RELATIONSHIP BETWEEN WEATHER AND CLIMATE IN SHAPING OF INFLUENZA

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ABSTRACT

The leading epidemiological hypothesis for the seasonality of influenza involves the crowding of sick and healthy individuals indoors in response to the winter season decrease in outdoor temperatures.India, located in northern hemisphere, has distinct seasonality that might be related to latitude and environmental factors. In this paper we discussed about the geographic location and its influenza seasonality.

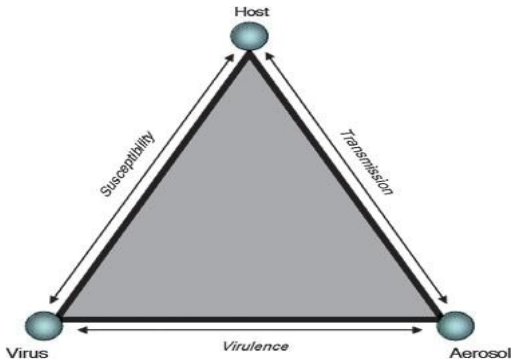
1. Introduction:

Influenza, commonly referred to as the 'flu', is a respiratory virus that causes significant mortality and morbidity during annual epidemics, and is capable of affecting nearly half of the global population during severe pandemics (e.g., the 1918–1920 'Spanish Flu'; Potter 2001). In temperate climate regions, influenza epidemics display a distinct seasonality, with widespread infection typically occurring during the winter season months: November to March in the Northern Hemisphere and May to September in the Southern Hemisphere (Hope-Simpson 1992). Despite the simplistic and unscientific nature of this hypothesis, it has continued to prevail almost unchallenged for the better part of half a century (Lofgren et al. 2007)[2]. There are, however, growing bodies of literature that reveal both biological and physical mechanisms involving the influenza virus and the susceptibility of the host immune system that may be responsible for the seasonality of epidemics. In general, these studies can be divided into two areas: (i) laboratory-based studies on viral etiology and host susceptibility and how they vary under different environmental conditions, and (ii) epidemiological studies relating large-scale

morbidity and mortality patterns to various climate signals and atmospheric conditions. The objective of this article is to synthesize the results of these studies in an effort to increase the transparency of what is currently known about the effects of weather and climate on influenza and what aspects of the relationship remain unexplored.

2. Conceptual Framework

The role of weather and climate in disease ecology and epidemiology may be illustrated in a variety of ways and with varying levels of complexity. The most comprehensive conceptual models include environmental, social, economic, and health care system conditions and the direct and indirect effects they have on human health. While the complexity of the climate–health problem is recognized, the full scope of these conditions is beyond the goal of this article.



As a means of emphasizing the weather and climate components of influenza, a diagrammatical construct adopted from the field of epidemiology[4] – the Triangle of Disease is utilized as a framework from which the components of disease can be identified and the pathways between them explored in the context of weather and climate (Timmerick 2002). The vertices of the triangle represent the major epidemiological components of influenza (i.e., host, virus, aerosol / droplet) while the arms of the triangle represent the dynamic processes, conditions, and pathways that facilitate and maintain the cycle of disease (i.e., transmission, susceptibility, virulence). It is important to note that the exact components and pathways of the triangle often vary depending on its use or the nature of the health conditions being studied (Comrie 2007).

3. EFFECTS OF WEATHER AND CLIMATE ON VIRAL TRANSMISSION:

The transmission of influenza involves the shedding of respiratory particles (i.e., droplets or aerosols) by an infected host into the ambient environment, generally through coughing and sneezing. A typical cough or sneeze can produce tens of thousands of individual respiratory particles, ranging in size from one-tenth of a micrometer (lm) to 100 lm in diameter (Hall 2007). There are three primary modes of viral transmission involving the shedding of respiratory particles: droplet, contact, and airborne (Brankston et al. 2007). Both the droplet and contact modes involve large respiratory particles (›100 lm) that are too heavy to remain suspended in the air. Droplet transmission occurs when these particles are expelleddirectly onto another person, usually within 1 m of the infected host and focused near the eyes, nose, and mouth. Contact transmission involves physical contact with respiratory particles that have settled on surfaces. Subsequent contact with the nose or mouth can result in inhalation of viral particles. Unlike droplet or contact transmission, airborne transmission involves smaller respiratory particles (i.e., aerosols ‹ 10 lm). Their smaller size allows them to remain suspended in the air and makes them more likely to be respired (i.e., pass into the lower respiratory tract) (Tellier 2006). Viral particles that pass into the lungs are likely to cause other airway infections, such as pneumonia, which are often closely associated with influenza infection[1,3,5]. There has been much debate as to which mode of transmission is most significant with respect to the epidemiology of influenza. For example, Lemieux et al. (2007) found that respiratory particles are most likely to settle in the upper respiratory tract, implicating droplet and contact modes (i.e., large particles) as the primary means of transmission. This is further supported by Brankston et al. (2007) who suggested that most viral infections occur over short distances through physical or direct contact. However, a later study by Lowen et al. (2008) on guinea pigs found that the relative role of contact and airborne transmission may be sensitive to changes in ambient temperature. Therefore, the dominant mode of transmission likely varies according to environmental conditions (Hall 2007). Earlier work by Schulman (1967) on virus transmission between mice corroborates this statement. While no study was found that quantified the size distribution of respiratory particles expelled during a typical cough or sneeze, it may be safe to assume that both small and large droplets, as defined above, are expelled simultaneously. If this is the case, then smaller respiratory particles may disperse and affect susceptible populations across a broader

geographical area (i.e., airborne mode), while larger particles may settle directly onto an individual or other surface in a more confined environment (i.e., contact or droplet mode) (Hall 2007). Over the course of a full seasonal epidemic, it is likely that all three modes of transmission contribute to infection across varying space and time scales (Weber and Stilianakis 2008). Until now, research on the environmental effects of influenza transmission has focused primarily on ambient humidity and temperature in laboratory-controlled settings. The ambient humidity is important in the transmission of influenza because it can affect the size of the respiratory particle (Weber and Stilianakis 2008). When the air is dry, large drops partially evaporate, creating smaller, lighter drops that are more likely to remain airborne for extended periods of time. Based on studies of aerosol dynamics, a typical respiratory particle exposed to an ambient relative humidity of 80% can remain airborne for up to 1 h. When the relative humidity is decreased to 20%, the same particle is able to remain airborne for more than 24 h (Weber and Stilianakis 2008). Dispersion studies of atmospheric particulate matter between 0.1 and 0.3 lm, which is comparable in size to the smallest respiratory particles, have revealed that such particles can in fact remain airborne for many days (Hammond et al. 1989). Therefore, even large particles expelled through coughing and sneezing may shrink to a size that favors long-range transport when the air is sufficiently dry. Viral transmission may also be sensitive to ambient temperature. Lowen et al. (2008) found that increasing the ambient temperature of guinea pig cages during transmission experiments appeared to prevent airborne transmission but not contact transmission. To simulate airborne transmission conditions, both inoculated and recipient guinea pigs were placed in separate cages in a chamber held at a temperature of 30 _C. After a week of exposure, no recipient guinea pigs were infected. However, when placed in the same cage to simulate contact transmission conditions at 30 _C, between 75% and 100% of the recipient guinea pigs became infected. Moreover, these results were insensitive to changes in ambient relative humidity. When considering the combined effects of ambient humidity and temperature on influenza transmission, it appears as though airborne transmission is more sensitive to changes in these variables than contact and droplet modes. As temperature and humidity fluctuate with the seasons in temperate climates, and as influenza epidemics exhibit a distinct seasonality in these areas, it is believed that the airborne route is the dominant mode of transmission in temperate regions (Lowen et al. 2008). The relative lack of seasonality in influenza prevalence in tropical regions, as well as less variability in temperature and humidity, suggests that contact and

droplet modes are dominant there. Further, the primary source for sporadic and isolated outbreaks that sometimes occur in temperate regions during the summer may be contact and droplet transmissions operating in optimal microclimate conditions. The role of weather and climate on influenza transmission may also extend to regions where the disease is endemic. The epicenter for most avian influenza strains and variants is in the Eastern Hemisphere, mainly Southeast Asia and China (Pyle 1986; Shortridgem 1997). Climate conditions in this part of the world have been linked to the migratory patterns of aquatic bird species (e.g., duck, goose, swan), which are a major reservoir for the virus (Gilbert et al. 2008). Their breeding and wintering patterns, however, extend northward into central Asia and much of Europe. Current projections of global climate change indicate that these regions will experience greater warming than lower latitude regions, which will likely affect the migration cycle of birds that help carry the virus (Gilbert et al. 2008). Within the epicenter regions of Southeast Asia and China, rainfall patterns connected with climate variability (e.g., the Asian Monsoon) and unmanaged agricultural practices (e.g., backyard duck farms, live markets) may widen the areas of possible contact (and transmission) between domestic water fowl and wild bird species (Henning et al. 2009). Changes in solar output and activity, as indicated by sunspot frequency, may also hold some predictive power in terms of forecasting global-scale pandemics (Hope-Simpson 1978). As the migratory patterns of most bird species are sensitive to changes in solar output and the resulting magnetism on earth, analyzing sunspot activity may provide an indicator of changing interspecies transmission cycles on a global scale. Recent work by Yeung (2006), who considered up to 15 global pandemics beginning in the 18th century, offers support for this theory.

4. EFFECTS OF WEATHER AND CLIMATE ON HOST SUSCEPTIBILITY

At the core of the debate over the causal mechanisms responsible for the seasonality of many infectious diseases is the role of population immunity and overall susceptibility to infection. One of the pioneers in this area of study was Edgar Hope-Simpson (Cannell et al. 2008). His proposed theory on viral transmission of infectious disease can be summarized as follows: the seasonal, epidemic influenza virus will tend to propagate through the environment via a series of transmissions from a small number of highly infectious but generally symptomless hosts who briefly become contagious as a result of a 'seasonal stimulus' (Hope-Simpson 1992). Hope-Simpson identified the seasonal stimulus as a deficiency in vitamin D levels because of seasonal

reductions in exposure to ultra-violet (UV) radiation. Low levels of vitamin D have been shown to impair the body's antimicrobial peptide system, which is responsible for regulating the immune response (Cannell et al. 2006, 2008). A corollary to the seasonal stimulus argument is the concept of photoperiod, which relates to seasonal oscillations in dark-light cycles in the middle and high latitudes (Dowell 2001). Seasonal changes in length of day can interfere with an individual's circadian rhythm, which is regulated largely by the release of the hormone melatonin. This interference can weaken the immune system and increase the risk of infection. Changes in photoperiod and sunlight exposure have been used to explain the observed latitudinal migration of influenza activity during the winter season (Cannell et al. 2008). On a global scale, the appearance and subsequent diffusion of influenza occurs along latitudinal belts, which coincide with changes in photoperiod and sunlight exposure because of changes in solar elevation, day length, and solar insolation. Effects of weather and climate on the seasonality of influenza least partially to the fact that the elderly are advised to limit exposure to the sun. One of the competing explanations for the seasonality of viral infectious disease was proposed by researchers at Cardiff University's Common Cold Centre in the UK and involves cooling of the nasal passageway (Eccles 2002). On a physiologic scale, the breathing of cold, dry air can slow mucociliary clearance of the nasal passage. Under normal conditions, the cilia in the nasal passage act to filter out pathogens and other aerosols from the upper respiratory tract. Experiments on the cilia of laboratory animals and humans indicate that exposure to cold air dramatically reduces mucus velocity (Baetjer 1967). This hypothesis has been used as a mechanism to explain the often observed rise in acute upper respiratory infections following a cold-air outbreak (Assaad and Reid 1971; Eccles 2002). In this case, a symptomless, subclinical infection may become clinical if the infected individual is exposed to cold air. The subsequent rise in clinical infections among a large population may then reveal itself, from a public health perspective, as an infectious outbreak. Although Eccles (2002) presents some compelling evidence for the nasal cooling hypothesis, it remains largely untested in the natural environment and among large populations. A more recent study by Johnson and Eccles (2005) found that chilling of the feet in cold water leads to vasco constriction of the upper respiratory system, which may increase susceptibility if exposed to viral particles. The susceptibility of an individual to influenza infection may also be mediated by exposure to other pathogens. In this case, modification of the host immune response to a given infection predisposes the individual to

infection from other pathogens (Fisman 2007). For example, recent work by Wong et al. (2009) in Hong Kong found that those infected with influenza were at greater risk for developing chronic obstructive pulmonary disease during periods of poor air quality. This was particularly the case for major pulmonary irritants, such as ozone. Prior expose to atmospheric pollutants can adversely affect mucociliary action in a manner similar to that experienced while breathing cold, dry air as described earlier. In addition to the innate immunity of healthy individuals, the host response to infection with respect to viral shedding has been shown to be sensitive to changes in ambient temperature. In their experimental studies involving viral transmission between guinea pigs, Lowen et al. (2007) found that infected hosts exposed to lower ambient temperatures shed significantly higher quantities of viral particles than those exposed to higher temperatures. Moreover, the period of peak shedding was extended by as many as 2 days when infection occurred at 5 _C compared with 20 _C. The abrupt rise in clinical infections typically associated with large-scale influenza outbreaks during the winter season may be at least partially related to the increased efficiency and duration of viral transmitters when the air is cold. Oddly, the efficiency and duration of viral shedding does not appear to be related to the degree of impairment of the host immune system because of cold stress (Lowen et al. 2007). This suggests that the environmental impacts on viral shedding may be acting at the level of the virus itself.

5. EFFECTS OF WEATHER AND CLIMATE ON VIRULENCE

In addition to factors such as transmission cycles and host susceptibility, the ability of the influenza virus to cause infection (i.e., virulence) is also important in the context of disease ecology and the seasonality of infection. Viral strains with a history of high infectivity, such as the H5N1 avian influenza, may indeed override any innate or adaptive Effects of weather and climate on the seasonality of influenza immunity (i.e., vaccination) regardless of environmental conditions. Virulence of the circulating viral strain, which often varies from year to year, has been used to explain why many influenza epidemics end rather abruptly, although there may be a large number of susceptible hosts[7]. There is evidence that the degree of infectivity of a circulating viral strain may be controlled at least partially by environmental factors. Recent work suggests that the survival of a virus is determined primarily by the characteristics of its outer casing, or envelope, which is composed of lipid compounds. Polozov et al. (2008) suggest that the lipid envelope encasing the virus remains intact longer when the air is sufficiently cold and dry. As the aerosolized viral particle enters the upper respiratory tract, the envelope melts, exposing the

virus to healthy host cells. In addition, air pollution can have adverse effects on the RNA sequence of the virus[5,6]. Weber and Stilianakis (2008) discuss this option, along with other forms of potential environmental inactivation of influenza virus. Interestingly, there remains a paucity of work on virus inactivation because of environmental factors. One possibility is prolonged exposure to UV radiation under clear sky conditions. Germicidal UV radiation produced by UV lamps has been shown to be highly effective at inactivating viruses on contaminated surfaces (Hall 2007). The effect to which this process occurs in the ambient environment is still unknown and warrants further investigation, particularly in light of the 'seasonal stimulus' hypotheses put forth by Hope-Simpson. Determining the environmental effects of virus infectivity requires a more complete understanding of how the virus exists in the ambient environment. Even today, there is still much debate as to lifecycle of the virus, particularly in the period between seasonal epidemics. Does the virus simply decay once temperatures and humidity levels rise to a critical point? Does the virus enter a dormant state between epidemics, only to be reactivated under a combination of biological and environmental triggers? Tracing the evolution of viral particles during and between epidemics using environmental modeling and atmospheric dispersion models may help solve this enigma. Recent work suggests that the influenza virus may in fact migrate from the tropics to temperate regions in both hemispheres during the winter (Alonso et al. 2007)[8,9,1011]. In their study, Alonso et al. (2007) found that the incidence of influenza mortality in Brazil exhibited a latitudinal shift from the northern, tropical regions in summer (November) to the southern, temperate regions of the country in winter (May). They suggest a strong environmental component to this pattern, as it flows against the expected population-driven diffusion pattern (i.e., south to north).

6. Influenza seasonality and Latitude differences:

Influenza surveillance is an important tool to identify emerging/re emerging strains, and defining seasonality. We describe the distinct patterns of circulating strains of the virus in different areas in India from 2009 to 2013. Patients in ten cities presenting with influenza like illness in out-patient departments of dispensaries/ hospitals and hospitalized patients with severe acute respiratory infections were enrolled. Naso pharangeal swabs were tested for influenza viruses by real-time RT-PCR, and sub typing; antigenic and genetic analysis were carried out using standard assays[12,13,14,15]. Geographically, India falls in the tropical region between the equator and Tropic of Cancer (23.4°N) and the subtropical region with latitude less than 40°N. We

examined the relationship between influenza positivity and the latitudes of the capital city of each state. Srinagar at the latitude of 34°N had an influenza peak in the winter, whereas most of the cities a latitude ‹30°N had influenza peaks during summer monsoon months (July-September), with Chennai and Vellore located at the south-west location have peaks in November-December. Rainfall correlated with influenza peaks in all cities except Srinagar

City, State	Latitude	Peak Seasonality	Proportion of Influenza Positive (%)	
			June-November	December-May
Srinagar, Jammu and Kashmir	34°0	Dec-Feb	13.3	86.7
Delhi	28°6	Jul-Sep	69.7	30.31
Dibrugarh, Assam	27°5	Jun-Jul	73.4	26.6
Lucknow, Uttar Pradesh	26°8	Jul-Sep	81.6	18.4
Kolkata, West Benga	22°6	Jun-Jul	68.2	31.8
Nagpur, Maharashtra	21°2	Jul-Aug	73.3	26.7
Pune, Maharashtra	18°5	Jul-Aug	61.2	38.8
Alappuzha, Kerala	9°5	May-Jul	70.9	29.1
Chennai, Tamil Nadu	13°1	Nov-Dec	37.7	62.3
Vellore, Tamil Nadu	12°9	Nov-Dec	58.4	41.6

Fig1: Relationship between geographic location, influenza seasonality

Of the 44,127 ILI/SARI cases, 6,193 (14.0%) were positive for influenza virus. Peaks of influenza were observed during July-September coinciding with monsoon in cities Delhi and Lucknow (north), Pune (west), Allaphuza (southwest), Nagpur (central), Kolkata (east) and Dibrugarh (northeast), whereas Chennai and Vellore (southeast) revealed peaks in October- November, coinciding with the monsoon months in these cities. In Srinagar (Northern most city at 34°N latitude) influenza circulation peaked in January-March in winter months. The patterns of

circulating strains varied over the years: whereas A/H1N1pdm09 and type B co-circulated in 2009 and 2010, H3N2 was the predominant circulating strain in 2011, followed by circulation of A/H1N1pdm09 and influenza B in 2012 and return of A/H3N2 in 2013. Antigenic analysis revealed that most circulating viruses were close to vaccine selected viral strains.

7. Conclusion:

Despite the increasing amount of research and knowledge on the seasonality of influenza, the seemingly simple question of why epidemics in temperate regions occur in the wintertime continues to remain elusive. Indeed, there are a number of characteristics regarding influenza prevalence that are still not fully understood in the context of prevailing weather and climate conditions: (i) the timing, or onset of an influenza epidemic; (ii) the severity of a given epidemic; (iii) the spatial distribution, or spread of influenza infection; (iv) the variability in timing and severity among individual epidemics; and (v) the apparent disappearance of influenza during the summer season, with only occasional isolated occurrences. Moreover, it is unclear why certain viral strains, such as the currently circulating novel H1N1, seem to emerge out of phase with the more common seasonal avian strains, such as H3N2 (Dowell and Ho 2004). India, located in northern hemisphere, has distinct seasonality that might be related to latitude and environmental factors. While cities with temperate seasonality will benefit from vaccination in September-October, cities with peaks in the monsoon season in July-September will benefit from vaccination in April-May.

8. References

1. Allan, R., Lindesay, J. and Parker, D. (1996). El Nino Southern oscillation and climatic variability. Victoria, Australia: CSIRO Publishing.
2. Alonso, W. J., et al. (2007). Seasonality of influenza in Brazil: a traveling wave from the Amazon to the Subtropics. American Journal of Epidemiology 165, pp. 1434–1442.
3. Assaad, F. A. and Reid, D. (1971). Some factors influencing mortality from influenza. Bulletin of the World Health Organization 45, pp. 113–117.
4. Baetjer, A. (1967). Effect of ambient temperature and vapor pressure on cilia-mucus clearance rate. Journal of Applied Physiology 23, pp. 498–504.

5. Brankston, G., et al. (2007). Transmission of influenza A in human beings. Lancet 7 (4), pp. 257–265.
6. Cannell, J. J., et al. (2006). Epidemic influenza and vitamin D. Epidemiology and Infection 134, pp. 1129–1140.
7. Cannell, J. J., et al. (2008). On the epidemiology of influenza. Virology Journal 5, pp. doi: 10.1186/1743-422X-5-29.
8. Choi, K. M., Christakos, G. and Wilson, M. L. (2006). El Nino effects on influenza mortality risks in the state of California. Public Health 120, pp. 505–516.
9. Comrie, A. (2007). Climate change and human health. Geography Compass 1/3, pp. 325–339. Confalonieri, U., et al. (2007)
10. P. J. and Hanson, C. E. (eds) Climate Change 2007: impacts, adaptation and vulnerability. contribution of Working Group II to the Fourth Assessment Report of the Intergovernmental Panel on climate change, Cambridge, UK: Cambridge University Press, pp. 391–431.
11. Davis, R. E., Knappenberger, P. C., Michaels, P. J. and Novicoff, W. M. (2004). Seasonality of climate-human mortality relationships in US cities and impacts of climate change. Climate Research 26, pp. 61–76.
12. Simonsen L. The global impact of influenza on morbidity and mortality. Vaccine. 1999; 17Suppl 1:S3–10. Epub 1999/09/02. PubMed PMID: 10471173.
13. Moura FE. Influenza in the tropics. Current opinion in infectious diseases. 2010; 23(5):415–20. Epub 2010/07/21. doi: 10.1097/QCO.0b013e32833cc955 PubMed PMID: 20644472
14. Chadha MS, Broor S, Gunasekaran P, Potdar VA, Krishnan A, Chawla-Sarkar M, et al. Multisite virological influenza surveillance in India: 2004–2008. Influenza and other respiratory viruses. 2012; 6(3):196–203. Epub 2011/10/01. doi: 10.1111/j.1750-2659.2011.00293.x PubMed PMID: 21955356.
15. Broor S, Krishnan A, Roy DS, Dhakad S, Kaushik S, Mir MA, et al. Dynamic patterns of circulating seasonal and pandemic A(H1N1)pdm09 influenza viruses from 2007–2010 in and around Delhi, India. PloS one. 2012; 7(1):e29129. Epub 2012/01/12. doi: 10.1371/journal.pone.0029129 PubMed PMID: 22235265; PubMed Central PMCID: PMC3250412.