Outbreaks report

A LARGE INCREASE OF SALMONELLA INFECTIONS IN 2003 IN THE NETHERLANDS: HOT SUMMER OR SIDE EFFECT OF THE AVIAN INFLUENZA OUTBREAK?

W van Pelt¹ , D Mevius², HG Stoelhorst³, S Kovats⁴, AW van de Giessen¹, W Wannet¹, YTHP Duynhoven¹

In June 2003, the NSC (Dutch National Salmonella Centre) reported a significant excess isolation rate of Salmonella Enteritidis when compared with earlier years in most regional public health laboratories. By the end of 2003, this amounted to an extra 540 laboratory confirmed cases for the whole of the Netherlands, which implies an estimated 7500 extra cases of gastroenteritis caused by S. Enteritidis in the general population, an increase of 50% on previous years. The hot summer could not explain the findings. Strong evidence has been found to suggest that the increase in importation of salmonella contaminated eggs, as a side effect of a concurrent avian influenza outbreak, was the most probable reason for this excess.

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Introduction

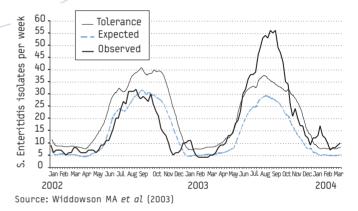
In June 2003, the Dutch National Salmonella Centre reported a significant excess Salmonella isolation rate compared to previous years in most regional public health laboratories (FIGURE 1). Beginning in May 2003, the number of laboratory confirmed cases clearly increased to above the level expected [1], and from June to November, and again since the beginning of 2004, to above the level of tolerance (a measure for the significance of an excess). This increase involved only Salmonella Enteritidis, and not S. Typhimurium (ST), or other Salmonella serotypes or Campylobacter spp. In this paper, we try to indicate the possible role in the 2003 excess of the hot summer compared with that of the increase of imports of (contaminated) eggs due to the concurrent avian influenza outbreak.

Salmonella surveillance

The data are from the National Salmonella Centre (NSC) and the National and European Reference Laboratory (CRL) for Salmonella at RIVM that performs the sero- and phage-typing of isolates taken from humans (mostly sent by regional public health laboratories,covering 64% of the Dutch population) and animals, from food, animal feed and from the environment [2]. The sensitivity to various antibiotics has been quantitatively determined by the minimal inhibitory concentration (MIC) at the Centraal Instituut voor DierziekteControle - Lelystad (CIDC- Central Institute of Animal Disease Control) [3].

FIGURE 1

Observed and expected laboratory confirmed cases of Salmonella Enteritidis infections since 2002 in the Netherlands



The excess isolation rate of S. Enteritidis since May 2003 amounted to an extra 540 laboratory confirmed cases for the whole of the Netherlands at the end of 2003 (FIGURE 2, adjusted for the 64% coverage of the laboratory surveillance). This is 50% higher than excesses found in previous years. Figure 2 shows that the large increase of cases involved S. Enteritidis only. Extrapolation using data from a 1999 study [4], then 540 extra laboratory confirmed cases would mean an estimated 7500 extra cases of gastroenteritis caused by S. Enteritidis in the total population. Denmark has a laboratory surveillance system comparable to that of the Netherlands, and a Danish study has shown that, when compared with controls, 1.5-2.1% of the laboratory confirmed patients with salmonellosis die within one year, probably due to the infection [5]. This would mean that the 2003 excess S. Enteritidis infections in the Netherlands caused 8-11 deaths.

Hot summer

The excess of SE cases in June and July was at first attributed to the exceptionally hot weather that lasted until August, when temperatures were far higher than normal for that time of year [FIGURE 2]. This was suggested by the findings in the WHO cCASHh (project (climate Change and Adaptation Strategies for Human health in Europe: http://www.who.dk/ccashh) of time series analysis of salmonellosis in 10 European countries. An additional effect of temperature was demonstrated clearly on the risk for food poisoning, apart from a general effect of season itself [6]. In the Dutch data (covering the period 1984-2001) this effect was exceptionally strong for S. Enteritidis (a linear 12.6% increase per oC). The largest effect of temperature is one week before onset of illness, with diminishing but positive effects up to 5 weeks [6]. Earlier calculations of our own, that more strongly adjust for season (covering 1990-1998), illustrate these findings [FIGURE 3].

^{1.} National Institute of Public Health and the Environment (RIVM),

Bilthoven, the Netherlands Central Institute for Animal Disease Control (CIDC), Lelystad, the Netherlands Product Boards for Livestock, Meat and Eggs, Zoetermeer, the Netherlands

London School of Hygiene and Tropical Medicine, London, United Kingdom

FIGURE 2

Cumulative number of laboratory confirmed cases of salmonellosis in excess of expected* and monthly average day temperatures for 2003 in the Netherlands

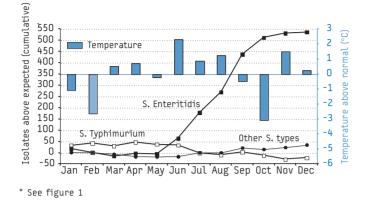
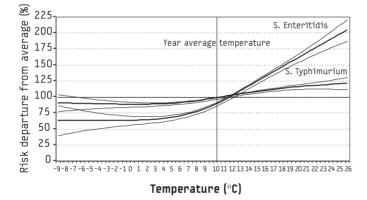


FIGURE 3

Temperature-salmonellosis relationship, adjusted for season. Analyses are based on Dutch Salmonella surveillance data covering 1990-1998. The Netherlands



Above a threshold of about 6°C the risk linearly increases; most strongly for S. Enteritidis but, in the Netherlands, hardly so for ST. The difference between S. Enteritidis and S. Typhimurium probably derives from the traditional food preparation of the main food vehicle for S. Enteritidis, eggs, sometimes processed and consumed raw, whereas S. Typhimurium is mainly associated with meat from pigs and cattle that normally get a proper heat treatment.

However, an inquiry among the members of the Enter-net surveillance network revealed that most European countries had not experienced an excess of Salmonella infections during the same time period, with the exception of Belgium, and England and Wales. Therefore the hot summer was unlikely to have had a major role in the excess.. Furthermore, figure 2 shows that the 'hot summer' occurred during the months of June, July and –August, when temperatures were on average between 1 and 2.5°C above normal. This period was followed by two months when temperatures that were below normal. Clearly, a 7-13% increase per °C cannot explain the 50% excess of cases at the end of the year. Note that due to the lag of about one week between temperature changes and the onset of disease and another three weeks until the laboratory results appear, the temperature findings in figure 2 should be compared with the surveillance findings of one month later.

Raw shell eggs

Surveillance programmes in the Netherlands show that the Salmonella control programme for poultry has been successful in reducing S. Enteritidis in broilers almost to exclusion [7]. However, in

commercial layers in 2003, more than 6% (9% in 2001 and 14% in 1997) of the flocks remained S. Enteritidis positive (7). This makes raw shell eggs the main suspect food vehicle for causing the 2003 excess of S. Enteritidis infections in humans. However, phage typing of S. Enteritidis, combined with antimicrobial resistance testing, showed remarkable differences between human and poultry isolates, pointing to a source from outside the Netherlands [3]. In 2003, twice as much phage type 1 (PT 1) was found among S. Enteritidis isolates from Dutch patients (14.5%) as between 1998-2002, 54% of them being resistant to nalidixic acid (Na) and with decreased susceptibility to ciprofloxacin. Between 1998-2003, PT 1 accounted for about 5% of all S. Enteritidis poultry isolates (SE isolates derive almost exclusively from layer flocks, but none of these were resistant to nalidixic acid. Human infections with PT 1(Na) in the Netherlands appeared to be travel-related three times more often than other S. Enteritidis phage types, and more than 50% of PT1(Na) infections were related to travel to Spain and Portugal.

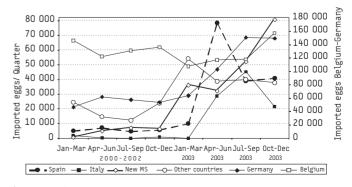
A series of outbreaks with S. Enteritidis in the United Kingdom (UK) in 2002 and again in 2003 [8] led to several investigations of raw shell eggs [9,10]. Among a range of other phage types, PT 1(Na) was found to be associated with Spanish eggs. Salmonella was found in 0.3% of the eggs produced in the UK and in 5.1% and 6.7% in two surveys of eggs imported from Spain and was high as well (7.7%) in other imports where the country of origin was unknown. Salmonella was found in only 0-0.03% of eggs produced in Holland [11], i.e. 10 and 160 times lower than eggs produced in the UK and Spain respectively. It is nevertheless estimated that about 35% human salmonellosis cases in the Netherlands are due to consumption of eggs [11].

Avian Influenza outbreak in poultry

The Netherlands experienced a major outbreak of avian influenza in poultry in the spring of 2003 that led to a shortage of eggs on the Dutch market. Data from EUROSTAT [FIGURE 4] shows that this shortage was compensated for with egg imports, mainly from Germany, Italy and Spain (>8-fold increase in the 2nd quarter of 2003 as compared to former quarters). In the fourth quarter of 2003, the number of imported eggs was still considerably higher than in former years. In fact the contribution of eggs imported from the new EU member states, negligible in previous years, continued to increase and doubled in the second half of 2003. Figure 1 shows that in the first months of 2004 there was still an excess of S. Enteritidis cases, now predominantly PT 8. PT 8 has been reported as a problem in the poultry industry in several new member states in central Europe (personal communication with NRL and ENTERNET colleagues). For several years, central European countries have been the number one destination of travellers that returned with a PT 8 infection.

FIGURE 4

EUROSTAT data on the number of imported eggs per quarter*



^{*} The data from 2000-2002 are averaged

Discussion

In June 2003, the Dutch National Salmonella Centre reported a significant excess isolation rate of S. Enteritidis when compared with previous years . The hot summer of 2003 could not explain the findings. Strong evidence was found to suggest that the increase in importation of contaminated eggs, as a result of the avian influenza outbreak, was the most probable reason for this excess.

The lesson is that with the low level of contamination in Dutch eggs, even small increases in imports of eggs that are relatively highly contaminated with S. Enteritidis, may have a large impact on the incidence of human salmonellosis, and may strongly affect both morbidity and mortality. Hence, major changes in market supply should initially be considered as a potential serious public health threat. Continuous surveillance, especially of imported eggs, is therefore strongly recommended. The approaching implementation of a harmonized system for monitoring and control of *Salmonella* spp. in flocks of laying hens in all EU Member States (EC Zoonosis Regulation 2160/2003 is an important, and constructive development in this respect.

Trace back of the source of salmonellosis cases, serotyping and phage typing of positive findings, together with testing for antimicrobial resistance, are essential for decision making and providing a basis for intervention.

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ORIGINAL ARTICLES

Euroroundup

BASIC SURVEILLANCE NETWORK, A EUROPEAN DATABASE FOR SURVEILLANCE DATA ON INFECTIOUS DISEASES

A Ternhag, A Tegnell, B Lesko, K Skaerlund, P Penttinen*

The Basic Surveillance Network was started in 2000 and is one of the networks on infectious diseases funded by the European Commission. The network collects and makes readily available basic surveillance data on infectious diseases from all the 'old' (pre-2004) European Union member states. The aim is to provide easy access to descriptive data that already exist in national databases, so that it is possible to monitor and compare incidence trends for infectious diseases in the EU member states.

The list of diseases covered by the network has recently been expanded from 10 initial 'pilot' diseases to over 40 diseases listed by the EU to be under surveillance. In the near future, the new member states will be invited to participate in the network.

Data are case-based and comprise date of onset of disease, age and sex. Only a very short list of disease specific additional variables, such as country of infection or immunisation status, is collected. Classification of cases (possible, probable, confirmed) is specified according to EU case definitions.

1. Swedish Institute for Infectious Disease Control, Stockholm, Sweden

The participants of the network have access to an internal web site were all the data is presented in tables and graphs. An open website is available for the public at https://www.eubsn.org./BSN/

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Introduction

In September 1998, a proposal from the European Commission was adopted as a Decision of the European Parliament and Council (2119/98/EC) to set up a network for the epidemiological surveillance and control of communicable diseases in the European Community.

With this legal document as a background, several projects to develop designated surveillance networks (DSN) have been funded by the Commission and they are now operating at the European level (for diseases such as salmonellosis, legionellosis, tuberculosis and HIV/AIDS). Each one of them is collecting data at a detailed level and most of them have objectives beyond routine surveillance