁴³ They point out that victims died from a severe form of pneumonia, sometimes in a matter of one or two days, suffering from symptoms similar to those seen in the outbreak of influenza in the autumn of 1918. Most unusually for plague, the disease spread sporadically and the case-fatality rate remained low, which casts doubt on the contemporary diagnosis. These researchers argue that it was not in fact plague at all but was actually the earliest outbreak of pandemic influenza, and conclude that the disease then spread outwards from China.⁴⁴ Again they cite the mobilization of the CLC as the main vector in transmitting the flu around the globe.

39 Ibid.

40 See, for example, Robert G. Webster, 'Wet Markets: A Continuing Source of Severe Acute Respiratory Syndrome and Influenza', *Lancet* CCCLXIII (17 January 2004), pp. 234–6.

41 Jordan, *Epidemic Influenza*, p. 75.

42 Barry, 'Site of Origin'.

43 See Christopher Langford, 'Did the 1918–19 Influenza Pandemic Originate in China?', *Population and Demographic Review* XXXI (2005), pp. 473–505, and Pettit and Bailie, *Cruel Wind*, pp. 27–8, 62–7, 231–2.

44 Pettit and Bailie, *Cruel Wind*, pp. 60–6; Langford, 'Did the 1918–19 Influenza Pandemic', pp. 492–3.

The Chinese origins theory has failed to gain acceptance among historians, mainly because of the circumstantial nature of the case and the contemporary identification of the disease as pneumonic plague. Langford, as a result, and Pettit and Bailie have been wisely cautious in their interpretation of a limited body of evidence, mainly drawn from public health reports, newspapers, and colonial office records. As part of a larger study of influenza in the Canadian Expeditionary Force and the pandemic in wartime Canada, new evidence has been uncovered in Canadian and British sources from China, North America, and Europe which builds on the case made by these other researchers to support a Chinese origin for the 1918 pandemic. This evidence, contextualized by a close reading of the military sources, allows us to construct an unbroken epidemiological chain from the interior of China to the battlefields of Europe.

In November 1917 a strange, contagious, and deadly disease of unknown origin began to spread in northern China. In mid-December, reports from Shansi province indicated that it was possibly an outbreak of pneumonic plague.⁴⁵ Pneumonic plague is the most virulent form of the disease caused by the bacillus *Yersinia pestis*.⁴⁶ Unlike bubonic plague, which is spread by bites from the fleas which live on rats and other small rodents, the pneumonic form of the disease spreads by aerosolized droplet infection, from human to human.⁴⁷ This more severe form of the illness, which most likely caused the Black Death of the 1300s, commonly arises as a secondary source of infection during an outbreak of bubonic plague.⁴⁸ If a person who contracts bubonic plague develops a lung infection, the bacteria can be coughed out and spread from human to human.⁴⁹ Plague was suspected in 1917 because the last eruption of that deadly illness in China occurred only a few years before, in 1911, when it killed tens of thousands of people. Prevention efforts by Chinese and Western doctors led to the isolation of the plague bacterium, and by 1917 it could be identified in the laboratory. When word of the new outbreak reached Peking

45 'Plague Raging in Shansi', *North China Herald*, 29 December 1917, p. 768. See also the reports cited by Pettit and Bailie, *Cruel Wind*, p. 62.

46 On the history of pneumonic plague in China, see Mark Gamsa, 'The Epidemic of Pneumonic Plague in Manchuria, 1910–1911', *Past & Present CXC* (2006), pp. 147–83.

47 CDC, 'Facts about Pneumonic Plague', www.bt.cdc.gov/agent/plague/factsheet.asp [accessed 14 July 2008].

48 The biological identity of the Black Death has been disputed by a number of scholars over the years. See David Herlihy, *The Black Death and the Transformation of the West* (Cambridge: Harvard University Press, 2007); Samuel K. Cohn, 'The Black Death: End of a Paradigm', *American Historical Review* CVII (2002), pp. 703–38; Samuel K. Cohn, *The Black Death Transformed: Disease and Culture in Early Renaissance Europe* (London: Edward Arnold, 2002). Recent investigations of DNA from plague victims in London suggest that the bacterium *Yersinia pestis* did indeed cause the Black Death, although it may have been a very different strain of the bacteria from what is commonly found today. See Verna J. Schuenemann et al., 'Targeted Enrichment of Ancient Pathogens Yielding the pPCP1 Plasmid of *Yersinia Pestis* from Victims of the Black Death', *Proceedings of the National Academy of Sciences of the United States of America* CVIII (2011), www.pnas.org/content/108/38/E746 [accessed 13 September 2013].

49 CDC, 'Facts about Pneumonic Plague'.

(Beijing) late that year, British and French doctors of the international legation assembled to confer on the steps they could take to stop the disease from spreading.⁵⁰ Their biggest fear was that it might reach the treaty ports and then be exported to the rest of the world.

To contain the disease the international legation doctors recommended that the Chinese government institute quarantines along the Great Wall and at various railway stations in the interior. It also recommended that the government send Western as well as Chinese doctors to the epicentre to investigate the source of the outbreak, manage containment efforts, and determine the identity of the disease in the laboratory.⁵¹ But the Chinese government was not so quick to respond as the legation doctors would have liked.⁵² Instead of taking the foreigners at their word, the Chinese government was listening to counter-reports from Chinese officials in the interior who said the disease was not pneumonic plague, but merely a severe form of 'winter sickness' endemic to the region.⁵³ European and American doctors believed this to be indicative of 'typical' Chinese backwardness rather than any familiarity or traditional knowledge about the signs and symptoms of a local illness.⁵⁴ In late December the legation sent a team of Western doctors to identify the outbreak, while the Chinese government dispatched Dr Wu Lien-Teh, a European-trained physician who had earned a reputation as China's premier 'plague fighter' during the 1911 outbreak.⁵⁵

The editors of the *North China Herald* applauded the decision to send Wu to investigate a disease whose identity was 'altogether too vague for any opinion to be formed'.⁵⁶ It proved to be a difficult disease to track. Initial reports indicated that it had spread almost 500 kilometres from a single epicentre in six weeks, and that most of those who came in contact with its victims later developed the disease.⁵⁷ But the messages reaching both the legation and the newspapers were confused and based largely on rumour.⁵⁸ Wherever the Western doctors went, they had difficulty finding the scientific evidence

50 'Peking Legation Memorandum', 1 January 1918, FO 228/3115, The National Archives (hereafter cited as TNA), Kew.

51 Ibid.

52 'Peking Legation Memorandum', 4 January 1918, *ibid.*

53 Pettit and Bailie, *Cruel Wind*, p. 64.

54 'Telegram, British Consul at Nanking to John Jordan', 19 March 1918', FO 228/3115, TNA. See also *North China Herald*, 26 January 1918, p. 185.

55 *North China Herald*, 5 January 1918. See also Wu Lien-Teh, *Plague Fighter: The Autobiography of a Modern Chinese Physician* (n.p.: W. Heffer, 1959), and Carsten Flohr, *The Plague Fighter: Wu Lien-Teh and the Beginning of the Chinese Public Health System* (London: Taylor & Francis, 1996).

56 *North China Herald*, 12 January 1918, pp. 49–50; Pettit and Bailie, *Cruel Wind*, p. 63.

57 *North China Herald*, 11 January 1919, p. 59.

58 The papers reported: 'People are dying on the roads and in the inns. Six cases occurred on January 5 at Sopingfu, 600 li North of Taiyuanfu, which city is by now probably affected. This means that the Kinhan Railway, Hankow and the Yangtze are menaced.' 'The Plague in Shansi', *Peking and Tientsin Times*, 12 January 1918, extra issue, p. 1.

59 Pettit and Bailie, *Cruel Wind*, p. 62.

necessary to confirm the identity of the disease.⁵⁹ When Wu Lien-Teh and his team appeared at one village to look for plague victims to autopsy and take samples from, they were mobbed.⁶⁰ Without any laboratory confirmation of the outbreak's cause, the Chinese government remained reluctant to enact quarantines and travel restrictions.⁶¹ In large part this was because those Chinese officials who had witnessed the outbreak denied that it was plague.⁶² The initial symptoms of pneumonic plague include fever, headache, weakness, pneumonia, shortness of breath, and coughing, sometimes producing purulent or bloody sputum, all of which were observed in 1917–18.⁶³ The main epidemiological indicator of plague would have been a massive case-fatality rate. The pneumonic form of the disease kills within a matter of a few hours or days, with its victims dying of severe lung infections and bronchopneumonia.⁶⁴ Almost no one recovers from infection. Chinese officials thus pointed to the mild nature of the disease and a low case-fatality rate as proof that it was merely a severe form of 'winter sickness' rather than pneumonic plague, claiming that there had been no more than 100 deaths in total.⁶⁵

In the end, though, Wu and his team were able to secure samples from several bodies, and on 12 January they announced that they had confirmed the presence of plague bacteria in the laboratory.⁶⁶ But there is good reason to doubt the certainty of this diagnosis.⁶⁷ Internal communications, preserved in the records of the British Foreign Office, between the legation and its doctors confirm that it was difficult for the team to obtain samples from plague victims and then to establish the cause of the disease with any degree of certainty.⁶⁸ Whenever plague appeared to have been confirmed in one location, reports from other sites cast doubt on the diagnosis. Chinese officials remained adamant throughout that the outbreak was minor and probably due to something other than pneumonic plague. In its investigation of plague in Nanking, Western doctors on the Plague Service of the Board of the Interior reported at the end of March 1918:

Based on observations made in the three northern plague provinces, we are surprised that there has been so little plague here, even allowing that some of it has been covered up. It is not three

60 Telegram, 12 January 1918, FO 228/3115, TNA; Pettit and Bailie, *Cruel Wind*, pp. 63–4.

61 'Report on "Plague"', 14–21 January 1918, FO 228/3115, TNA; *North China Herald*, 12 January 1918, pp. 54 and 65.

62 *North China Herald*, 12 January 1918, p. 57.

63 Ibid. 'Report of the Shansi Plague Prevention Bureau, 1918', FO 228/3115, TNA, pp. 2, 31.

64 Gamsa, 'Epidemic of Pneumonic Plague', p. 147.

65 'Report on "Plague"', 14–21 January 1918. British doctors also concluded that the 'mortality is not very great on the whole'. See Telegram 67, 9 February 1918, FO 228/3115, TNA; Pettit and Bailie, *Cruel Wind*, p. 63.

66 *North China Herald*, 12 January 1918, p. 57; Pettit and Bailie, *Cruel Wind*, p. 63.

67 Pettit and Bailie, *Cruel Wind*, pp. 62–5.

68 By 18 March, three months into the investigation, the most certain pronouncement that the team could make was to say that an outbreak in Nanking was 'now practically established to be pneumonic plague' See Telegram, British Consul in Nanking to John Jordan, 19 March 1918, FO 228/3115, TNA.

weeks since the first death, and not over twenty deaths are on record. We are also surprised that the authorities did not earlier avail themselves of the services of the foreign physicians here, freely offered ... [Some] twenty-three deaths were reported and investigated today, but not one of them proved to be from plague. Two children who are on the sick list are suspicious.⁶⁹

Clearly something was killing people in Nanking, causing doctors to investigate dozens of deaths a day, but it was not pneumonic plague.⁷⁰

Chinese officials remained adamant that it was Western fear-mongering and that the plague diagnosis had been erroneous. After causing a riot while attempting to obtain samples for testing from one site, Dr Wu was relieved of his duties at the head of the Chinese investigation, officially because of angina pectoris.⁷¹ Pettit and Bailie believe it more likely that the doctor was fired because he refused to toe the government line.⁷² They argue that Wu's announcement to the press that he had identified the disease may have been designed to force the Chinese government to implement protective measures that would have restricted movement of people and trade. According to later news reports at the time, when two Chinese doctors were asked to independently test Wu's samples for the presence of the plague bacteria, their results were negative for the bacterium *Yersinia pestis*. One of those doctors, Dr Huang, also had significant experience of working with plague bacteria in the laboratory in the 1910–11 outbreak, and he concluded from his study that 'it is doubtful whether [Wu's] cases were suffering from pneumonic plague'. If Wu's diagnosis was correct, it is difficult to understand why the 1917–18 outbreak of pneumonic plague did not receive more attention from plague researchers after the disease subsided. Instead it was soon forgotten.⁷³

This first 1917–18 epidemic in China ended as quickly as it began in the early spring of 1918, before the diagnostic riddle could be solved. However, it reappeared the following autumn with renewed force. In the second week of November the international legation again received reports that an epidemic of pneumonic plague was raging in northern China. The symptoms and epidemiology were identical to those described the previous year, but the mortality was higher: the epidemic was said to be causing 20 to 30 deaths a day in some towns.⁷⁴ Again, the European legation in Peking requested an investigation. A request was made to General Pao, the Tuchun or warlord of Heilongjiang, asking him to look into a severe outbreak of pneumonic plague at Tsitsihar.⁷⁵ 'I at once sent officers from the army medical department in this office to make careful enquiries,' he reported, and

it was found that a kind of very sudden influenza was prevalent in the country round the provincial capital. The epidemic had been very virulent at first and fatal cases had occurred

69 Note from the Plague Service of the Board of the Interior, 29 March 1918, FO 228/3115, TNA.

70 Ibid.

71 Pettit and Bailie, *Cruel Wind*, p. 63.

72 Ibid., pp. 63–4.

73 Ibid., pp. 63–4; quoted from p. 63.

74 Wai Chiao Pu to H.M. Minister, 14 November 1918, FO 228/3115, TNA.

75 Ibid.

among those attacked but lately conditions had improved. The complaint is not pneumonic plague and it is not so grave as reported by the British Legation.⁷⁶

At the top of the report a British official at the legation wrote his conclusions: 'Plague: alleged outbreak was really influenza.'⁷⁷ Nevertheless, Chinese officials had been similarly dismissive the previous year, so the international legation remained determined to make its own diagnosis. But a report from one of its doctors in the interior proved conclusive:

I have the honour to report that there has been an epidemic of Spanish Influenza in this Province similar probably to that which is reported from other parts of China; but the number of deaths has been very great and the sickness would appear to have been more serious in Sinkiang than elsewhere.

He continued:

The sickness was first reported from over the Russian border whence it was brought into Sinkiang by Chinese returning to their homes after a season of poppy cultivation in Russian territory: of these there are said to have been thirty thousand last year. The epidemic spread quickly from Tacheng towards Urumchi. The first cases reported in this neighbourhood occurred at a Mongol camp to the north of the city, and I am told by officials and missionaries that not one of the two hundred Mongols in the camp survived. It then raged in Urumchi and spread thence eastwards via Kucheng to Hami, and also via Turfan along the South Road to Kashgar ... I first heard of the epidemic on my arrival at Hami. It was there spoken of as plague and I thought at first, from the number of deaths which I knew were occurring that it might be an outbreak of plague ... On my arrival at Kucheng the worst seemed to be over but it was said that a great many deaths were still occurring among children: and the sickness was past before I reached Urumchi. The officials have shown their usual apathy ... The epidemic at Urumchi was so great that for a time nearly all the shops were closed and business was at a standstill. The symptoms were described by Chinese as headache, pain in the bones, weakness, and sometimes fever and vomiting ... it was quite a common sight to see carters and others drop down in the streets of Urumchi ... When the epidemic was at its worst, there were seventy to eighty deaths a day at Kucheng. At Hami I believe the death rate was decidedly lower. It was, as usual in cases of this sort, very difficult to get definite information, and many wild rumours were circulating.⁷⁸

This description of the epidemic and its elusive nature is identical to descriptions provided the previous year. It too had seemingly come from Mongolia with travellers from beyond the Great Wall. This time, however, a more conclusive diagnosis could be attached to the disease: Spanish influenza.⁷⁹ In the minds of Western and Chinese officials at the time, the two outbreaks were clearly linked and caused by the same disease.

76 Ibid.

77 Ibid.

78 [Signature illegible], 'Spanish Influenza in Sinkiang: Heavy Death Toll', 7 December 1918, FO 228/3115, TNA.

79 Ibid.

Comprehensive analysis of the available flu data by Christopher Langford and Wataru Iijima reveals that, while China did experience an epidemic, it was much less virulent than in the rest of the world – which is surprising, given the size and density of the Chinese population.⁸⁰ At Shanghai the crude mortality ratio was lower among the Chinese population in 1918 than it was for any other year between 1913 and 1919.⁸¹ According to statistics compiled by Langford, during that period the average mortality for all causes was 14.62 deaths per 1,000 persons.⁸² In 1918 and 1919 the observed mortality was actually significantly lower than was expected: 12.8 and 14.3 per 1,000 persons, 12.4 per cent and 2.2 per cent below the expected average.⁸³ A similar pattern is evident for Hong Kong, where officials recorded that it was an

extremely noteworthy fact that Hong Kong enjoyed an astonishing immunity to influenza during the worldwide pandemic of the closing months of 1918 ... Had the disease prevailed in Hong Kong in similar form and with the same intensity as it did in many parts of British India, the mortality that it would have caused in the crowded parts of Hong Kong City is terrible to contemplate.⁸⁴

Between 1913 and 1917 average mortality was 22.46 deaths per 1,000 persons. In 1918 and 1919 it was slightly elevated to 24.5 and 23.3 deaths per 1,000, increases of only 8.3 per cent and 3.6 per cent respectively. As Christopher Langford has shown, this is consistent with the evidence for other port cities in China.⁸⁵

In comparison, in New York, mortality rose by 24.8 per cent in 1918 from an expected rate of 13.62 per 1,000 persons (1913–17) to 18.1 deaths per 1,000.⁸⁶ In England and Wales the average number of deaths for the eight-year period preceding the pandemic was 14.04 deaths per 1,000 persons. In 1918 it rose 18.8 per cent to 17.3 deaths per 1,000.⁸⁷ In Sri Lanka the death rate rose 17.3 per cent in 1918 from an average of 28.2 per 1,000 persons (1913–17) to 34.1 per 1,000. In Bombay, the average mortality for the period 1910 to 1917 was 32.83 deaths per 1,000 persons. In 1918 those numbers climbed

80 Langford, 'Did the 1918–19 Influenza Pandemic', pp. 478–88. His evidence is detailed in charts and several appendices. See especially the table on p. 478. Wataru Iijima, 'Spanish Influenza in China, 1918–20: A Preliminary Probe', in Howard Phillips and David Killingray, eds, *The Spanish Influenza Pandemic of 1918–19: New Perspectives* (New York: Routledge, 2003), pp. 101–9; Mark Osborne Humphries, 'The Duty of the Nation: Public Health and the Spanish Influenza in Canada, 1918–19', unpublished PhD dissertation, University of Western Ontario, 2008, ch. 2.

81 Langford, 'Did the 1918–19 Influenza Pandemic', p. 478.

82 Ibid.

83 Ibid.

84 F. Norman White, *The Prevalence of Epidemic Disease and Port Health Organization and Procedure in the Far East* (Geneva: Health Committee of the League of Nations, 1923), p. 78.

85 Langford, 'Did the 1918–19 Influenza Pandemic', pp. 480–1; see also White, *Prevalence of Epidemic Disease*, pp. 79, 83.

86 Langford, 'Did the 1918–19 Influenza Pandemic', p. 478.

87 Ibid.

82 per cent to 59.61 deaths per 1,000; in 1919 they were higher still at 113 per cent of expected mortality.⁸⁸

In examining why flu deaths should be so low in Chinese treaty ports, which had a population density comparable to Sri Lanka and Bombay, and much lower than either New York or London, Christopher Langford proposed an intriguing explanation. 'The most obvious explanation is that many people in China had some previous exposure to the virus responsible for the outbreak,' he writes, 'or one closely related to it, and so had a degree of immunity to the disease ... It could well be, then, that the influenza virus responsible for the 1918–19 pandemic originated in China.'⁸⁹ Yet given a lack of evidence to confirm an early outbreak of flu-like disease until 1919, he concludes: 'if so ... it developed there fairly unobtrusively, since no serious outbreak of influenza seems to have occurred in China in the years leading up to 1918–19'.⁹⁰ But had the outbreak which was reported as plague in 1917–18 actually been influenza, this would help to explain why the number of deaths was so low in China in 1918–19. When a population is exposed to a virus, those who survive infection develop a degree of immunity to subsequent outbreaks of the same or similar viruses. If the flu originated in China, diffused around the globe, and then returned the following year in a modified form, this would explain why Langford found that flu mortality did not peak in Shanghai or Hong Kong until February and June of 1919 respectively.⁹¹

While this presents a compelling, albeit circumstantial, case for the misidentification of flu as plague in China during the winter of 1917–18, only DNA testing of samples from these earlier outbreaks can truly confirm or deny the theory. But if flu did originate in China during the autumn and winter of 1917–18, then the available evidence must also prove that it travelled quickly and undetected to Europe while at the same time accounting for the known spring outbreaks in North America in March and April 1918.⁹² According to Langford and Pettit and Bailie, the most likely vector was the mobilization and deployment of the CLC from the British treaty port of Weihaiwei.⁹³ Port Edward, as it was also known, was leased by the British government in 1898 as a countermeasure to the Russian acquisition of Port Arthur on the opposite side of the Gulf of Chihli.⁹⁴ The territory was located on the eastern coast of the Shandong peninsula and included both the island fortress-city and a small territory on the mainland. As the Chinese government was concerned about remaining neutral in the eyes of the Central Powers, recruiting took place in the interior of northern China under the direction of local Chinese

88 The remaining statistics are derived from Langford, 'Did the 1918–19 Influenza Pandemic', and White, *Prevalence of Epidemic Disease*.

89 Langford, 'Did the 1918–19 Influenza Pandemic', p. 491.

90 Ibid.

91 Ibid., p. 476.

92 Langford presents a good overview of the questions which must be answered to make this case. See *ibid.*, pp. 490–5.

93 Ibid., pp. 488–90; Pettit and Bailie, *Cruel Wind*, pp. 65–7.

94 Pamela Atwell, *British Mandarins and Chinese Reformers: The British Administration of Weihaiwei (1898–1930) and the Territory's Return to Chinese Rule* (New York: Oxford University Press, 1985), pp. 1–31.

95 Telegram No. 280, 'China, Military', Sir J. Jordan to Foreign Office, 26 September 1918, WO 106/33, TNA.

agents hired on contract.⁹⁵ The recruits were then funnelled to Weihaiwei and into British leased territory, where they were inducted into the CLC.⁹⁶ In the winter of 1917 the number of Chinese labourers in barracks at Weihaiwei and at the various depots on the mainland swelled in preparation for their movement to Europe.⁹⁷ The problem was that not enough transports had been secured to ship the more than 20,000 labourers a month who were arriving at the island.⁹⁸ The crowded conditions provided a perfect breeding ground for disease as Chinese workers from many different parts of the interior of northern China were suddenly thrown together in hastily arranged quarters on the coast.

During 1917–18, 94,000 Chinese workers were shipped by the British from China.⁹⁹ Initially they were transported from Weihaiwei to Europe via Singapore, Durban, and Cape Town, or through the Mediterranean via the Suez Canal. Both routes tied up vitally important troop transports and surface escorts which were most needed in the Atlantic.¹⁰⁰ The fastest and safest way to Europe was instead to move the CLC via Vancouver, across Canada to Halifax, and then on to France.¹⁰¹ In the spring of 1917 the Foreign Office requested that Ottawa allow the Chinese labourers to pass through the Dominion of Canada and that the government of Robert Borden arrange for their transportation. The Canadian prime minister agreed, but it was not a simple matter.¹⁰² Chinese immigration was a sensitive subject in Canada, and there were nativist fears that the labourers might try to escape while travelling across country.¹⁰³ To ensure that they did not do so, special Railway Service Guards from the army were placed on the trains, and guards were also stationed at the camps encased in barbed wire fences.¹⁰⁴ Newspapers were banned from reporting on the movement of the CLC and the whole enterprise was kept secret.¹⁰⁵

96 Telegram No. 131, 'China, Military', Sir J. Jordan to Foreign Office, 30 September 1918, WO 32/11345, TNA.

97 Telegram No. 76, 'China, Military', Mr. Alston to Foreign Office, 19 February 1917, WO 106/33, TNA.

98 Telegram No. 77, 'China, Military', Mr. Alston to Foreign Office, 19 February 1917, WO 106/33, TNA.

99 Langford, 'Did the 1918–19 Influenza Pandemic', pp. 489–90.

100 Telegram No. 553, SNO Hong Kong to Admiralty, 8 February 1917, MT 23/818, TNA.

101 Telegram No. 510, Admiralty to Commodore Hong Kong, 24 February 1917, MT 23/818, TNA; Telegram No. 627, Admiralty to Commodore Hong Kong, 3 March 1917, MT 23/818, TNA; Pettit and Bailie, *Cruel Wind*, pp. 66–7.

102 Colonial Office Memorandum, T22497, 10 March 1917, MT 23/818, TNA.

103 See Patricia E. Roy, *A White Man's Province: British Columbia Politicians and Chinese and Japanese Immigrants, 1858–1914* (Vancouver: UBC Press, 1989); idem, *The Oriental Question: Consolidating a White Man's Province, 1914–1941* (Vancouver: UBC Press, 2003); idem, 'British Columbia's Fear of Asians', *Histoire Sociale / Social History* XIII (1980), pp. 161–72. See also Peter S. Li, *The Chinese in Canada* (Toronto: Oxford University Press, 1988).

104 E.P., 'Chinese Coolies: Great War', 30 April 1940, GAQ 8–36, vol. 1833, RG 24, Library and Archives Canada (hereafter LAC), Ottawa.

105 See various documents in the files of the Chief Press Censor, file 331, vol. 620, RG 6, LAC. See also Elizabeth A. Tancock, 'Secret Trains Across Canada, 1917–1918', *Beaver* (October–November 1991), pp. 39–43.

At Christmas, stories began to appear in the Canadian papers about a strange new epidemic in China. 'Epidemic of Pneumonia in Chinese Province,' read the *Toronto Globe's* headline of 5 January 1918. 'The epidemic of pneumonia which broke out about a week ago along the Shansi–Mongolia border has reached Fengchengting, Province of Shansi, 160 miles northwest of Pekin [*sic*], and is causing alarm among the foreigners there.'¹⁰⁶ On the 17th the paper reported that in China '[people are] dying by scores from pneumonia'.¹⁰⁷ At the end of the weekend, readers found that the epidemic had worsened yet again.¹⁰⁸ Censorship regulations meant that the *Globe* was unable to report that during the outbreak alone, 25,000 Chinese workers were being transported to Europe via Canada, many coming from the plague-affected areas in China.¹⁰⁹ British officials at Weihaiwei wondered whether it was advisable to continue transporting the labour corps under such dangerous circumstances. 'I need hardly point out that if cases of plague were to break out in the coolie depot, the consequences might be grave in the extreme,' wrote the governor of Weihaiwei to T.J. Bourne, the head British recruiter at Weihaiwei, in mid-January; 'perhaps the ... Canadian government would also object to Chinese emigrants from plague-infected areas passing through their ports or travelling on their railways'.¹¹⁰ Nothing was said about the potential health effects for the workers, and transportation continued all the same.

Officials' worst fears were confirmed when plague did in fact break out among the Chinese workers waiting to embark for Canada at Weihaiwei. On 1 March 1918 a recruiting officer at the island city sent a telegram to the director of transports and shipping in London which read: '1,600 coolies are ready to embark at Chengyang in *Conconada*. Recruiting there is stopped by plague. At Weihaiwei there are 2,480 coolies not yet free from infection and probably ready to embark in about six weeks time.'¹¹¹ Despite the presence of plague, the *Conconada* loaded 1,899 members of the labour corps the next day, and set sail for Vancouver.¹¹² The ship arrived at William Head on 23 March, with one death from pneumonia having occurred at sea.¹¹³ Soon after they arrived, it was clear that the workers would not be healthy enough to send on to Europe for some time. By early April, of the 3,660 Chinese labourers then housed at William Head, as many as 300, or roughly 8 per cent, were under medical

106 'Epidemic of Pneumonia in Chinese Province', *Globe*, 5 January 1918, p. 7.

107 'Dying by Scores from Pneumonia', *Globe*, 17 January 1918, p. 1.

108 'Pneumonia Plague Sweeping China', *Globe*, 20 January 1918, p. 9.

109 WO 32/11345, TNA; see also Pettit and Bailie, *Cruel Wind*, pp. 65–7.

110 R.F.J., Office of the Adjutant General to T.J. Bourne, War Office Recruiter, 14 January 1918, FO 228/3115, TNA.

111 Telegram P9151-SR1, Unnamed sender to Director of Transports and Shipping, 1 March 1918, MT 25/13, TNA.

112 'Statement Showing Movement of Vessels Conveying Chinese Workers to France and Numbers Reported on Each Vessel', WO 32/11345, TNA.

113 Telegram, Canadian General Staff to London, 25 March 1918, file c2276, vol. 2554, RG 24, LAC. The cause of death was provided to me by Gregory James, Hong Kong University of Science and Technology, on the basis of his detailed research for *Foch's Reserves: The Chinese Labour Corps, 1917–1921* (Hong Kong: Proverse, 2013).

treatment.¹¹⁴ In April, Canadian Surgeon General Guy Carleton Jones sent an order to the commander of Military District 11 (Victoria, British Columbia) asking him to ensure that precautions be taken to guarantee that, when the remaining members of the CLC were ordered overseas, no contagious diseases be allowed to come in contact with the civilian population.¹¹⁵ The disease which kept more than 3,000 Chinese labourers in quarantine is not described in the surviving records, but, according to the research of Gregory James, at least one of those quarantined from the *Conconada* died of pneumonia.¹¹⁶ We also know that, not long after the surgeon general's request, London decided to cancel the CLC programme because of the danger posed by plague in China.¹¹⁷ The British government, anxious to end its commitments to those labourers already in Canada, decided to transfer the remaining workers, healthy or not, from Vancouver to New York and on to Europe.¹¹⁸

While medical inspections of embarking CLC labourers were carried out at Weihaiwei, they were not taken seriously. The European military doctors who inspected recruits were most concerned about looking for what they regarded as 'Asian' diseases like trachoma.¹¹⁹ Some doctors boasted of having examined 3,000 'patients' in a single day – an impossibility but an exaggeration that speaks to the cursory nature of the medical inspections carried out on the docks in China.¹²⁰ At the same time Canadian physicians tended to downplay or ignore common 'European' diseases among the labourers, instead emphasizing the 'inferior' and 'lazy' nature of their patients when they came down with colds and sore throats. For example, H.D. Livingstone, a young Canadian Army Medical Corps doctor from Listowel, Ontario, who was sent to oversee the passage of labourers from Weihaiwei in the autumn of 1917, described how he and other European doctors dealt with common complaints such as sore throats and persistent coughs which plagued the CLC contingents on their voyages:

A coolie would come in and we would say to him 'what sickness have you?' He would reply (taking the most common complaint) 'throat sore' – how long have you had it? He would usually exaggerate and say 20 days or 15 days or some such big number to try and impress on us how sick he really was. Then after depressing the tongue and examining the throat I'd give him some Pat. Cholr. Tablets at the same time saying 'Take two every two hours and suck them' then perhaps if he complained very much we would force down two ounces of castor oil and

114 Internal Memorandum for Naval Secretary, Interdepartmental Committee, 30 March 1918, file c2276, vol. 2554, RG 24, LAC.

115 Surgeon General Jones to GOC MD 11, 2 April 1918, file c2276, vol. 2554, RG 24, LAC.

116 Personal communication with Gregory James, based on his research for *Foch's Reserves*.

117 See MT 25/13, TNA.

118 The Military Secretary for the Interdepartmental Committee to the Naval Secretary, Interdepartmental Committee, 9 May 1918, file 48-45-2 part 12, vol. 3738, RG 24, LAC.

119 See Anon., 'The Necessity of a Recognition and Isolation of Trachomatous Patients in Canada', *Canadian Practitioner and Review* XXVI (1901), pp. 508–9.

120 H.D. Livingstone, 'Chinese Labour Corps' Medical Officer's Journal, 5th Contingent.' The handwritten original is in the possession of Mr David Livingstone of Waterloo, Ontario, who kindly allowed me to quote from his father's papers.

then practically shoved him out of the room saying at the same time ‘come back tomorrow if no better’ but the thought of the castor oil usually kept them away.¹²¹

The Canadian civilian quarantine service took little interest in the Chinese labourers, despite long-standing fears which linked disease and immigration on Canada’s west coast. In the case of the CLC, they were understood to be transients moving across the country to a further destination. As long as the military took precautions to isolate them from the general Canadian population and ensure that they did not ‘escape’ while in transit, quarantine officials were satisfied.¹²²

In Canada the medical records of those soldiers detailed to guard Chinese labourers – the Railway Service Guards – provide evidence of increased respiratory disease activity in the winter of 1917–18. Because the viruses and bacteria that affect the human respiratory system are always in circulation, when examining hospital or health records for any period one would expect to find evidence of disease activity. But large, sustained spikes in the number of admissions for respiratory complaints over several days or weeks would signify the presence of a disease-causing agent within the population – although it is impossible to determine from the existing records whether such activity was the result of a normal seasonal flu or a new type of virus. An examination of the unit’s daily orders nevertheless shows that in the winter of 1917–18 there were 32 cases of influenza reported among Canadian soldiers assigned to the Chinese workers between November 1917 and April 1918 (Figure 1).¹²³

As the members of the CLC arrived in France from southern England, they were sent to the ‘Coolie Camp’ outside Étaples, near Noyelles-sur-Mer – close to the base where Oxford noted the outbreak of purulent bronchitis.¹²⁴ There, Number 3 Native Labour General Hospital, or the Chinese Hospital as it was also known, served the needs of the CLC in France and Belgium. The hospital’s commanding officer, Colonel Gray, typified the attitudes displayed by the British War Office towards Chinese labourers during the war: he was far more interested in the temperature of his stove than in the hundreds of ill Chinese workers who filled his hospital. As Gray recorded the hospital’s official daily war diary in the winter of 1917–18, he usually took care to note the exact heat at which his stove burned. It seems he was trying to invent a more efficient fuel mixture, as he also recorded how various alterations to the coke would affect the longevity of the fire and its temperature. He tried soaking the coke in oil and letting it dry. He experimented with adding a number of chemicals to the marinade and putting it into the stove when it was

121 ‘Livingstone Memoir’, pp. 14–15. This is a typed memoir, a copy of which is in possession of the author while the original remains with Mr David Livingstone of Waterloo, Ontario. I would like to thank Mr Livingstone for allowing me to consult the memoir and photograph it in the summer of 2008. According to Pettit and Bailie, sore throats were rampant in the camps: see *Cruel Wind*, pp. 65–7.

122 See E.P., ‘Chinese Coolies: Great War’, and Surgeon General Jones to GOC MD 11, 2 April 1918, file c2276, vol. 2554, RG 24, LAC.

123 Compiled from daily orders in ‘Military District 11 – No. 43 – Railway Service Guard’, vol. 1447, series II-F-9, RG 9, LAC.

124 ‘Livingstone Memoir’, pp. 22–3.

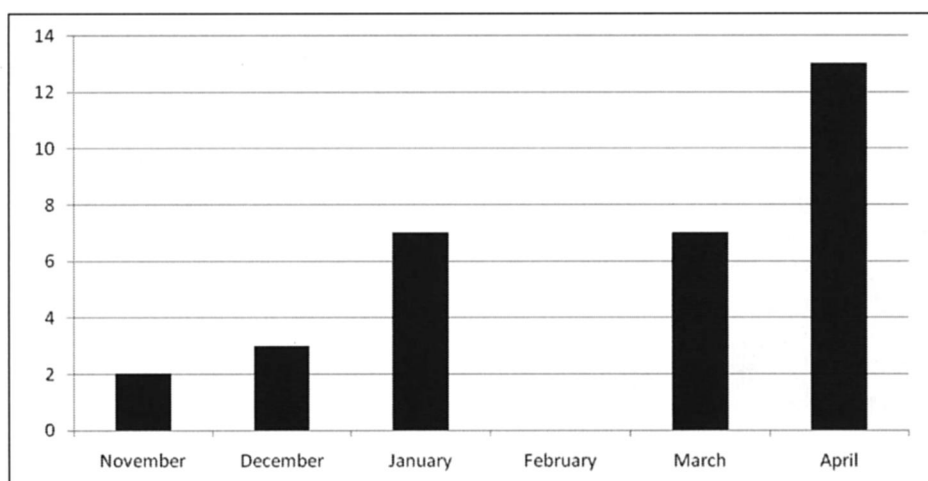


Figure 1. Railways Service Guard admissions for influenza-like illness, winter 1917–18.

Source: Daily orders in 'Military District 11 – No. 43 – Railway Service Guard', vol. 1447, series II-F-9, RG 9, Library and Archives Canada, Ottawa.

still wet. He even tried adding additional fuels. But while Gray studied his stove, a remarkable number of his patients at the hospital were dying from severe pneumonia infections – providing the first evidence to support Jordan's 1927 report of a strange respiratory infection among Chinese workers in France.¹²⁵

In February 1918 there were 9 deaths in the Étaples hospital, recorded as being from either pneumonia or tuberculosis, with 1,360 admissions during the month, mostly for trachoma. In March the hospital reported 14 deaths from lung conditions, including 3 deaths from 'acute bronchitis' within 4 days, from a total of 967 admissions. In April there were 5 deaths from various lung conditions. In May there were a total of 25 deaths from respiratory diseases, including 4 from pneumonia and acute bronchitis on 1 May and 5 from what was assumed to be tuberculosis on 5 May. On 2 May the war diary noted that an epidemic of influenza had begun.

The respiratory epidemic was preceded on 14 January 1918 by the death of the hospital's two pigs.¹²⁶ Unlike the many Chinese patients who died in hospital, an autopsy was performed on the second pig to determine its cause of death. The pathologist's report suggested that the animal had died of a generic 'swine fever', which in 1918 simply meant that it died of an unknown disease. While today swine fever is used a synonym for hog cholera, in 1918 that disease was indistinguishable from a host of other illnesses, including swine flu.¹²⁷ While it is impossible to establish what the pigs died of, influenza

125 See various entries in War Diary, Number 3 Native Labour General Hospital, WO 95/4115, TNA.

126 Ibid., 14 January 1918. Pettit and Bailie also note animal co-morbidity in China: see *Cruel Wind*, pp. 65–7.

127 On the diagnosis of swine diseases before the identification of influenza, see W.W. Dimock, 'Differential Diagnosis of Diseases of Swine', *Journal of the American Veterinary Medical Association* LIV (1919), pp. 321–30, and J.S. Koen, 'A Practical Method for Field Diagnosis of Swine Disease', *American Journal of Veterinarian Medicine* XIV (1919), pp. 468–70.

is one of the few diseases which can kill both humans and pigs. While Oxford and Barry cite human and animal cohabitation in Europe and in Kansas as evidence that a disease had the opportunity to cross the species barrier, here we find unusual evidence that both humans and pigs were dying of epidemic disease at the same time and in the same place.¹²⁸

It would seem that these same respiratory diseases were circulating among Chinese labourers elsewhere in Europe around the same time. The records of the majority of CLC units do not appear to have survived in the archives, but references to the CLC in the files of other units are informative. The first reports of influenza in Canadian military records, for example, actually refer directly to members of the CLC. On 13 April 1918, at Number 9 Canadian General Hospital in Shorncliffe, England, 'one Chinese patient [was] admitted in [a] dying condition from [the] Labour Camp'.¹²⁹ The 'Labour Camp', also known as the 'Chinese Camp', provided temporary housing for Chinese workers as they made their way to France en route from Canada.¹³⁰ A few days later, on 25 April, the war diary recorded that 'several Chinamen [*sic*] [were in a] special ward sick with Purulent Bronchitis'.¹³¹ The following day the number of cases increased and a second ward had to be opened for the labourers. According to the Assistant Director of Medical Services, Shorncliffe, the cases of purulent bronchitis seem to have first appeared among the members of the CLC, as it was not then circulating among Canadian troops in the area.¹³²

Meanwhile, Chinese labourers continued to die at Noyelles-sur-Mer – some diagnosed with tuberculosis, others with respiratory infections. In June there were 4 deaths: 3 from bronchitis and 1 from pneumonia. In July there were a total of 16 deaths: although 11 had no cause of death specified in the war diary, 3 deaths were from bronchitis, 1 was from pneumonia, and, most significantly, 1 was from influenza. In August there were another 3 deaths, one again being listed as due to acute bronchitis. Between January and the beginning of September there were a total of 64 deaths from respiratory diseases (including tuberculosis) when typically the hospital only admitted between 500 and 1,000 patients per month, most of whom were treated for trachoma. The deaths stopped at Noyelles just as the severe wave of the influenza pandemic began.¹³³

In September, when the fatal wave of flu was beginning to decimate populations around the world, there were no deaths reported in the Chinese Hospital as being due to respiratory illness of any kind – the first month this had happened since December 1917. In October, as the flu was sweeping across Canada, killing thousands in Montreal and Toronto, there was one death reported from pneumonia even though the

128 Oxford et al., 'Hypothesis', pp. 940–5; Barry, *Great Influenza*, pp. 91–2.

129 War Diary, Number 9 Canadian General Hospital, Shorncliffe, 13 April 1918, file 858, vol. 5036, series III-D-3, RG 9, LAC.

130 'Livingstone Memoir', p. 22.

131 War Diary, Number 9 Canadian General Hospital, Shorncliffe, 25 April 1918, file 858, vol. 5036, series III-D-3, RG 9, LAC.

132 'Status of Infectious Diseases', War Diary, Assistant Director of Medical Services, Shorncliffe, 17, 30 April 1918, *ibid*.

133 See various entries in War Diary, Number 3 Native Labour General Hospital, February–August 1918, WO 95/4115, TNA.

hospital continued to admit a typical 596 patients. In November, 661 patients were admitted to hospital and there were a total of 11 deaths, none of which were recorded as being due to influenza, pneumonia, or another respiratory illness. The December war diary is missing, but even if the epidemic did suddenly erupt in the hospital, it did not rage into January 1919 as there were only 5 deaths from flu, pneumonia, or bronchitis, and a total of 36 deaths from all causes. A possible explanation is that members of the CLC – or significant portions of the workers – had acquired immunity to a similar virus at some point before the autumn of 1918.¹³⁴ If so, they may even have spread it to the neighbouring British camp at Étaples.

This pattern of diffusion is supported by an analysis of how the second known wave of flu spread outwards from an epicentre on the English Channel. Alfred Crosby argues that the original virus mutated in midsummer 1918, appearing almost simultaneously in France, the United States, and Africa at the end of August, and exploded from those three locations around the world.¹³⁵ He writes:

In the latter part of August 1918, the Spanish influenza virus mutated, and epidemics of unprecedented virulence exploded in the same week in three port cities thousands of miles apart: Freetown, Sierra Leone; Brest, France; and Boston, Massachusetts. Whether these explosions were three manifestations of a single mutation of the virus which originated in one of the three ports and almost simultaneously traveled to the other two or were three different simultaneous mutations will never be known. All we can say is that the first hypothesis is improbable and the second extremely improbable. Perhaps the truth is something entirely different.¹³⁶

We can now say that the second wave of influenza spread out from southern England, specifically the ports of Plymouth and Devonport. Plymouth was a land and sea transportation hub, connected by railway to Shorncliffe and the nearby port facilities at Folkestone. Chinese labourers had frequently been offloaded at Plymouth and shipped by rail to Kent, and the port's logbooks record the arrival and departure of dozens of units from the interior of England.¹³⁷ Just as the death rate was beginning to subside in Noyelles-sur-Mer, on 26 July the admiral in charge of Devonport issued general order 653, which read:

Epidemic of Influenza: Surgeon-General Sir Humphry D. Rolleston and Fleet-Surgeon Robert W.G. Stewart arrived at the Port on 25th July, and will remain for such time as may be necessary, to investigate cases of serious illness arising from influenza. Every facility is to be afforded these officers in their investigations.¹³⁸

134 See *ibid.*, various entries, September 1918 – April 1919.

135 Crosby, *America's Forgotten Pandemic*, pp. 37–40.

136 *Ibid.*, p. 37.

137 See the log of Coolie Transportation in WO 32/11345, TNA, various correspondence in MT 23/818, TNA, and the log of Plymouth Harbour in ADM 53/48308, TNA.

138 'Devonport General Order 653', 26 July 1918, ADM 130/27, TNA.

As Rolleston and Stewart made their way to Plymouth to investigate reports of a new and more deadly iteration of the disease, HMS *Mantua* set sail for Freetown, Sierra Leone.¹³⁹

The *Mantua* was a 10,000-ton ship built in 1909 to service the Australian run by the Peninsular and Oriental Steam Navigation Company. With room for 600 passengers in peacetime, it was refitted as an armed merchant cruiser for service during the Great War. Before sailing from port, it took on 10,000 pounds of fresh meat and 365 tons of fresh water.¹⁴⁰ On 1 August the *Mantua* set sail from Plymouth Harbour (across the bay from Devonport) for the British West African colony. On 8 August an epidemic recorded as being due to 'influenza' began on board. On the 10th there were 25 sick; by the 12th, 74; and on the 13th, 103. When the ship made port in Freetown on 14 August, there were 124 sick aboard. The epidemic did not peak, however, until the 18th, when 176 sailors were reported as sick in hospital or their bunks. Over the next two weeks ten crewmen died of pneumonia following influenza. We know this new and more deadly strain of flu was taken on board in southern England because the ship remained isolated from the time it set sail on 1 August until it made landfall on the 14th. According to its log, the *Mantua* took on no water, supplies, or crew during its two weeks at sea. Once it arrived at Freetown, the virus quickly spread to the mainland.¹⁴¹ From Freetown it diffused into the interior and down the coast of Africa. Ships that called in for coal at the British colony brought influenza to Cape Town in South Africa as well as the Gold Coast.¹⁴² New Zealand soldiers on their way to France also caught it there.¹⁴³ This means that the mutated fatal strain of the virus must have come aboard at Plymouth just before 1 August.¹⁴⁴

Plymouth harbour is located directly across the Channel from Brest, France, from where numerous transports carrying British and American troops plied the waters between the two ports during the summer of 1918.¹⁴⁵ As the *Mantua* was carrying influenza from England to Africa, American ships had been making similar voyages back across the Atlantic. One of those ships probably arrived in Boston at about the same time as the *Mantua* made landfall in Africa.¹⁴⁶ On 27 August the first cases of the severe autumn wave of influenza were reported among American soldiers stationed at the city's Commonwealth Pier.¹⁴⁷ From there it began a rapid expansion outwards from Boston's waterfront. Soon it was spreading through the city and into the neighbouring army

139 Ship's Log, HMS *Mantua*, 31 July 1918, ADM 53/48308, TNA.

140 Ibid.

141 Sandra M. Tomkins, 'Colonial Administration in British Africa during the Influenza Epidemic of 1918–19', *Canadian Journal of African Studies* XXVIII (1994), pp. 68–70.

142 Phillips, *Black October*, p. 8.

143 Rice, *Black November*, p. 58.

144 Ship's Log, HMS *Mantua*, ADM 53/67788, TNA. This is a significant finding, in part because previous historians have argued that there were no deaths from influenza on board the ship and thus the illness that it was carrying must have been of the earlier, more mild type, rather than the fatal autumn strain. See Crosby, *America's Forgotten Pandemic*, p. 37.

145 See the log of Plymouth Harbour for July and August 1918, ADM 53/48308, TNA.

146 Barry, *Great Influenza*, pp. 192–3.

147 Crosby, *America's Forgotten Pandemic*, p. 39.

camps. By the second week of September, Spanish flu had already spread across Massachusetts and upper New York State.¹⁴⁸ In that week local newspapers across the north-east reported that the disease was widespread. By the 14th, it was raging in Newport News and Philadelphia.¹⁴⁹ By then there were thousands of cases among Camp Devon's soldiers outside Boston.¹⁵⁰ Over the next few months, 675,000 Americans died of influenza or pneumonia.

While there are several theories which explain the origins of the 1918 influenza pandemic, the Chinese hypothesis makes the most convincing case and is supported by the strongest epidemiological and historical evidence. To be sure, the evidence remains circumstantial, but it can be nothing else in the absence of viable DNA evidence. Nevertheless, the Chinese origins theory connects many of the loose ends left by the European and American theories, while also linking history with our modern understanding of flu virology. Thanks to the work of Langford and of Pettit and Bailie, we know that in late 1917 a virulent respiratory disease of unknown origins with symptoms approximating both pneumonic plague and Spanish influenza erupted in the interior of northern China. The disease spread rapidly through terrain marked by few roads – 500 km in 43 days.¹⁵¹ While Foreign Office records show that Western officials on the ground assumed it to be pneumonic plague because of previous outbreaks of that disease (not in that region but in Manchuria), local Chinese officials denied that this was the case. The Western investigators dispatched to identify the microbes had difficulty obtaining samples of the plague bacteria, even from those who had died during the outbreak. Suddenly the outbreak ended in late spring, only to reappear the following autumn. When it did, internal correspondence shows that both Chinese and Western officials identified the disease as Spanish influenza rather than pneumonic plague – and they both linked it to the previous year's outbreak. When flu reappeared in China the following year, it killed far fewer there than elsewhere in the world, indicating the possibility that a previous epidemic had provided some form of survivor immunity.

At the same time the war gave an opportunity for a localized outbreak of disease to spread beyond China. The mobilization of the Chinese Labour Corps moved tens of thousands of individuals from the region where the outbreak occurred, across North America to Europe. We know that the plague-like illness in China did appear among Chinese workers bound for Vancouver, while, at the same time, hundreds of labourers were put into quarantine for an unspecified disease in British Columbia during the winter of 1917–18. Finally we have the Chinese Hospital at Noyelles-sur-Mer in France which confirms the existence of a strange and deadly respiratory disease among Chinese workers in Europe – providing records that were unavailable to Edwin Jordan when he noted this same 'curious' respiratory disease in 1927.¹⁵² While the plague-like disease spread

148 Ibid., pp. 197–9.

149 'Influenza Is Now Spreading', *Marion Daily Star* (Ohio), 14 September 1918, p. 9.

150 '2000 Cases of Influenza Found among Soldiers', *Syracuse Herald* (New York), 15 September 1918, p. 11.

151 Pettit and Bailie, *Cruel Wind*, pp. 66–7.

152 Jordan, *Epidemic Influenza*, p. 75.

across China, humans and pigs died at the hospital. The final link in the epidemic chain was the diffusion of the more virulent mutation of the disease from the Channel coast in the summer of 1918 which began just as deaths among Chinese labourers at the hospital subsided.

A Chinese origins theory also best explains conflicting evidence uncovered by other researchers such as John Oxford and John Barry. Chinese labourers could easily have spread flu much earlier to Europe, linking the outbreak which Oxford identified at Étaples to a much larger chain of disease events. By the same token, the cases in Haskell, Kansas, could have developed as part of a much larger and earlier pattern, representing the diffusion of flu from military to civilian paths of infection. While this is entirely speculative, it suggests that ‘origin’ evidence might be seen as documenting separate moments when a new and emerging virus reached the public consciousness for a brief time only to disappear again. It also reminds us that new viral threats may emerge anywhere, and can develop over relatively long periods of time, erupting when long-entrenched patterns of socio-economic activity begin to change.¹⁵³

The Chinese origins theory best explains evidence that only falls into place when the disease is placed within its proper military context and the mobilization of peoples necessitated by the global nature of the Great War. For the first time massive numbers of people from previously isolated populations converged on the battlefields of Europe. The mobilization of the CLC may have allowed a new disease to spread in fits and starts from China, across North America, to Europe, where it mutated and then exploded along the sinews of war. In this way influenza followed the same path carved by previous epidemics. The result was the most deadly disease event in history.

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153 John Barry told NPR in an interview published online on 20 February 2006: ‘Ultimately, I don’t think it matters where it began ... The most important thing is that it can begin anywhere.’ See Vikki Valentine, ‘Origins of the 1918 Pandemic: The Case for France’, www.npr.org/templates/story/story.php?storyId=5222069 [accessed 28 February 2012].