

areas reduces but does not eliminate the risk for disease associated with consumption of raw or undercooked oysters (3).

Oysters from many parts of the world have been implicated in previous norovirus outbreaks (3–6), including similar norovirus outbreaks in New Zealand in 2004 and 2006 caused by consumption of frozen oysters from South Korea (6). In addition, norovirus was detected in 10% of imported oysters in Hong Kong (7), and adenoviruses or enteroviruses were identified in 80% of oyster samples from popular harvest areas in South Korea (8).

Although widely distributed commercial foods are rarely implicated as a source of norovirus infections, oysters (3) and raspberries (9) are notable exceptions. Without timely subtyping of virus specimens and a PulseNet-like data-sharing system, cluster linkage is unlikely. Norovirus infections are rarely confirmed by laboratory tests, and sporadic cases are rarely considered notifiable. The outbreak we described was recognized and reported because illnesses clustered in 1 workplace. However, even when outbreaks are reported, they are not always investigated thoroughly. The conventional wisdom that many, if not most, foodborne norovirus outbreaks are caused by contamination at the point of service (10) may discourage thorough epidemiologic investigations of these outbreaks.

Because thorough outbreak investigations are time-consuming and gastroenteritis outbreaks are common, resource issues often affect decisions about how intensively to pursue investigations. Our use of integrated questionnaire, data entry, and analysis templates (www.oregon.gov/DHS/ph/acd/keene.shtml) facilitated a quick and efficient response to the outbreak described here. Questionnaire design, interviews, data entry, and analysis were completed within 6 hours of the initial report, and distributors and regula-

tory agencies worked quickly to recall other oysters from the same source, thus probably preventing additional illnesses. We believe that widespread use of such templates would increase the number of outbreaks that could be investigated thoroughly.

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Epidemiologic Questions from Anthrax Outbreak, Hunter Valley, Australia

To the Editor: Anthrax was introduced into Australia in 1847 near Sydney, New South Wales, and spread along stock routes throughout New South Wales and southern Queensland (1). Anthrax was considered endemic to the Hunter Valley, New South Wales, during the 1890s. The last recorded anthrax-related stock losses there occurred on 3 properties in the Upper Hunter Valley in 1939 (1).

During the past 4 decades, anthrax has become uncommon in Australia. Clinical cases are seen only spo-

radically in sheep, cattle, and (rarely) horses. Annually, 6–12 properties are affected in unrelated incidents; where cattle are involved, generally only 1–3 animals per property are affected (2). Anthrax is confined almost exclusively to a belt running through the center of New South Wales (3).

From December 14, 2007, through January 3, 2008, a total of 53 cattle (*Bos taurus*) with peracute anthrax and 1 horse died on 11 properties in the Rouchel area, 20 km east of Aberdeen in the Hunter Valley and 350 km from the anthrax belt (Figure). The area is hilly, rising to ≈ 550 m, with alluvial soils alongside a stream and rocky basaltic and sandy soils on the slopes. Most properties feature gullies that flow intermittently after rain. The affected properties covered ≈ 60 km².

The animals that died in this area were of all ages. Anthrax was not suspected because of a long history of no local activity, but *Bacillus anthracis* was initially confirmed by microscopy of blood smears and subsequently by PCR of blood or other carcass fluid smear scrapings taken when animals were decomposed and

microscopy was unreliable (4). Dates of discovery of the index case on each property ranged from December 14 to December 29; 1–26 deaths (median 2) occurred per property. Property attack rates varied from 0.9% (1/110 cattle) to 10.7% (3/28 cattle). All stock on infected and 24 neighboring properties were vaccinated in late December; carcasses were burned to ash; movement control, including quarantine, was implemented; and all subsequent stock deaths in the area were investigated to rule out anthrax. One subsequent case occurred when an unvaccinated bull was introduced onto an infected property in late May 2008.

Detailed record review excluded importation of infected feed from known anthrax-endemic areas before the outbreak, and no deaths occurred in stock introduced from these areas during the previous month. Many of the animals died near streams, and waterborne spore dispersal with infection was initially hypothesized. However, the temporal pattern of properties affected, with downstream properties affected before upstream properties; the fact that properties without contiguous

streams were affected; and the dilution effect of rapidly flowing streams argued against this transmission route. Because they are septicemic, terminally ill animals with anthrax often seek water (5).

The mysterious contemporaneous reemergence of anthrax in this area is unlikely to be explained by mechanical vector-borne transmission because only 1 animal had eye damage, suggesting a crow attack. There was no additional evidence of scavenger attack. No tabanid species (biting) flies were seen on any carcass, and the small number of carcasses and relatively large distances between some properties made mechanical transmission with ocular inoculation by nonbiting flies unlikely (4).

Both the remarkable survival capability of anthrax spores and a 1-in-100-year rain event probably contributed to the near-simultaneous reemergence of anthrax on multiple properties in this area. Anthrax spores are resistant to biological extremes of heat, cold, pH, desiccation, chemicals, and irradiation, persisting in this state for decades awaiting conditions that favor germination and multiplication (6). In June 2007, drought-ravaged Hunter Valley experienced intense flooding; most rain fell in just 3 days (259 mm in the Aberdeen area, compared with the previous 3-year June average of 43 mm), and massive amounts of topsoil washed into gullies and streams. During late 2007, rainfall also was excessive: 132 mm and 129 mm in November and December, respectively, compared with the 3-year average of 87 mm and 65 mm.

The June floods are likely to have unearthed anthrax spores in the area. The question remains whether these spores had been present for >6 decades, concentrating in depressions that collected water and dead vegetation, potentially providing a milieu for germination and multiplication (i.e., incubator areas), a mechanism that has been implicated in wildlife epidemics

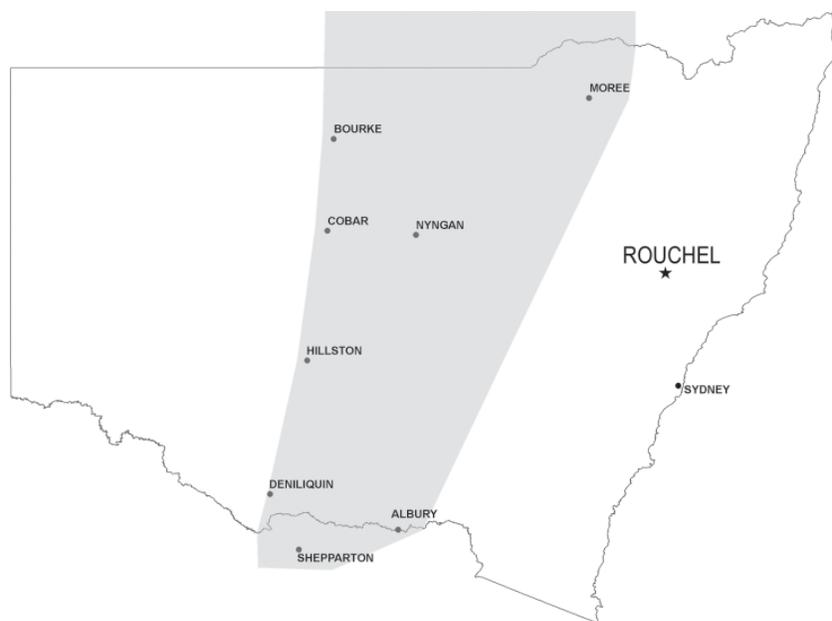


Figure. Map of New South Wales, Australia, showing the anthrax belt (gray shading) and the Rouchel location where anthrax reemerged during December 14, 2007–January 3, 2008.

of anthrax (7,8). Alternatively, low-grade sporadic infection may have been ongoing since the 1940s and infrequent stock mortality may not have been investigated for anthrax because of a low local index of suspicion, resulting in environmental contamination. The extreme weather conditions in the area may have unearthed spores from undiagnosed carcasses, providing simultaneous exposures on multiple properties.

We are currently unable to resolve this epidemiologic conundrum. However, our experience is a timely reminder that veterinary public health authorities should be on high alert for possible anthrax when unexpected livestock deaths follow flooding in areas where anthrax has historically occurred.

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Distinct Ecologically Relevant Strains of *Anaplasma phagocytophilum*

To the Editor: *Anaplasma phagocytophilum* was defined to include *Ehrlichia phagocytophila*, *E. equi*, and the agent of human granulocytic ehrlichiosis. Nevertheless, we and others have found phenotypic and genetic differences from diverse regions and hosts and conclude preliminarily that ecologically separate strains might exist that should be distinguished. Two precedents include ruminant strains of *A. phagocytophilum* in Europe and the Ap-Variant 1 from ruminants and ticks of North America and Europe.

In Europe, *A. phagocytophilum* infects livestock, rodents, and

humans, with some species such as European cattle showing severe disease and high antibody prevalence. In contrast, cattle infection is rare in the United States, despite being common in other species. Experimental infection of cattle with California equine-origin strain MRK failed to induce disease or marked rickettsemia (1). Thus, even though European strains have ruminant tropism, an equine strain does not.

Ap-Variant 1 is found in ticks and deer in North America. This strain is distinctive in the 16S rRNA, major surface protein 4 (*mSP4*), *mSP2*, and *ankA* genes (2). Deer, goats, and tick-derived cell lines can be infected with Ap-Variant 1, but rodents cannot (3). Our recent data examining *A. phagocytophilum* in western North America show at least 2 phenotypes: strains originating from sciurids (chipmunks and tree squirrels) and strains from woodrats (the previously postulated reservoir). In a survey of 2,121 small mammals in areas of California with enzootic Ap-Variant 1, seroprevalence was highest in tree squirrels (71%), woodrats (50%), and chipmunks (up to 28%), and PCR prevalence was highest in tree squirrels (16%) and chipmunks (34%) (4). We showed that chipmunks were competent reservoirs for *A. phagocytophilum* through exposure in the field, successful inoculation with strain MRK, and transmission through *Ixodes pacificus* to mice. However, discrepancy in the phenotype of strains originating from woodrats and chipmunks is substantial when these strains are inoculated into horses. One chipmunk strain can infect both rodents and horses (important laboratory animal models for human infection), whereas woodrat strains show restricted rodent-only tropism.

A naturally infected redwood chipmunk was trapped in Mendocino County, California, exsanguinated, and documented to be positive for *A. phagocytophilum* by using real-time