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Influenza seasonality affected by the 2009 pandemic episode in Senegal

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In Senegal, the seasonality of influenza epidemics is well defined with a clear peak around August and September (in rainy season which occurs from July to October). Surprisingly, the first detection of the A(H1N1)pdm09 virus was in January 2010 and rapidly reached a high detection peak between January and February, indicating a real shift in the influenza seasonality in Senegal. Therefore, climatic factors, the host susceptibility seem insufficient to explain the epidemiology of this virus which is quite different as compared to that of seasonal viruses. Intrinsic properties of this virus may play a role in its seasonal behavior.

Key words: influenza, A(H1N1)pdm09, pandemic, seasonality, epidemiology.

INTRODUCTION

In late March of 2009, a new influenza virus, a quadruple reassortant H1N1 virus, emerged in Mexico (Neumann et al., 2009; New South Wales Public Health Network, 2009). The virus, A(H1N1)pdm09, spread rapidly around the world, prompting the World Health Organization (WHO) to declare the first influenza pandemic of the 21st century on June 11, 2009 (Enserink and Cohen, 2009). However, this virus was not detected in the African continent till lately, notably in West Africa (Nzussouo et al., 2012). Senegal was probably one of the last West African countries where the virus was detected, despite the well-established network for influenza virus monitoring. Indeed the pandemic virus was not detected in Senegal until the first week of 2010, months after being reported by other West African countries (e.g. Ivory Coast, Cape Verde, etc.). The analysis of the circulation profile of the new virus showed differences as compared to seasonal influenza viruses, a situation which has significantly altered the seasonality of influenza in Senegal in the following years (Niang et al., 2012).

MATERIALS AND METHODS

A prospective observational study was conducted during 2009,

2010 and 2011. Senegal, a Western Africa country, has a well-established surveillance system with several Influenza sentinel sites located in urban, sub-urban and rural. At each sentinel site trained physicians identified all influenza-like illness (ILI) cases presenting at the clinics from Monday to Friday. An ILI cases were identified as an outpatient presenting with sudden onset of fever (≥38°C) and cough or sore throat accompanied or not by myalgia, prostration, headache or malaise, with the onset of symptoms occurring within the previous three days. A standardized form was used to collect demographic and clinical information from the enrolled patients.

Nasal-pharyngeal and oral-pharyngeal swabs were collected from all enrolled ILI cases, placed in 2 ml cryovials containing viral transport medium (Universal Transport Medium, COPAN Diagnostics Inc., Murrieta, CA, USA) and stored at 4°C on site. Upon arrival at the laboratory the specimens were processed immediately for virus detection, identification and characterization.

RNA was extracted from 200 µl of each sample using the QIAamp Viral RNA kit (QIAGEN, Valencia, CA, USA**)** according to the manufacturer's instructions. One step real-time RT-PCR was performed using the ABI 7500 platform according to the CDC protocol for the identification of influenza A(H1 and H3) and B viruses (courtesy of the Centers for Disease Control, Atlanta) during the pandemic episode. Influenza A positives samples are run in a second real time RT-PCR for the subtyping using primers targeting haemagglutinin genes of seasonal (H1 and H3) viruses and A(H1N1)pdm09 (CDC, 2009a).

The weekly rainfall data over the 3 years were collected from the National Meteorological Department of Senegal with the aim to

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Table 1. Detection rates of influenza virus by type and subtype in Senegal during the years 2009, 2010 and 2011.

Influenza virus	Years (tested samples number)			
Virus number (%)	2009 (936)	2010 (1328)	2011 (922)	Total (3186)
Total/an	274 (29.3%)	443 (33.4%)	246 (26.7%)	963 (30.2%)
Inf B	0	76 (5.7%)	109 (11.8%)	185 (5.8%)
Inf A	274 (29.3%)	367 (27.6%)	137 (14.9%)	778 (24.4%)
H1N1	3 (0.3%)	0	0	3 (0.1%)
H3N2	271 (28.9%)	22 (1.7%)	81 (8.8%)	374 (11.6%)
H1N1pmd09	0	345 (26%)	56 (6.1%)	401 (12.6%)

analyze the behavior of the different influenza viruses with respect to rainfall.

RESULTS

During this study period 3186 samples from patients with ILI were analyzed (Table 1): 936 (29.4%) during 2009, 1328 (41.7%) during 2010 and 922 (28.9%) during 2011. Patient ages ranged from 1 month to 96 years with a mean age of 3 years. 63% of the ILI cases during this period are children between 0 and 5 years age. The male to female ratio was 1:1 (1512 females versus 1674 males).

Of the 3186 samples analyzed, 963 (30.2%) were positive for influenza virus using the real-time RT-PCR method (Table 1). Among these positive, 778 (80.8%) were influenza A, and 185 (19.2%) were influenza B. Of the influenza A positives, 3 (0.4%) were seasonal H1N1, 374 (48.1%) were H3N2 and 401 (51.5%) were H1N1pdm09. The three H1N1 are detected in 2009. The pandemic virus was detected only from 2010 in Senegal (no case detected in 2009).

With regard to the temporal distribution of influenza positives samples during the 3 years of surveillance, we observed different profiles (Figure 1). For the year 2009 (Figure 1A) no influenza virus was detected until week 20 from which the first H3N2 cases were detected. The H3N2 subtype was the major influenza virus detected during this year with a peak around week 27 (beginning of July). The virus continued to circulate at a significant level until week 43 and then gradually disappeared. The end of year 2009 is marked by three cases of seasonal H1N1 (detected at weeks 51 and 52). If we consider the rainfall curve, we note that significant virus circulation coincides with the onset of rains.

The year 2010 (Figure 1B) began with the first cases of the pandemic virus in Senegal (with 2 cases at the week 1). H1N1pdm09 cases increase gradually and reach a peak around the weeks 6, 7 and 8 and disappear at the week 12. The influenza B type appeared at the week 17 with a low circulation level during the following weeks and disappeared at the week 46. Influenza B virus circulates mainly during the raining period. The pandemic virus reappeared at week 38 and circulated at a low level until week 45. This reappearance coincided with the end of the rainfall. Few cases of H3N2 are detected during this period between weeks 32 and 44.

For 2011 (Figure 1C), only the pandemic virus circulated between weeks 5 and 27 and disappeared. The influenza B type circulation started at week 31 and reached a peak around weeks 37, 38 and 39. During these weeks, it co-circulated with the H3N2 subtype which attained a circulation peak at week 42. Circulation peaks of these two viruses are preceded by rainfall peak recorded at week 34. The pandemic virus reappeared at the week 37 and circulated at a very low level when compared with the others (Flu B and H3N2 subtype) until week 43.

DISCUSSION

In Senegal, the pandemic began in the first week of year 2010 and rapidly reached peak detection during weeks 6, 7 and 8. It is well known that influenza epidemics occur annually worldwide, and display a seasonal pattern in temperate areas, with marked peaks in the winter (typically December-April in the Northern Hemisphere and June-September in the Southern Hemisphere) (Viboud et al., 2006). Tropical and subtropical regions are also subjected to seasonal oscillations in influenza incidence, which have been linked to rainy seasons (Shek and Lee, 2003), even if the seasonal pattern was generally less pronounced than in temperate areas (Viboud et al., 2006). In Senegal, a sub-Saharan country, the seasonality of influenza epidemics has been well defined after 16 years of regular influenza monitoring (Niang et al., 2012).

Indeed there is a clear peak of influenza cases around August and September (in the middle of the rainy season which occurs from July to October) and a very low level of detection for the rest of the year. The first pandemic wave (March 2009) coincided with the end of the influenza season in the Northern Hemisphere and prior to the beginning of the influenza season in the Southern
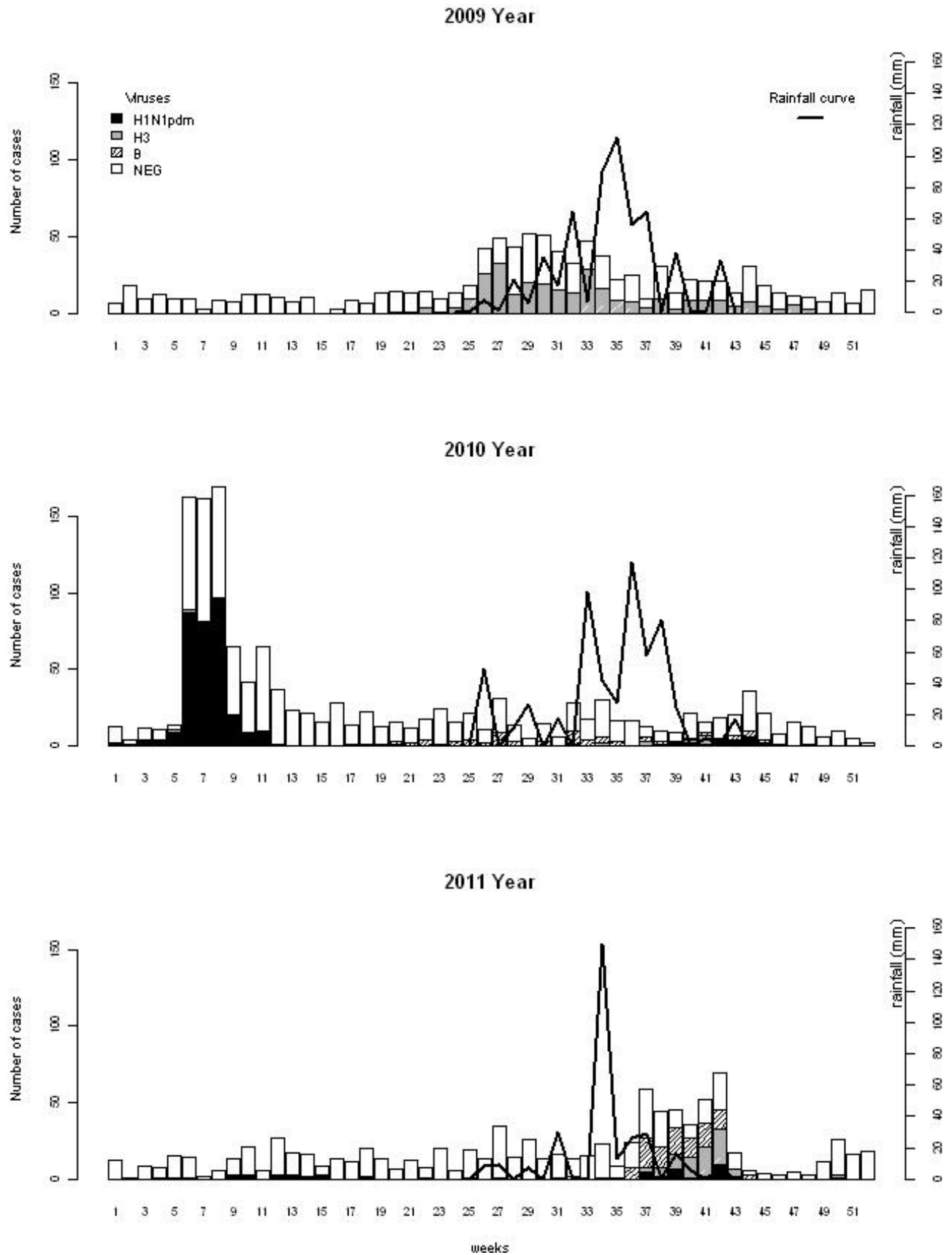
2009 Year

2010 Year

2011 Year

Figure 1. Distribution of weekly influenza positives and pluviometry in Senegal during years 2009, 2010 and 2011.

hemisphere. It would be anticipated that the pandemic virus would have sufficient time to reach Senegal for the regular influenza season; however, this did not occur. No cases of influenza A(H1N1)pdm09 were detected in Senegal during 2009. The 2009 influenza season in Senegal displayed a normal seasonal pattern with a peak between July and September which coincided with the rainy period.

The influenza A(H3N2) virus was the only circulating subtype during this period, although some seasonal A(H1N1) strains were detected at the end of the year (week 51 and 52). The A(H1N1)pdm09 arrived months after the end of the 2009 influenza season in the Senegal.

The reasons for the delayed circulation of A(H1N1)pdm09 in Senegal (and other West African countries) are not clear but unfavorable temperature and humidity conditions are indexed to explain some of the observed patterns (Lowen et al., 2008; Steel et al., 2010). However, this does not seem applicable to Senegal, as the conditions were favorable for seasonal influenza viruses. Although the international air transportation network was evoked by some authors in the spread of influenza viruses (Viboud et al., 2006), it would be inappropriate to use this argument to justify the delay in circulation of the pandemic virus in a city like Dakar (capital city of Senegal).

Considering the well-defined seasonal pattern of influenza circulation in Senegal (Mbayame et al., 2013), the arrival of the A(H1N1)pdm09 virus in January 2010, with the early seasonal peak detection (between January and February), was an atypical and yet unexplained event in the influenza seasonality in Senegal. Thus, for a complete description of the Senegal influenza season in 2010, it should be noted that the rainy season (typical peak influenza circulation) was marked by a disappearance of the pandemic virus and low level circulation of seasonal viruses (influenza B and A(H3N2)). The pandemic virus reappeared in early October, at the end of the typical rainy season. The A(H1N1)pdm09 virus continued to circulate the most in 2011, especially before the rainy season. During this rainy season, the pandemic virus co-circulated with seasonal viruses (influenza B and A(H3N2)), with a lower rate of detection.

With regards to the A(H1N1)pdm09 virus circulation and the annual rainfall distribution, we holistically observed a better circulation of the pandemic virus outside periods of heavy rain. The virus tended to slip away and to reappear at the end of rainy season. It should be noted that the rainy season in Senegal in 2011 started late, with a short duration (with a very low amount of rainfall), and certainly this would explain the circulation of the virus at this rate.

Thus it is clear that the pandemic virus has a seasonal flow profile significantly different from that of other seasonal viruses (the seasonal A(H1N1) having disappeared since the onset of the pandemic virus). To explain this lag in the circulation of the pandemic virus, we cannot rely entirely on the weather and climate factors although their influence on the epidemiology of influenza viruses is very clear and undeniable (Altizer et al., 2006; Christopher, 2010).

The proposed theory of Edgar Hope-Simpson based on the solar radiation influence on the host susceptibility (depletion of the innate immune system) also cannot be either the argument that may explain this difference (Hope-Simpson, 1992). Indeed, in the Senegalese climatic context the solar radiation levels remained normal despite the cold winter period.

We believe that the intrinsic properties of the pandemic virus may have contributed to the difference in circulation patterns as compared to seasonal viruses, allowing the virus to circulate normally outside of the rainy season to such a high level.

It is now well known that the survival of an influenza virus is determined primarily by the characteristics of its outer casing, or envelope, which is composed of lipid compounds, suggesting that the lipid envelope encasing the virus remains intact longer when the air is sufficiently cold and dry (Polozov et al., 2008). With regards to its high capacity to circulate outside influenza season (rainy season), the pandemic virus seems to have less demand in terms of air humidity, and therefore is able to survive longer in the air as compared to seasonal viruses.

Conclusion

As the pandemic period had been well-described, the post period also need to be explored to collect epidemiologic and biologic information on respiratory viral infections. A better global surveillance would certainly aid in providing insights into understanding the factors contributing to the circulation, transmission and virulence of influenza viruses.

REFERENCES

Altizer S, Dobson A, Hosseini P, Hudson P, Pascual M and Rohani P (2006). Seasonality and the dynamics of infectious diseases. Ecol. Lett. 9:467-84.

CDC (2009a). 2009 H1N1 flu: international situation update. Centers for Disease Control and Prevention, Atlanta, GA.

Christopher F. (2010). The Effects of Weather and Climate on the Seasonality of Influenza: What We Know and What We Need to Know. Geography Compass 4/7:718-730, 10.1111/j.1749-8198.2010.00343.x

Enserink M, Cohen J (2009). Virus of the year. The novel H1N1 influenza. Science 326:1607.

Hope-Simpson RE (1992). The transmission of epidemic influenza. New York:Plenum Press.

Lowen AC, Steel J, Mubareka S, Palese P (2008). High temperature (30 degrees C) blocks aerosol but not contact transmission of influenza virus. J. Virol. 82:5650-2.

Neumann G, Noda T, Kawaoka Y (2009). Emergence and pandemic potential of swine-origin H1N1 influenza virus. Nature 459:931-939.

New South Wales Public Health Network (2009). Outbreak of swine-origin influenza A (H1N1) virus infection - Mexico, March-April 2009. Morbidity and Mortality Weekly Report 58:467-470.

Niang MN, Dosseh A, Ndiaye K, Sagna M, Gregory V, Goudiaby D, Hay A, Diop OM (2012). Sentinel surveillance for influenza in Senegal, 1996-2009. J. Infect. Dis 206 (Suppl 1):S129-S135.

Nzussouo NT, Michalove J, Diop OM, Njouom R, Monteiro Mde L, Adje HK et al (2012). Delayed 2009 pandemic influenza A virus subtype H1N1 circulation in West Africa, May 2009-April 2010. J. Infect. Dis. 206 (Suppl 1):S101-S107.

Polozov IV, Bezrukov L, Gawrisch K and Zimmerberg J (2008). Progressive ordering with decreasing temperature of the phospholipids of influenza virus. Nat. Chem. Biol. 4:248-255.

Shek LP, Lee BW (2003). Epidemiology and seasonality of respiratory tract virus infections in the tropics. Paediatr. Respir. Rev. 4:105-11.

Steel J, Palese P, Lowen AC (2010). Transmission of a 2009 pandemic influenza virus shows similar sensitivity to temperature and humidity as an H3N2 seasonal strain. J. Virol. 85:1400-1402.

Viboud C, Alonso WJ, Simonsen L (2006). Influenza in tropical regions. PLoS Med. 3:e89.