Climate, Severe Acute Respiratory Syndrome (SARS) and Avian Flu

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Background

This report is written following a recommendation of the 56th session of the WMO Executive Council (2004) to expand climate and human health studies to include emerging diseases, and the invitation of the president of the WMO Commission for Climatology to report on the climate aspect of Severe Acute Respiratory Syndrome and Avian Flu.

Introduction

Severe Acute Respiratory Syndrome (SARS) is a previously unknown coronavirus. In a few months between November 2002 and summer 2003, SARS infected more than 8,000 people and claimed more than 900 lives worldwide. Hong Kong, China was among the hardest hit having a total of 1,755 cases with 300 deaths out of a population of 6.8 million (SARS Expert Committee, 2003). There were several outbreaks in hospital and a serious community outbreak at the Amoy Gardens residential estate. In Guangdong Province in southern China, a large proportion of the first SARS patients worked in kitchens or wildlife markets. In Canada, transmission was largely confined to health care settings. Details can be found in SARS Expert Committee (2003), Chau and Yip (2005) and WHO (2003).

A number of investigations have found that SARS is a disease that is spread by direct contact or by virus-laden droplets that travel only a few metres (WHO, 2003). There are also suggestions that SARS can be spread by lighter airborne particles (Yu et al., 2004; Booth et al., 2005). As the causative agent of SARS is a virus of the corona family, and as about one third of all common colds which show a winter and spring seasonality are caused by viruses from the same family, seasonality may be a contributing factor for SARS (Abdullah, 2003). This report provides a review of studies in this regard.

In recent years, it has emerged that the Avian Flu associated with the H5N1
virus has the ability to infect humans. The activity of H5N1 is known to peak from November to March (WHO, 2005). The seasonal characteristics of human infections by this virus are also reviewed here.

SARS

Regional scale

Outbreaks in different regions over the world followed one another closely in time, starting in southern China in November 2002. SARS affected 26 countries in Asia, North America, the South-West Pacific, Africa and Europe between November 2002 and July 2003. There is evidence to show that the disease had spread from one region to another by travelling individuals. The introduction of screening and other related measures by various countries at airports, ports and border points was instrumental in containing the disease.

Local scale

Zhang et al. (2004) showed that for the two cities Beijing and Hong Kong, the incidence of SARS is highly correlated with air temperature and pressure, with cold air outbreaks quite likely serving as a triggering mechanism. They also found that high incidences of SARS were associated with reduced solar radiation, probably on account of the resulting decrease in UV radiation. Further, they devised a SARS High Danger Meteorological Index based on the daily number of confirmed SARS cases and the daily temperature to indicate the risk. This showed that both Beijing and Hong Kong shared the attribute of having large indices when the maximum temperature was between 24°C and 27°C.

Similarly, in their initial investigation using data from the four cities Hong Kong, Guangzhou, Beijing and Taiyuan, Tan et al. (2005) found that the optimum environmental temperature associated with SARS cases was between 16°C to 28°C, a range which might be favourable for virus growth. They also found that the average number of SARS patients increased markedly with the cumulative decrease in the maximum temperature in the previous 10 days, mostly due to surges in the winter monsoon. Human vulnerability to temperature change has been discussed by Hales et al. (2003) and the seasonality of diseases such as Influenza by Dowell (2001) among others.
**Microscale**

At the microscale (horizontal dimension of 2 km or less, see Orlanski 1975) a serious community outbreak of SARS in Hong Kong occurred in late March 2003 at Amoy Gardens, a private housing estate. By mid-April some 320 had become affected by SARS out of a total of some 19 000 residents (SARS Expert Committee, 2003). Such a high degree of clustering suggested the likelihood of an environmental point source outbreak (Lai et al., 2004). At Amoy Gardens it was probable that an index patient (often called a super virus carrier) in one residential block initially infected a relatively small group of residents in that block and subsequently the rest of the residents in that block through the sewerage system, person to person contact and the use of shared communal facilities such as lifts and staircases. The bathroom floor drains with dried-up U-traps provided a pathway through which residents came into contact with small droplets containing viruses from the contaminated sewage. These residents subsequently transmitted the disease to others both within and outside the block through person to person contact and environmental contamination. DH (2003) found that transmission of the disease by airborne and waterborne routes was not supported by the epidemiological picture and laboratory results.

On the other hand, using airflow-dynamics modelling Yu et al. (2004) hypothesized that this large community outbreak might be explained by the spread of the virus carried on aerosols from one building to another under the action of the north-easterly winds. It was also possible that after the physical decay of the virus-laden aerosol plume, the prevalent mode of exposure could still be contact with inanimate materials or objects contaminated by the infection-competent virus, rather than airborne exposure (Nicastri et al., 2004).

The mean incubation period for SARS in Hong Kong was determined to be 6.37 days (WHO, 2003). Figure 1 shows that this community outbreak coincided with a mark drop in temperature in Hong Kong due to the passage of a cold front some six days previously. This is in line with the observations of Zhang et al. (2004) and Tan et al. (2005). The out-break also coincided with a period of comparatively higher relative humidity due to the rainy and misty conditions brought by the front, as well as with a period of northeasterly winds which as noted by Yu et al. (2004) might have carried the virus downwind to cause infections in some of the residents living in other blocks.
Figure 1. Epidemiology curve of SARS outbreak at Amoy Gardens and time series of maximum temperature (top panel), mean relative humidity (middle panel) and wind direction (lower panel) 6 days before the outbreak.
Li et al. (2004) and Yu et al. (2005) have used computational fluid dynamics to provide environmental evidence for an airborne transmission route for SARS in a hospital ward in Hong Kong. Booth et al. (2005) and Tong (2005) also suggest that SARS could be an opportunistic airborne infection in hospital wards.

**Predictability**

While it is not possible to determine the seasonal nature of SARS with limited data from only one global outbreak, the studies of Zhang et al. (2004) and Tan et al. (2005) suggest that, at the local scale, SARS outbreaks are significantly related to the air temperature and its variation. At the microscale the Amoy Gardens community outbreak in Hong Kong provides an indication of the possible influence of the weather in aiding transmission when a highly pathogenic source is introduced into specific building settings. The reduction in SARS incidences with warmer weather suggests that seasonality might play a contributing role, although should it recur its epidemiology might be different (Abdullah, 2003). The investigations of Yu et al. (2005), Li et al. (2004) and Booth et al. (2005) also point to the potential for an airborne spread of the SARS virus in hospital settings.

Notwithstanding opposing views from different studies, there is tentative evidence to suggest that SARS is capable of opportunistic airborne transmission, and that the special circumstances of the outbreak at Amoy Gardens may be a harbinger of unorthodox transmission patterns associated with emerging infective agents in the modern urban environment (Chad and Milton, 2004).

However, seasonality if confirmed cannot by itself be used to predict the next outbreak of SARS. This is because the occurrence of SARS epidemics depends on the interplay between the three factors host, agent, and environment (Lee, 2003).

**Avian Flu**

**Documented outbreaks**

The world’s first case of human infection by the H5N1 strain of Avian Influenza was documented in 1997 in Hong Kong, China (WHO, 2004(a)), involving the death of a three-year old boy who was infected in May 1997.
After a lull of five months, the infections resurfaced with four cases occurring in November, and another 13 in December. There was thus a total of 18 cases of confirmed H5N1 infections in humans in Hong Kong, China in 1997, with 17 of them occurring between November and December. Six of these 18 cases were fatal (Choi and Tsang, 1998; Sims et al., 2003).

Two more cases of H5N1 infections in humans in Hong Kong, China were documented in February 2003, and one of the two infected patients died (WHO, 2005). These occurrences coincide with the peak influenza season in Hong Kong, China which is usually between January and March (DH, 2004).

In late 2003 and early 2004, outbreaks of the H5N1 virus caused lethal illness in a number of Asian countries. In January 2004 human H5N1 cases were confirmed in Thailand and Viet Nam (WHO 2004(a)). Between January and October 2004 Thailand recorded 17 confirmed cases among which there were 12 fatalities; Viet Nam 27 cases of which 20 were fatal. Following a lull of about two months, a new outbreak seems to have occurred in mid-December 2004 in Viet Nam and Cambodia and is still ongoing. It has so far claimed two lives in Cambodia out of the two cases reported there and 15 lives out of 33 cases in Viet Nam (CIDRAP, 2005). Regular updates on the status of the Avian Flu are given by WHO on its Website: http://www.who.int/wer/2005/wer8013/en/.

Predictability

Avian Flu outbreaks in humans appear to demonstrate a tendency to occur in winter and spring months. Available evidence points to an increased risk of transmission in humans when the H5N1 Influenza is widespread in poultry (WHO, 2004(b)). The seasonality in outbreak in humans is probably a reflection of the peak activity of the H5N1 virus from November to March. This link remains to be confirmed, however.
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