**Original Articles**

**Outbreaks report**

**A large increase of Salmonella infections in 2003 in the Netherlands: hot summer or side effect of the avian influenza outbreak?**

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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.

**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].

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Therefore the hot summer was unlikely to have had a major role in the period, with the exception of Belgium, and England and Wales. Surveillance network revealed that most European countries had not exceeded 1 and 2.5°C above normal. This period was 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 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 0.03% 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.

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...
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.

**References**


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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.