

Enteric Yersiniosis on the north coast of NSW – an epidemiological review

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Epidemiological Features – review of literature

Yersinia is a genus of gram negative bacteria in the family Enterobacteriaceae. Both *Yersinia pseudotuberculosis* and *Yersinia enterocolitica* can be associated with enteric disease (Carter 1995). *Yersinia pseudotuberculosis* is considered the main agent for outbreaks of enteric Yersiniosis in domestic cattle of New South Wales (Thompson 2003). The epidemiology of enteric yersiniosis in Australian bovidae is complex (Hum, Slattery & Love 1997). Causal factors remain unsubstantiated. Observations on disease outbreaks on the North coast of NSW suggest a complex interaction between the agent, host and environment.

Outbreaks often occur under stressful environmental circumstances (Hum, Slattery & Love 1997). Cold weather, flooding, marshy lowlands, frosts, transport and poorly nourished animals may be typical stressors. Outbreaks are widely reported to follow winter flooding. This has given rise to the common name of “flood mud scours”. Callinan et al (1988) described key features of the “flood mud scour” syndrome during outbreaks in 1983 to 1985. During these years the syndrome was observed during winter and early spring. Typically, affected cattle “had access to low lying poorly drained pastures which were waterlogged either by recent flooding or persistent heavy rain. In many cases, fine silt remained on the pasture after flood waters had receded” (Callinan et al 1988).

In the observed syndrome all cattle were older than 6 months of age. If found alive animals had all or some of recumbency, lethargy, inappetance, dehydration, pyrexia and diffuse diarrhea. Faeces were found to be watery, smelly and sometimes tinged with blood. Sometimes animals were simply found dead. On biochemical analysis the majority of animals were hypoproteinaemic and at necropsy macroscopic features of enterocolitis were observed. On histopathology the tips of small intestinal villi were replaced with gram negative bacteria and neutrophils. *Yersinia pseudotuberculosis* was cultured as the dominant organism from 85% of intestinal samples. When administered early in the course of the disease animals responded well to tetracycline antibiotics. During the outbreaks the average morbidity rate was estimated at 8% and the case fatality rate at 58% (Callinan et al 1988).

Callinan et al (1988) used culture and histopathological information to define a pathogenic association between the agent *Yersinia pseudotuberculosis* and the disease syndrome being observed. They described the colonisation of distinct macroscopic lesions with bacteria consistent with *Yersinia* and the culture of *Yersinia pseudotuberculosis* from these intestinal lesions. They

concluded that *Yersinia* is important in the pathogenesis of the syndrome but that its role as a primary pathogen still required further confirmation.

There is limited information in later literature that further explores the causal relationship between *Yersinia* and the NSW “flood mud scour syndrome”. Slee et al (1988) discuss a similar syndrome in Gippsland between 1985-1988 where *Yersinia* was cultured from 222 scouring cattle. They concluded from clinical, haematology, serology, bacteriology and histopathology data that cattle are a common host for *Yersinia pseudotuberculosis* and that clinical and fatal disease occur occasionally from the bacterium. They comment that the factors leading to clinical disease are unknown. Earlier Hodges and Carmen (1985) had emphasised the fact that *Yersinia pseudotuberculosis* can be isolated from clinically normal cattle.

Papers from a NSW veterinary conference in 2003 indicate that NSW government pathologists accepted specific “intestinal microscopic lesions as being virtually pathognomonic” for Yersiniosis and that history, clinical signs and culture of the organism provides “additional support for the diagnosis” (Thompson 2003).

Epidemiological features – laboratory data

The author analysed laboratory reports dated between January 1st 1999 and 24th August 2009 in the Lismore office of the North Coast Livestock Health and Pest Authority. These reports primarily related to cases of disease within the Tweed Lismore district.

During this period there were 25 records where Yersiniosis was listed as the final conclusion for enteric disease. This was on the basis of microbiology alone (19 cases) or both microbiology and histopathology (6 cases). It is important to recognise that 25 is likely to be only a very small proportion of the number of outbreaks that have occurred in the Tweed-Lismore district over the last ten years. Some outbreaks would not have had veterinary attendance. There has also been good awareness regarding Yersiniosis and many producers and private veterinarians treat for the condition without laboratory confirmation.

Of the 25 cases, 22 were selected because in these *Yersinia pseudotuberculosis* was cultured as a profuse population without the need for selective enrichment media. The three cases were discarded because the culture of *Yersinia* seemed at odds with the history, clinical signs or other pathology. In the 22 cases the history, clinical signs and histopathology were very similar to the syndrome described by Callinan et al (1988).

Exceptions to the typical syndrome of acute disease in adults included one case of disease in a younger calf and one case where clinical disease extended over 2 weeks. In most histories there is mention of either low condition score of the animals or poor available pasture.

The case fatality rate of 48.3% is similar to that estimated by Callinan et al (1988) of 58%.

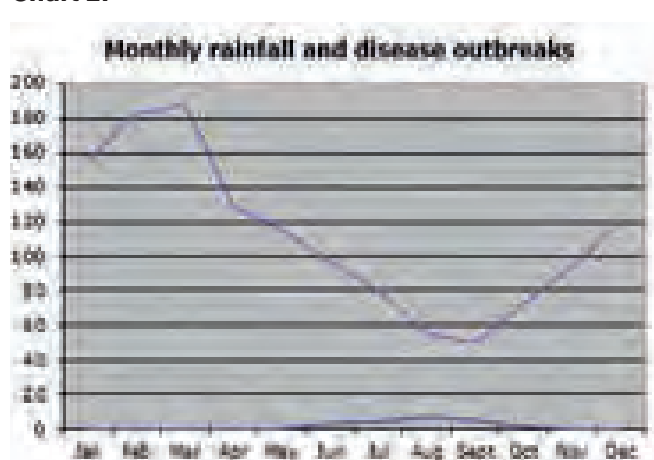
Analysis of the laboratory data supports the strong seasonal nature typically described for *Yersinia* outbreaks. Over the ten year period cases were only reported between June and October (Chart 1). August 1999 had 5 outbreaks alone which has pushed the seasonal trend towards the right in Chart 1. If cases from 1999 are excluded from the data the peak in the seasonal trend occurs more in July than in August. The seasonal nature of the syndrome strongly suggests that environmental influences are important.

Chart 1:



There is an association between rainfall and the disease outbreaks (Chart 2). The greatest number of cases occurred when mean monthly rainfall is low.

Chart 2:



Heavy rainfall, floods and general pasture inundation with water can occur in the Lismore region in late autumn and early winter. The ideal environmental conditions for a *Yersinia* outbreak may occur some time after heavy rainfall and flooding. For example, in 2009, 4 disease outbreaks occurred in July and August when an extreme flooding

event occurred in late May. A causal association between flooding and disease is suggested by the flooding preceding the disease event and by the biological plausibility of *Yersinia* being available at a high infective dose following its multiplication in anaerobic and moist conditions. But is this association being seen consistently?

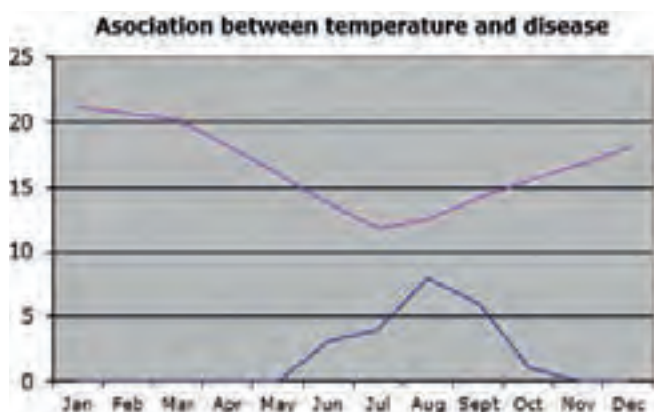
Lismore City Council summarises significant flood events on its webpage (Lismore City Council 2008). There is not a consistent association with these flood events and timing of disease outbreaks (Table 1). Between 1999 and 2009 six years had confirmed cases of Yersiniosis. In only three of these years was there a significant flood event. In 2005 a large flood occurred in June but there were no confirmed cases of disease that season.

Table 1: Lismore floods and disease outbreaks by year

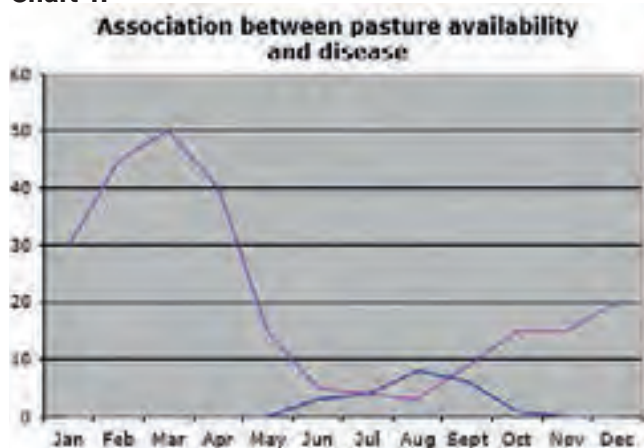
Year	Number of outbreaks	Significant flood event	Month of flood
1999	8	No	
2000	2	No	
2001	0	Yes	Feb
2002	0	No	
2003	3	No	
2004	0	No	
2005	0	Yes	June
2006	1	Yes	Jan
2007	0	No	
2008	4	Yes	Jan
2009	4 (up to 24 August)	Yes	May

Recognition of the local variance in weather conditions is also important. Some areas of the Tweed-Lismore region will receive much higher rainfall than average while others will receive less. Localized flooding can also occur. Collecting rainfall data any history of localised flooding for the paddock locations of each outbreak would be useful. Particular paddocks where disease occurred may have experienced wet, swampy or recent heavy floods when the wider region did not. For example, one outbreak in 2009 was very localized and exposure was assumed to be from an area where the 'bowl' shape of the land retained water and left a heavy layer of silt. Cattle had chosen to preferentially graze in this area. Similar data could be obtained from other outbreaks by retrospective studies.

A number of authors have described an association between outbreaks of Yersiniosis and low temperature (Radiostits 1997). This association is supported from analysis of the lab data and mean monthly minimum temperatures in Lismore (Chart 3). Because there is moisture throughout most of the year temperature is probably the limiting climatic factor for Yersiniosis outbreaks. If the temperature is too high *Yersinia* cannot survive in the environment. (Chart 3)

Chart 3:

Nutritional status of cattle also follows a seasonal pattern on the North Coast. Nutrition is largely derived from pasture which has a seasonal decline in winter. There is an association between the seasonal decline of pasture and the seasonal peak in *Yersinia* cases (Chart 4). Pasture availability is at its lowest when cases are most common. Radostits (1997) discussed that good nutrition may have a role in preventing Yersiniosis. This may also explain why cases do not occur in summer.

Chart 4:

Trace element deficiencies and internal parasitism have also been suggested as being factors associated with Yersiniosis (McLennan and Kerr 2000). McLennan and Kerr specifically discuss copper and selenium deficiencies. The Tweed-Lismore district has well recognised copper and selenium deficiencies and internal parasites are the most common syndrome diagnosed. These associations may simply be confounding factors. It may be that any host stress may predispose to clinical expression of yersiniosis.

Summary of likely epidemiological factors

Yersiniosis on the North Coast of NSW is a multifactorial disease. *Yersinia pseudotuberculosis* is a "necessary" cause of Yersiniosis on the North Coast of NSW. You cannot get disease if it is not present. However it will not cause disease on its own. *Yersinia pseudotuberculosis* combines with a number of host and environmental factors to produce "sufficient" cause for disease. It is likely that the most important sufficient cause is composed of *Yersinia pseudotuberculosis*, host stress and ambient temperatures beneath a certain threshold.

Animals become exposed to *Yersinia* by oral ingestion of any *Yersinia* that have survived and multiplied in water and feed. This survival and multiplication is probably assisted if the paddocks have been covered in water and if environmental temperatures are low. Exposure would happen to a range of species and in all age groups. Unweaned calves may have a lower rate of exposure simply because they are not grazing. This may be one factor that explains why disease is more common in cattle over 6 months of age. It is possible that a low level of exposure in unweaned animals may lead to a protective immunity.

Following exposure *Yersinia* becomes a common inhabitant of the intestine in both domesticated and wild animals. With 99,000 cattle and a diversity of native and pest animals there is likely to be a very large animal reservoir on the North Coast ensuring survival of the bacterium and continued environmental contamination.

Wild animal reservoirs and cattle "carriers" excrete *Yersinia* organisms in their faeces onto pasture and into waterways leading to environmental contamination. Radostitis et al (1997) report this as the "major method of transmission". The seasonal nature of the disease on the North Coast of NSW suggests that this passive pasture contamination will not lead to an outbreak unless it is combined with environmental factors favouring survival and multiplication of the agent in soil, pasture and/or water.

It is assumed that a massive environmental reservoir of *Yersinia* builds up when the survival and multiplication of the organism is favoured by wet and low oxygen conditions. *Yersinia* is facultatively anaerobic and has an ability to survive and multiply in water at low temperatures. The wet and anaerobic conditions in localised North Coast paddocks after flooding would favour the growth of *Yersinia* compared to most other bacteria. Such growth can continue unchecked if ambient temperatures are not too high.

Yersinia multiplies to a level on pasture that infection and clinical disease is more likely. Radostitis et al (1997) describes this as "heavy infection pressure". Once a small number of cattle have succumbed to this heavy infection pressure they will further increase environmental contamination by excretion of bacteria in their faeces.

The history from Case 22 suggests that cattle became exposed to the organism at a point source in time. Out of the 8 affected animals 4 died within 48 hours of each other and the other 4 had clinical signs within the next few days. Transmission from one animal to another animal is probably not the major factor compared to all affected animals having ingested a large infective dose from pasture.

Following oral exposure to *Yersinia* cattle can have a bacteraemia and then a full spectrum of gastrointestinal syndromes:

1. septicemia and rapid death
2. acute severe hemorrhagic diarrhoea, dehydration and death
3. chronic subtle signs of ill thrift and diarrhoea and
4. infection without clinical signs but shedding organism in faeces (Hum, Slattery & Love 1997; Callinan et al 1988).

The range in severity of clinical signs is likely to be related to a number of host factors. Not all cattle succumb to the “heavy infection pressure” described by Radostitis et al (1997). They suggest that this is because infection mainly occurs in those “debilitated from other influences”. Herds will be more likely to have an outbreak if their immune status is compromised by stressors such as lactation, pregnancy, weather conditions, energy and/or protein imbalance, micronutrient deficiency, transport, change of feed or concurrent disease. Persistent pestivirus infection and uncontrolled parasites are examples of concurrent diseases on the North Coast that could predispose to both *Yersinia* infection and clinical disease. Path diagram of causality for Yersiniosis on North Coast of NSW



Disease frequency and other measurements

Frequency calculations are important for diseases within a district as they can assist management decisions for field veterinary services. For example, is a specific program on Yersiniosis justified for the North Coast.

Some measures of frequency for *Yersinia* outbreaks have already been identified in Table 1. For each outbreak the proportion of the population at risk that became affected with disease (attack rate) was calculated. The average attack rate was found to be 12.8%. The proportion of animals affected with the disease that die (case fatality rate) was also calculated for each outbreak and on average found to be 48%. Outbreaks of Yersiniosis are significant for individual producers.

Caution is needed in the interpretation of the frequency calculations from laboratory reports. The mortality and morbidity data on the laboratory reports is only a reflection of the situation at the time of sampling. True mortality and morbidity figures are probably significantly higher as outbreaks would continue for a period of time after sampling.

In addition mild clinical cases would not be detected on many properties where stock are not frequently or closely examined. The actual morbidity is likely to be higher compared with the mortality. Estimated case fatality rates for this disease are probably too high. The accuracy of mortality and morbidity data on the laboratory reports could be checked by retrospective interviews with affected producers.

While *Yersinia* outbreaks are a significant problem for affected producers how much are they a problem for the district as a whole?

The total population at risk from the laboratory data was 1592 and the total mortalities was 61. The risk of developing disease (Incidence risk) can be measured by calculating the proportion of non-diseased animals at the start of a period that became diseased during the period (Dohoo et al 2003). There was a 4% chance of an individual cow dying from Yersiniosis during the ten-year period.

29 (47.5%) of the mortalities occurred within a single year (1999). There was a 3% chance of an individual animal dying in this year which can be extrapolated to 30% for 10 years. It is difficult to determine whether the higher number of cases in 1999 represents a true increase in the incidence risk of the disease. Around this time there were changes in policy relating to the charging of laboratory services by the NSW Government. This may have lowered the rate of submissions by veterinarians. It may be that the number of laboratory reports for 1999 is closer to the actual number of outbreaks when compared to subsequent years. A 3% chance of developing disease is relatively significant. If the number of cases was truly lower in 2000-2009 it may be because control measures were more effective. Effective awareness campaigns may have led to earlier detection and treatment of suspicious cattle by producers and private veterinarians. This could have reduced the number of mortalities and request for laboratory confirmation.

Overall any of the calculated figures on incidence risk may not be particularly meaningful as many *Yersinia* mortalities will have occurred without attendance by a veterinarian and laboratory confirmation.

A proportional disease measure may be useful to assess the relative importance of *Yersinia*. Between 1 January and 27 August 2009 the author investigated 26 mortality events. One of those events was confirmed by laboratory testing to be *Yersinia*. Therefore over the 8-month period 3.85% of mortalities referred to the author for investigation were *Yersinia*. In comparison 19% of the mortalities were diagnosed by the author as black leg. If a specific objective was to reduce mortalities in the district then an awareness program for correct use of vaccines may be more effective than one for the early treatment of Yersiniosis.

Controlling Yersinia

Because *Yersinia* are facultative intracellular parasites and because the enteric infections they cause can be acute and fatal early and adequate treatment is important (Carter 1995).

Producers need to increase observation of their stock once an index case is identified. Any further clinical cases need to be detected early and treated immediately with oxytetracycline. Ideally clinical cases should be separated from the rest of the herd. For some herds preventative treatment of at risk cattle with antimicrobials may be appropriate.

Recognizing the likely source of infection is important and if possible cattle should be moved to a new area.

Preventing Yersinia

The suggested role that poor nutrition and other stress have in causing Yersiniosis indicate that prevention should be based on stress minimisation. Hum et al (1997) comment that control of stress factors is important for prevention of Yersiniosis.

Disease from Yersiniosis on the North Coast can probably be prevented by good husbandry that minimises nutritional and other stressors. Producers should set suitable stocking rates, manage pastures appropriately and supply supplementary feed in winter. When identified selenium and copper deficiencies should be addressed. A monitoring program for gastrointestinal parasites and strategic drenching is needed.

The high-risk time for the disease on the North Coast is in winter. Producers can identify the water holding and flood prone areas of their properties and if possible graze cattle on other paddocks in winter. Some properties may be able to fence off high risk areas.

Good farm biosecurity and management to control other endemic disease such as pestivirus and internal parasites may also be preventative. Controlling pests such as rodents would be sensible.

Awareness by government and private veterinarians is an important prevention strategy. The clustering of cases within a year suggests that following an index outbreak for the year an "early warning" could be provided to producers considered at risk.

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