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Human vs. Animal Outbreaks of the 2009 swine-origin H1N1 influenza A epidemic

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Abstract

The majority of emerging infectious diseases are zoonotic in origin, including recently emerging influenza viruses such as the 2009 swine-origin H1N1 influenza A epidemic. The epidemic that year affected both human and animal populations as it spread globally. In fact, before the end of 2009, 14 different countries reported H1N1 infected swine. In order to better understand the zoonotic nature of the epidemic and the relationship between human and animal disease surveillance data streams, we compared 2009 reports of H1N1 infection to define the temporal relationship between reported cases in animals and humans. Generally, human cases preceded animal cases at a country-level, supporting the potential of H1N1 infection to be a "reverse zoonosis", and the value of integrating human and animal disease report data.

Keywords

Influenza A Virus; H1N1 Subtype; Population Surveillance; Zoonoses; Disease Outbreaks

The majority of emerging infectious diseases are zoonotic in origin, including recently emerging influenza viruses such as the 2009 swine-origin H1N1 influenza A epidemic (Krauss 2003). The virus contained viral sequences related to human, avian and swine lineages (Garten et al. 2009) (Smith et al. 2009). It had a greater transmissibility than seasonal flu (Fraser et al. 2009), and caused significant fear around the world. While Mexico was one of the earliest countries with known outbreaks, the precise origin of the 2009 swine-origin H1N1 influenza A epidemic remains unclear (Fraser et al. 2009). The epidemic affected both human and animal populations as it spread globally. In fact, before the end of 2009, 14 different countries indicated that they had swine infected with the virus (Mathieu et al. 2010).

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To better predict and prepare for future epidemics, greater integration of human and animal disease surveillance has been recommended (Institute of Medicine (U.S.). Committee on Achieving Sustainable Global Capacity for Surveillance and Response to Emerging Diseases of Zoonotic Origin. and Keusch 2009). In fact, a systematic review by Vbova et al. found that of surveillance systems that collected human or animal data, only around 19% collected data on both (Vrbova et al. 2010). In order to better understand the zoonotic nature of the epidemic and the relationship between human and animal surveillance data streams, we compared for 2009, by country, the date of the initial report of human cases of the 2009 swine-origin H1N1 influenza A epidemic infection to the first report of disease in animals in that country.

Our source for human case reports of the 2009 swine-origin H1N1 influenza A epidemic infection was the HealthMap project (Brownstein and Freifeld 2007; Brownstein et al. 2008), a real-time monitoring system for emerging infectious diseases that had created a global online database for the H1N1 epidemic. To find the first report of human disease in a particular country, we used the first date identified by HealthMap from any source (formal or informal). For reports of the 2009 swine-origin H1N1 influenza A epidemic in animals, we used the EMPRES-i database, a global animal surveillance system maintained by United Nations Food and Agriculture Organization (FAO) (Welte and Vargas Teran 2004; FAO 2010), and noted the date when infection was first reported in an animal population in a particular country.

We also examined the effect of two additional country-level covariates, Gross Domestic Product (GDP) and density of swine in each country, on the difference in days between first animal and human case reports in that country. We chose to analyze density of swine because swine were an infected host in 83% of the first animal reports, by country, in EMPRES-i (Table 1). Data on country-level 2008 GDP was obtained from the United Nation's website of demographic and social statistics (2010). 2007 Swine density data was obtained from the Food and Agriculture Agency's Global Livestock Production and Health Atlas (GLiPHA) (2011). Data from 2008 or 2009 were not available from this source. For each covariate, categorical variables were created based on the statistical distribution of the underlying data, and were added to a linear regression model using SAS 9.2 (Cary, NC).

A total of 157 countries reported human cases. Twenty-one countries reported both animal and human outbreaks (Figure). No countries reported only animal cases.

The time between first human and animal cases varied from almost simultaneous to more than seven months (Table 2). The mean of the time lag was 143 days with a standard deviation of 65 days. In the case that was almost simultaneous (Canada), the subsequent investigation revealed early cases of human H1N1 infection had returned to the country from Mexico and then contacted livestock that became ill (OIE 2009). The fact that the animal case was reported before the human index case could be interpreted as an indicator of a very sensitive and timely animal surveillance network in that country. Countries where the time lag was relatively smaller (such as Chile and Argentina) may have sensitive surveillance, increased contact between humans and animals, or both.

We also analyzed the effect of our two covariates, country GDP and swine density on the difference in days between animal and human case reports. The results of the linear regression model indicate that swine density had a statistically significant impact with the difference in days ($p = .01$), while country GDP was not a significant predictor of the time lag ($p = .86$). When swine density was broken up into the quartiles low, medium, medium-high, and high density, three of the four categories were significantly associated with the time lag between human and animal cases (with greater swine density associated with a longer lag). The medium density category was not significant (p = .778).

There has been little very little integration in general between human and animal disease surveillance of the 2009 swine-origin H1N1 influenza A epidemic. Our comparison of a human and animal surveillance databases for infection indicates that as the epidemic spread, human cases preceded animal cases in countries, supporting the assumption that human-to-human transmission was driving disease spread. At the same time, many countries reported subsequent animal cases, indicating that “reverse zoonotic” transmission may have been occurring. However, additional work is needed to confirm this notion including the use of variables such as information related to human movement within infected areas as well as biological evidence that virus was not circulating before the dates reported. In addition, since there is evidence to suggest that the origin of the 2009 swine-origin H1N1 influenza A epidemic is from swine (Smith et al. 2009), another characterization of this phenomena is that of a *spillback,* where a virus is transmitted back into the original host from intermediate hosts (humans in this case). The comparison of human and animal surveillance data streams may also point out gaps in surveillance systems and delays in reporting. For example, Hosseini et al (Hosseini et al. 2010) found that countries with lower resource settings tended to have delays in reporting. Our analysis showed GDP not to impact the difference in days between reporting of animal and human cases. In addition other papers have examined GDP and reporting and found mixed results. For example, Reperant et al. (Reperant et al.) compared GDP per inhabitant of countries in Europe that reported cases of H5N1 in wild birds versus all of Europe and found no significance. However, Brownstein et al. (Brownstein et al.) used their HealthMap system to examine reports of 2009 swine-origin H1N1 influenza A and found that wealthier countries had shorter lag times between suspected and confirmed reports of human cases. Finally, there is the potential for GDP to bias reporting time of animal cases because of the perceived economic impact to livestock trade.

We found that country-level swine density was positively associated with a longer time lag between the reporting of human and swine cases. Explanations for this could include greater biosecurity in countries with higher swine density, less effective surveillance, or alternatively, a reluctance to report swine cases due to economic considerations.

In attempting to compare animal and human disease surveillance data, a key issue is the different factors driving the effectiveness of such surveillance in humans versus animals. Animal disease surveillance is often closely tied to economic factors and international trade in animal products (Ndiva Mongoh et al. 2008), while human disease surveillance is primarily a public health function. Concerns over infected food could cause significant financial impact for many years after an outbreak. Therefore, responses of the animal and human surveillance networks to an epidemic with zoonotic potential may differ for a number of reasons. Our analysis shows the feasibility of making such comparisons and the need for further investigation when there are large discrepancies between the data streams. Finally, creating an integrated surveillance system requires involvement of agencies at different levels from the local community to global agencies of health (Perry et al. 2007). These agencies must be involved at different levels of the surveillance processes from identification of a health issue to the response and feedback on that issue (Perry et al. 2007). Thus for zoonotic disease surveillance including influenza there needs to be strong integration of animal and human agencies of health at the local, state, and country-level including wildlife, agriculture, and public health, as well as a shared willingness to overcome barriers to reporting in all species.

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References

[Accessed on Apr 26, 2011] United Nations Statistics Division - Demographic and Social Statistics. 2010. http://unstats.un.org/unsd/demographic/products/socind/inc-eco.htm.

[Accessed on Apr 26, 2011] Global Livestock Production and Health Atlas (GLiPHA). 2011. http://kids.fao.org/glipha/.

Brownstein JS, Freifeld CC. HealthMap: the development of automated real-time internet surveillance for epidemic intelligence. Euro Surveill. 2007; 12:E071129–E071125. [PubMed: 18053570]

Brownstein JS, Freifeld CC, Chan EH, Keller M, Sonricker AL, Mekaru SR, et al. Information technology and global surveillance of cases of 2009 H1N1 influenza. N Engl J Med. 362:1731–1735. [PubMed: 20445186]

Brownstein JS, Freifeld CC, Reis BY, Mandl KD. Surveillance Sans Frontieres: Internet-based emerging infectious disease intelligence and the HealthMap project. PLoS Med. 2008; 5:e151. [PubMed: 18613747]

FAO. [Accessed on Jul 6, 2010] EMPRES-i: Global Animal Disease Information System. 2010. http://empres-i.fao.org/empres-i/home.

Fraser C, Donnelly CA, Cauchemez S, Hanage WP, Van Kerkhove MD, Hollingsworth TD, et al. Pandemic potential of a strain of influenza A (H1N1): early findings. Science. 2009; 324:1557–1561. [PubMed: 19433588]

Garten RJ, Davis CT, Russell CA, Shu B, Lindstrom S, Balish A, et al. Antigenic and genetic characteristics of swine-origin 2009 A(H1N1) influenza viruses circulating in humans. Science. 2009; 325:197–201. [PubMed: 19465683]

Hosseini P, Sokolow SH, Vandegrift KJ, Kilpatrick AM, Daszak P. Predictive power of air travel and socio-economic data for early pandemic spread. PLoS One. 2010; 5:e12763. [PubMed: 20856678]

Keusch, G. Institute of Medicine (U.S.). Committee on Achieving Sustainable Global Capacity for Surveillance and Response to Emerging Diseases of Zoonotic Origin. Sustaining global surveillance and response to emerging zoonotic diseases. Washington, DC: National Academies Press; 2009.

Krauss, H. Zoonoses : infectious diseases transmissible from animals to humans. 3rd edition. Washington, D.C: ASM Press; 2003.

Mathieu C, Moreno V, Retamal P, Gonzalez A, Rivera A, Fuller J, et al. Pandemic (H1N1) 2009 in breeding turkeys, Valparaiso, Chile. Emerg Infect Dis. 2010; 16:709–711. [PubMed: 20350395]

Ndiva Mongoh M, Hearne R, Khaitsa ML. Private and public economic incentives for the control of animal diseases: the case of anthrax in livestock. Transbound Emerg Dis. 2008; 55:319–328. [PubMed: 18786071]

OIE. [Accessed on Dec 23, 2010] A/H1N1 influenza, Canada (Immediate notification: 05/05/2009). 2009. http://web.oie.int/wahis/reports/en_imm_0000008065_20090505_191855.pdf.

Perry HN, McDonnell SM, Alemu W, Nsubuga P, Chungong S, Otten MW Jr, et al. Planning an integrated disease surveillance and response system: a matrix of skills and activities. BMC Med. 2007; 5:24. [PubMed: 17697387]

Reperant LA, Fuckar NS, Osterhaus AD, Dobson AP, Kuiken T. Spatial and temporal association of outbreaks of H5N1 influenza virus infection in wild birds with the 0 degrees C isotherm. PLoS Pathog. 6:e1000854. [PubMed: 20386716]

Smith GJ, Vijaykrishna D, Bahl J, Lycett SJ, Worobey M, Pybus OG, et al. Origins and evolutionary genomics of the 2009 swine-origin H1N1 influenza A epidemic. Nature. 2009; 459:1122–1125. [PubMed: 19516283]

Vrbova L, Stephen C, Kasman N, Boehnke R, Doyle-Waters M, Chablitt-Clark A, et al. Systematic review of surveillance systems for emerging zoonoses. Transbound Emerg Dis. 2010; 57:154–161. [PubMed: 20202176]

Welte VR, Vargas Teran M. Emergency Prevention System (EMPRES) for transboundary animal and plant pests and diseases. The EMPRES-livestock: an FAO initiative. Ann N Y Acad Sci. 2004; 1026:19–31. [PubMed: 15604466]

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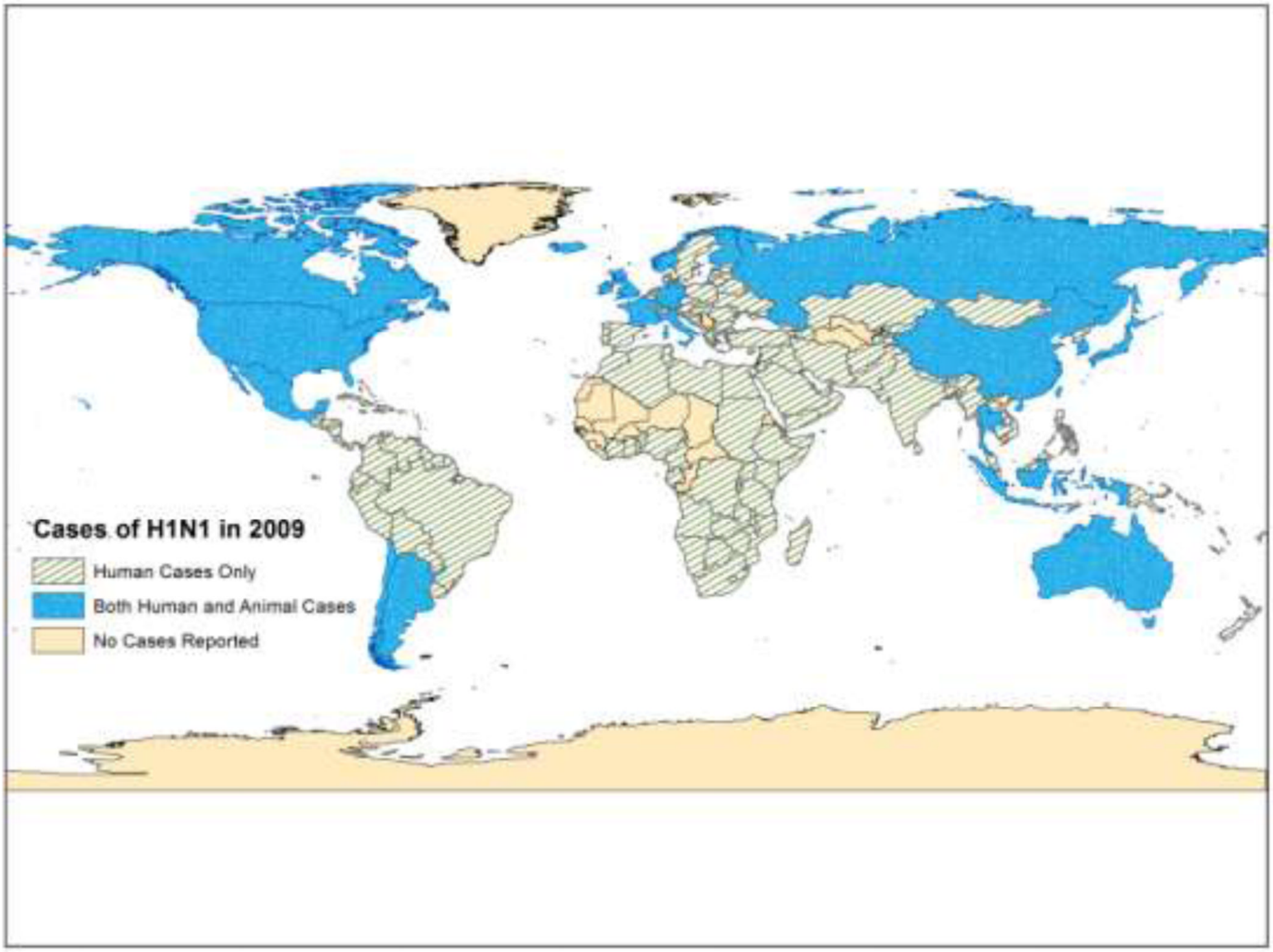

Figure.
Countries reporting cases of the 2009 swine-origin H1N1 influenza A epidemic, 2009.

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Table 1

Species of animals involved in reported cases of the 2009 swine-origin H1N1 influenza A epidemic, in 2009.

Species	# of recorded outbreak events	Percent
Domestic swine	100	82.64
Domestic cats	9	7.44
Domestic turkey	6	4.96
Domestic ferret	3	2.48
Domestic dogs	2	1.65
Cheetah (captive)	1	0.83
Total	121	100%

Table 2

Date of first reported cases of the 2009 swine-origin H1N1 influenza A epidemic in humans.

Country	Human Date Reported (First Date)	Animal Date Reported	Days Difference	Per Capita GDP in 2008	Swine Density in 2007#
Mexico	4/1/2009	4/30/2009	29	9,964	14.5
United States	4/21/2009	8/31/2009	132	45,230	15
Canada	4/23/2009	4/21/2009	−2	45,166	22
Australia	4/26/2009	7/24/2009	89	48,253	0.6
United Kingdom	4/26/2009	8/18/2009	114	43,544	27.3
France	4/26/2009	11/26/2009	214	44,675	50
Ireland	4/27/2009	9/25/2009	151	61,314	37.1
Argentina	4/28/2009	6/15/2009	48	8,358	1.7
Chile	4/28/2009	7/23/2009	86	10,091	18.7
Singapore	4/28/2009	8/27/2009	121	39,423	32500
Norway	4/28/2009	10/9/2009	164	94,791	81.1
China	4/28/2009	10/19/2009	174	3,292	75.8
Italy	4/28/2009	11/23/2009	209	38,640	66.8
Thailand	4/28/2009	12/4/2009	220	4,187	42.4
South Korea	4/28/2009	12/14/2009	230	555	522
Indonesia	4/29/2009	8/27/2009	120	2,247	13.8
Germany	4/29/2009	11/21/2009	206	44,363	160
Japan	4/30/2009	10/2/2009	155	38,578	209.8
Russia	5/5/2009	11/10/2009	189	11,858	7.3
Finland	5/12/2009	11/18/2009	190	51,409	63
Iceland	5/23/2009	10/24/2009	154	52,490	1.8
and animals.					

Number of swine divided by agricultural areas expressed as kilometers squared.