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PART I

'No one has yet succeeded in achieving a satisfactory definition of what is meant by influenza, much less a regulation of the usage of the word.'

– F.G. Crookshank, *Influenza: essays*⁹

1

influenza: a primer

On 24 June 1918 the young war poet Wilfred Owen crawled into his Army-issue bell tent in a windswept field near Scarborough and began composing a letter to his mother. Then a 20-year-old lieutenant in the Second Manchesters, Owen had just been deemed fit for duty after a lengthy convalescence in Scotland following an attack of neurasthenia, a nervous condition brought on by the stresses and strain of the war. But as Owen waited in North Yorkshire for the orders that would return him to France and the Front his thoughts were seemingly on another disease entirely.

'STAND BACK FROM THE PAGE! and disinfect yourself,' he begins his letter to Susan Owen. 'Quite 1/3 of the Batt and about 30 officers are smitten with the Spanish Flu. The hospital overflowed on Friday, then the Gymnasium was filled, and now all the place seems carpeted with huddled blanketed forms... The boys are dropping on parade like flies in number...'¹⁰

At first glance, Owen's bold capitals and self-conscious italics read like genuine alarm. But as the next passage makes clear Owen is being ironic and far from taking the disinfectant measures seriously, considers influenza something of a joke. 'The thing is much too *common* for me to take part in. I have quite decided not to! Scottie [a regimental friend], whom I still see sometimes, went under today, & my servant yesterday. Imagine the work that falls on unaffected officers.'¹¹

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Owen's wry remarks, though calculated to amuse, were typical of British attitudes to influenza that summer. Though at times influenza can resemble a plague, it is most usually regarded as benign and unthreatening. This misconception about influenza results from our tendency to confuse it with the common cold. But while the symptoms of a cold can include a sore throat, cough, and headache, as well as muscle aches and fatigue, colds are caused by rhinoviruses or coronaviruses and only rarely result in serious respiratory complications. Influenza on the other hand is caused by viruses in the Orthomyxoviridae family. Some of these viruses have specific adaptations which enable them to penetrate deep into the respiratory tract and the lungs, and even a mild infection can cause intense muscle and joint pains, headache, and prostration – hence the French term *grippe* from the verb *gripper* meaning to 'grasp or hook.' In the case of pandemic strains of influenza, they can also trigger grave pneumonic complications in otherwise healthy young adults. Having said that, Owen's lack of concern is understandable as the majority of influenza victims usually recover within a week to ten days. Even the less familiar pandemic variety is not an automatic death sentence – up to half a population may be affected but in the developed world at least the case fatality rate rarely exceeds 3 per cent. By comparison a disease like cholera, if untreated, typically has a case fatality rate of 50 per cent and is truly fearful to observe, producing agonizing retching and diarrhoea that leaves its victims dehydrated and dying in watery rice stools.

It is the apparently benign nature of flu, coupled with its familiarity and ubiquity, that breeds complacency. But the ubiquity of flu is exactly why we *should* fear it. Even where outbreaks do not reach epidemic proportions, influenza infects so many people that even the mildest viruses almost always kill. In Britain, for instance, the Department of Health estimates

that normal 'seasonal' influenza, which occurs predominantly during a six to eight week period in winter and affects between 5–10 per cent of the population accounts for 12,000 deaths annually, the majority of its victims being the very young and elderly or those with underlying chest and heart conditions. But during the 1918–19 pandemic, according to some estimates, up to a third the British population, or some ten million people, were affected.¹² Although the case fatality was just 2.5 per cent, the result was 228,000 deaths – 25 times more than in a normal flu season.¹³

All mammalian influenzas begin as viruses of birds but though we know some of the ways in which these avian viruses mutate so as to infect pigs and humans we still do not know the precise evolutionary and environmental pressures driving the mutations. Nor do we know whether the 1918 virus originated in a bird or pig, or some other as yet unidentified animal host, and precisely how it became infectious in people (for further discussion of these issues see chapter six).

Without intact viral genetic material definitive retrospective diagnoses of past influenza epidemics are impossible, but from historical records it's safe to say that highly contagious, acute respiratory illnesses have afflicted mankind since the beginning of civilization. Both the Roman historian Livy and Hippocrates, the Greek father of medicine, describe a disease that sounds very much like influenza sweeping northern Greece in 412 BC and in 1411 the French applied the name 'tac' to an epidemic disease whose features included a dry hacking cough accompanied by a chill and extreme lethargy.

In September 1485 a similarly mysterious disease suddenly appeared in London. Dubbed the *sudor Anglicus*, or the 'English sweat,' it was thought to have been imported to the English capital by Henry Tudor following his victory over Richard III at

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Bosworth Field. According to contemporary medical chroniclers, the symptoms included 'grete swetyng,' a 'contynual thirst' and an intense headache or 'pricking [of] the brains.' But the most unusual characteristic was the suddenness of the attacks and the way that the disease seemed to target the rich and better-off members of society. By the time the outbreak had subsided in late October, it had dispatched two London mayors and four city aldermen.¹⁴ Writing in 1551 after the fifth and final visitation of the sweats to the British Isles (the other outbreaks occurred in 1508, 1517 and 1528), John Caius, physician to Henry VIII and Edward VI, said that those who were 'sore with peril of death were either men of wealth, ease or welfare, or of the poorer sort, such as were idle persons, good ale drinkes and taverne haunters.' He also had little doubt that the disease was of foreign origin, blaming it on Henry's army of French mercenary archers recruited in Rouen.¹⁵

However, the English Sweat does not appear to have been accompanied by a cough or other marked respiratory symptoms, a key hallmark of pandemic influenza. The same cannot be said of '*coqueluche*' (from the French term for a monk's hood because those so afflicted wrapped themselves in a hood to contain their shivering) a severe pulmonary disease which in 1510 swept across Europe from Africa 'not missing a family and scarce a person'. The disease, which was accompanied by earthquakes and a volcanic eruption in Iceland, was almost certainly influenza, the key symptoms being a sharp headache, chill and 'a terrible tarring cough' so violent that one observer reported many patients were 'in danger of suffocation.'

In 1562 another disease dubbed the 'Newe Acquayntance' swept through Queen Mary's court in Edinburgh at the end of November – the classic period for the onset of seasonal winter

flu. Lord Randolph writing to Lord Cecil from Edinburgh described it as:

... a plague in their heads that have yt, and a soreness in their stomaches, with a great cough, that remayneth with some longer, with others shorter tyme... the Queen kept her bed six days. There was no appearance of danger, nor manie that die of the disease, except some olde folks.¹⁶

It is difficult to say when the first true influenza pandemic occurred. Many medical historians argue the most likely candidate is the disease that swept northward from the Mediterranean to the Baltic in 1580. Within six weeks it had infected every country in Europe and was so ubiquitous that contemporary commentators remarked that 'hardly the twentieth person was free of the disease.' In Rome alone it left 9,000 dead and during the war against the Desmonds in Ireland it decimated the ranks of the English Army. 'For three daies,' reported one observer, 'they laie as dead as stockes, looking still when they should die; but yet such was the will of God that ... they all recovered.' There are also contemporary accounts from Asia and Africa, suggesting that this was a disease that affected people on every continent at the same time, which is the very definition of a pandemic (by contrast, an epidemic is merely a disease that affects people at the same time in a particular community, institution or country).

In the 17th century we can pinpoint at least three further candidates, including an epidemic that struck England, Ireland and Virginia in 1688, the year of the Glorious Revolution, causing people to die 'as in a plague.' Five years later, in 1693, influenza was again raging in Europe, attacking people of all conditions and ages, including 'those that were robust and hardy, as well as those that were weak and tender.'¹⁷

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At this time it was common to ascribe outbreaks of epidemic catarrhs and coughs accompanied by chills and great lassitude to volcanic eruptions and the passage of meteors through the night sky – a conception that persists in some scientific circles to this day.¹⁸ Indeed, the term influenza most likely derives from the Italian phrase *influenza coeli*, meaning ‘influence of the heavens.’ However, by the mid-18th century it had become more common in Italy to talk of *influenza di freddo* – ‘influence of the cold’ – and it was probably in this sense that the term first entered the English language in 1743.¹⁹

In the 18th century it is thought that there were at least three, and possibly as many as eight, influenza pandemics and once again contemporary commentators noted the democratic nature of the victims. During the 1732–33 outbreak in Plymouth, for instance, it was reported that ‘scarce anyone had escaped,’ while in 1781–82 it is said that two-thirds of Rome’s population were attacked.²⁰ However, these outbreaks pale in comparison with the 1836–37 and 1847–48 pandemics. The first, which swept west across Europe from Russia, claimed 3,000 lives in Dublin alone – more than the 1832 cholera epidemic – and in London the 1847–48 pandemic triggered 5,000 more deaths than in a normal flu season. But while a London doctor described the 1837 pandemic as one of the ‘most direful scourges’ he’d ever experienced, and in 1847 the ravages of influenza were once again compared to cholera, these early 19th century outbreaks apparently occasioned little overt panic.

‘Do you know what it is to succumb under an insurmountable daymare ... an indisposition to do anything, or to be anything; a total deadness and distaste; a suspension of vitality,’ asked the English essayist Charles Lamb laconically in a letter to his friend the Quaker poet Bernard Barton in 1824. ‘This has been for many weeks my lot, and my excuse; my

fingers drag heavily over this paper, and to my thinking, it is three and twenty furlongs from here to the end of this demisheet.²¹

Thomas Carlyle, writing to his sister at the height of the 1837 pandemic, was similarly non-plussed describing influenza as ‘a dirty, feverish kind of cold.’ In common with his contemporaries Carlyle blamed the outbreak on excess humidity, in particular the damp fogs that arrived in London from the northeast. ‘Printing offices, Manufactories, Tailor shops and such like are struck silent, every second man lying sniffling in his respective place of abode,’ he complained.²²

In fact, Carlyle’s observation is based on a misconception. Unlike the viruses which cause colds and which spread primarily by direct person-to-person contact, influenza viruses spread aurally, usually in small droplets expelled when someone coughs or sneezes, and tend to be more stable in cool, dry conditions. At room temperature, influenza peaks at a relatively low humidity – around 20 per cent – but at over 80 per cent humidity the droplets become too heavy and fall to the ground. Researchers have also discovered that at around 5° Celsius the virus transmits for about two days longer than at 20°.²³ In other words, the children’s rhyme about ‘in-flu-enza’ entering via an open window may be spot on.

By the 1890s many Britons had lulled themselves into thinking that the wonders of medical science could vanquish any foe, no matter how microscopic. In Germany Robert Koch had isolated the bacilli of tuberculosis and cholera while in Paris Louis Pasteur had developed vaccines against anthrax and rabies and discovered the process of pasteurization. But, for all their achievements, neither had the least notion what a virus was.

True, bacteriologists had begun to suspect that some diseases might be spread by what they called 'filter-passers' – microbes small enough to pass through the filters used in the laboratory isolation of bacteria – but as far as most scientists were concerned there was no mystery about influenza. Influenza, they thought, was spread by a bacillus like cholera, a belief that seemed to be confirmed in 1892 when one of Koch's most brilliant disciples, Richard Pfeiffer, announced in Berlin that he'd isolated the infective agent, *Bacillus influenzae*, later known as Pfeiffer's bacillus. In fact, *Haemophilus influenzae*, as Pfeiffer's bacillus is known today, is a fellow traveller and like other bacteria commonly found in the throats and lungs of influenza patients (principally pneumococci, streptococci and staphylococci) is not the primary cause of the disease.²⁴

It would not be until long after WW1, in 1933, that a team of British scientists working for the Medical Research Council (MRC) would demonstrate that influenza was actually transmitted by a virus, and it was not until the 1940s that researchers would see the influenza virion for the first time. Viewed through an electron microscope it resembled nothing so much as the surface of a dandelion bristling with tiny spikes and mushroom-like spines. The spikes, we now know, are made of a protein called haemagglutinin (HA) which derives its name from its ability to agglutinate to red blood cells. When a person inhales an air droplet containing the virus it is these spikes that stick to the receptors on the surface of the cells in the respiratory tract, much as a prickly seed case catches on the fibres of clothing in tall grass. The square-headed mushroom-like protrusions, fewer in number, are a powerful enzyme, neuraminidase (NA). It is the combination of these proteins and enzymes that enable the virus to evade the body's immune defences, much as a burglar equipped with a set of skeleton keys prises open a lock.

There are three types of influenza viruses: A, B and C. Type C viruses rarely cause disease in humans, while type Bs produce classic winter flu. Neither pose an epidemic or pandemic threat. That is the preserve of type A viruses, the primary reservoir of which is wildfowl such as geese and ducks. Unlike the building blocks of the human body – the double-stranded helix spirals of DNA (deoxyribonucleic acid) – influenza viruses, including type A viruses, are composed of eight delicate strands of RNA (ribonucleic acid). It is these RNA strands that code for the proteins and enzymes on the surface of the virus and determine the particular configurations of H's and N's.

Unfortunately, RNA does not possess an accurate proof reading mechanism. During replication, when the virus invades and colonizes animal cells, manufacturing hundreds of thousands of copies of itself, the RNA makes small copying errors, resulting in genetic mutations to the surface antigens – the combination of H's and N's which dictate the production of antibodies and the body's immune response. These mutations are known as 'antigenic drift'.

In addition, type A viruses can also 'swap' or reassort genetic material with other viruses. This process, known as 'antigenic shift', usually occurs when an avian or swine strain of influenza A exchanges genes with a human version of the virus, producing a completely new subtype. Inside the host, the eight RNA gene segments are shuffled randomly, like the symbols in the window of a slot machine. The result is a new virus that codes for proteins that may be new to the immune system and to which the body has no antibodies. It is these strains that historically have been the cause of pandemics.

The genetic identity of pre-modern pandemic strains of influenza are lost to history, but in the modern era there have been four major shifts. The first, H1N1, is the name given to the 1918 strain and is currently found only in pigs. This was

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also the dominant strain in humans until 1957 when a new subtype, H2N2, suddenly emerged in Asia triggering a pandemic that killed an estimated one million people worldwide. In 1968 there was a third shift, with the sudden emergence of H3N2 in Hong Kong, which also caused some one million global deaths. This is the strain currently circulating in human populations today. In addition, in 1977 there was a puzzling re-emergence of H1N1 – probably as the result of laboratory error – but it failed to trigger a pandemic or replace the prevailing H3N2 subtype.

Since the middle of the 19th century most pandemics have been thought to originate in the ‘silent spaces’ of Asia and the Far East – a suitably broad and imprecise sweep of territory stretching from southern China and southwestern Siberia to Kazakhstan and the Urals. But while many flu’s have been described as ‘Asiatic’ they have also been attributed to other peoples and nations, including in 1918 the Spanish, and in 1889 the Russians.

The ‘Russian’ flu pandemic came in three waves over a period of as many years sweeping west from Bokhara in Tsarist-controlled Uzbekistan to St Petersburg via the new Trans-Caspian railway. From St Petersburg commercial and diplomatic travellers conveyed the disease to the Baltic ports while rail passengers spread it to Warsaw, Berlin and Vienna. By December the disease was in Paris, and by the early winter of 1890 it had reached London from where it was disseminated to the rest of Britain. The speed with which infections spread and the way the disease appeared to follow the lines of communication and commerce attracted widespread comment, as did the democratic nature of the attacks. ‘From emperors to potboys, no one has been exempt,’ commented one journal, ‘we have all ached in common.’²⁵



Figure 1.1 'Everyone has influenza.' Wood engraving for *Le Grelot*,
12 January 1890.

Credit: Wellcome Library, London

In London alone it is estimated that the first wave of Russian flu sickened 10–15 per cent of the population or in excess of 400,000 people. The official death toll was low – just 4,523 recorded deaths from influenza for the whole of England and Wales. But medical health officers, whose job it was to monitor local disease outbreaks and report back to government, noticed that this was not the whole story and that when deaths from associated respiratory complaints, such as bronchitis and pneumonia, were taken into account, the true excess death toll was 27,000. Leading London teaching hospitals such as St Bartholomew's in the City were particularly hard hit with doctors arriving at morning surgery to find upwards of a 1,000 patients already awaiting treatment. This flu was nothing like a cold but seemed to strike at the heart of late Victorian industrial society, seizing men, the main family breadwinners, in their places of work. Some patients could even name the exact hour of an attack saying, 'I went to work all right this morning, but was suddenly taken ill at eleven o'clock, and had to leave off.' The disease was characterized by extreme prostration, weakness and nervous depression. Frontal headache, pain in the eyeballs and muscular pains were also common, and, in the worst cases, a peculiar 'creeping pneumonia.'²⁶

Public apprehension deepened when it was reported that two soldiers had died at the Guards Hospital in Rochester Row and that Lord Salisbury, the Prime Minister, was laid up at his country seat at Hatfield. The Queen asked for regular updates and saw her Prime Minister recover. But nearly a year later, in the spring of 1891, the flu returned sweeping south along the Pennines to Sheffield from where it was carried to London and back to the Houses of Parliament. One of the first victims was the Archbishop of York who had been in Westminster pressing the case for child insurance. As Lords, MPs and even the Prince of Wales were taken ill, fumigation squads were brought in to

spray the Houses of Parliament in the hope of chasing the 'microbe' from Westminster's insanitary nooks and crannies. In Sheffield, meanwhile, the death rate in the first week of May reached 70 per 1,000 – the highest rate in the town's history, exceeding even the 1832 cholera epidemic.²⁷

By the end of the Whitsuntide holiday, however, the epidemic was on the wane, and although the Russian flu returned for a third time in the autumn and winter of 1891–92, this time it was nothing like as severe. Nevertheless, the spring 1891 wave alone had caused nearly 58,000 excess deaths, and including the follow-on waves in 1893 and 1894 it is estimated that more than 100,000 Britons lost their lives.²⁸

The Russian flu pandemic awakened interest in the disease as never before. Whereas before 1889 most doctors regarded flu as relatively mild and harmless, after 1889 medical professionals had little choice but to take it seriously. In particular, doctors were struck by the association between the Russian flu and what they described as 'a low and insidious form of pneumonia.' It was these pneumonias, which lodged deep in the lobes of the lungs so as to mimic the symptoms of catarrh, that had caused most of the deaths, though whether these 'lobar pneumonias' were due to the 'microbe' itself or secondary bacterial infections no one could be certain. The other alarming feature was that while, as in a normal seasonal flu, the elderly and the very young had been the worst affected – a graph of mortality by age showing the usual U-shaped curve – men and women aged 30–40 had suffered an abnormally high number of lung complications. Not only that but after the initial attack had passed and patients were thought to be on the road to recovery 'nervous complications' had supervened sparking depression, lethargy, and, in some cases, suicides. Finally students of epidemiology observed how the pandemic had taken the form of three distinct waves of infec-

tion with the second wave – which arrived in the spring of 1891 – being more severe than the first, and the third and final wave in the winter of 1891–2 being the least severe.²⁹

In 1918 this pattern of a mild primary wave followed by a severe secondary wave would be repeated but this time the mortality would fall disproportionately on young adults – men and women aged 20–40 – producing a W-shaped mortality curve with the tallest peak in middle life. This was a phenomenon that had never been seen before. Nor has it been repeated since. But the most alarming feature was the manner in which people died. ‘Spanish’ influenza struck suddenly and without warning: one moment a person was up and about, the next they would be lying incapacitated coughing up greenish-yellow sputum. In some cases, a frothy, blood-stained fluid gushed from their nose and mouth. As pneumonia set in their temperature would soar to 40 or 41° C and they would slip into a delirium. The final stage came when their lungs filled with fluid prompting their heart to leech oxygen from the blood vessels supplying the head and feet. This was the condition known as heliotrope cyanosis. It must have felt like drowning. Typically, a blue or dark-purple stain would spread across the lips and cheekbones. Then the victim would turn a mahogany colour and die. In the words of one historian of the 1918 pandemic, the flu ‘turned people the color of wet ashes.’³⁰

But in January 1918 such horrors still lay in the future. After nearly four years of ‘total warfare’ during which Britons had suffered Zeppelin raids, food and fuel shortages, and the loss of hundreds of thousands young men in the ‘killing fields’ of Flanders and northern France, the prospect of being struck down by an epidemic disease, let alone something as familiar as the flu, was not uppermost in most people’s minds. In those cold and hungry years pneumonia and other respiratory

diseases, such as tuberculosis, were a far more present threat. Perhaps that is why when reports of a strange new epidemic disease first emerged from Spain in May 1918 the *Times's* Glasgow correspondent reported that the man in the street dismissed the threat 'and cheerfully anticipated its arrival here.'³¹

In the United States, however, attitudes were different. There, General Pershing was on the point of dispatching a million 'doughboys' to Europe and US Army medics emboldened by their victories over yellow fever in New Orleans were on the look out for unusual disease outbreaks. The result was that when hundreds of soldiers at an Army encampment in Kansas were suddenly struck down by a flu-like disease accompanied by severe pneumonia in March 1918 a telegraph was sent to Washington. It was the first report anywhere in the world of what would later erroneously be labelled 'Spanish' influenza. Within weeks the flu had spread to other US Army camps and by April it had reached Bordeaux, courtesy of the American Expeditionary Force landings at Brest. By May 'three-day fever' and 'grippe' was rife throughout the Allied lines and the Germans were complaining of 'Blitzkattarrh.' By now there were also reports from Scapa Flow in Scotland that 9,000 seaman – 10 per cent of the total fighting force of the British Grand Fleet – were laid up. Then, in June, came rumours that some 3,000 Tommies had been taken by hospital trains to a massive British Army hospital complex on the northern French coast near Boulogne. The official diagnosis was 'P.U.O.' – Pryexia of Unknown Origin – but most medics at the camp were convinced it was the same disease that had accompanied the AEF to Bordeaux. What is more they thought they had seen a very similar disease two years before.

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