Impact factor 6.15

Eurosurveillance

Europe's journal on infectious disease epidemiology, prevention and control

Vol. 19 | Weekly issue 18 | 9 May 2013

Rapid communications

Surveillance and outbreak reports

News

ecdc EUROPEAN CENTRE FOR DISEASE PREVENTION AND CONTROL

www.eurosurveillance.org

Preliminary inferences on the age-specific seriousness of human disease caused by avian influenza A(H7N9) infections in China, March to April 2013

B J Cowling (bcowling@hku.hk)[1,2], G Freeman[1,2], J Y Wong[1], P Wu[1], Q Liao[1], E H Lau[1], J T Wu[1], R Fielding[1], G M Leung[1]
1. School of Public Health, Li Ka Shing Faculty of Medicine, The University of Hong Kong, Hong Kong Special Administrative Region, China
2. These authors contributed equally to this work

Citation style for this article:
Cowling BJ, Freeman G, Wong JY, Wu P, Liao Q, Lau EH, Wu JT, Fielding R, Leung GM. Preliminary inferences on the age-specific seriousness of human disease caused by avian influenza A(H7N9) infections in China, March to April 2013 . Euro Surveill. 2013;18(19):pii=20475. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20475

Article submitted on 25 April 2013 / published on 9 May 2013

Between 31 March and 21 April 2013, 102 laboratory-confirmed influenza A(H7N9) infections have been reported in six provinces of China. Using survey data on age-specific rates of exposure to live poultry in China, we estimated that risk of serious illness after infection is 5.1 times higher in persons 65 years and older versus younger ages. Our results suggest that many unidentified mild influenza A(H7N9) infections may have occurred, with a lower bound of 210–550 infections to date.

Introduction

In recent weeks, increasing numbers of avian influenza A(H7N9) virus infections have been identified in humans in China [1,2]. Laboratory-confirmed cases of influenza A(H7N9) infection have typically suffered serious illness [3,4], and there is a notable excess of confirmed cases in the elderly [3,5]. In the present analysis, we compared the incidence of serious influenza A(H7N9) infections with data on age-specific patterns in exposure to domestic poultry and live poultry markets to estimate the relative seriousness of influenza A(H7N9) and obtain a lower bound on the number of human infections to date.

Methods

Poultry exposures in China

We obtained unpublished data on poultry exposures in Shenzhen, a city in Guangdong province on the border with Hong Kong, and in Xiuning, a rural county in Anhui province in eastern China. In each location, a two-stage household-based cluster survey was conducted to assess poultry exposures based on average annual visits to poultry wet markets (Shenzhen, n=2,058), and ownership of backyard poultry (Xiuning, n=2,892). Trained investigators conducted each face-to-face interview with selected households, and every family member who met the inclusion criteria (aged at least five years, and resident in the study area for at least three months) was interviewed. Poultry wet markets were defined as places where small animals and poultry may be purchased alive or slaughtered just before purchase. The surveys were conducted from July to September 2007.

Data on poultry exposures in urban and semi-rural areas of Guangzhou, the capital of Guangdong province in Southern China, were obtained through face-to-face interviews, from January through March 2006 [6]. Households were selected for interview through stratified cluster sampling in the ten urban districts (n=1,363) and two satellite towns (n=187) of Guangzhou. One adult per selected household was interviewed. We assessed household exposures to retail and domestic poultry in both urban and semi-rural locations based on average annual visits to poultry wet markets to purchase live poultry, and ownership of backyard poultry [6].

Avian influenza A(H7N9) cases

Information on laboratory-confirmed human infections with influenza A(H5N1) and A(H7N9) was obtained from official notifications, including age, geographic location, and seriousness of disease (mild/serious). The definition for an influenza A(H7N9) case is given elsewhere [3]. A serious case was defined as a laboratory-confirmed influenza A(H7N9) case that required hospital admission for medical reasons, i.e. with a complication such as pneumonia, rather than merely for isolation. Cases defined as serious included all fatal laboratory-confirmed cases. The age-specific populations of provinces in China were obtained from the 2010 population census of the People's Republic of China [7].

Statistical analysis

We specified a model for the observed number of serious influenza A(H7N9) infections under the assumption that the risk of infection was directly proportional to the risk of exposure, while the seriousness of infection varied by age. Specifically, we modelled X_{ij}, the number

of serious influenza A(H7N9) infections in age group i and area j, as following a Poisson distribution with mean $A_{ij} \times p_{ij} \times r_i$, where A_{ij} is the population of persons in age group i (i=1 for 0–14 years, 2 for 15–24 years, 3 for 25–34 years, 4 for 35–44 years, 5 for 45–54 years, 6 for 55–64 years, 7 for ≥65 years) and area j (1 for Anhui-urban, 2 for Beijing-urban, 3 for Henan-rural, 4 for Jiangsu-urban, 5 for Jiangsu-rural, 6 for Shanghai-urban, 7 for Zhejiang-urban, 8 for Zhejiang-rural), p_{ij} represents the incidence rate of infection by age and area over the time period covered by our analysis, and r_i represents the age-specific risk of serious illness if infected. For urban areas ($\delta j=1$) and rural areas ($\delta_j=0$), we specified $p_{ij}=\delta_j \times U_i \times \theta_j + (1-\delta_j) \times V_i \times \theta_j$, where U_i and V_i represent the age-specific rates of exposure in urban and rural areas, respectively, while θ_j represents the area-specific risk of infection. In our main analysis, we modelled the risk of serious illness conditional on infection as r_i taking value r_{old} for age≥65 years and r_{young} for age<65 years. We explored other parameterisations for *ri* such as $r_i=r_7 \times exp(\beta \times (i-7))$ in sensitivity analyses.

We used a Bayesian inferential framework to fit the model to observed data on X_{ij}, A_{ij} and δ_j, incorporating U_{ij}, and V_{ij} as parameters with strong prior distributions from the survey data to retain uncertainty (as is standard in Bayesian evidence synthesis [8]), and r_{old} as a parameter with a strong prior based on observed mild and serious influenza A(H7N9) cases. We estimated θ_j and r_{young} using independent uninformative uniform priors on the positive real line for each θ_j and on the (0,1) interval for r_{young}. Models were fitted with the Hamiltonian Monte Carlo sampler NUTS [9] using the Stan modelling language in R version 3.0.0 (R Foundation for Statistical Computing, Vienna, Austria). Convergence of the simulations was assessed using the potential scale reduction statistic [10].

After fitting the models, posterior estimates of the model parameters were used to estimate $q_{ij}=A_{ij} \times p_{ij}$ as the total number of influenza A(H7N9) infections for each age group *i* and area *j*. This estimate can be

Figure 1

Geographical location of officially announced serious cases of influenza A(H7N9) virus infection in mainland China, 31 March–21 April 2013 (n=98)

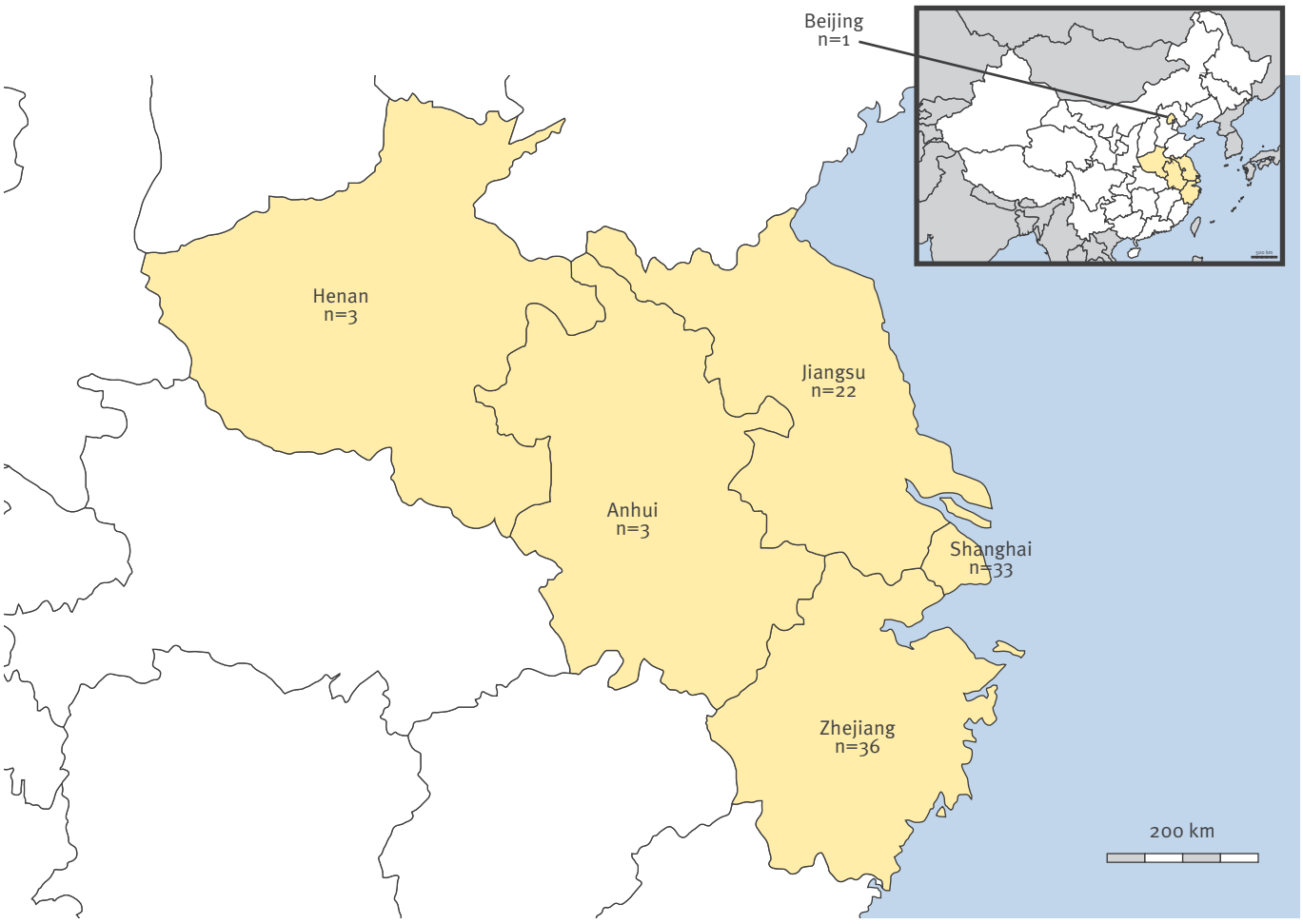
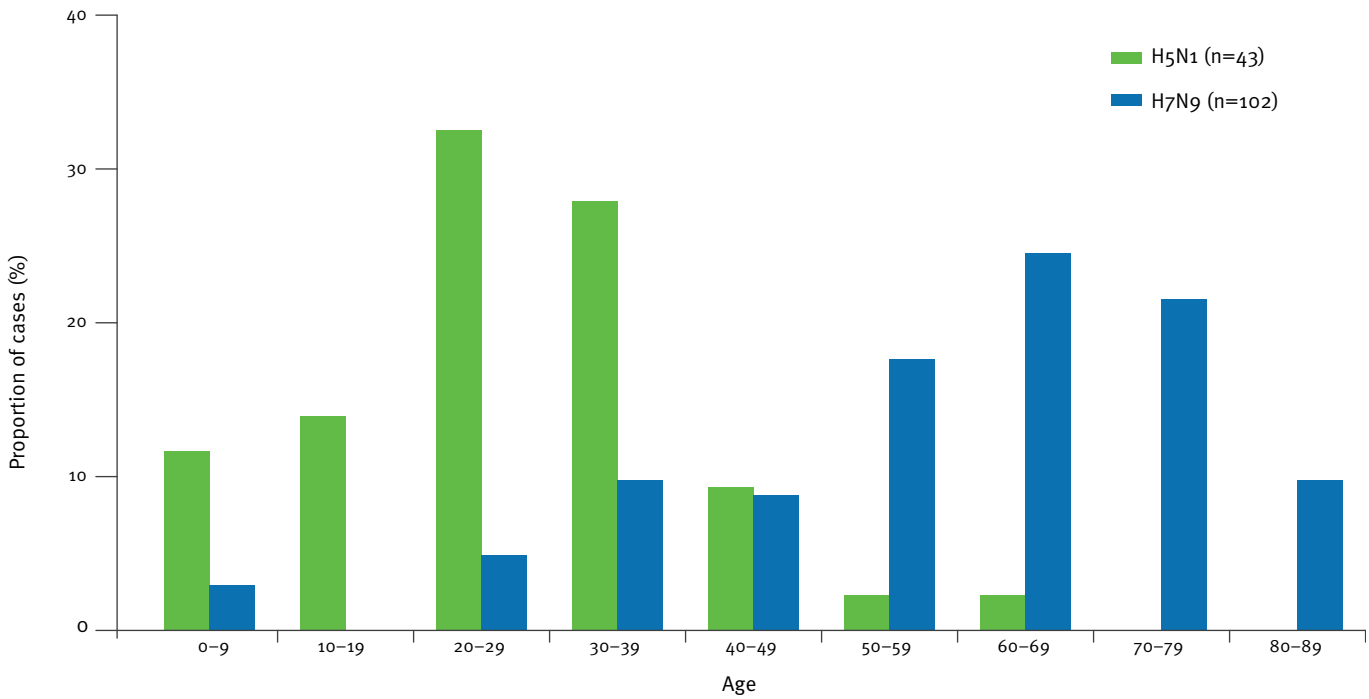


Figure 2

Age distribution of laboratory-confirmed human infections with avian influenza A(H5N1) in 2003–2013 (n=43) and A(H7N9) notified between 31 March 2013 through 21 April 2013 (n=102), mainland China



Table

Serious influenza A(H7N9) cases reported in six provinces of mainland China, and corresponding population denominators, 31 March–21 April 2013 (n=98)[a]

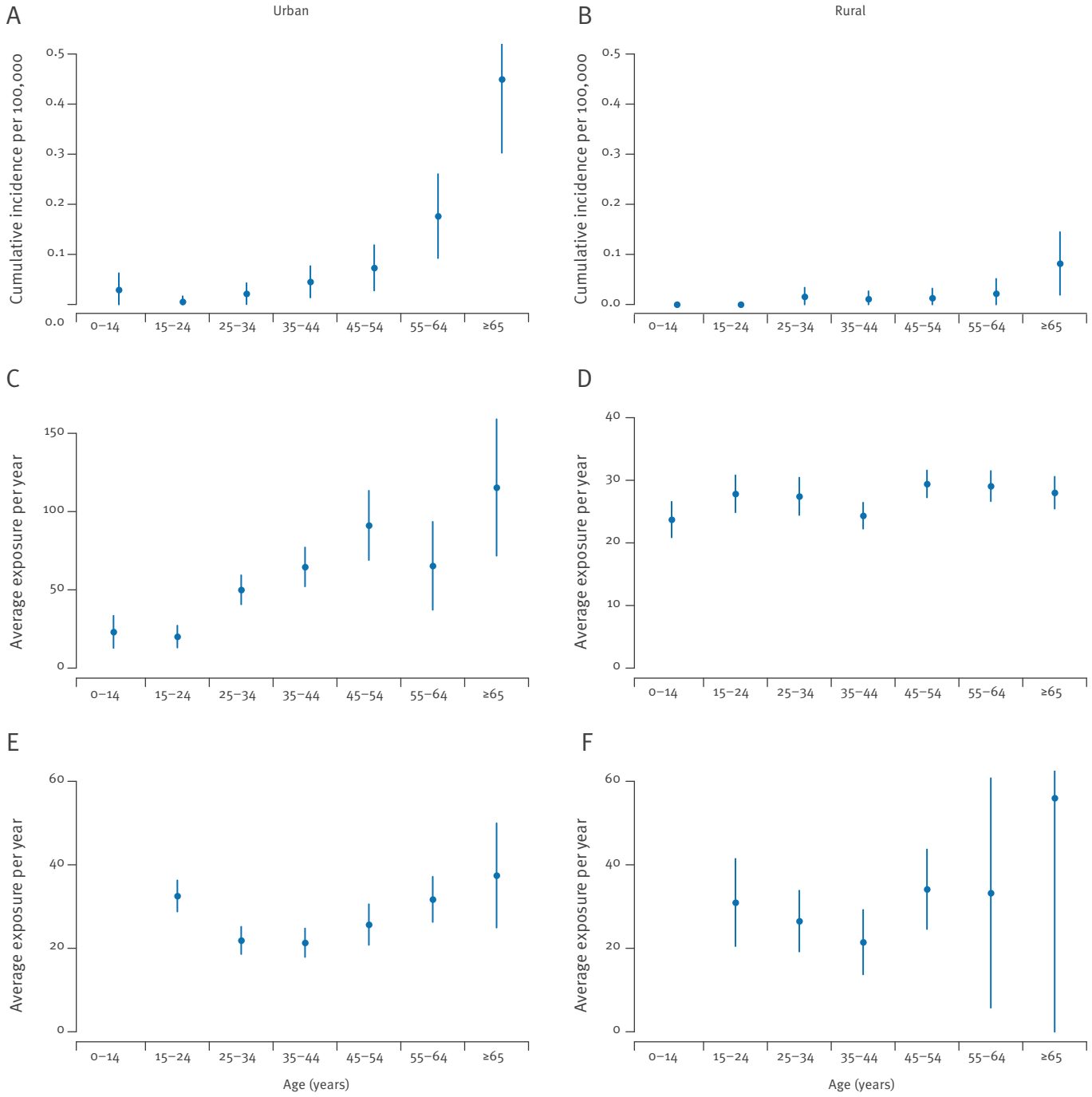
Province-type	Age group (years)						
	0–14	15–24	25–34	35–44	45–54	55–64	≥65
Number of serious influenza A(H7N9) cases							
Anhui-urban	0	0	0	1	0	2	0
Beijing-urban	1	0	0	0	0	0	0
Henan-rural	0	0	1	1	0	0	1
Jiangsu-urban	0	1	3	1	3	4	6
Jiangsu-rural	0	0	1	0	1	0	2
Shanghai-urban	2	0	1	1	5	5	19
Zhejiang-urban	0	0	0	5	2	6	11
Zhejiang-rural	0	0	0	1	1	3	7
Population size[b]							
Anhui-urban	1,617,392	2,299,994	1,965,849	2,512,466	1,671,583	1,135,834	979,469
Beijing-urban	1,311,411	2,968,261	3,513,686	2,657,513	2,278,771	1,485,603	1,347,970
Henan-rural	13,341,020	9,674,352	6,711,837	9,040,458	7,264,034	6,516,703	5,261,768
Jiangsu-urban	3,390,036	6,004,427	5,389,879	5,658,879	4,150,560	3,042,830	2,529,855
Jiangsu-rural	4,421,789	4,517,515	3,459,924	5,406,568	4,789,994	4,443,849	4,249,814
Shanghai-urban	1,483,687	2,821,598	3,660,496	2,797,231	2,809,896	2,267,794	1,800,140
Zhejiang-urban	2,449,320	4,004,494	4,044,383	4,062,503	2,768,791	1,707,271	1,349,532
Zhejiang-rural	2,888,769	2,448,074	2,676,907	3,835,297	3,477,935	2,840,965	2,708,735

[a] The four mild cases among the total of 102 cases are not shown in this Table.

[b] Population sizes obtained from the 2010 population census of the People's Republic of China, published on the official website of National Bureau of Statistics of China [7].

Figure 3

Comparison of age-specific cumulative incidence of serious illness associated with laboratory-confirmed influenza A(H7N9) virus infection, 31 March–21 April 2013, and age-specific poultry exposures, 2006 and 2007, China



Panels A and B show cumulative incidence and 95% confidence intervals of serious influenza A(H7N9) cases in (A) urban and (B) rural populations, based on 98 serious cases reported by 21 April 2013. Panels C to F show rates of exposures to retail and domestic poultry in (C) urban Shenzhen in 2007, (D) rural Xiuning in 2007, (E) urban Guangzhou in 2006, and (F) semi-rural Guangzhou in 2006.

regarded as a lower bound on the number of influenza A(H7N9) infections because it relies on complete ascertainment of all serious influenza A(H7N9) cases, and complete ascertainment of all influenza A(H7N9) infections in people aged 65 years and older. We also estimated $\beta_{age}=r_{old}/r_{young}$, the relative risk of serious illness conditional on infection in those aged 65 years and older compared with those younger than 65 years.

Results

Between 31 March and 21 April 2013, 102 laboratory-confirmed human influenza A(H7N9) cases were officially announced in six provinces of China. The affected areas were the cities and provinces around the city of Shanghai on the eastern coast of mainland China (Figure 1).

The age distribution of influenza A(H7N9) cases was very different to the age distribution of the 43 influenza A(H5N1) cases reported between 2003 and 2013 in mainland China (Figure 2). In particular, 56% of the influenza A(H7N9) cases were persons aged 60 years or older, whereas the majority of influenza A(H5N1) cases were young adults aged 20 to 39 years. In the eight affected areas, there were a total of 98 serious influenza A(H7N9) cases in a total population of 206 million persons (Table). The cumulative number of serious influenza A(H7N9) cases increased substantially with age particularly in urban locations (Figure 3).
We fitted the model described above to data on the incidence rates of serious influenza A(H7N9) cases in the six provinces, along with poultry exposures in urban and rural locations (Figure 2). In the age group of at least 65 years there were 46 serious and one mild infection, so we used a beta(47,2) distribution for the parameter r_{old}.

Based on the exposure data from Shenzhen and Xiuning to reflect exposures in affected urban and rural areas, we obtained the estimate β_{age}=5.06 (95% credibility interval (CI): 2.99–8.15), corresponding to a 5.06-fold increase in the risk of serious illness for those aged 65 years and older versus those younger than 65 years. The estimated values of p_{ij} and the observed values of A_{ij} were then used to estimate that there have been at least 323 (95% CI: 214–475) total influenza A(H7N9) infections in the population, including those reported. When we used the exposure data from Guangzhou to reflect exposures in affected urban and rural areas, we estimated β_{age}=5.95 (95% CI: 3.37–10.00), and an estimated minimum number of 352 (95% CI: 225–541) total influenza A(H7N9) infections in adults (because we did not have exposure data for children in Guangzhou).

In sensitivity analyses, results were similar using alternative simple parameterisations for the effect of age. For example when we used $r_i=r_7\times\exp(\beta\times(i-7))$, we obtained an estimated 1.83-fold (95% CI: 1.56–2.18) increase in the risk of serious illness for every ten-year increase in age, and an estimate of at least 334 (95% CI: 239–461) total influenza A(H7N9) infections in the population. The small sample size did not allow us to examine more complex functional forms for r_i. All analyses reported above were based on data available until April 25; we repeated the analyses based on data available until May 6 and the relationship between age and seriousness of disease was essentially the same.

Discussion

Our results suggest that the seriousness of influenza A(H7N9) infections increases with age. Previous reports also identified increases with older age in the seriousness of seasonal influenza [11] and H1N1pdm09 [12,13], although this may partly be due to the role of secondary bacterial pneumonia, whereas many of the influenza A(H7N9) deaths have been associated with primary viral pneumonia [4]. However, the age distribution of serious human infections with avian influenza A(H5N1) is very different (Figure 1). The patterns of exposure to avian influenza A(H5N1) and A(H7N9) viruses by age may not be identical because of the high degree of pathogenicity of influenza A(H5N1) in poultry compared with the absence of disease in poultry with influenza A(H7N9) infections [4], at least before to the national influenza A(H5N1) vaccination programme in poultry was introduced in 2006–07. Exposures to sick or dead poultry would be more frequent in farms and backyards, compared to live poultry markets. In addition, healthcare seeking behaviours may also have changed over the past 10 years. There are various potential explanations for an increased risk of serious illness for influenza A(H5N1) infections in young adults compared to other ages, and these hypotheses deserve further investigation [14].

We estimated that a minimum of 210–550 influenza A(H7N9) infections have occurred by 21 April 2013, assuming that almost all influenza A(H7N9) infections are serious in the elderly and that all serious infections have been identified. This estimate is therefore a lower bound on the number of total influenza A(H7N9) infections, and for these two reasons the real figure may be substantially higher. There could be some under-ascertainment of serious influenza A(H7N9) infections through failure to seek care or failure to be tested early enough in the course of disease to permit identification of the influenza infection [5]. Our estimate is also dependent on the assumption that age-specific patterns of exposure to retail and domestic poultry in affected areas of China in 2011 are similar to the patterns measured in Guangzhou, Shenzhen and Xiuning in 2006 and 2007. We are not aware of data on age-specific patterns in poultry exposures from eastern China other than our unpublished data from Xiuning, and future collection of such data from across China (and across South-east Asia) in urban and rural settings would be extremely useful.

Our estimates are limited by the lack of data on exposures in affected urban and rural areas. In particular, the higher risk for infection in males compared to females could be due to variation in sex-specific rates

of exposure by region [5]. Without data on such differences, we did not include sex in our models. Most confirmed cases report exposure to live poultry [3] and this remains the most likely source of infection for the majority of influenza A(H7N9) cases. However, the exposure distributions used in our analysis may not fully capture the age-specific risk profile, if there are other sources of infection apart from retail and domestic poultry. As of April 25, we are not aware of provinces in China with laboratory-confirmed A(H7N9) cases in poultry but not in humans. Finally, no published information is available on population levels of immunity to influenza A(H7N9), although preliminary investigations suggest very low antibody levels against influenza A(H7N9) virus in all ages, and we assumed there was no heterogeneity in immunity by age. If older persons had some degree of immunity against influenza A(H7N9) through potential past exposures to avian influenza viruses, this would imply an even higher number of undetected infections in adults based on our method.

In conclusion, we estimated a lower bound for the number of influenza A(H7N9) infections based on the possible age distribution of exposures and varying seriousness of infection by age. More accurate estimates of the risk of influenza A(H7N9) infection and the age-specific seriousness of infection could be provided by detailed seroepidemiological studies in affected areas [15].

Acknowledgements

We thank Vicky Fang, Angel Li, Michael Ni and Hoi Wa Wong for technical assistance.

This research was supported by the Harvard Center for Communicable Disease Dynamics from the National Institute of General Medical Sciences (grant no. U54 GM088558), the Research Fund for the Control of Infectious Disease, Food and Health Bureau, Government of the Hong Kong Special Administrative Region, and the Area of Excellence Scheme of the Hong Kong University Grants Committee (grant no. AoE/M-12/06). The funding bodies had no role in study design, data collection and analysis, preparation of the manuscript, or the decision to publish.

Potential conflicts of interest

BJC reports receipt of research funding from MedImmune Inc., and consults for Crucell NV. GML has received speaker honoraria from HSBC and CLSA. The authors report no other potential conflicts of interest.

Authors' contributions

Designed the study: BJC. Collected, synthesised and analysed data: BJC, GF, JYW, PW, QL, RF. Wrote the first draft: BJC. Interpreted the results and revised the article: GF, JYW, QL, PW, JTW, EHYL, RF, GML. All authors read and approved the final manuscript.

References

1. Gao R, Cao B, Hu Y, Feng Z, Wang D, Hu W, et al. Human infection with a novel avian-origin influenza A (H7N9) virus. N Engl J Med. 2013;Apr 11. [Epub ahead of print]. http://dx.doi.org/10.1056/NEJMoa1304459
2. Uyeki TM, Cox NJ. Global Concerns Regarding Novel Influenza A (H7N9) Virus Infections. N Engl J Med. 2013;Apr 11. [Epub ahead of print]. http://dx.doi.org/10.1056/NEJMp1304661
3. Li Q, Zhou L, Zhou M, Chen Z, Li F, Wu H, et al. Preliminary report: epidemiology of the avian influenza A (H7N9) outbreak in China. N Engl J Med. 2013;Apr 24. [Epub ahead of print]. http://dx.doi.org/10.1056/NEJMoa1304617
4. Chen Y, Liang W, Yang S, Wu N, Gao H, Sheng J, et al. Human infections with the emerging avian influenza A H7N9 virus from wet market poultry: clinical analysis and characterisation of viral genome. Lancet. 2013;Apr 25. pii: S0140-6736(13)60903-4. http://dx.doi.org/10.1016/S0140-6736(13)60903-4
5. Arima Y, Zu R, Murhekar M, Vong S, Shimadaa T, World Health Organization Regional Office for the Western Pacific Event Management Team. Human infections with avian influenza A(H7N9) virus in China: preliminary assessments of the age and sex distribution. Western Pac Surveill Response J. 2013;4(2). doi:10.5365/wpsar.2013.4.2.005.
6. Liao Q, Lam WW, Jiang CQ, Ho EY, Liu YM, Zhang WS, et al. Avian influenza risk perception and live poultry purchase in Guangzhou, China, 2006. Risk Anal 2009; 29:416-24. http://dx.doi.org/10.1111/j.1539-6924.2008.01157.x
7. National Bureau of Statistics of China. Tabulation of the 2010 population census of the People's Republic of China. Beijing: National Bureau of Statistics of China. [Accessed 25 April 2013]. Available from: http://www.stats.gov.cn/english/statisticaldata/censusdata/rkpc2010/indexch.htm
8. Presanis AM, De Angelis D, New York City Swine Flu Investigation Team, Hagy A, Reed C, Riley S, et al. The severity of pandemic H1N1 influenza in the United States, from April to July 2009: a Bayesian analysis. PLoS Med. 2009; 6(12):e1000207. http://dx.doi.org/10.1371/journal.pmed.1000207
9. Hoffman MD, Gelman A. The no-U-turn sampler: adaptively setting path lengths in hamiltonian monte carlo. arXiv:1111.4246v1 [stat.CO]. 18 Nov 2011.
10. Gelman A, Rubin DB. Inference from iterative simulation using multiple sequence. Statist Sci. 1992;7(4):457-72. http://dx.doi.org/10.1214/ss/1177011136
11. Simonsen L, Fukuda K, Schonberger LB, Cox NJ. The impact of influenza epidemics on hospitalizations. J Infect Dis. 2000;181(3):831-7. http://dx.doi.org/10.1086/315320. PMid:10720501
12. Van Kerkhove MD, Vandemaele KA, Shinde V, Jaramillo-Gutierrez G, Koukounari A, Donnelly CA, et al. Risk factors for severe outcomes following 2009 influenza A (H1N1) infection: a global pooled analysis. PLoS Med. 2011;8(7):e1001053. http://dx.doi.org/10.1371/journal.pmed.1001053 PMid:21750667 PMCid:3130021
13. Wong JY, Wu P, Nishiura H, Goldstein E, Lau EH, Yang L, et al. Infection Fatality Risk of the Pandemic A(H1N1)2009 Virus in Hong Kong. Am J Epidemiol. 2013;177(8):834-40. http://dx.doi.org/10.1093/aje/kws314
14. Liem NT, Tung CV, Hien ND, Hien TT, Chau NQ, Long HT, et al. Clinical features of human influenza A (H5N1) infection in Vietnam: 2004-2006. Clin Infect Dis. 2009;48(12):1639-46. http://dx.doi.org/10.1086/599031
15. Van Kerkhove MD, Broberg E, Engelhardt OG, Wood J, Nicoll A, CONSISE steering committee. The consortium for the standardization of influenza seroepidemiology (CONSISE): a global partnership to standardize influenza seroepidemiology and develop influenza investigation protocols to inform public health policy. Influenza Other Respi Viruses. 2013;7(3):231-4. http://dx.doi.org/10.1111/irv.12068

A major outbreak of gastroenteritis in Réunion Island in 2012: first identification of G12 rotavirus on the Island

N Caillère (nadege.caillere@ars.sante.fr)[1], P Vilain[1], E Brottet[1], J Kaplon[2], K Ambert-Balay[2], D Polycarpe[3], L Filleul[1]
1. Regional Office of the French Institute for Public Health Surveillance (Institut de Veille Sanitaire), Saint-Denis, Réunion, France
2. National Reference Centre for Enteric Viruses, Dijon, France
3. Health Agency of the Indian Ocean, Saint-Denis, Réunion, France

Citation style for this article:
Caillère N, Vilain P, Brottet E, Kaplon J, Ambert-Balay K, Polycarpe D, Filleul L. A major outbreak of gastroenteritis in Réunion Island in 2012: first identification of G12 rotavirus on the Island. Euro Surveill. 2013;18(19):pii=20476. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20476

Article submitted on 08 April 2013 / published on 09 May 2013

Between August and November 2012 a severe outbreak of gastroenteritis occurred on Réunion Island, affecting more than 50,000 cases, particularly young children. Virological analyses showed that the virus responsible for this epidemic was rotavirus. Genotyping of stool samples indicated circulation of rotavirus type G3P[8] but also G12P[8], highlighting the risk of global emergence of this genotype in the coming years.

On Réunion Island, a French overseas administrated territory located in the south-western Indian Ocean, gastroenteritis outbreaks are usually observed during the austral winter, between the months of August and November. While outbreaks of gastroenteritis had been of moderate severity on the island between 2008 and 2011 [1], an intense epidemic occurred in 2012. Monitoring has been in place for several years, involving many professionals and coordinated by the Indian Ocean Regional Office (Cellule de l'InVS en region Océan Indien: Cire OI) of the French Institute for Public Health Surveillance. This system allowed to detect in mid-August 2012 an unusual increase in gastroenteritis cases that exceeded seasonal averages, and to inform the public health authorities and the population in a reactive way.

Population and healthcare system on Réunion Island

Réunion Island is located in the south-western Indian Ocean, at 5,900 miles from France and 500 miles from Madagascar, with a population of 830,000 inhabitants in 2012. The population is younger than in mainland France. In 2010, 41% of the population was younger than 25 years, and only 11% were older than 60 years. Medical facilities are similar to those in France, and there are more than 890 general practitioners and more than 80 pediatricians distributed throughout the island, as well as four hospitals and six emergency departments.

Surveillance system

Gastroenteritis surveillance on Réunion Island is based on different complementary systems:

A syndromic surveillance system is based on all emergency departments (ED) on the island (Organisation de la surveillance coordonnée des urgences (OSCOUR) network). Data are collected daily directly from patients' computerised medical files that are filled in during medical consultations at ED [1-3]. Among the collected variables, the diagnosis is categorised according the 10th revision of the international Classification of Diseases (ICD-10)[4]. Several indicators are routinely monitored, including the number of ED visits for gastroenteritis (ICD-10 codes A08 and A09).

A network of sentinel general practitioners of Réunion Island reports every week the numbers of consultations for acute diarrhoea [1,5]. A case of acute diarrhoea is defined as a patient having more than three liquid stools per day during the past 15 days and motivating consultation.

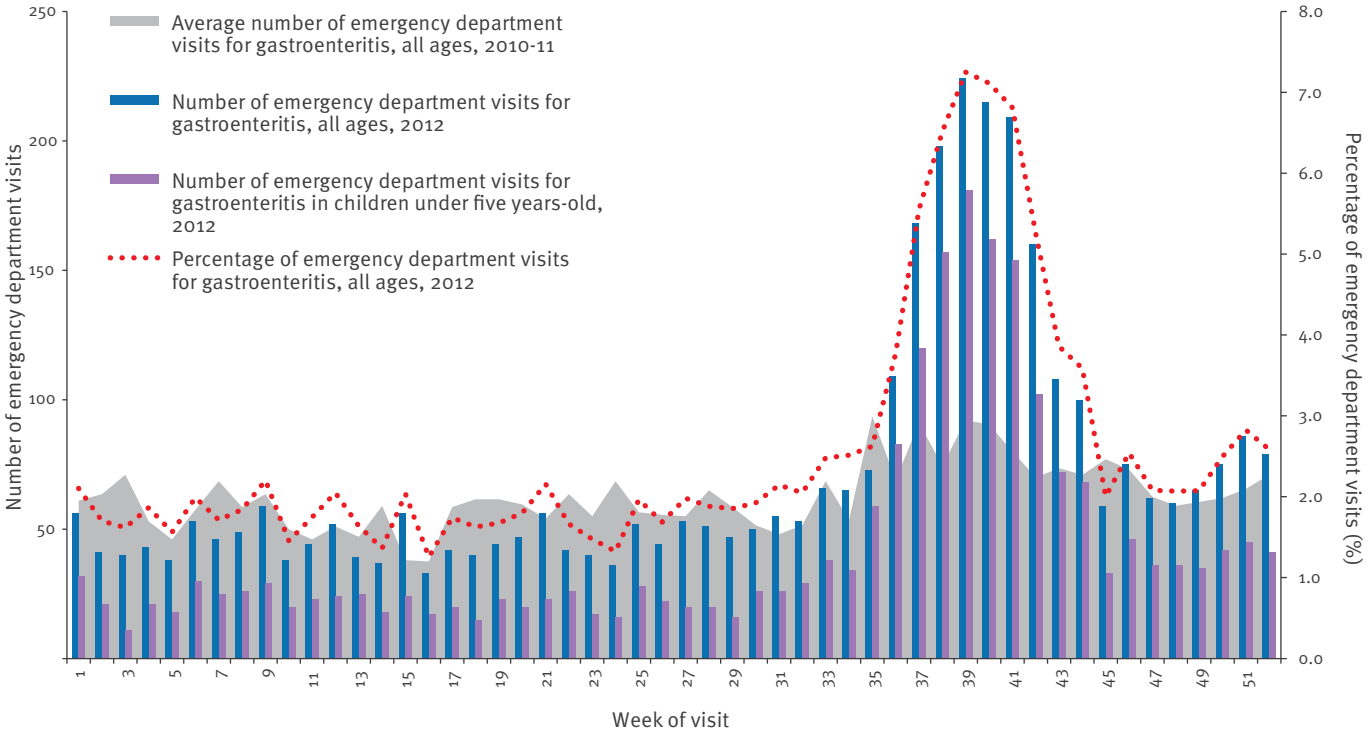
The national health insurance centre of Réunion Island (Caisse générale de sécurité sociale; CGSS) sends to the Cire OI every week the numbers of consultations made by general practitioners and paediatricians of the island. These consultation data, coupled with those from sentinel practitioners, allow to estimate by extrapolation the total weekly number of consultations for acute diarrhoea on the whole island [1,6,7].

Three sentinel hospital laboratories report to the Cire OI the monthly percentage of samples positive for rotavirus, adenovirus and norovirus to ensure virological surveillance of viral gastroenteritis. In addition, a selection of rotavirus-positive samples was sent, as part of this outbreak investigation in 2012, by the laboratories of the hospitals of Saint-Denis and Saint-Pierre to the National Reference Centre for Enteric Viruses in order to determine the G and P genotypes of rotavirus strains. Genotyping was performed by RT-PCR according to the EuroRotaNet protocol [8-14] and confirmed by sequencing the partial VP7 and VP4 coding genes.

A surveillance of deaths possibly related to gastroenteritis is achieved via monitoring of death certificates

Figure 1

Weekly visits to emergency departments for gastroenteritis, Réunion Island, 2012 versus mean numbers in 2010 and 2011 (n=3,732)



Sources: Institut de veille sanitaire (InVS) – Cellule de l'InVS en région (Cire) océan Indien / Organisation de la surveillance coordonnée des urgences (OSCOUR).

received by the Health Agency of the Indian Ocean (Agence de Santé Océan Indien; ARS OI).

Each week, data from these different surveillance systems are collected, validated, analysed and interpreted. During outbreaks, a weekly epidemiological report is written, presenting the results of these analyses and the appropriate recommendations.

Outbreak description

In 2012, an increase in gastroenteritis cases on Réunion Island was detected in week 33 (week starting 13 August) by the OSCOUR network (Figure 1). During that same week, the percentage of consultations for acute diarrhoea reported by sentinel practitioners was about 2.1%, exceeding the seasonal average of 1.6%. Analysis of the data by age showed that children five years-old and younger were most affected throughout the epidemic period (Figure 1). In fact, 73% of ED visits for gastroenteritis were observed among children of this age.

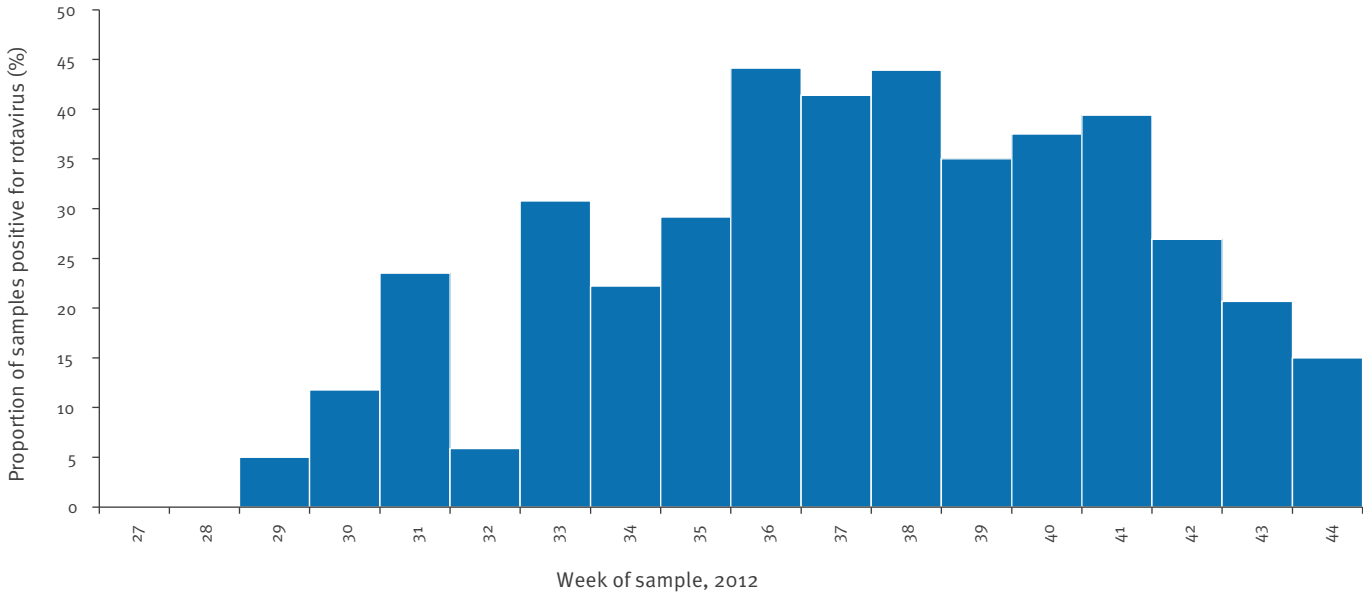
According to the two surveillance systems, the outbreak peak was reached in week 39 (last week of September). During that week, more than 230 ED visits for gastroenteritis were recorded, representing more than 7% of the total attendance. Among these 230, 187 involved children aged five years and younger, representing 27% of the total attendance for this age group. Moreover, for the same week, the percentage of consultations for acute diarrhoea identified by sentinel practitioners was 6% and the total number of consultations for this pathology on the whole island was estimated to be nearly 8,000.

Over the entire outbreak period from week 35 to week 44, nearly 1,600 ED visits for gastroenteritis were recorded, and it was estimated that more than 53,500 general practitioner consultations for acute diarrhoea took place on the whole island. Among the ED visits for gastroenteritis, 74% were concerning children aged five years and younger; 56% of them were boys.

The virological surveillance rapidly revealed high rates of samples positive for rotavirus. Retrospective analysis of weekly data showed an increase in this rate at the end of July, the rate exceeded more than 30% in week 33 (week starting August 13). The peak was reached in week 36 (first week of September), with a value of 44%

Figure 2

Weekly percentage of samples positive for rotavirus, hospital laboratories Saint-Denis, Saint-Paul, Saint-Benoît, Réunion Island, week 27–44 2012



Sources: Institut de veille sanitaire (InVS) – Cellule de l'InVS en région (Cire) Océan Indien / Laboratories of the hospitals of Saint-Denis, Saint-Paul, Saint-Benoît, Réunion Island.

(Figure 2). This rate was maintained at a level greater than 35% before declining gradually after week 41.

Percentages of samples positive for adenovirus and norovirus were lower (7% and 3%, respectively, from July to October), suggesting that the outbreak of gastroenteritis was mainly due to the circulation of rotavirus on the island. In December, genotyping of 20 rotavirus-positive samples, randomly selected, was carried out by the National Reference Centre for Enteric Viruses. Four of them were genotype G12P[8], one was a co-infection of genotypes G12, G1 and G3 associated with P[8], and the 15 others were G3P[8] strains. Phylogenetic analysis of the partial VP7 coding gene revealed that the G12 strains from the Réunion Island clustered in lineage III (Figure 3).

During the whole outbreak period, six death certificates mentioning gastroenteritis as one of the potential causes of death were identified. Three of them involved children younger than two years, and the other three affected individuals older than 70 years. One child and one elderly person died of dehydration, the other two children had underlying medical conditions, and the other two elderly people were suffering from comorbidities such as diabetes and hypertension.

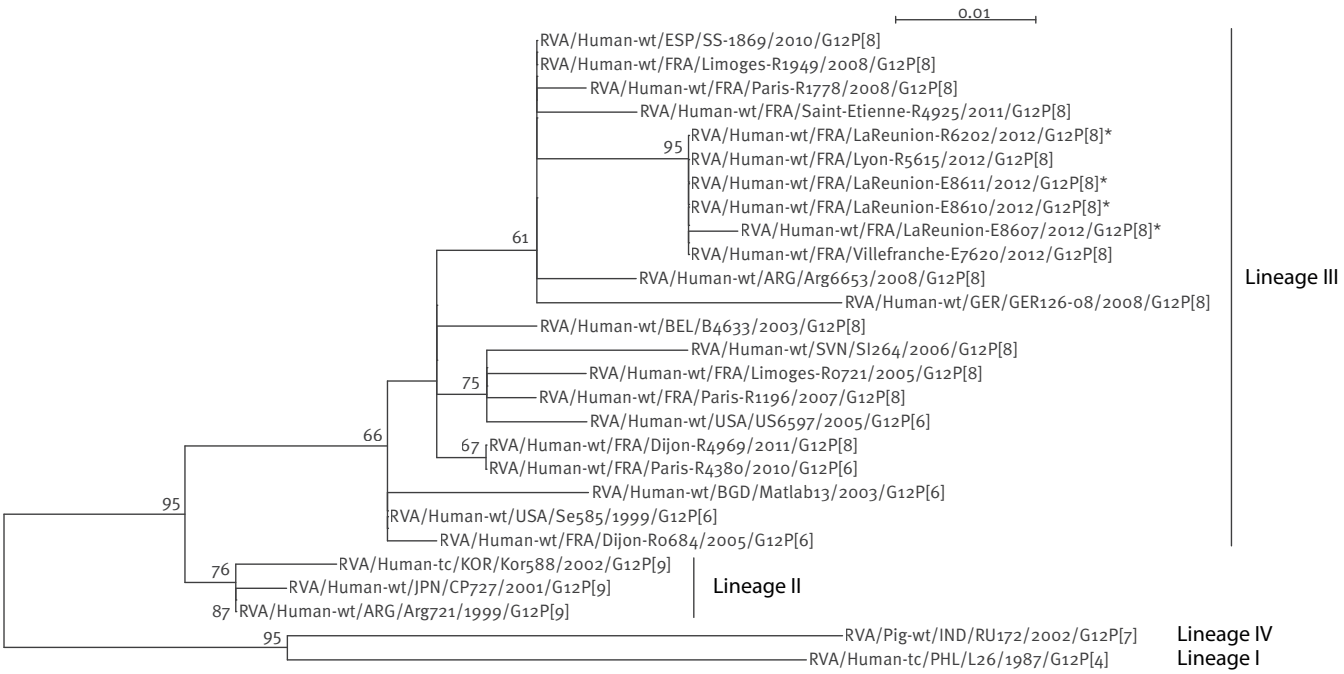
Discussion

The surveillance based on networks from different health providers on Réunion Island highlighted an outbreak of gastroenteritis of unusual intensity, which lasted about 10 weeks, extending from late August to early November 2012. This outbreak was characterised by intense circulation of rotavirus and a high proportion of young cases, as well as by its severity, reflected in the occurrence of several deaths. This underlines the importance of not neglecting this risk when such epidemics occur.

This study reports for the first time the occurrence of the G and P genotypes of rotavirus strains on Réunion Island and revealed circulation of rotavirus genotype G12 at a significant level (5/20), although the high activity of rotavirus observed on the island does not seem to be the result of a high prevalence of this emerging genotype only. The G12 strains from Réunion Island clustered in lineage III, as previously observed for G12 strains from other European countries and worldwide. According to the National Reference Centre for Enteric Viruses, G12 genotypes circulated with a low prevalence of 3.5% during the previous season in mainland France (data not shown), but their emergence as the most prevalent rotavirus genotypes has

Figure 3*

Phylogenetic tree based on the partial amino acid sequences (280 aa) of the rotavirus G12 VP7 coding gene, gastroenteritis outbreak Réunion Island, 2012



Phylogeny was reconstructed using the maximum-likelihood method implemented in the PhyML programme with the Jones Taylor Thornton substitution model. Number of substitutions per site is indicated by the scale bar. Bootstrap values were calculated for 500 replicates and are indicated at each node when ≥60%. Strains from Réunion island are indicated with an asterisk.

Sequences of the French RVA G12 strains were submitted to the European Nucleotide Archive database (http://www.ebi.ac.uk/ena/) under the following accession numbers: HF952906 to HF952917, HM035517 and HM035518.

GenBank accession numbers of the reference strains used for this analysis are: RVA/Human-tc/PHL/L26/1987/G12P[4]: M58290; RVA/Human-wt/ARG/Arg721/1999/G12P[9]: EU496254; RVA/Human-wt/JPN/CP727/2001/G12P[9]: AB125852; RVA/Human-tc/KOR/Kor588/2002/G12P[9]: EU496259; RVA/Human-wt/ESP/SS-1869/2010/G12P[8]: JQ410021; RVA/Human-wt/ARG/Arg6653/2008/G12P[8]: JN088450; RVA/Human-wt/GER/GER126-08/2008/G12P[8]: FJ747618; RVA/Human-wt/BEL/B4633/2003/G12P[8]: DQ146643; RVA/Human-wt/SVN/SI264/2006/G12P[8]: DQ995173; RVA/Human-wt/USA/US6597/2005/G12P[6]: FJ152121; RVA/Human-wt/BGD/Matlab13/2003/G12P[6]: DQ146676; RVA/Human-wt/USA/Se585/1999/G12P[6]: AJ311741; RVA/Pig-wt/IND/RU172/2002/G12P[7]: DQ204743.

been recently demonstrated in several regions of the world such as Nepal [15], Spain (Basque Country) [16], Argentina (Tucuman, Catamarca and Rio Gallegos) [17] and Nigeria [18], and we can assume that this genotype could emerge globally in the coming years. The impact of the rotavirus G12 is not documented in these countries, except in the Spanish study, where it is claimed that the impact of the rotavirus G12 epidemic in the Basque Country was high, with the rate of hospitalisation similar to previous seasons in which rotavirus G1 or G9 were dominant [16].

Efficacy trials of the two licensed rotavirus vaccines (Rotarix, GlaxoSmithKline and RotaTeq, Merck) focused on the rotavirus G genotypes most prevalent across the world, i.e. G1, G2, G3, G4 and G9, and data on their efficacy against rotavirus G12 are very limited. In a recent study, Rotarix vaccine demonstrated efficacy against severe gastroenteritis caused by G12 rotavirus [19]. Nevertheless, it should be emphasised that knowledge about vaccine efficacy against the G12 genotype is limited and there is a need for further study. However, in our study conducted in Réunion Island, G12 genotype was associated with P[8]. Since the P[8] genotype is present in both licensed vaccines, these vaccines should be effective against these G12P[8] strains.

Acknowledgements

We acknowledge all the partners involved in the surveillance of gastroenteritis in Réunion Island: The sentinel general practitioners, Dr Arnaud Bourdé, Emergency Department, University Hospital Centre of Saint-Denis, Réunion, France, Dr Yves Jacques Antoine, Emergency Department, Hospital Group of Saint-Benoît, Réunion, France, Dr Katia Mougin Damour, Emergency Department, Hospital Centre of Saint-Paul, Réunion, France, Dr Pierre-Jean Marianne Dit Cassou, Emergency Department, University Hospital Centre of Saint-Pierre, Réunion, France, Dr Marie-Christine Jaffar Bandjee, Microbiology Laboratory, University Hospital Centre of Saint-Denis, Réunion, France, Dr Pierre Colbalchini, Microbiology Laboratory, Hospital Group of Saint-Benoît, Réunion, France,

Dr Gérard Martigny, Biology Laboratory, Hospital Centre of Saint-Paul, Réunion, France, Dr Alain Michault, Laboratory for Bacteriology, Parasitology, Virology and Hospital Hygiene, University Hospital Centre of Saint-Pierre, Réunion, France, GCS Tesis, Brigitte Ristor, National Health Insurance Center of Réunion Island, Céline Fremy, National Reference Center for enteric viruses, Dr Nathalie Jourdan-Da Silva, French Institute for Public Health Surveillance.

Authors' contributions

Nadège Caillère has made contributions to the epidemiological analyzes, to the interpretation of results and to the writing of the manuscript. Pascal Vilain has made contributions to the epidemiological analyzes, to the interpretation of results and to the writing of the manuscript. Elise Brottet has made contributions to the epidemiological analyzes and interpretation of results and has been involved in revising the manuscript critically for important intellectual content. Jérôme Kaplon has made contributions to the virological analyzes, to the interpretation of results of virological analyzes and to the writing of the manuscript. Katia Ambert-Balay has made contributions to the virological analyzes, to the interpretation of results of virological analyzes and to the writing of the manuscript. Dominique Polycarpe has been involved in revising the manuscript critically for important intellectual content. Laurent Filleul has made contributions to the interpretation of results of epidemiological analyzes, and to the writing of the manuscript.

Conflict of interest

None declared.

*Authors' correction

Bootstrap values and lineages were added to the figure on 13 May 2013.

References

1. Filleul L. Le développement de la surveillance épidémiologique à la Réunion et à Mayotte. [The development of epidemiological surveillance in Reunion Island and Mayotte]. Bulletin de veille sanitaire. 2012;15:8-17. French. Available from: http://www.invs.sante.fr/fr/Publications-et-outils/Bulletin-de-veille-sanitaire/Tous-les-numeros/Ocean-indien-Reunion-Mayotte/Bulletin-de-veille-sanitaire-ocean-Indien.-N-15-Numero-special-Fevrier-2012
2. Caillère N, Fouillet A, Henry V, Vilain P, Mathieu A, Raphaël M, et al. La surveillance des urgences par le réseau OSCOUR® (Organisation de la surveillance coordonnée des urgences). [The surveillance of emergencies by the OSCOUR® network (Organisation of the coordinated surveillance of emergencies)]. Saint-Maurice: Institut de Veille Sanitaire; 2012. 12 p. French. Available from: http://www.invs.sante.fr/fr/Publications-et-outils/Rapports-et-syntheses/Autres-thematiques/2012/La-surveillance-des-urgences-par-le-reseau-OSCOUR-R-Organisation-de-la-surveillance-coordonnee-des-urgences
3. Carrillo-Santisteve P, Ambert-Balay K, Arena C, Poujol I, Caillère N, Delmas G, et al. Epidémies hivernales de gastro-entérites aiguës en France, bilan de trois saisons (2006/2007, 2007/2008 et 2008/2009). [Winter outbreaks of acute gastro-enteritis in France, results for three seasons (2006/2007, 2007/2008 and 2008/2009)]. French. Bull Epiémiol Hebd. 2010;31-32:349-52. Available from: http://www.invs.sante.fr/beh/2010/31_32/beh_31_32_2010.pdf
4. World Health Organization (WHO). International Statistical Classification of Disease and Related Health Problems, Tenth Revision (ICD-10). Geneva: WHO; 2004. Available from: http://www.who.int/classifications/icd/en/
5. Brottet E, Renault P, Larrieu S, Jaffar-Bandjee MC, Rachou E, Polycarpe D, et al. Implication du réseau de medecins sentinelles dans la surveillance des maladies infectieuses à la Réunion. [Involvement of the network of sentinel general practitioners in the surveillance of infectious diseases in Réunion Island]. Bulletin de veille sanitaire. 2010;7:2-4. French. Available from: http://www.invs.sante.fr/fr/Publications-et-outils/Bulletin-de-veille-sanitaire/Tous-les-numeros/Ocean-indien-Reunion-Mayotte/Bulletin-de-veille-sanitaire-Ocean-Indien-Reunion-Mayotte-n-7-Septembre-2010
6. Vilain P, Caillère N, Larrieu S, Ristor B, Filleul L. Utilisation des données de l'assurance maladie pour la surveillance syndromique à la Réunion. [Using data from health insurance fo syndromic surveillance in Réunion Island]. Bulletin de veille sanitaire. 2012;17:10-14. French. Available from: http://www.invs.sante.fr/Publications-et-outils/BEH-Bulletin-epidemiologique-hebdomadaire/Derniers-numeros-et-archives/Archives/2011/BEH-n-31-2011
7. Baroux N, Ristor B, Ferdinand P, Renault P, Filleul L. Utilisation des données de la Caisse Générale de Sécurité Sociale de la Réunion dans le cadre de la surveillance non spécifique. [Using French national health insurance data for non-specific monitoring on Reunion Island]. Rev Epidemiol Sante Publique. 2011;59(5):319-25. http://dx.doi.org/10.1016/j.respe.2011.04.005 PMid:21940126
8. European Rotavirus Network. European Rotavirus Network 2013 [Accessed 5 Apr 2013]. Available from: http://www.eurorota.net/docs.php
9. Gómara MI, Cubitt D, Desselberger U, Gray J. Amino acid substitution within the VP7 protein of G2 rotavirus strains associated with failure to serotype. J Clin Microbiol. 2001;39(10):3796-8. http://dx.doi.org/10.1128/JCM.39.10.3796-3798.2001 PMid:11574622 PMCid:88438
10. Gouvea V, Glass RI, Woods P, Taniguchi K, Clark HF, Forrester B, et al. Polymerase chain reaction amplification and typing of rotavirus nucleic acid from stool specimens. J Clin Microbiol. 1990;28(2): 276-82. PMid:2155916 PMCid:269590
11. Iturriza-Gomara M, Kang G, Gray J.. Rotavirus genotyping: keeping up with an evolving population of human rotaviruses. J Clin Virol. 2004;31(4): 259-65. http://dx.doi.org/10.1016/j.jcv.2004.04.009 PMid:15494266
12. Simmonds MK, Armah G, Asmah R, Banerjee I, Damanka S, Esona M, et al. New oligonucleotide primers for P-typing of rotavirus strains: Strategies for typing previously untypeable strains. J Clin Virol. 2008;42(4):368-73. http://dx.doi.org/10.1016/j.jcv.2008.02.011 PMid:18378188
13. Gentsch JR, Glass RI, Woods P, Gouvea V, Gorziglia M, Flores J, et al. Identification of group A rotavirus gene 4 types by polymerase chain reaction. J Clin Microbiol. 1992;30(6):1365-73. PMid:1320625 PMCid:265294
14. Iturriza-Gomara M, Green J, Brown DW, Desselberger U, Gray JJ. Diversity within the VP4 gene of rotavirus P[8] strains: Implications for reverse transcription-PCR genotyping. J Clin Microbiol. 2000;38(2):898-901. PMid:10655412 PMCid:86240
15. Ansari S, Sherchand JB, Rijal BP, Parajuli K, Mishra SK, Dahal RK, et al. Characterization of rotavirus causing acute diarrhoea in children in Kathmandu, Nepal, showing the dominance of serotype G12. J Med Microbiol. 2013;62(Pt 1):114-20. http://dx.doi.org/10.1099/jmm.0.048124-0 PMid:23038804
16. Cilla G, Montes M, Gomariz M, Alkorta M, Iturzaeta A, Perez-Yarza EG, et al. Rotavirus genotypes in children in the Basque Country (North of Spain): rapid and intense emergence of the G12[P8] genotype. Epidemiol Infect. 2013;141(4):868-74. http://dx.doi.org/10.1017/S0950268812001306 PMid:22873952
17. Stupka JA, Degiuseppe JI, Parra GI. Increased frequency of rotavirus G3P[8] and G12P[8] in Argentina during 2008-2009: whole-genome characterization of emerging G12P[8] strains. J Clin Viro.l 2012;54(2):162-7. ttp://dx.doi.org/10.1016/j.jcv.2012.02.011 PMid:22410133
18. Oluwatoyin JM, Adeyemi AO, Famurewa O, Svensson L, Nordgren J. Molecular epidemiology of rotavirus and norovirus in Ile-Ife, Nigeria: high prevalence of G12P[8] rotavirus strains and detection of a rare norovirus genotype. J Med Virol. 2012;84(9):1489-96. http://dx.doi.org/10.1002/jmv.23343 PMid:22825829
19. Steele AD, Neuzil KM, Cunliffe NA, Madhi SA, Bos P, Ngwira B, Witte D, Todd S, Louw C, Kirsten M, Aspinall S, Van Doorn LJ, Bouckenooghe A, Suryakiran PV, Han HH. Human rotavirus vaccine Rotarix™ provides protection against diverse circulating rotavirus strains in African infants: a randomized controlled trial. BMC Infect Dis. 2012 Sep 13;12:213. http://dx.doi.org/10.1186/1471-2334-12-213 PMid:22974466 PMCid:3462149

Unusual increase of psittacosis in southern Sweden linked to wild bird exposure, January to April 2013

M Rehn (moa.rehn@smi.se)[1,2], H Ringberg[3], A Runehagen[4], B Herrmann[5], B Olsen[6], A C Petersson[7], M Hjertqvist[1], S Kühlmann-Berenzon[1], A Wallensten[1]

1. Swedish Institute for Communicable Disease Control, Solna, Sweden
2. European Programme for Intervention Epidemiology Training (EPIET), European Centre for Disease Prevention and Control (ECDC), Stockholm, Sweden
3. Department of Communicable Disease Control, Skåne county, Malmö, Sweden
4. Department of Communicable Disease Control, Kronoberg county, Växjö, Sweden
5. Department of Medical Sciences, Clinical Bacteriology, Uppsala University, Uppsala, Sweden
6. Department of Medical Sciences, Infectious Diseases, Uppsala University, Uppsala, Sweden
7. Clinical Microbiology, University and regional Laboratory, Lund, Sweden

Citation style for this article:
Rehn M, Ringberg H, Runehagen A, Herrmann B, Olsen B, Petersson AC, Hjertqvist M, Kühlmann-Berenzon S, Wallensten A. Unusual increase of psittacosis in southern Sweden linked to wild bird exposure, January to April 2013. Euro Surveill. 2013;18(19):pii=20478. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=2047

Article submitted on 30 April 2013 / published on 09 May 2013

Free-living wild birds worldwide act as reservoir for *Chlamydia psittaci*, but the risk of transmission to humans through contact with wild birds has not been widely documented. From 12 January to April 9 2013, a total of 25 cases of psittacosis were detected in southern Sweden, about a threefold increase compared with the mean of the previous 10 years. A matched case–control study investigating both domestic and wild bird exposure showed that cases were more likely than controls to have cleaned wild bird feeders or been exposed to wild bird droppings in other ways (OR: 10.1; 95% CI: 2.1–47.9). We recommend precautionary measures such as wetting bird feeders before cleaning them, to reduce the risk of transmission of *C. psittaci* when in contact with bird droppings. Furthermore, *C. psittaci* should be considered for inclusion in laboratory diagnostic routines when analysing samples from patients with atypical pneumonia, since our findings suggest that psittacosis is underdiagnosed.

Introduction

From 12 January to 9 April 2013, 25 cases of psittacosis were detected in southern Sweden. Only one case had been reported during the preceding months in 2012. Psittacosis has been a notifiable disease in Sweden since 1969. A mean of seven cases (SD: 3.3) per year have been reported in Sweden over the last 10 years [1]. The cases were found in the counties of Skåne, Kronoberg, Kalmar and Östergötland, in the south of the country. Of the 25 cases, 23 were in Skåne and Kronoberg. In order to identify the source of the outbreak, county medical officers and the Swedish Institute for Communicable Disease Control (SMI) conducted an outbreak investigation.

Free-living wild birds worldwide act as reservoirs for *Chlamydia psittaci*. The bacterium mainly infects birds but can also be transmitted to mammals. In addition to humans, *C. psittaci* has also been found in cattle, sheep and rodents [2] In humans, the disease is termed psittacosis or parrot disease, as it was first recognised in 1929, when about 800 cases of pneumonia were reported around Europe in an outbreak caused by exposure to *C. psittaci*-infected parrots imported from South America [3]. Although domestic birds are the most common source for infection in humans [4], wild bird species have also been shown to be a source of *C. psittaci*, leading to infection in humans, spanning from single cases to outbreaks. In the 1930s, fulmars (Fulmarus glacialis) that were probably infected with *C. psittaci* from dead parrots thrown overboard during transport from South America to Europe were hunted for food; this resulted in large psittacosis outbreaks in humans on the Faroe Islands and in Iceland [3]. In recent years, outbreaks of psittacosis in Australia have been linked to lawn mowing in gardens polluted by wild bird droppings [5,6]. However, the risk of transmission of *C. psittaci* through contact with wild birds has not been widely documented.

The preferred name for the disease in birds is avian chlamydiosis, although the names ornithosis and psittacosis are commonly used. The epizootiology in wild birds has not been studied extensively, but to date, *C. psittaci* has been detected in around 460 bird species within 30 orders [7]. Passerine birds (including species most frequently visiting bird feeders in gardens) have formerly not been considered to play an important role as major hosts, but there have been reports that, in the Passeriformes order, many species are carriers of the bacterium [8,9]. A wide prevalence variation is seen in studies of *C. psittaci* in wild birds [8,10,11] and can partly be explained by different diagnostic techniques and even methodological problems. Swedish studies based on DNA detection have found prevalence rates

between 1% and 3% in falcons, eagles [11], wetland birds [10] and passerines [8].

C. psittaci is excreted by infected birds in their faeces and other body fluids, and the microorganism can remain infectious for several months outside the host [12,13]. Birds get infected through inhaling or ingesting the bacteria, and the infection may persist for months, although the birds may only excrete the bacteria intermittently. Stress factors such as migration, crowding, weather changes and breeding can activate the excretion. Overt disease is unusual in wild birds [13].

Humans become infected by inhaling the bacteria through contact with contaminated bird secretions, dried-out droppings or dust from feathers [12]. Human-to-human transmission has been suggested and thought to be rare [12,14], but it has not been extensively documented. The incubation period is one to four weeks, and clinical symptoms are compatible with influenza-like illness and include fever, rigors, sweats, headache, myalgia and mild cough [15,16]. Most infected people do not show any symptoms or only experience a mild influenza-like illness. However, some develop systemic illness with severe atypical pneumonia, which can be serious and sometimes fatal. The infection can be treated with antibiotics with intracellular action.

Methods

The 23 cases in Skåne and Kronoberg were investigated further by the county medical officers. The investigation identified a cluster of eight cases (four probable and four confirmed), all linked to one index patient treated in a hospital and thus nosocomial human-to-human transmission was suspected. Due to the rarity of this event, the human-to-human transmission cluster will be described in a separate publication.

A confirmed case was defined as a person with a clinical diagnosis of psittacosis and laboratory confirmation either by detection of *C. psittaci* in respiratory secretions by polymerase chain reaction (PCR) or by an antibody (IgG or IgM) titre of 256 or greater.

A probable case was defined as a person with a clinical diagnosis of psittacosis with an IgG titre between 64 and 256.

Case–control study

We conducted a case–control study among the 23 cases in Skåne and Kronoberg to investigate potential risks of contracting psittacosis that were associated with different bird exposures, in particular wild bird exposures. All 23 cases (19 confirmed and 4 probable), with symptom onset between 31 December and 27 March and notified before 9 April, were invited to take part in the study. Eight cases (four probable and four confirmed) from the human-to-human transmission cluster were excluded. Consequently, 15 confirmed cases were included in the case–control study.

Six controls per case were selected from the population registry (Infotorg) and were matched to the cases by postal code, sex and age (± 5 years). Matching was performed partly to facilitate control selection but also to adjust for disparity in age and sex distribution of cases and differences in behaviour related to living in or outside urban areas. We asked cases and controls to fill out a web-based or paper questionnaire on the following: exposure to domestic and wild birds; history of visits to pet shops; exposure to rodents; and habits of outdoor activities.

The exposure period for cases was defined as one to four weeks before symptom onset. For controls, the exposure period was defined as the calendar months corresponding to the calendar months of the matched case's exposure period.

A variable for exposure to wild birds (yes/no) was defined as either feeding wild birds or having other contact with wild birds that was not feeding. Other contact included activities such as cleaning bird feeders or other areas covered with bird droppings and contact with dead birds.

All exposures to domestic birds were grouped into one single variable. Domestic bird exposure included history of visiting a pet shop, contact with domestic birds at home or contact with domestic birds outside of the home.

Contact with rodents was included as a question as *C. psittaci* has been identified in rodents.

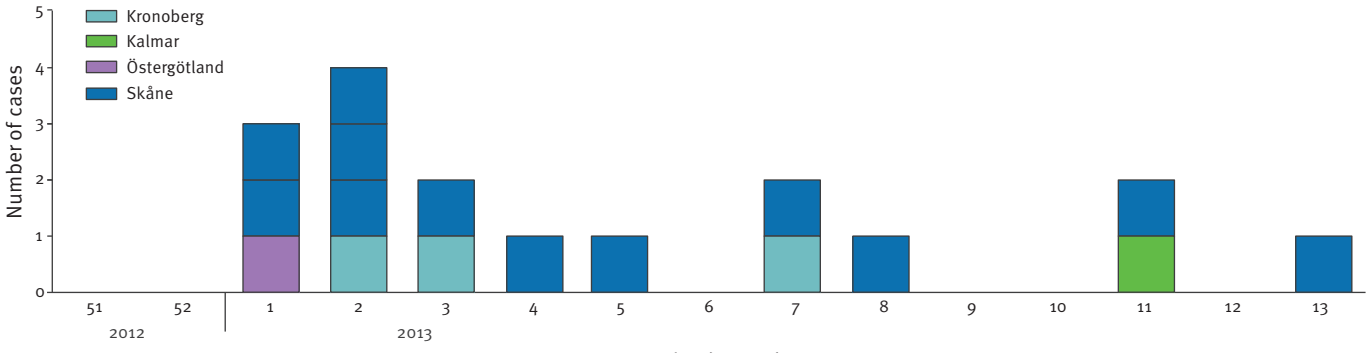
All statistical analyses consisted of conditional logistic regression from which odds ratios (OR) and 95 % CIs were obtained. We first performed univariate analysis to explore possible individual risk factors and obtain crude ORs. In the multivariable model, we included those exposures with a p value ≤0.2 in the univariate analysis, as well as age and exposure to domestic birds. Age was included to control for residual confounding since the age difference between controls that were matched to the same case could be up to 10 years. As exposure to domestic birds is known to be associated with psittacosis, it was included to correct for its potential effect on the other included associations. Statistical analysis was performed using Stata 12.

Laboratory investigation

C. psittaci was identified in respiratory samples by amplification of an 84 base pair (bp) fragment of the outer membrane protein A gene (*ompA*) according to Heddema et al. [17]. The assay was run as a duplex real-time PCR including screening for Legionella species and an internal amplification control. In order to determine the genotype of *C. psittaci*, all PCR-positive samples were further investigated by amplification and sequence analysis of a 560 bp fragment of *ompA* covering variable domain I and II. IgG and

Figure 1

Sporadic psittacosis cases in southern Sweden by week of symptom onset, reported between 12 January and 9 April 2013 (weeks 1–13) (n=17)[a]



[a] Cases from the human-to-human transmission cluster (n=8) are not shown.

IgM antibodies specific to *C. psittaci* were shown by microimmunofluorescence.

Two bird dropping samples from a parrot and a hen, kept by two cases respectively, were analysed for *C. psittaci* using the MagAttract Viral RNA M48 extraction kit (Qiagen, Hilden, Germany) and real-time PCR detection of the 23S gene, as previously described [18].

No samples were collected from cases' bird feeders.

Results

The cases' symptoms started between 31 December 2012 and 27 March 2013 (weeks 1–13) (Figure 1). The cases were found in the counties of Skåne (12 confirmed cases), Kronoberg (7 confirmed cases, 4 probable cases), Kalmar (1 confirmed case) and Östergötland (1 confirmed case) (Figure 2).Among the 23 cases in Skåne and Kronoberg, 16 were men. The median age was 66 years (range: 37–88) for men and 47 years (range: 34–72) for women. The majority of cases (n=21) had verified pneumonia. Two cases were diagnosed after recovery, thus no clinical verification was performed. A total of 19 cases were hospitalised: one case had a fatal outcome.

Case–control study results

All 15 sporadic cases and 51 of the 90 controls responded to the questionnaire (response rates of 100% and 57%, respectively).

The crude ORs for examined exposures and adjusted ORs for exposures included in the final model are presented in the Table. The univariate analysis identified that there was no statistically significant association between psittacosis and any type of exposure to wild birds (OR: 7.8; 95% CI: 1.0–64.0). When looking at more specific types of exposures to wild birds, a stronger association was identified between psittacosis and contact with wild birds other than feeding (OR: 10.1; 95% CI: 2.1–47.9), whereas the association with feeding wild birds was weaker and not statistically significant (OR: 2.6; 95% CI: 0.6–11.0). Of the 15 cases, 10 had been exposed to wild birds in other ways than feeding.

The final conditional logistic model included any contact with domestic birds, feeding wild birds, contact with wild birds other than feeding and age. When adjusting for the other exposures in the model, contact with wild birds other than feeding was the only exposure that remained statistically associated with psittacosis (adjusted OR: 26.4; 95 % CI: 2.0–348.6).

Laboratory findings

All 15 patient samples that were analysed by PCR were positive. Genotyping by sequence analysis of an approximately 500 bp fragment of the *ompA* gene was attempted for 12 samples and was successful in four. The obtained sequences were identical and clustered within genotype A. These patient samples originated from different parts of Skåne and Kronoberg. Eight cases were diagnosed using serology.

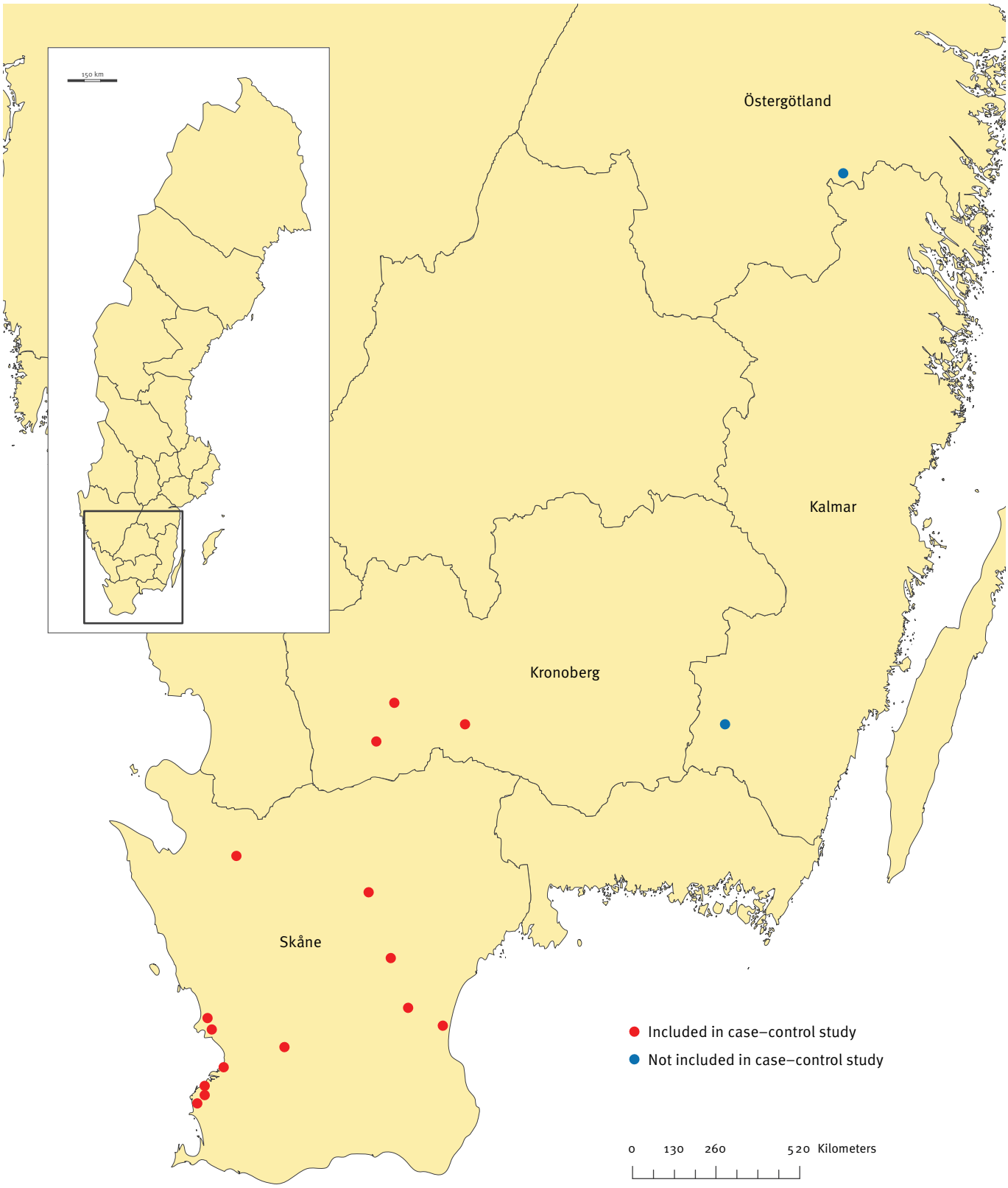
C. psittaci could not be detected in bird droppings from the parrot and the hen.

Control measures

Due to the increase in the number of psittacosis cases this winter in Sweden, county medical officers and the Swedish Institute for Communicable Disease Control (SMI) notified the public and healthcare services on 20 March 2013 about potential risks associated with handling bird feeders. The purpose was to increase awareness about the illness and about preventive measures. On 22 March, the situation was summarised in a ProMED article [19], resulting in some international correspondence, (personal communication Ander Wallensten) but no reports of an increased number of human cases in

Figure 2

Distribution of psittacosis cases in southern Sweden, reported between 12 January and 9 April 2013 (n=17)[a]



[a] Cases from the human-to-human transmission cluster (n=8) are not shown.

TABLE

Potential risk factors associated with psittacosis among psittacosis cases (n=15) and matched controls (n=51) in southern Sweden, December 2012–March 2013

Potential risk factor	Exposed		Crude		Adjusted	
	Number of cases	Number of controls	OR[a] (95 % CI)	P value	OR[a,b] (95 % CI)	P value
Age[c]	–	–	0.7 (0.4–1.2)	0.200	0.6 (0.4–1.2)	0.145
Any contact with domestic birds	4	7	2.2 (0.5–9.2)	0.296	7.0 (0.5–95.6)	0.147
Visit to shop with caged birds	2	2	3.4 (0.5–24.5)	0.229	–	–
Contact with domestic birds at home	3	4	2.8 (0.5–14.8)	0.281	–	–
Any contact with wild birds	12	25	7.8 (1.0–64.0)	0.054	–	–
Feeding wild birds	10	25	2.6 (0.6–11.0)	0.190	0.6 (0.1–4.9)	0.625
Contact with wild birds other than feeding	10	10	10.1 (2.1–47.9)	0.004	26.4 (2.0–348.6)	0.013
Contact with rodents	6	14	2.1 (0.5–8.3)	0.313	–	–
Outdoor activities	12	39	1.1 (0.2–4.8)	0.920	–	–

CI: confidence interval; OR: odds ratio.

[a] Missing values excluded.

[b] Adjusted for all the other exposures in the model. Conditional logistic regression model included any contact with domestic birds, feeding wild birds, contact with wild birds other than feeding and age.

[c] Age was included in the multivariable model to control for residual confounding since the age difference between controls that were matched to the same case could be up to 10 years.

other countries, apart from a ProMED report on 5 April describing a sixfold increase in psittacosis incidence over the past 10 years in Saint Petersburg, Russia, according to the Department of the Federal Service on Supervision of Protection of Consumer Rights and Human Well-Being in Saint Petersburg [20].

The Swedish National Veterinary Institute (SVA) did not receive any reports of increased mortality among wild birds during this winter [21].

To date, one additional case of psittacosis has been reported from Skåne, on 10 April.

Discussion

It was apparent after the initial investigation of cases conducted by the county medical officers that the only bird exposure among most cases was exposure to wild birds, mainly by tending bird feeders in their gardens. The results of our study showed that cases were more likely than controls to have been cleaning bird feeders or exposed to bird droppings in other ways. However, assuming that wild bird feeding, handling of bird feeders and exposure to droppings and dead birds have been relatively constant activities over the years, we still need an explanation as to why more people have been diagnosed with psittacosis this year.

One explanation could be that weather conditions were unfavourable for wild birds this winter, which may have induced increased excretion of the bacteria in *C. psittaci*-carrying birds and thereby caused abnormal contamination of bird feeders. This year's national bird count at bird feeders noted a general decline in numbers for most species, indicating that many birds may have succumbed this winter [22]. The Swedish Meteorological and Hydrological Institute summarised the winter as having been relatively usual, but it had prolonged periods of low temperatures and fast weather changes [23-25]. Feeding wild birds may enhance the risk of local epizootics as the birds congregate in potentially contaminated feeders.

There is no surveillance system in place for avian chlamydiosis in wild birds in Sweden although unusual findings of sick or dead birds are usually reported by the public to the Swedish National Veterinary Institute. There was no increase in the number of such reports this winter [21], but this does not rule out an increased infection prevalence or increased excretion in the wild bird population, as birds rarely become overtly ill. It may also be difficult to notice such an increase in sick and immobilised birds, as they become easy prey, e.g. for sparrow hawks, pygmy owls and cats.

A second explanation could be that a *C. psittaci* strain more pathogenic or transmissible in humans circulated in the wild bird population this year. That there are differences in the virulence of a strain for different hosts has been long known. In the Faroe Islands' outbreak of the 1930s, the strain was found to be of low pathogenicity to the fulmars, while it caused severe disease

in the humans handling them [3]. Partial genotyping of *ompA* showed that four of the cases in this current outbreak were caused by *C. psittaci ompA* type A, suggesting that they could have been infected from the same source. Further genotyping including complete sequence analysis of *ompA* combined with multi-locus sequence typing (MLST) [26] may provide insight into whether a common source explains the current outbreak. However, currently MLST requires access to isolates of *C. psittaci*, which is difficult to obtain in clinical routine diagnostics and requires expertise and equipment of biosafety level 3. Type A of *ompA* – the type causing the majority of reported human psittacosis cases [27] – is primarily associated with psittacine birds, but has also been found in other bird species including tits [8], fulmars [28] and poultry [29]. Although *C. psittaci* genotyping has been predominantly determined by *ompA* analysis, recent investigations show that type A strains with identical *ompA* genes can differ considerably in virulence and that the virulence is confined to a few single nucleotide polymorphisms [30]. To rule out the possibility of a more pathogenic strain occurring in this outbreak, further genome analysis is needed.

The clinical picture of psittacosis may be similar to disease caused by other agents such as Mycoplasma pneumoniae, Chlamydia pneumoniae and Legionella species [31] as well as to that of respiratory illness caused by influenza, which was also circulating at the time [32]. However, a *C. psittaci*-positive PCR result in combination with relevant symptoms is, in our opinion, robust evidence that the infections were caused by *C. psittaci*, especially since the other agents were in most cases excluded by testing.

One likely explanation as to why cases mainly occurred in two counties in southern Sweden is that different diagnostic routines are in place in the clinical laboratories. Since 2005, the laboratory that diagnosed most of the cases in this outbreak has routinely tested for *C. psittaci* when a sample from a patient with atypical pneumonia is submitted for Legionella analysis. This is not the case in most other Swedish laboratories. Therefore it is likely that cases in other counties may have been missed. A partial review of four cases of atypical pneumonia in Kronoberg during this winter showed that two had not been tested for Legionella or *C. psittaci*, even though they tested negative for M. pneumoniae. Retesting of these undiagnosed cases revealed two additional suspected psittacosis cases, thus supporting the theory that cases may have been missed. This is an important observation, which indicates that psittacosis is an under-diagnosed disease and should be considered more often in cases of atypical pneumonia. Apart from this explanation, it is difficult to understand why cases occurred only in southern Sweden. The bird species most often visiting bird feeders in gardens in the southern part of Sweden (e.g. great tit, tree Sparrow, blue tit, green finch, bullfinch) [22] can be found in almost all parts of Sweden. These species are relatively short-lived and migrate only within short distances during winter, depending on access to food.

Regardless of the small size of the study, a significant association between psittacosis and exposure to wild birds was shown. However, the risk may have been underestimated, as it could be assumed that controls who kept bird feeders in their gardens were more likely to have responded to the questionnaire, leading to a bias towards bird feeders among the controls. Almost half of the controls in this study had fed birds with bird feeders in their garden during December to March. Cases and controls often lived in small towns or in the country side, where bird feeding might be more frequent than in cities. Even if the population studied may not be representative of the Swedish population in general, this high proportion indicates that wild bird feeding is common in non-urban parts of southern Sweden.

The risk for psittacosis when exposed to wild birds is difficult to assess. Large outbreaks with 20% mortality were seen on the Faroe Islands in the 1930s [3], whereas contemporary studies found no cases of psittacosis among people exposed to birds with a high prevalence (10%) of infection [28]. Additionally, another study could not detect antibodies to *C. psittaci* among 65 bird ringers, a group with close exposure to a variety of birds, mainly passerines [8]. In general, the risk of human infection from contact with wild birds is likely to be low [33]. However, it can be expected to be higher if exposed to birds carrying *C. psittaci* that are suffering from stress or sickness leading to increased excretion of the bacteria.

Public health implications

This study shows that there is an association between exposure to wild birds through handling wild bird feeders and bird droppings during winter and psittacosis. Bird feeding alone, however, was not associated with disease. The actual risk is likely to vary geographically and over time due to epizootics in the wild bird population that may be aggravated by harsh weather conditions.

Wild bird feeding is a common and appreciated pastime in Sweden and should not be discouraged based on the results of this study. However, people feeding birds should be informed of the risk of handling bird droppings and how to minimise the risk, for example, by using safe practices when cleaning bird feeders and using bird feeders constructed to limit birds from defecating on the feeder. The Swedish Institute for Communicable Disease Control has previously recommended wetting the areas covered with bird droppings before removing them, hand washing after contact with bird droppings and only cleaning bird feeders in well-ventilated areas [34]. For people working in heavily contaminated and confined rooms or areas, respiratory air filters should be used. On the basis of the findings

from this current investigation, we support these recommendations.

Finally, an important recommendation from this study is that *C. psittaci* should be considered as a potential causative agent of atypical pneumonia. Inclusion of the pathogen in laboratory diagnostic routines when analysing samples from patients with atypical pneumonia is likely to identify more cases, and should therefore be considered.

Acknowledgements

We thank Caroline Bröjer at the Swedish national Veterinary Institute for information regarding observations in the wild bird population, as well as Hans Fredlund at the Department for Communicable Disease Control in Örebro for information about serological diagnostics. We would also like to acknowledge the contribution of EPIET coordinator Marion Muehlen, in reviewing this manuscript.

Authors' contributions

Moa Rehn was responsible for conducting the case–control study, data analysis and drafted the manuscript together with Anders Wallensten, who had the overall responsibility for the study. All authors revised and approved the final report submitted. Arne Runehagen and Håkan Ringberg were responsible for investigation of cases. Björn Herrmann performed analysis on bird samples and contributed with his expertise in the field. Björn Olsen contributed with his expertise in the field. Ann-Cathrine Petersson performed the laboratory diagnostics and genotyping of human samples. Sharon Kühlmann-Berenzon supervised the statistical analysis. Marika Hjertqvist is responsible for the surveillance of psittacosis in Sweden.

Conflict of interest

None declared.

References

1. Smittskyddsinstitutet (SMI). Parrot fever. Solna: SMI. Updated 8 Apr 2013. [Accessed 16 Apr 2013]. Available from: http://www.smittskyddsinstitutet.se/in-english/statistics/parrot-fever/
2. Andersen AA. Serotyping of Chlamydia psittaci isolates using serovar-specific monoclonal antibodies with the microimmunofluorescence test. J Clinical Microbiol. 1991;29(4):707-11.
3. Rasmussen-Ejde R. Ueber eine durch Sturmvögel übertragbare Lungenerkrankung auf den Färöern. [About a fulmar related transmission of lung disease in the Faroe Islands]. Zentralblatt für Bakteriologie, Parasitenkunde und Infektionskrankheiten, Erste Abteilung Originale. 1938;143:89-93. German.
4. Smith KA, Bradley KK, Stobierski MG, Tengelsen LA; National Association of State Public Health Veterinarians Psittacosis Compendium Committee. Compendium of measures to control Chlamydophila psittaci (formerly Chlamydia psittaci) infection among humans (psittacosis) and pet birds, 2005. J Am Vet Med Assoc. 2005;226(4):532-9.
5. Telfer BL, Moberley SA, Hort KP, Branley JM, Dwyer DE, Muscatello DJ, et al. Probable psittacosis outbreak linked to wild birds. Emerg Infect Dis. 2005;11(3):391-7. http://dx.doi.org/10.3201/eid1103.040601 PMid:15757553 PMCid:3298256
6. Williams J, Tallis G, Dalton C, Ng S, Beaton S, Catton M, et al. Community outbreak of psittacosis in a rural Australian town. Lancet. 1998;351(9117):1697-9. http://dx.doi.org/10.1016/S0140-6736(97)10444-5
7. Kaleta EF, Taday EM. Avian host range of Chlamydophila spp. based on isolation, antigen detection and serology. Avian Pathol. 2003;32(5):435-61.
8. Olsen B, Persson K, Broholm KA. PCR detection of Chlamydia psittaci in faecal samples from passerine birds in Sweden. Epidemiol Infect. 1998;121(2):481-4. http://dx.doi.org/10.1017/S0950268898001320 PMid:9825803 PMCid:2809549
9. Simpson VR, Bevan R. Chlamydia psittaci infection in robins. Veterinary Rec. 1989;125(21):537. http://dx.doi.org/10.1136/vr.125.21.537-b
10. Blomqvist M, Christerson L, Waldenström J, Herrmann B, Olsen B. Chlamydia psittaci in Swedish wetland birds: a risk to zoonotic infection? Avian Dis. 2012;56(4):737-40. http://dx.doi.org/10.1637/10105-022812-ResNote.1 PMid:23397847
11. Blomqvist M, Christerson L, Waldenström J, Lindberg P, Helander B, Gunnarsson G, et al. Chlamydia psittaci in birds of prey, Sweden. Infect Ecol Epidemiol. 2012;2.
12. Compendium of measures to control Chlamydia psittaci infection among humans (psittacosis) and pet birds (avian chlamydiosis), 2000. Centers for Disease Control and Prevention. MMWR Recomm Rep. 2000;49(RR-8):3-17.
13. Thomas NJ, Hunter DB, Atkinson CT. Infectious diseases of wild birds. Oxford: Blackwell publishing; 2007.
14. McGuigan CC, McIntyre PG, Templeton K. Psittacosis outbreak in Tayside, Scotland, December 2011 to February 2012. Euro Surveill. 2012;17(22):pii=20186. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20186
15. Stewardson AJ, Grayson ML. Psittacosis. Infect Dis Clin North Am. 2010;24(1):7-25. http://dx.doi.org/10.1016/j.idc.2009.10.003 PMid:20171542
16. Yung AP, Grayson ML. Psittacosis--a review of 135 cases. Med J Aust. 1988;148(5):228-33.
17. Heddema ER, Beld MG, de Wever B, Langerak AA, Pannekoek Y, Duim B. Development of an internally controlled real-time PCR assay for detection of Chlamydophila psittaci in the LightCycler 2.0 system. Clin Microbiol Infect. 2006;12(6):571-5. http://dx.doi.org/10.1111/j.1469-0691.2006.01417.x PMid:16700707
18. Ehricht R, Slickers P, Goellner S, Hotzel H, Sachse K. Optimized DNA microarray assay allows detection and genotyping of single PCR-amplifiable target copies. Mol Cell Probes. 2006;20(1):60-3. http://dx.doi.org/10.1016/j.mcp.2005.09.003 PMid:16330186
19. ProMED-mail. Psittacosis - Sweden: (Skane, Kronoberg) RFI. Archive Number 20130322.1599238. 22 Mar 2013. Available from: http://www.promedmail.org/
20. ProMED-mail. Psittacosis - Russia: (Saint Petersburg). Archive Number 20130405.1622310. 5 Apr 2013. Available from: http://www.promedmail.org/
21. Statens Veterinärmedisinska Anstalt (SVA). Papegojsjuka i södra Sverige. [Parrot fever in southern Sweden]. Uppsala: SVA. Updated 21 Mar 2013. [Accessed 23 Apr 2013]. Swedish. Available from: http://www.sva.se/sv/Djurhalsa1/Zoonoser/Psittakos-papegojsjuka-som-zoonos/Papegojsjuka-i-sodra-Sverige/
22. Sveriges Ornitologiska Förening (SOF). Vinterfåglar inpå knuten. [Winter birds around your house]. Lilla Brunneby: SOF. [Acccessed 25 Apr 2013]. Swedish. Available from: http://www.turgor.se/net_sofnet_vinter_oldasp/uuser/Valj_lan_AllaArt_ej_kommun.aspx
23. Swedish Meteorological and Hydrological Institute (SMHI). Februari dominerades av kraftiga temperatursvängningar. [February was dominated by strong fluctuations in temperature]. Norrköping: SMHI. Updated 1 Mar 2013. [Accessed 22 Apr 2013]. Swedish. Available from: http://www.smhi.se/nyhetsarkiv/februari-dominerades-av-kraftiga-temperatursvangningar-1.29014?search=yes&month=3&year=2013
24. Swedish Meteorological and Hydrological Institute (SMHI). Vårvärmen har låtit vänta på sig i mars. [The arrival of the spring is late]. Norrköping: SMHI. Updated 28 Mar 2013. [Accessed 22 Apr 2013]. Swedish. Available from: http://www.smhi.se/nyhetsarkiv/varvarmen-har-latit-vanta-pa-sig-i-mars-1.29626?search=yes&month=3&year=2013
25. Swedish Meteorological and Hydrological Institute (SMHI). Vintern 2012/2013 – ganska normal svensk vinter. Norrköping: SMHI. Updated 4 Mar 2013. [Accessed 6 May 2013]. Swedish. Available from: http://www.smhi.se/nyhetsarkiv/vintern-2012-2013-ganska-normal-svensk-vinter-1.29088?search=yes&month=3&year=2013

26. Pannekoek Y, Dickx V, Beeckman DS, Jolley KA, Keijzers WC, Vretou E, et al. Multi locus sequence typing of Chlamydia reveals an association between Chlamydia psittaci genotypes and host species. PloS One. 2010;5(12):e14179.
27. Harkinezhad T, Geens T, Vanrompay D. Chlamydophila psittaci infections in birds: a review with emphasis on zoonotic consequences. Vet Microbiol. 2009;135(1-2):68-77.
28. Herrmann B, Persson H, Jensen JK, Joensen HD, Klint M, Olsen B. Chlamydophila psittaci in Fulmars, the Faroe Islands. Emerg Infect Dis. 2006;12(2):330-2. http://dx.doi.org/10.3201/eid1202.050404 PMid:16494766 PMCid:3373105
29. Gaede W, Reckling KF, Dresenkamp B, Kenklies S, Schubert E, Noack U, et al. Chlamydophila psittaci infections in humans during an outbreak of psittacosis from poultry in Germany. Zoonoses Public Health. 2008;55(4):184-8. http://dx.doi.org/10.1111/j.1863-2378.2008.01108.x PMid:18387139
30. Miyairi I, Laxton JD, Wang X, Obert CA, Arva Tatireddigari VR, van Rooijen N, et al. Chlamydia psittaci genetic variants differ in virulence by modulation of host immunity. J Infect Dis. 2011;204(4):654-63. http://dx.doi.org/10.1093/infdis/jir333 PMid:21791668 PMCid:3203387
31. Thibodeau KP, Viera AJ. Atypical pathogens and challenges in community-acquired pneumonia. Am Fam Physician. 2004;69(7):1699-706.
32. Smittskyddsinstitutet (SMI). Influensarapport, vecka 17 (22/4 - 28/4), 2013. [Influenza report week 17 (22/4-28/4) 2013]. Solna: SMI. Updated 6 May 2013. [Accessed 7 May 2013]. Swedish. Available from: http://www.smittskyddsinstitutet.se/publikationer/veckorapporter/influensarapporter/sasongen-20122013/influensarapport-vecka-17-224---284-2013/
33. Tsiodras S, Kelesidis T, Kelesidis I, Bauchinger U, Falagas ME. Human infections associated with wild birds. J Infect. 2008;56(2):83-98. http://dx.doi.org/10.1016/j.jinf.2007.11.001 PMid:18096237
34. Smittskyddsinstitutet (SMI). Papegojsjuka i södra Sverige 2013. [Parrot fever in southern Sweden]. Solna: SMI. Updated 18 Mar 2013. [Accessed 7 May 2013]. Swedish. Available from: http://www.smittskyddsinstitutet.se/nyhetsarkiv/2013/papegojsjuka-i-sodra-sverige/

Updated information and map of areas where the risk of contracting tick-borne encephalitis is largest in Germany published

Eurosurveillance editorial team (eurosurveillance@ecdc.europa.eu)[1]
1. European Center for Disease Prevention and Control, Stockhom, Sweden

Citation style for this article:
Eurosurveillance editorial team. Updated information and map of areas where the risk of contracting tick-borne encephalitis is largest in Germany published. Euro Surveill. 2013;18(19):pii=20477. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20477

Article published on 09 May 2013

On 6 May 2013 the Robert Koch Institute (RKI) published up-to-date information on the incidence of tick-borne encephalitis (TBE) in Germany [1].

Every year data on the number of cases of TBE is reported in Germany. The data is broken down by 'Kreis' or district, which is a defined geographic area. The total number of TBE cases reported in 2012 was 195, down by 54% from 2011, when 424 cases were reported. Annual case numbers in previous years since 2001 fluctuated between a low of 239 and high of 546, with no clear trend. These fluctuations are most likely related to ecological factors influencing natural TBE foci as well as the risk of human-tick contact.

In 2012, a total of 141 districts were defined as areas where a significantly increased incidence had been noted. A district is defined as a TBE risk area if the number of TBE cases in the district or district region (consisting of the district plus all adjoining districts) in any 5-year-interval since 2002 was significantly greater ($p < 0.05$ according to the poisson distribution) than the number of cases required to reach an incidence of 1/100000 inhabitants. The primary preventive measure is TBE vaccination.

The Standing Commission on Vaccination Recommendations (STIKO) at RKI recommends vaccination against TBE for (i) people who live or work in areas at risk of TBE and there are at risk of tick bites, and (ii) for people in endemic areas for other reasons if they are at risk of tick exposure.

References

1. Tick-borne encephalitis – risk areas in Germany. Epidemiologisches Bulletin 18/2013. 6 May 2013, Germany. In German. Available from: https://www.rki.de/DE/Content/Infekt/EpidBull/Archiv/2013/Ausgaben/18_13.pdf?__blob=publicationFile