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# Individual and household-level risk factors for sporadic salmonellosis in children☆

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**Summary** *Objectives:* To explore risk factors for sporadic salmonellosis at the individual and household level in children in tropical Darwin, where animal faeces contaminated with *Salmonella* is thought to be common.
*Methods:* A 2-year community based case—control study of children aged 0—4 years residing in Darwin and Palmerston from June 2006. Variables included behaviour, health, food, family and housing characteristics. Environmental samples were taken from houses of case and control children.
*Results:* Of children whose parents were contacted, 59/131 cases and 95/222 controls were included. *Salmonella* was isolated from 41/56 (73%) case houses and 18/29 (62%) control houses ($p = 0.29$). Multivariate analyses showed breastfeeding 0.16 ($p = 0.02$), increasing age (months) 0.89 ($p = 0.00$) and daily vacuuming 0.18 ($p = 0.06$) were protective; consuming powdered formula milk 4.88 ($p = 0.02$), pet ownership 4.86 ($p = 0.02$), oral contact with animals 7.85 ($p = 0.05$), recent antibiotic use 10.01 ($p = 0.03$) and sweeping in the presence of children 3.73 ($p = 0.04$) were associated with sporadic salmonellosis.
*Conclusions:* Salmonellosis in children under 5 years of age is associated with potentially modifiable risk factors other than food. Breastfeeding beyond 6 months, careful hygiene when preparing formula milk and around pets, frequent cleaning of infant play areas especially quick removal of animal faeces are behaviours likely to reduce childhood sporadic salmonellosis.


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## Introduction

Salmonellosis is a significant contributor to morbidity and mortality worldwide with an estimated 93 million cases and 155,000 deaths each year.[1] More than 2500 serovariants of *Salmonellae* have been isolated from humans or animals and more than 99.5% of these are capable of causing disease in humans.[2,3] Most serovars are of zoonotic origin, have a broad host spectrum and can infect humans.[4]

Salmonellosis in children has been linked to the contamination of surfaces and environmental sources such as soil, reptiles, pets and livestock.[5–14] Other risk factors include the consumption of undercooked eggs and beef[15–21] and exposure to drugs, such as antibiotics and antacids, and particular food items, such as infant formula.[22] Conversely, breastfeeding has been found to confer protection.[19–21] The mode of transmission of salmonellosis varies with age: in infants, the likely transmission route is through passive contact, such as exposure to dust, aerosols or contaminated surfaces in the home, in older children through active contact, such as playing with infected animals and in adults through eating contaminated foodstuff.[23–28]

Even though *Salmonellae* do not multiply significantly outside digestive tracts, they can survive in a wide range of environments if temperature and humidity are favourable, such as in tropical climates.[29,30] In Australia, the state or territory with the highest rate of salmonellosis is the Northern Territory (NT) where the rate of notifications in 0–4 year olds between 1996 and 2005 is 6.8 fold higher than the national age specific rate (1366 vs 201 per 100,000 child-years) and 16.5 times the rate in the NT 5–9 age group (83 per 100,000 child-years). The median age for salmonellosis is 13 months and the peak age of notification is 10 months.[31] The majority of cases among children are sporadic and point source outbreaks are rare.

The Northern Territory of Australia has a population of 230,000 living in an area of 1.35 million square kilometers, with a climate that varies from desert and semi-arid in the south to sub-tropical in the north. Darwin (population 120,000), the major urban centre, lies on the north coast. About 30% of the population of the Northern Territory and 11% of Darwin are comprised of Indigenous Australians.[32]

Subsequent to an earlier study showing that *Salmonellae* are present in animal faeces in Darwin houses,[33] we designed a case–control study to explore risk factors for sporadic salmonellosis in this environment at the individual and household levels. Our hypothesis was that environmental contamination is a source of sporadic salmonellosis in children and that the risk of salmonellosis is influenced by childhood behaviour, household cleaning behaviours and direct contact with animals, contaminated food, water, fomites or dust.

## Materials and methods

A community based case–control study was conducted from June 2006 to June 2008 among children aged 0–4 years residing in the Darwin and Palmerston municipalities. The study was conducted following the STROBE protocol[34] and ethics approval was obtained from the Menzies School of Health Research Human Research Ethics Committee.

Notification of cases was by laboratories according to the NT Notifiable Diseases Act using the Australian national case definition: isolation or detection of *Salmonella* species in any specimen.[35] The parents of cases were contacted by telephone and verbal informed consent was obtained to partake in the study. Cases were excluded if: (i) an adult household member had diarrhoea in the preceding 7 days or was diagnosed with salmonellosis in the preceding 30 days; (ii) a child household member had diarrhoea or was diagnosed with salmonellosis in the preceding 30 days; (iii) the case had another enteric pathogen isolated during the same illness; (iv) the case had the same *Salmonella* serovar isolated within the preceding 6 months; (v) the case was part of an outbreak investigation; (vi) the case had been in hospital for any of 7 days prior to illness onset; (vii) the interview could not be conducted within 30 days of illness onset; (viii) the household could not be contacted within 6 attempts to call.

Controls were selected randomly from a list of all children born after May 01, 2002 who were on the Department of Health and Families health information system and who lived in the study area. Control children were frequency matched on the basis of being less than 5 years of age. Controls were excluded if: (i) they could not be reached within 6 attempts to call; (ii) the parent reported a child household member had diarrhoea in the prior 30 days. If they reported that an adult household member had diarrhoea in the prior 30 days they were included in the study but excluded from household-level analysis.

A questionnaire was administered to the parents of all participants by one of five trained staff members either by phone or in the household. Interviewers were not blinded to the case or control status of the participant. The questionnaire included exposure variables relating to the 7 day period before onset for cases or 4 week period before interview for controls, except for behavioural traits when 7 days before interview was used. The variables included behaviour, health, food, family and housing characteristics.

Environmental sampling was carried out for all cases and 1 in 3 controls. Sampling was done from the house where the case spent most time in the 3 days before symptom onset, and was guided by parental description of pets and the child's play areas and behaviours. Sampling included all visible animal faeces in the living, kitchen and play areas. No live animals were tested as part of this study. Sample jars were filled with up to 80 mls of animal faeces or environmental samples (such as vacuum dust, pond water, sandpit sand) and were stored in the dark at 4 °C until arrival in the laboratory. The culturing techniques used were consistent with WHO recommendations for isolating *Salmonella*.[36] Human and environmental *Salmonella* isolates were serotyped according to the Kauffmann–White scheme by the Public Health Unit, Microbiology & Infectious Diseases, SA Pathology, Adelaide, South Australia.

Analysis was performed using Intercooled Stata 8®. The characteristics of individual cases and controls were compared first, followed by a comparison of household characteristics. The two-sample Wilcoxon rank-sum test was used to compare continuous variables, while the Chi-square test was used for dichotomous variables (or Fisher's exact test where cell numbers were small). Odds Ratios