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Have changes to meat and poultry food safety regulation in Australia affected the prevalence of *Salmonella* or of salmonellosis?

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Abstract

During the 1990s, there was radical change in regulation of meat and poultry hygiene in Australia, and Australian Standards were developed for each sector of the meat industry. Systems for industry/government co-regulation and company-employed meat inspection were introduced based on company HACCP programs approved and audited by the Controlling Authority. However, in the 5 years since regulatory changes took full effect, rates of salmonellosis have not decreased (surveillance and reporting systems have remained unchanged). Using statistics gathered by the National Enteric Pathogens Surveillance Scheme, an attempt was made to link *Salmonella* serovars isolated from meat and poultry with those causing salmonellosis. Two periods were studied, 1993/1994, before regulations were introduced, and 2000/2001, when regulations should be having an effect. For red meat, the same serovars were prominent among the top 10 isolates both before and after regulation, and there was little linkage with salmonelloses. For poultry, frequently isolated serovars differed pre- and post-regulation, however, in both periods there was some linkage between serovars isolated from poultry and those causing salmonelloses. Using published and unpublished survey data, it was concluded that there had been improvements in microbiological quality of red meat and poultry over the same timeframe as regulatory changes. That these improvements apparently have not carried through to reduced case-rates for salmonellosis may be due to numerous causes, including lack of control in the food processing, food service and home sectors. The present paper illustrates difficulties faced by governments in measuring public health outcomes of changes to food hygiene regulation.

Keywords: Salmonellosis; Food hygiene regulation; Australia; Serovar matching

1. Introduction

In Australia, over the past decade, reported salmonelloses have averaged around 6000 annually, ranging between 4600 in 1992 to 7700 in 1998. After accounting for under reporting, food borne salmonelloses are estimated between 240,000/annum (Sumner et al., 2000a,b) and 650,000 (ANZFA, 1999). Traditionally, it has been thought that, because warm-blooded animals harbour *Salmonella* in their intestines, these organisms are transmitted during slaughter and dressing to the animal product and thence to consumers.

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Thus, meat and poultry have long been considered the primary entry point for *Salmonella* into human populations, a proposition supported by pandemics such as *S. agona* and *S. hadar* during the 1970s and 1980s involving poultry. More recently, eggs have become an additional primary source of salmonellosis via *S. enteritidis*, particularly Phage Type 4. In each of these pandemics there were clear microbiological and epidemiological linkages between food types and serovars.

During the past decade, there has been macrochange in regulation of meat and poultry hygiene in Australia, most notably the introduction of industry/government co-regulation and company-employed meat inspection. Australian Standards have been developed for each sector of the meat industry and all meat processors operate a HACCP program approved and audited by the Controlling Authority. In the agri-meat/poultry–food–retail–service–home continuum, hygiene regulation embraces slaughter/dressing, further processing and transport, plus wholesale and retail distribution. Regulation is underpinned by outcomes-based Australian Standards detailing minimum requirements and mandating HACCP.

The basis for regulation is to maintain public health standards, or in this case, to provide for the wholesomeness of meat and poultry meat and to reduce the incidence of food-borne illness. Government continues to invest heavily in meat hygiene regulation and, in doing so, places significant compliance costs on industry. A critical question is: have changes to regulation of meat and poultry hygiene in Australia reduced the prevalence of disease from consumption of these products?

In this paper, we attempt to answer this question using all published data in Australia surrounding salmonellosis and prevalence of *Salmonella* in meat and poultry. We have followed two approaches. Firstly, we have collated annual case rates of notified salmonelloses and secondly, we have attempted to match serovars isolated from meat and poultry before and after, regulatory changes were made (1993/1994) and (2000/2001), respectively.

2. Data sources and analysis

In Australia, salmonellosis is a notifiable disease and cases are investigated to establish the serovar and, where applicable, the phage type. Case rates for these pathogens are recorded in the National Notifiable Diseases Surveillance System (NNDSS). When serovars of *Salmonella* are isolated from humans and from foods they are recorded in the National Enteric Pathogens Surveillance Scheme (NEPSS) (National Enteric Pathogens Surveillance Scheme, 2000a,b). Two industry monitoring programs are important sources for the NSPSS. In the case of red meat, serovars are isolated from carcases produced at export establishments as part of the *Escherichia coli* and *Salmonella* Monitoring (ESAM) program overseen by the Australian Quarantine and Inspection Service (AQIS) according to AQIS Notice 2003/06 (Australian Quarantine and Inspection Service, 2003). For poultry, serovars from a monitoring program maintained by the Australian Poultry Industry Association (APIA) are a major contributor to the NEPSS.

Data from these sources were analysed for the years 1993 and 1994 (as exemplifying the pre-regulatory change period) and 2000 and 2001 (for the post-regulatory change period). As well, published and unpublished reports on prevalence of *Salmonella* in red meat and poultry were obtained to supplement NEPSS and NNDSS data.

3. Results

3.1. Trends in salmonellosis

One broad measure of regulatory impact (i.e. public health outcomes) is to evaluate trends in disease prevalence caused by target pathogens and, in Table 1, are presented annual salmonelloses in Australia over the period 1991–2001. Despite regulatory changes, which took full effect by late-1997, salmonellosis generally trended higher over time, both in number of cases and rate/100,000 population. It is possible that this trend could reflect enhanced surveillance capability but we can find no significant changes either to laboratory or to reporting systems over the investigation period. Large outbreaks can inflate rates of foodborne salmonelloses. In 1997, there were several outbreaks linked with cooked meats (Lester et al., 1997) which accounted for around 1000 cases and, in 1999 there were around 500 cases from unpasteurised orange juice (Anonymous, 1999a). In

Table 1
Salmonellosis in Australia, 1991–2001 (after NNDDS data)

	Salmonellosis	
	Number of cases	Rate/100,000 population
1991	5440	31.9
1992	4614	26.2
1993	4731	27.5
1994	5327	31.2
1995	5895	34.0
1996	5819	33.2
1997	7005	38.2
1998	7700	41.8
1999	6834	38.3
2000	6121	31.9
2001	7147	35.8

other years, however, there were no large outbreaks of foodborne salmonellosis to raise the annual total significantly above the baseline of sporadic cases.

3.2. Serovar matching between meat, poultry and patients

Another approach to evaluating whether regulation has been effective is to investigate whether there is concurrence between serovars isolated from foods and those which cause salmonellosis. Accordingly, serovars isolated from raw meat and poultry were matched with those isolated from patients for 1993/1994 (pre-regulatory change) and for 2000/2001 (post-change).

During 1993/1994, there were 82 isolations from meat and meat products largely reflecting investigation of food poisoning incidents, compared with 374 isolations during 2000/2001, which also included serovars from the *E. coli* and *Salmonella* Monitoring (ESAM) program carried out on beef, sheep and goat carcases produced at export establishments since 1998. There was a wider range of serovars (81) isolated during 2000/2001, compared with 15 serovars isolated in 1993–1994. In Table 2, the most-frequently isolated serovars from meat are presented. All 10 most-frequently isolated serovars in 1993–1994 were also isolated in 2000–2001. The relative frequency with which these serovars caused salmonellosis also was similar between 1993/1994 and 2000–2001. Based on the foregoing it may be concluded that there was little difference in the suite of serovars isolated from meat before and after regulatory changes.

For poultry, there were 500 isolations in 1993 and 1994 compared with 1153 in 2000 and 2001. In Table 3, the most-frequently isolated serovars from poultry are presented, most obvious of which is *S. sofia*, which accounted for 80% and 36% of isolations in the two periods studied. This serovar apart, there was a qualitative difference between the most-frequently isolated serovars with, in 2000–2001, *S. virchow*, *S. infantis*, *S. mbandaka* and *S. kiambu* displacing *S.*

Table 2
Serovars isolated from patients and from meat in Australia during 1993/1994 and 2000/2001

1993 and 1994			2000 and 2001		
Serovar	Isolation from meat	Human isolations	Serovar	Isolation from meat	Human isolations
	Number (%)	Frequency ranking[a]		Number (%)	Frequency ranking[a]
S. havana	14 (17.1)	17	*S. anatum*	43 (11.5)	18
S. anatum	9 (11.0)	14	*S. infantis*	31 (8.3)	11
S. adelaide	9 (11.0)	20+	*S. chester*	31 (8.3)	8
S. bovismorbificans	7 (8.5)	9	*S. derby*	30 (8.0)	20+
S. chester	5 (6.1)	7	*S. typhimurium*	22 (5.9)	1
S. typhimurium	5 (6.1)	1	*S. adelaide*	21 (5.6)	20+
S. derby	4 (4.9)	20+	*S. bovismorbificans*	17 (4.5)	9
S. infantis	3 (3.7)	12	*S. agona*	17 (4.5)	20
S. agona	2 (2.4)	17	*S. heidelberg*	14 (3.7)	20+
Other	24 (29.3)		*S. havana*	12 (3.2)	20+
	82 (100)		Other	136 (36.4)	
				374 (100)	

[a] Ranking indicates frequency with which serovar is isolated from patients; "1" is most frequent cause of salmonellosis.

Table 3
Serovars isolated from patients and from poultry meat in Australia during 1993/1994 and 2000/2001

1993 and 1994			2000 and 2001		
Serovar	Isolation from poultry meat	Human isolations	Serovar	Isolation from poultry meat	Human isolations
	Number (%)	Frequency ranking[a]		Number (%)	Frequency ranking[a]
S. sofia	403 (80.6)	20+	*S. sofia*	422 (36.6)	20+
S. hadar	18 (3.6)	10	*S. virchow*	130 (11.3)	3
S. typhimurium PT 135	12 (2.4)	1	*S. infantis*	126 (10.9)	11
S. typhimurium PT 179	12 (2.4)	20+	*S. mbandaka*	55 (4.8)	20+
S. singapore	11 (2.2)	19	*S. kiambu*	49 (4.2)	20+
S. typhimurium PT 9	9 (1.8)	2	*S. typhimurium* PT 126	44 (3.8)	6
S. anatum	8 (1.6)	15	*S. bovismorbificans*	40 (3.5)	9
S. typhimurium PT 64	7 (1.4)	20+	*S. typhimurium* PT 135	39 (3.4)	1
S. typhimurium PT 44	5 (1.0)	5	*S. typhimurium* PT 108	37 (3.2)	20+
S. agona	3 (0.6)	17	*S. agona*	30 (2.6)	20+
Others	12 (2.4)		Others	181 (15.7)	
	500 (100)			1153 (100)	

[a] Footnote as for Table 2.

hadar, *S. singapore* and *S. anatum*, which were among the 10 most-frequently isolated serovars during 1993/94. In both periods studied, *S. typhimurium* was frequently isolated from poultry, and the phage types were also frequently associated with salmonellosis. Although there were differences in serovars most-frequently isolated from poultry before and after introduction of regulatory change, on both occasions those serovars were also involved in salmonellosis. This, however, may merely reflect the fact that sampling is based on investigating food poisonings, rather than on statistically based surveys.

4. Discussion

The approach of measuring case rates for salmonellosis has been used by USA authorities to link the effectiveness of their Pathogen Reduction Program (PRP) in meat and poultry with reduced numbers of reported foodborne disease (FSIS, 1996). This approach merits circumspection because other contemporaneous changes (livestock husbandry, process control, ability to detect pathogens, consumer education and behaviour) may also have had their effect, e.g. enhanced control of *S. enteritidis* in egg and poultry products. As Tauxe (2002) states in commenting on a fall in numbers of salmonelloses following introduction of mandatory HACCP in the USA meat and poultry industries, "Year to year variation may be substantial, but declines in the incidence of infections caused by several foodborne zoonotic pathogens may be early returns on... HACCP in the meat industry, better egg safety, and food safety eduction."

Serotype matching has been used in two USA studies. Schlosser et al. (2000), as part of the implementation of the USA Pathogen Reduction Program, documented the prevalence of *Salmonella* serovars in carcase and ground beef, pork and poultry. For each product there was some commonality between serovars on carcase surfaces and in ground products. However, there was little commonality between serovars in raw meats (carcase/ground meats) and those from patients. For example, the most common serovar from chicken, *S. heidelberg*, was the third most common human isolate; *S. montevideo* (most common from beef) was 7th and *S. derby* (pork) was 27th most common human isolate. The authors speculate that this discordance between serovar prevalence in food and in humans may reflect differences in pathogenicity and/or concentration.

Sarwari et al. (2001) compared serovars isolated from meat and poultry over the period 1990–1996 with serovars involved in salmonellosis in USA. Like Schlosser et al. (2000) these authors found little concurrence when attempting to match serovar prevalence in meats with human cases. For example, *S. kentucky* was found in beef, pork and chicken, some-

times at high prevalence, and yet caused only 0.1% of cases compared with an "expected" involvement of 14%. Sarwari et al. (2001) modelled the "ability to cause human illness" for each serovar. Assigning an arbitrary ability to cause human illness of 1 to *S. typhimurium*, the researchers found that the observed involvement with human illness could be accounted for only if other serovars were less likely to cause illness. For example, *S. heidelberg* was four times less likely and *S. kentucky* 200 times less likely to cause illness than *S. typhimurium*.

The present study has parallels with the findings of Schlosser et al. (2000) and Sarwari et al. (2001). Firstly, there was no great concurrence between the suite of serovars isolated most frequently from red meat and those most commonly associated with salmonellosis in Australia. Secondly, *S. sofia* was isolated from 50% of poultry samples and yet caused only 0.3% of salmonelloses, pointing to a very low ability to cause human illness.

In the case of poultry meat, however, phage types of *S. typhimurium* isolated from poultry were responsible for a significant proportion of salmonelloses, a situation which pertained both before and after regulatory change.

Prima facie data do not suggest a positive public health outcome associated with the inception of new meat and poultry hygiene standards and regulations. This could be due to regulatory changes not being effective, either because they have not been implemented, or/and because their implementation has not been effective. However, there is evidence that the microbiological status of both red meat and poultry have improved over the past decade.

For beef carcases, historical data are available from national baseline surveys in 1994 and 1998 (Vanderlinde et al., 1998, 1999; Phillips et al., 2001a,b), respectively, and the *E. coli* and *Salmonella* Monitoring (ESAM) database. Combining data from these sources, there have been improvements in TVC and prevalence of *E. coli* and *Salmonella* over the period 1994–2002 (Table 4). For beef and sheep carcases, mean TVCs have fallen by 2 log scales, while prevalence of *E. coli* has also fallen markedly. Prevalence of *Salmonella* on sheep carcases is also much lower in 2002 compared with 1994, though on beef carcases there has been little change from the low (0.34%) prevalence in 1994.

It should be explained firstly that 1994 data were based on excision sampling while other data were based on sponge sampling and secondly, that 1994 and 1998 data are based on samples from both domestic and export abattoirs while 2000–2002 data are solely for export abattoirs. However, around half of all meat slaughtered in the export sector is consumed domestically and there are several reports which indicate little difference in microbiological quality of product from domestic and export abattoirs (Anonymous, 1999b, 2000; Phillips et al., 2001a,b; Sumner et al., 2003).

For poultry, historical data collated by the Australian Poultry Industry Association (APIA), were obtained for prevalence of *Salmonella* at the processing plant level (Dr. Jeff Fairbrother, pers. comm.). It

Table 4
Microbiological profile of beef and sheep carcases in Australia, 1994–2002

	National baseline surveys		ESAM data		
	1994 (Vanderlinde et al., 1998, 1999)	1998 (Phillips et al., 2001a,b)	2000	2001	2002
Beef carcases					
TVC (mean log cfu/cm^2)	3.02	2.43	0.89	0.90	0.91
E. coli[a]	168/881 (19.1)	131/1275 (10.3)	1199/21492 (5.6)	876/21294 (4.1)	1065/21791 (4.9)
Salmonella[a]	3/882 (0.34)	3/1275 (0.24)	12/4338 (0.28)	12/4583 (0.26)	12/4687 (0.26)
Sheep carcases					
TVC (mean log cfu/cm^2)	3.92	3.55	1.79	1.79	1.68
E. coli[a]	352/470 (75)	269/921 (29.2)	5295/19552 (27.0)	3860/19315 (19.9)	4562/18480 (24.7)
Salmonella[a]	27/470 (5.7)	1/921 (0.1)	33/3910 (0.84)	30/3863 (0.78)	11/3695 (0.30)

[a] Number positive/tested (%).

should be noted that the poultry sector began the process of installing HACCP systems in the mid-1980s, where it is more instructive to begin the time-frame comparison for this sector. Since 1981, when testing by whole-bird rinse at the processing plant began, around 150,000 samples have been analysed for a prevalence of *Salmonella* around 30% and annual incidence varying between 25% and 35% (APIA data). Since 1981, detection methods for *Salmonella* have improved markedly and the fact that APIA detections in 2000 and 2001 were 28% and 29%, respectively, points to process improvement over the past two decades.

However, the most significant aspect of *Salmonella* detection from poultry can be seen in Table 5, with the dominant serovar becoming *S. sofia*, and *S. typhimurium* being reduced to less than 5% of serovars. The APIA data are supported by a survey of poultry at processing plants in South Australia (Sumner et al., in press) in which *S. sofia* accounted for 131/146 (90%) of isolations; the other serovars were *S. infantis* (eight isolations), *S. anatum* and *S. zanzibar* (two each), and *S. mbandaka*, *S. chester* and *S. typhimurium* PT8 (one each).

Interestingly, in Table 3, the prevalence of *S. sofia* in 2000/2001 appears to have fallen dramatically to 36.6%. It is understood that laboratories at processing plants are now capable of identifying this serovar, which is no longer sent for serotyping and therefore will not appear in the NEPSS data. This is a further complication for anyone wishing to review serovars from poultry and from salmonellosis.

Table 5
Distribution of *Salmonella* serovars from chicken meat in Australia (after Australian Poultry Industry Association)

Serovar	1981–1985	1986–1990	1991–1994
S. typhimurium	17.7[a]	18.2	4.1
S. sofia	32.8	56.8	82.8
S. infantis	1.3	5.1	2.3
S. anatum	1.8	5.8	0.7
S. agona	4	1.7	0.8
S. singapore	5.8	2.4	0.2
4,12:d	7.5	0.8	0.2
S. virchow	1.2	1.4	1.8
S. bovismorbificans	2.4	0.4	0.2
Subtotal	83.5	92.6	93

[a] Percentage of isolations.

There is evidence, then, of improvement in the microbiological status of red meat and poultry at the processing level over the same period that regulatory changes were imposed in each sector. That this improvement has not led to any apparent reduction in case-rates for salmonellosis may be due to numerous causes.

Firstly, loss of control in food processing plants can amplify prevalence and concentration of *Salmonella*. Thus, a specific *Salmonella* may be present in only a few individual animals or rare in a raw produce overall, but if it colonises a manufacturing line, then the proportion of cases caused by that specific *Salmonella* may increase. In Australia, such amplification has been shown to occur, including contamination from a mixing vat in ice cream production (Anonymous, 1998), an orange waxing bath (Anonymous, 1999a). As well, processes in the poultry industry like immersion chilling of chickens can intermittently cause significant outbreaks of salmonellosis such as the *S. typhimurium* PT126 outbreaks of 2001 (Anonymous, 2001). These examples follow investigations of food poisoning incidents where the aetiology was ascertained.

Secondly, while food safety plans dictate processing regimes in the processing sector, at the food service level, the uptake of effective food safety plans is far from complete. This is especially so in the small restaurant and take-away businesses that prepare a significant proportion of the meals eaten away from home each year in Australia. Thirdly, handling of foods in the home provides an unknown quantum of salmonellosis. Fourthly, non-food sources such as contaminated water or contact with pets and farm animals also have an unknown impact on salmonellosis.

From a Government (regulatory) viewpoint, and also for industries, there is a clear imperative to be able to assess the impact of regulatory change on food-borne illness in terms of a public health burden. At the same time, current Government policy dictates that the level of regulation be appropriate to the risk. But if we cannot quantify the level of risk (i.e. the cause of illness), how can we demonstrate to industry, markets and the public that we have appropriate regulation. At present in Australia, there are data which point to regulatory change having been effective at the processing level but ineffective in improv-

ing the overall health of consumers. The vast majority of reported salmonelloses are sporadic cases for which the aetiology is not followed. It is hoped that the introduction in 2001 of a food network, OzFoodNet, will provide an effective tool in investigating foodborne illness in Australia and, by extension, to allow assessment of regulatory change.

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