

Seasonality of Respiratory Viruses Including SARS-CoV-2

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Abstract

We propose that a reservoir of respiratory viruses in clumps of micro-sized dust exists in tropospheric clouds from which virions can be seasonally released into the lower atmosphere and thence to ground level. Respiratory Syncytial Virus (RSV), Seasonal Influenza and Human Para Influenza Virus (HPIV) are all diseases that fall in this category, including SARS-CoV-2. The seasonal incidence of disease at ground level would appear to be patchy over distance scales that are largely dictated by viral-laden dust cloud size modulated by scales of atmospheric turbulence. This could produce clustering of cases in space and time that has given rise to 'contagion' concepts of community spread and of superspreaders.

Keywords: Seasonal respiratory viruses • Troposphere • COVID-19

Introduction

The presence of microorganisms in the troposphere (8-10 km), their role in the nucleation of ice crystals as well as the seeding of rain clouds has been known for many years [1]. However, the role they play in atmospheric physics and biological processes, more generally, still remains poorly understood. Recent studies by Rodriguez et al. [2] have shown that this region of the troposphere actually constitutes a substantial microbiome with a large fraction of micrometre-sized aerosols that are actually associated with bacteria. More recently Smith et al. [3] have reported the existence of a more or less homogeneous distribution of bacteria extending into the stratosphere up to 12 km so defining what can be described as an extended terrestrial biosphere. It is evident that a largely unknown and diverse microbial habitat including bacteria and viruses (possibly embedded in electrically charged clumps of dust) is present in the upper tropospheric clouds. This microbial ecology would inevitably have a continuing connection and interchange with regions of the atmosphere both above in the stratosphere and below through the troposphere closer to the surface. This connection would involve the periodic recycling of viruses between the tropospheric reservoir and the biosphere at ground level.

The most recent study by Reche et al. [4] does in fact show such a connection and implies a steady deposition and/or re-deposition of viruses numbering $\sim 10^9$ m⁻² day⁻¹. However, the work of Reche et al. [4] quantified

the virus deposition using a previously used FACS algorithm [5] and not by direct analysis of infection. There are other independent studies [6] that show viruses being involved in atmospheric recycling that can remain viable and infective after long-range atmospheric transport and circulation. Against this backdrop the emergence from time to time of disease-causing viruses in the human population should occasion no surprise.

Seasonal Deposition of Atmospheric Viruses

It has been known for some time that many respiratory viruses have a distinctly seasonal pattern of incidence. These include RSV (Respiratory Syncytial Virus), seasonal influenza and HPIV (Human Para Influenza Virus), as well as a number of common cold viruses. The reason for their predominance in the winter seasons in both northern and southern temperate latitudes has remained a mystery from the time of the earliest discussions by Sir Andrewes C [7]. The current status of this dilemma is summarised in a recent paper by Price et al. [8]. Although social and behavioural causes, such as people tending to stay indoors in winter have been discussed, there is no compelling case for such causal associations. On the other hand, physical environmental factors such as ambient temperature, humidity and the flux of ultraviolet radiation that vary through the seasonal cycle have shown promising signs of a correlation. This is illustrated in the humidity-

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HPIV-3 virus correlation given by Price et al. [8], but decisive proof of a causal connection with any environmental factor has been much harder to establish (Figure 1).

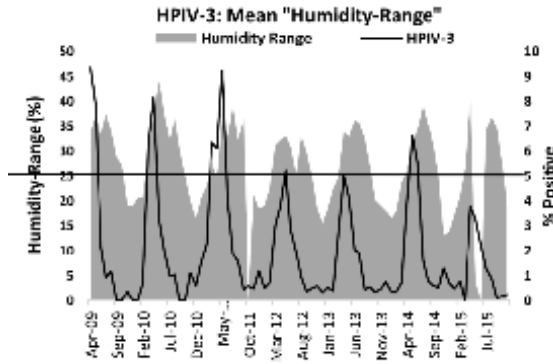


Figure 1. The seasonality of HPIV incidence compared with percent humidity (from Price et al. [8]).

However, what happens during the winter months in both the northern and southern hemispheres, 6 months apart, is the phenomenon of large-scale atmospheric recycling and turbulence leading to mixing through different layers in the atmosphere. This is in remarkable correspondence with the worldwide seasonality of non-pandemic influenza as seen in the data collated in 1977 by Hoyle and Wickramasinghe [9] for two northern and southern latitude countries, as well as for an island in the tropics (Sri Lanka) where no distinct seasonality is seen Figure 2.

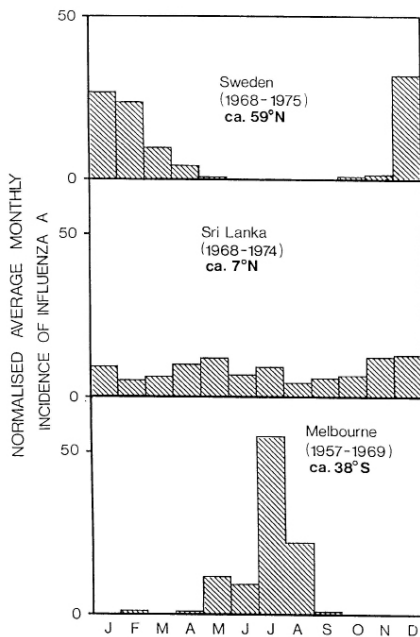


Figure 2. Normalised average influenza incidence showing seasonality – Sweden, Sri Lanka, Melbourne (from Hoyle and Wickramasinghe, Diseases from Space [7]).

Reche et al. [1] report that the viruses reaching the peaks of the Sierra Nevada mountain range were associated with organic aerosols of sizes less than $<0.7 \mu\text{m}$. In the case of respiratory viruses the aerosols could well have been contained in virus/cell aggregates. Within such aggregates it is possible that viruses may have remained viable in the upper tropospheric clouds over several months. In this way a circulation of viable virus of the type Ground \leftrightarrow Troposphere [1] might be imagined, and the seasonality of certain respiratory viral diseases could thus be explained.

Evidence of seasonal deposition of micron-sized dust from the stratosphere

The evidence for the existence of a transient biosphere, capable of harbouring bacteria and viruses that extends even into stratospheric clouds,

can be linked to early studies by Kalkstein [10]. In the last of a series of atmospheric nuclear bomb tests on August 11, 1958 a radioactive tracer, Rh-102 was introduced into the stratosphere at a height above 50 km and the incidence of the tracer was measured annually thereafter by means of airplane flights at altitudes ~ 10 km. It was discovered that the tracer took nearly a decade to clear itself through repeated seasonal downdrafts as seen in Figure 3. Noting that the ordinate scale in Figure 3 is logarithmic, we find that the fall-out of Rh-102 is very much greater in temperate latitudes than elsewhere, with the period January to March showing up as the dominant months. The notion that a similar incidence would be seen in southern temperate latitudes with an expected 6-month time lag was unfortunately not investigated by Kalkstein [10].

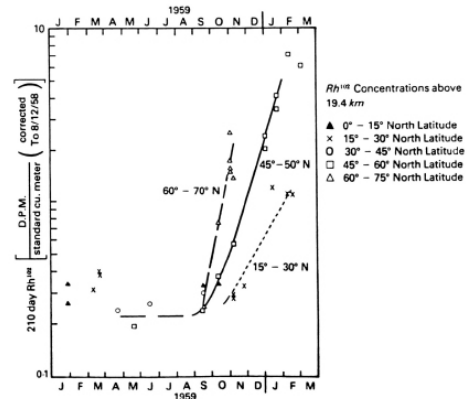


Figure 3. The fall out of Rh-102 at various latitude intervals from the HARDTACK atmospheric nuclear bomb that was exploded on 11 August 1958.

Since a large fraction of the viruses discovered by Reche et al. [1] At 3 km altitude was thought to be associated with wind-borne dust, the question arises as to whether during the COVID-19 pandemic a substantial population of suitably protected SARS-CoV-2 virions would have been lofted into the tropospheric reservoir. Tropospheric clouds may thus be regarded as a transitional inter-seasonal reservoir from which further depositions may occur in a seasonal cycle. This circuit requires a significant degree of survival of viruses in the stratosphere for ~ 6 months, and such survival within cryogenically preserved electrically charged dust clumps appears plausible. If this is indeed the manner by which seasonal influenza, the RSV and common cold viruses maintain their seasonal character it is not surprising to find SARS-CoV-2 behaving in a similar manner. The second wave of the COVID-19 pandemic can thus be understood in these terms with primary incidence at ground level displaying patchiness over scales of turbulence ranging from hundreds of kilometers to tens of meters. This is schematically illustrated in Figure 4, the shaded patches representing the scale of turbulence defining incidence at the ground.

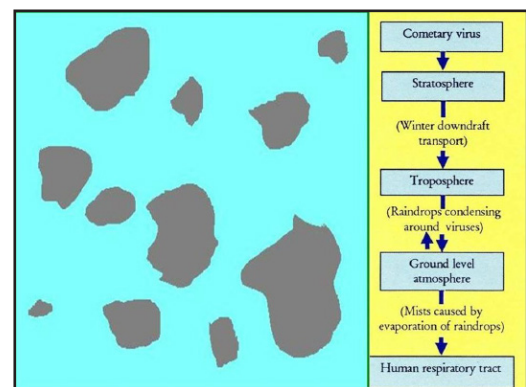


Figure 4. Schematic distribution of patches containing infective dust at ground level, and flow chart of presumed route from the atmosphere. The up-down arrows define the cycle leading to a transient tropospheric biosphere.

Patchiness of incidence over diverse distance scales

This is the pattern of incidence that was seen in many of the pandemics and epidemics of influenza that were studied by Hoyle and Wickramasinghe [9] in the book *Diseases from Space*. One crucial study related to the H1N1 (Red Flu) pandemic of 1976. This subtype of influenza was not in circulation for over 20 years, so its re-emergence as a pandemic strain gave Hoyle and one of us (Wickramasinghe) an opportunity to consider school children with “virgin” immunities as detectors of the virus. This study revealed a pattern of exceedingly variable attack rates in schools across England and Wales showing a patchiness of incidence over distance scales ranging from tens of kilometres to hundreds of metres. At the time of the epidemic at Eton College (a famous English boarding school near Windsor) there were 1248 pupils residing in 25 houses. The total number of influenza cases at this school was 441 giving an average attack rate 35%. The disparities of attack rates from house to house were striking. College House which had a total of 70 pupils had only 1 case, whereas another house with a population of 51 pupils had 46 cases with the scale of separation between houses being typically hundreds of metres. The fluctuations from the mean attack rate in units of the sample standard deviation are shown in Figure 5.

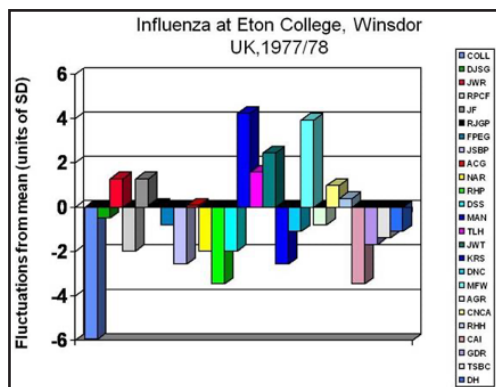


Figure 5. Fluctuation in attack rates from house to house, in terms of a sample standard deviation.

The probability of this distribution arising from a person-to-person infective process was estimated as 1 in 10^{10} (Hoyle and Wickramasinghe, [9]). Simultaneity of onset as displayed in the schematic patchiness in Figure 4, combined with the statistics of the type laid out in Figure 5 would give rise to a concept of a “superspreader” starting off each outbreak within a “patch”, particularly evident in the school houses at Eton with attack rates that are 4 standard deviations above the mean.

Whereas an approximate simultaneity of initial outbreak within each patch (Figure 4) is to be expected by virtue of the cloud fragmentation/infall process that we have considered, person-to-person spread would then take over as a secondary process of transmission that has the possibility of limitation through human interventions. Strong evidence for such a model of patchy incidence is to be found in the many instances of outbreaks of COVID-19 reported in ships at sea as documented in Howard et al. [11] (And reviewed further in detail in Steele et al. [12] and Supplementary File showing further recent evidence for patchy incidence COVID-19 outbreaks). While in some cases a shore connection of a primary infected source/individual have been identified, very many cases still remain unexplained. These constitute examples akin to the cases of the two Eton school houses where the attack rate is 4 sample standard deviations above the mean. For our more detailed analysis of the COVID-19 pandemic based on a primary atmospheric infall followed by secondary person to person infection we refer the reader to our recent earlier publications [13-17]. In a forthcoming paper we shall discuss the largest scales of patchiness of incidence of COVID-19, across continents and specifically within Australia and individual states in the USA, that can at least partly be explained by the largest scales of patchiness such as displayed in Figure 4.

Conclusion

Turning to smaller scales of patchiness, we draw attention to breaking news of a new large cluster of COVID-19 that was reported recently in Sri Lanka. More than 1,000 workers out of a total 1700 in a garment factory in the island have tested positive for COVID-19 within a two-day period in October this year. Until this episode there had been no recently reported incidents of “community spread” (infective infall in our view) in Sri Lanka. The COVID 19 that was present was confined to only a few cases directly traceable to workers returning to the island from foreign countries.

Clearly the new very large and localised cluster displayed in the data of Figure 6 can only be consistent with a patchiness of incidence in an infall process such as we have discussed. Unsurprisingly the authorities have been unable to identify the source of the outbreak, nor indeed a superspreader to account for this event.

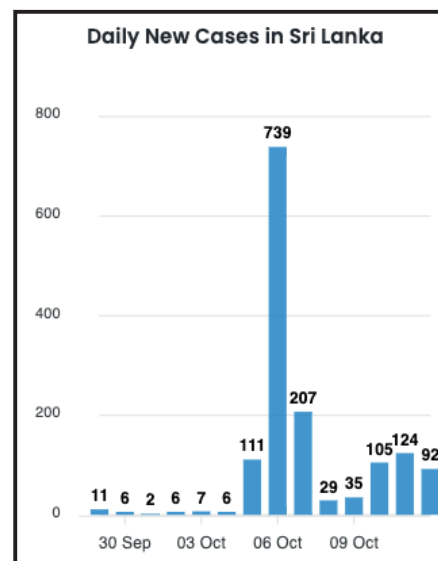


Figure 6. The Sri Lanka Health Department Covid 19 Live situational analysis dashboard of October 2020 give the data displayed above.

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