Special Review

Salmonellosis – a cause of concern and a challenge

H.-J. Sinell

Institut für Lebensmittelhygiene, Freie Universität Berlin

Salmonellose – Grund zur Betroffenheit und Herausforderung

Key words: pathogenicity; epidemiology; food as a vehicle; prevention and control; animal production; food processing

Schlüsselwörter: Pathogenese; Epidemiologie; Vorkommen in Lebensmitteln; Prävention und Kontrolle; Tierische Lebensmittel; Lebensmittelverarbeitung

Worldwide zoonosis

The Nutrition Report („Ernährungsbericht“) of the Federal Government recorded 91,237 cases of salmonellosis in 1990. This is more than double the number registered in 1980. Compared to 1970, the figure has increased by the factor 7.6. Intensified investigations in connection with the Salmonella Surveillance Program of the Federal Government might have resulted in an increase of reported incidents before 1980. The situation then appeared to stabilize, the number of cases even dropping for a few years. However, hopes of having attained a plateau or possibly of having the problem under control were disappointed. The rapid spread of the infection during the late 1980s and, above all, the unparalleled record of 1990 exceeded the worst expectations. The system of sampling and investigation as well as interpretation of the data has not changed for many years. Consequently, the recent dramatic increase in the frequency of salmonellosis is certainly not a result of intensified investigations.

As far as recording systems exist, the increase of salmonellosis is a world-wide phenomenon. Reports from WHO at the beginning of the 1980s, have shown the incidence of salmonellosis in Third World countries to vary from a figure comparable to Germany (Kenya, Mexico, Turkey) to 30 times (Senegal) or nearly 100 times that figure (Guinea-Bissau, Angola). The rise in salmonellosis is therefore not a specific German or European problem. We are just approaching the international level.

Taxonomy and pathogenicity

To enable a better understanding of what should be done, some details will be recalled. Salmonellae are gram-negative, and usually (with one exception) motile rod-shaped bacteria that belong to the family Enterobacteriaceae. *S. choleraesuis*
and *S. enterica* are regarded as species of their own, the latter one including seven subspecies and 2200 immunologically distinct serovars. Salmonellae cause disease that can be naturally transmitted between vertebrates and also to humans. A very few serovars are more or less host-adapted, but the vast majority is transmitted between many host species. True host specificity exists solely with regard to *S. typhi* infection in humans. Salmonellosis is the classical zoonosis.

Infection occurs usually via the oral route. An incubation period of 12 to 36 (rarely, 5 to 72) h and an unspecific prodromal stage are followed by abdominal pain, fever (usually \( \leq 38 \) °C) and diarrhea. In view of the short incubation period and of the clinical picture similar to poisoning, the disease is often termed a toxico-infection or toxico-infection. The acute stage of illness is overcome in most cases after 8 to 14 days. There are also severe progressive forms that are complicated by cardiovascular disorders requiring intensive care. *S. choleraesuis* infection – fortunately rarely found – may often assume a septic course and end fatally. The overall lethality is about 0.1 % and involves, in particular, the newborn and the elderly.

A minimum number of cells (i.e., the minimum infective dose) must be ingested in order to cause overt illness. It was believed for many years that about \( 10^5 \) living Salmonella cells must be ingested to cause disease in man. However, several more recent reports have shown that as few as 3 to 10 cells/g may lead to manifest infection. Accordingly, every finding of salmonellae in a sample of ready-to-eat food – irrespective of their actual number – must be regarded at least as a potential health hazard. To introduce tolerance levels or sampling schemes with acceptance numbers ("c")1 would indeed be a disastrous mistake.

Knowledge about the mechanisms of virulence has been considerably extended during the past decade. Chromosomal determinants and plasmids have been detected that encode adhesion and invasion in the host cell. Fimbriae, adherence factors and hemagglutinins have also been identified. Certain polypeptides that are synthesized in the bacterial cell enhance adhesion to the intestinal mucosa, lipopolysaccharides hamper phagocytosis of macrophages, and iron-chelating siderophores compete with the host cell for bio-available iron. Hydrophobic proteins of the bacterial cell surface, so-called porins, are said to interfere with transmembrane diffusion and to inhibit phagocytosis of polymorphonuclear leukocytes and macrophages. Their action has still not been elucidated. It has not yet been established whether the mechanisms of virulence of salmonellae are controlled by the same principle in all serovars.

Enterotoxin has been detected in a number of serovars. The toxin activates adenylate cyclase in the cytoplasm membrane, thereby inducing formation of 3',5'-cyclo-AMP and excretion of fluid. This correlates well with the mode of action of LT produced by ETEC and the mode of action of the cholera toxin. Salmonella enterotoxin is immunologically closely related to both of the latter.

### Spread, chains of infection, epidemiology

The organism persists outside the host and may multiply if temperature and environmental conditions are adequate. International trade with contaminated feedstuffs has contributed to the spread of the infection in livestock and in pets. Multifold chains of infection link feeds, animals, foods, humans, sewage and waste materials. Besides livestock and pets, "animal" includes also game and other wild