Geographical and Biological Origin of the Influenza Pandemic of 1918[1]

John M. Saul[2]

Abstract: During a possibly unique sequence of events in early 1918, an initially mild influenza virus became stranded within a human population composed of healthy young adults, virtually all whose members had well-functioning immune systems. Instead of dying out, the virus adapted to this hostile environment. Available information suggests that the influenza virus of 1918 originated in the U.S. military training center at Camp Funston, Kansas, was then taken to a sparsely populated civilian area in Haskell County, Kansas, 500 km away, and was returned to Camp Funston before spreading abroad.

Key Words: 1918 pandemic, influenza, natural selection, evolutionary selection of 1918 influenza virus, Patient Zero 1918

"Explaining the extraordinary excess influenza mortality in persons 20–40 years of age in 1918 is perhaps the most important mystery of the pandemic" (Morens and Fauci 2007). The overall severity of the pandemic and its uncertain place of origin are also unresolved puzzles. Here I argue for a sequence of events that permitted the virus to evolve an adaptive trick that would enable it to preferentially infect healthy young adults with well-functioning immune systems. To do so, an initially mild viral strain had to make a round trip between two significantly different populations separated from one another by five hundred kilometers. As reconstructed here, the events of early 1918 do not include a meaningful or definable role for a Patient Zero 1918.

Brief Background

The influenza pandemic of 1918 was like none other. It was especially virulent and deadly, killing an estimated 50 to 100 million people worldwide (Osterholm 2005), specifically targeting healthy people in the prime of life, and killing perhaps as many as 8–10% of all young adults then living (Barry 2004). It also killed infants, children, and the elderly but proportionately far less than might have been expected (Barry 2004). This held true worldwide (Barry 2004) and continued through 1920 (Crosby 2003). One Swiss doctor "saw no severe cases in anyone over 50" (Barry 2004, p. 239).

[1] Submitted on October 23, 2018. Accepted on October 26, 2018. Last revisions received on October 30, 2018.
[2] ORYX, 16 rue du Pré-aux-Clercs, 75007 Paris, France. E-mail: john.saul@wanadoo.fr . ORCID 0000-0002-2282-2430.

DOI: 10.9784/LEB6(1)Saul.01
Electronically available on October 31, 2018. Mailed on October 31, 2018.

The elderly may have acquired partial immunity during the Russian Flu of 1889-1890 or some earlier unidentified influenza outbreak (Barry 2004, Crosby 2003, Spinney 2017) but, if so, which has not been demonstrated, this must have been worldwide (Barry 2004). In any case, such immunity would have provided no explanation for the relative resistance of children, many of whom fell sick with relatively few dying. The disease came in three waves. During the second wave, which was the great killer, feverish newly orphaned children wandered dazed in the streets, both parents having succumbed. In Blackpool, England, influenza ran through a boys school with 700 students, but only one died (Honigsbaum and Mawdsley 2018). In parts of Shansi, China, mortality during the winter of 1917-1918 attained 80-90%. Most survivors were young children but there is doubt concerning the identity of the epidemic, even whether it was influenza (Spinney 2017).

From the first wave in March 1918 through the end of the third, the pandemic was most efficient at killing men and women whose immune systems were the best *Homo sapiens* can produce. There are no usable statistics but here and there doctors in hospitals, nurses in wards, sailors on ships, and soldiers in barracks reported that it was the most robust, strongest, most fit, disease-free athletic sorts who suffered the worst (Barry 2004, Crosby 2003). Student nurses were greatly affected but retired nurses called up because of the emergency fared far better. In Japan, the large number of wrestlers who withdrew from the May 1918 tournament in Tokyo brought the term "Sumo flu" (*sumō kaze*) into common use (Hayami 2015)

Those in the 15–34-year age bracket who came down with influenza in 1918 or with the quasi-ubiquitous pneumonia-like complications were about twenty times more likely to die than had been the case in 1917, a non-epidemic year for influenza. In Philadelphia, the death rate reached 700 times the normal rate (Hoehling 1961).

Crowding

The risk of catching the flu in 1918 was far higher in the crowded military staging camps and in congested supply depots than in Europe's muddy trenches (Crosby 2003) and this was not something it took a statistician to detect. French soldiers who left crowded barracks for windblown trenches reduced their chances of getting the flu by a factor as high as twelve (Crosby 2003). Some of the most awful accounts are those of outbreaks on overcrowded troop transports on their way across the Atlantic (Barry 2004, Crosby 2003). A sailor's diary reads, "'October 5 – fifteen more bodies have just been buried from the *President Grant*'" (Crosby 2003, p. 137). During the last two months of the war, approximately 4000 American servicemen died at sea or after being put ashore for hospitalization at Halifax (Crosby 2003). Troopships disembarking large numbers of sick and dying men in French ports hindered the Allied effort (Crosby 2003) but such things were only written after the war (Crosby 2003).

Death rates from influenza as high as those recorded in 1918 are exceptional. But in large, crowded, homogeneous populations of immobile

human hosts, natural selection favors the most rapidly acting strains, whether they kill their hosts or not (Ewald 1991).

Pigs and Poultry

In late 1917 and early 1918 before the initial outbreak, young men were crowded into military camps throughout the United States. Swine and poultry were raised in some camps and horses were present in all, with wild birds attracted by the bounty of waste fodder and droppings. The initial human version of the 1918 flu may have started in such a setting with pigs – many of which suffered from "swine flu" in 1918 (Spinney 2017) – as mixing-bowls in which segments of the genes of avian and human influenza viruses were reassorted to produce strains with new characteristics (Wenjun et al. 2009). In 1918, the avian component in the genetic mix included indications of a North American origin (Worobey et al. 2014).

Camp Funston, Kansas

There is a degree of cautious agreement that the first cases of the pandemic influenza of 1918 occurred in Camp Funston, an army training camp now part of Fort Riley, in Kansas on March 4, 1918 (Barry 2004), though the exact date in early March has been contested. [For other hypotheses concerning the geographic origin of the pandemic of 1918, see Crosby (2003), Barry (2004), and Spinney (2017).] Funston was not just crowded. It was *overcrowded* (Barry 2004) and it was also underheated, causing men to huddle together around stoves during the record cold Kansas winter of 1917-1918 (Barry 2004).

Shortly before breakfast on the day in question, a Company Cook, Private Albert Gitchell, reported to the infirmary with a "bad cold". Corporal Lee W. Drake was right behind with similar symptoms and by noon the Camp Surgeon had 107 flu patients on his hands (Hoehling 1961). Within three weeks the number of sick at Funston was above a thousand, though the number of deaths, 38, was insufficiently high to draw national attention (Barry 2004).

New incidences of the flu at Funston bobbed up as new groups of draftees and volunteers arrived. And as troops were moved around the country in crowded trains, the flu spread. It spread throughout March and April 1918 reaching Camps Oglethorpe, Gordon, Grant, Lewis, Sherman, Doniphan, Fremont, Hancock, Kearney, Logan, McClellan, Sevier and Shelby (Barry 2004, Crosby 2003), a nasty epidemic that at this stage had not spread into the general civilian population. Civilians had been healthy during the summer of 1918, a matter established by later investigators peering through the haze of wartime censorship and self-censorship (Crosby 2003). Exceptions included San Quentin Prison where over 500 (of 1900) prisoners came down sick, 3 of whom died (Crosby 2003), as well as workers in the crowded Ford factory in Detroit, a thousand of whom were sent home (Crosby 2003). Soldiers in various camps died as they had at Funston, but their numbers were limited, and their deaths

were generally attributed to pneumonia rather than to influenza with which it is closely linked. For although the Funston version of the 1918 flu – "the first wave" – was extremely contagious, it was not itself a great killer.

The second wave, from which the most appalling accounts and statistics were drawn, did not come until early September 1918 when the disease broke out from military camps on the East Coast of the United States and entered the general population (Crosby 2003). But the second and third waves are not examined here, where the focus is on the origin of the first wave, hence of the pandemic itself.

Rapidity

The influenza of 1918 was extraordinarily quick. According to Jessie Lee Brown Foveaux who had worked in the quartermaster laundry at Camp Funston at age 18: "We'd be working with someone one day, and they'd go home because they didn't feel good, and by the next day they were gone" (Foveaux 1997).

During the second wave, the time from apparent good health to complete prostration was an hour or two (Crosby 2003) with no time to just feel unwell. On September 17 five doctors and fourteen nurses suddenly collapsed in Philadelphia: "None had exhibited any prior symptoms whatsoever. One moment they felt normal; the next, they were being carried in agony to hospital beds" (Barry 2004, p. 201). The following month, a student nurse wrote she "never saw a patient walk into the ward or come in a wheel chair; victims came on stretchers, often propped up for breathing ease" (Hoehling 1961, p. 82). "'They'd be sick one day and gone the next, just like that, fill up and die', wrote the physician and poet William Carlos Williams" (Crosby 2003, p. 216). Many people could recall the exact moment they knew they were sick (Barry 2004).

Haskell County, Kansas

John M. Barry suggested that the origin of the first wave may have been in Haskell County, Kansas, approximately 500 km southwest of Camp Funston, a month or more before Private Gitchell's visit to the infirmary (Barry 2004). Barry's contention focused on the work of Dr. Loring V. Miner (1860-1935), who in 1918 had had a decades-old medical practice in sparsely populated Haskell County (Barry 2004). Making his rounds from farm to farm and town to town in late January and February 1918, Dr. Miner encountered a new ailment that he recognized as influenza, and which he signaled to the U.S. Public Health Service as "influenza of severe type". This disease was "rapid in its progress through the body" (Barry 2004) and, with 18 severe cases and 3 deaths (Crosby 2003) in a county population of 1720, was far more lethal than the first wave of the pandemic, still a month in the future, and which was mild at the outset (Barry 2004). The Haskell flu attacked everyone, including many children, but notably with the "most robust people in the county… struck down as suddenly as if they had been shot" (Barry 2004, p. 93).

On February 14, 1918, Haskell County's weekly *Santa Fe Monitor* (Barry 2004), reported: "Mrs. Eva Van Alstine is sick with pneumonia. Her little son Roy is now able to get up... Ralph Lindeman is still quite sick... Goldie Wolgehagen is working at the Beeman store during her sister Eva's sickness... Homer Moody has been reported quite sick... Mertin, the young son of Ernest Elliott, is sick with pneumonia... Pete Hesser's children are recovering nicely... Ralph McConnell has been quite sick this week." A week later the same paper reported, "Most everybody over the country is having lagrippe or pneumonia" (Barry 2004; *The Santa Fe Monitor* 1918 February 21). Most of the military-age men from Haskell County trained at Camp Funston (Barry 2004) along with 50,000 to 60,000 other recruits (Barry 2004). The issue of *The Santa Fe Monitor* with the news that "most everybody over the country is having lagrippe or pneumonia" also reported that "Dean Nilson surprised his friends by arriving at home from Camp Funston on a five days furlough" and that Ernest Elliott left "to visit his brother at Funston just as his child fell ill" (Barry 2004; *The Santa Fe Monitor* 1918 February 21). The issue of February 28 recorded the departure of John Robert Bottom for Funston and other comings and goings were indicated in various issues of *Monitor*. Yet there was nothing exceptional about Haskell County except the presence of a severe and unusual influenza somewhat before the beginning of March 1918.

Camp Funston, however, was special. First, there was the overcrowding. Then, there were the swine and poultry. In addition, there was the select presence of fit young men and women to the near exclusion of anyone else. Enough young women were at Funston for weekend dances but presumably few infants, children, or older people other than some senior officers and their wives.

Alfred Crosby (2003) and Barry (2004) gave March 4th as the day the first soldiers at Camp Funston reported ill with the symptoms of influenza. This fits Barry's repeated suggestion that the epidemic originated in Haskell County in January or early February 1918 (Barry 2004). If so, as Barry contended, the disease might have died out for lack of susceptible human hosts in sparsely populated Haskell where the epidemic was so short-lived that school reopened with healthy children by mid-March (Barry 2004). "No one is absent from school [in Santa Fe] this week for the first time in six weeks" reported the *The Santa Fe Monitor* for March 14 and "Just a few are absent from school now [in Sublette, also in Haskell County] … after so much sickness" (*The Santa Fe Monitor* 1918 March 14).

If the original 1918 flu virus had somehow sprung up in the general population of Haskell County and then been brought to Camp Funston, as Barry suggests, it would have found itself in a far less receptive human population, a hostile environment in which most people possessed well-functioning immune systems. A soldier who caught an early case of this hypothetical "Haskell-origin flu" might have got barely sick without passing his flu on to anyone else, he might have endured a few nasty non-productive coughs, or he might have got

quite sick. But none of these scenarios can easily account for the abrupt arrival in the infirmary of 107 sick soldiers during a single morning (Hoehling 1961) unless they had all been in contact the same day with a contagious person from Funston (a possibility that cannot be entirely excluded, given that Private Gitchell was a "Company Cook").

There is nothing obviously wrong with Barry's suggestion that the epidemic started in Haskell County, but the idea is of limited value because it leaves the most interesting questions unanswered. Why in the unexceptional Haskell County? Why did the mild first wave kill some people with extreme rapidity (Foveaux 1997)? Why were later waves especially severe worldwide among individuals who were fit? And what of Barry's assertion, contested below, that "if the virus did not originate in Haskell, there is no explanation for how it arrived there" (Barry 2004, p. 455)?

Round trip of the virus: Camp Funston to Haskell County and back to Camp Funston

The origin of the pandemic, as reconstructed here, starts at Camp Funston (Kansas) in very early 1918 with a mixing-bowl event involving swine, North American wild birds or poultry, and humans, producing mutant influenza viruses that passed through numerous soldiers, none of whom got sufficiently sick to report to the camp hospital. In mid- or late January 1918, soldiers on leave or returning visitors brought this new strain of influenza to Haskell County.

Children and other civilians in Haskell County got ill from a mutant of the Funston flu, which, according to Dr. Miner's experience, was of a new, unusual, and severe type (Barry 2004). Adults in good health were affected as well. The death rate as a percentage of the county's small population was greater than it would be the next month at Camp Funston, but only a fraction of the rate for the overall U.S. population during the second wave later that year (Barry 2004). But the numbers are uncertain because Haskell County's "local paper, *The Santa Fe Monitor*, apparently worried about hurting morale in wartime, said little about deaths" (Barry 2004, p. 94), which was in keeping with the self-censorship ubiquitous in the U.S. throughout the war. The disease disappeared from Haskell County in late February or early March 1918, presumably after running out of susceptible people to infect. Yet mutants of the virus were still surely brought from Haskell back to Camp Funston until the very last arrivals of contagious visitors around the turn of the month.

Following their return to Camp Funston, few mutant viruses would have survived in the absence of their usual preferred hosts, infants, children and the elderly. But one strain, which had originated earlier in the Funston mixing-bowl and had then affected some adults in Haskell County, did survive, adapting to the one ecological niche available to it at Camp Funston, a niche in which the potential human hosts were all in the prime of life. It did so in very late February or the first days of March 1918 after evolving an adaptive "trick". For around

that date, a viral mutant emerged that could act with *extreme* rapidity, apparently more rapidly than any other influenza strain before or since. And "by late April, the essential character of the new strain seems to have been established" (Barry 2004, p. 178) and would be maintained worldwide through 1920.

There are several ways by which the virus could have obtained the extreme rapidity it needed to survive in the environment of Camp Funston. Some possibilities include concealing the evidence that it had hijacked the cell's reproductive machinery, thus giving it a head start over the immune response (Spinney 2017, p. 192, referring to work by Jeffery Taubenberger and collaborators); packing more individual viruses into each infected cell before breaking out; shortening the time between viral generations; or acquisition of an unusual degree of viral stability so that a greater proportion were of a single type.

Extremely rapid viral swarming would nevertheless evoke a rapid and forceful immune response. *In the general population*, infected individuals would get very sick very rapidly, and would then either recover or succumb to influenza or to secondary complications. For individuals *in the prime of life,* such as those in the military, yet another outcome was possible. The initial viral swarming would at first outrun the initial immune response, just as it did in members of the general population, and individuals would also get very sick very rapidly. The immune response of these fitter individuals would then be rapid and massive. In many cases in 1918 it would be too massive, producing cytokine storms that flooded the lungs with cell debris, blood, white blood cells and collagen in "a springtide of fluids" (Crosby 2003, p. 222), blocking or exploding capillaries, and producing never-seen effects unique to the disease of 1918 (Barry 2004). In attempting to breath with damaged lungs, some patients would forcefully expel fluids. Earaches could be violent, and blood might emerge from ears and eyes. Female victims had symptoms mistaken for menstrual bleeding. Those who died most rapidly showed the greatest damage to the lungs (Barry 2004). In Fort Devens, Massachusetts, fluids poured out of the nostrils of young victim in the autopsy room when "he was moved in the slightest degree" (Barry 2004, p. 190). As put by Barry, "strength became a weakness". "The immune response killed" (Barry 2004, pp. 249-250).

Conclusion

The influenza pandemic of 1918 emerged from a multi-step natural evolutionary process that selected for an exceptionally fast-acting viral strain in a population in which a slower acting strain would have rapidly died out. We are again reminded of Theodosius Dobzhansky's teaching that "nothing in biology makes sense except in the light of evolution" (Dobzhansky 1973).

Acknowledgements

I thank six reviewers for useful comments on earlier versions of this paper. This work received no external funding. The author has no conflicts of interest.

Literature Cited

Anonymous contributors. 1918. *The Santa Fe Monitor* (Santa Fe, Haskell County, Kansas, USA). February 14, February 21, February 28, March 14.

Barry, J. M. 2004. *The Great Influenza: The Epic Story of the Deadliest Plague in History*. Viking Books. New York, NY, USA. 547 pp.

Crosby, A. W. 2003. *America's Forgotten Pandemic: The Influenza of 1918*. Second Edition. Cambridge University Press. Cambridge, England, UK. 337 pp. https://doi.org/10.1017/CBO9780511586576

Dobzhansky, T. 1973. Nothing in biology makes sense except in the light of evolution. *American Biology Teacher* 35(3):125-129. https://doi.org/10.2307/4444260

Ewald, P. W. 1991. Transmission modes and the evolution of virulence, With special reference to cholera, influenza, and AIDS. *Human Nature* 2(1):1-30. https://doi.org/10.1007/BF02692179

Foveaux, J. L. B. 1997. *Any Given Day: The Life and Times of Jessie Lee Brown Foveaux*. Warner Books. New York, NY, USA. 224 pp.

Hayami, A. 2015. The influenza pandemic in Japan 1918-1920: The first world war between humankind and a virus. *Nichibunken Monograph Series* 18. International Research Center for Japanese Studies. Kyoto, Japan. http://id.nii.ac.jp/1368/00006047/

Hoehling, A. A. 1961. *The Great Epidemic*. Little, Brown and Company. Boston, Massachusetts, USA and Toronto, Ontario, Canada. 217 pp.

Honigsbaum, M. and H. Mawdsley. 2018. *Going Viral 5. Global Impacts, Local Traces* http://goingviralthepod.libsyn.com/5-global-impacts-local-traces . 23'44".

Morens, D. M. and A. S. Fauci. 2007. The 1918 influenza pandemic: Insights for the 21st century. *Journal of Infectious Diseases* 195(7):1018-1028. https://doi.org/10.1086/511989

Osterholm, M. T. 2005. Preparing for the next pandemic. *New England Journal of Medicine* 352(18):1839-1842. https://doi.org/10.1056/NEJMp058068

Spinney, L. 2017. *Pale Rider. The Spanish Flu of 1918 and How It Changed the World*. Vintage. London, England, UK. 332 pp.

Wenjun, M, R. E. Kahn, and J. A. Richt. 2009. The pig as a mixing vessel for influenza viruses: Human and veterinary implications. *Journal of Molecular and Genetic Medicine* 3(1):158-166.

Worobey, M., G.-Z. Han, and A. Rambau. 2014. A synchronized global sweep of the internal genes of modern avian influenza virus. *Nature* 508:254-257. https://doi.org/10.1038/nature13016

Interior of laundry in Camp Funston, Kansas (1917). Photographer unknown. National Archives and Records Administration (Washington, District of Columbia, USA). Record 165-WW 267E89. https://catalog.archives.gov/id/45498983