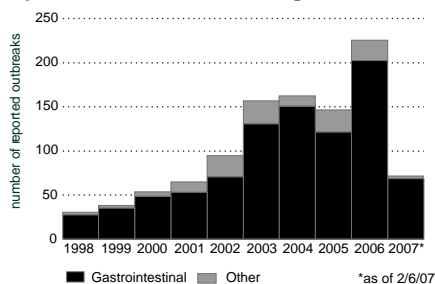


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AN OVERVIEW OF OUTBREAK INVESTIGATIONS, 2006

Two-hundred twenty-five outbreaks were logged in Oregon in 2006. To help you visualize that number, if every outbreak was 1000 miles long, and you stacked them end-to-end, they would reach to the moon. That's a lot for a state with a fairly modest population of ~3.7 million—only some of whom are public health workers. More disconcertingly, 225 represents an exponential increase from the 2005 total.* Already in 2007 we have been notified of an unprecedented 63 separate clusters. Are events spiraling out of control? In this issue of the *CD Summary*, we review highlights from our 2006 files.

Reported Outbreaks – Oregon, 1998–2007



OUTBREAK RECOGNITION

Outbreaks are usually identified in one of three ways. The majority are handed to us on a plate: someone contacts a health department to say, in effect, that “a bunch of people have gotten sick.” Others arise from routinely collected surveillance data—the case reports of salmonellosis, listeriosis, shigellosis, etc., that flow in daily from labs and physicians—analyzed against the “normal background.” Subtyping of bacterial isolates at the public health lab may also uncover outbreaks. Lastly, we learn about outbreaks from public

health colleagues outside Oregon.

Outbreaks of almost *any* kind of illness are reportable under Oregon law,[†] and over the past few years we have investigated clusters of influenza, hepatitis, skin infections and rashes, measles, mumps, and any number of mystery illnesses. That said, the vast majority of outbreaks that are reported and investigated in Oregon involve one or more flavors of gastroenteritis—in 2006, 202 (90%) of 225 outbreaks. To avoid conflating disparate phenomena, we will confine the following discussion to clusters of GI illness.

2006 HIGHLIGHTS

While the descriptor “food-borne” is often carelessly applied to almost all GI outbreaks, food-borne transmission was identified in only 38 (19%) of 202 GI clusters. Over half of these outbreaks (110; 54%) were predominantly “person-to-person,” without any obvious point source. The mode of transmission was indeterminate for 50 clusters (25%).

It is unclear from the historical record[‡] whether Disraeli was ever intimate with epidemiologists, but the elephant in the outbreak statistics room is “nursing home Norwalk.” Of all reported GI outbreaks, 116 (57%) occurred in nursing homes, assisted living centers, or similar facilities, and at least 95 (82%) of those were due to noroviruses (no specimens were submitted from another 9 clusters). These outbreaks rarely get the analytic scrutiny that goes into, say, foodborne outbreaks. Delayed reporting is a common problem, and often resi-

dents are difficult to interview. The emphasis in these investigations is curtailing further transmission, rather than identifying sources.

The granddaddy of the 2006 foodborne outbreak investigations was the spinach-associated outbreak of *Escherichia coli* O157:H7 infections, in which Oregon contributed 6 or 7 cases and lead the charge to identify the food item. Shigellosis was on the menu at a popular Yamhill County restaurant in January 2006. The investigation implicated food handlers with inadequate hygiene. At least 35 victims were identified, 7 hospitalized. Two small listeriosis outbreaks were noted, with 60% of 5 cases dying. One cluster was traced to consumption of a pasteurized artisanal sheep cheese; the other went unsolved.

We usually get one or two sprout-associated outbreaks, and 2006 was no exception. A small salmonellosis outbreak in February was traced to a Portland grower with cases following meals in 3 different restaurants. The grower was using a FDA-recommended protocol for seed disinfection and product testing, but once again we saw proof of the superior sensitivity of the human bioassay. An outbreak of *E. coli* O157:H7 infections was linked to meals at a Portland sushi restaurant. A specific vehicle could not be implicated, although the daikon radish sprouts seemed a better bet than the tuna.

Consumption of raw oysters is a good source of business for

*log_e = 0.4325

†ORS 431.110.

‡aka Wikipedia.



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epidemiologists: hot weather or cold, there's always something on the plate. At least 4 outbreaks were linked to raw oysters in 2006—more depending on how you count. In July what started as one restaurant outbreak investigation led to the identification of a widespread problem with oysters harvested in Washington and British Columbia, with cases turning up as far away as New York City.¹ Once the source was pinpointed (oysters from Washington and probably B.C. with higher-than-usual levels of naturally occurring *Vibrio parahaemolyticus*), later cases from other restaurants and from recreational harvest got less scrutiny. A November investigation following a Marion County event identified raw oysters as the source of a noroviral outbreak with an unusual twist. The oysters were from Korea—frozen on the half shell 7 months earlier. Tons of product from the same lot were recalled, preventing thousands of illnesses.

Zoonotic disease is a staple in Oregon. Nine people in Crook county developed cryptosporidiosis after contact with some sick calves; three of the cases were hospitalized. Every spring we see cases of salmonellosis associated with handling baby chicks. A hatchery near Walla Walla has

been linked to outbreaks of illness in 2006, 2005, 2004, 2003, 1996, and 1995; the 2007 season will start soon. Control of “the chick problem” is difficult. Some people like to buy and handle chicks with their inevitable contamination, and some don't wash their hands well enough.

Outbreak investigations represent opportunities for preventing illness, and they help us to keep tabs on where the pathogens are coming from. Keep those cards and letters coming.

REFERENCES

1. CDC. *Vibrio parahaemolyticus* infections associated with consumption of raw shellfish—three states, 2006. *MMWR* 2006; 55:854-6.

UPDATE: MUMPS TESTING

With lessons learned during the Great Midwest Mumps Outbreak of 2006, CDC recently updated its recommendations for mumps testing.

Both PCR testing and culture for the mumps virus have proven to be insensitive (i.e., these tests are usually negative even when we're pretty sure that the virus was there—such as in patients with parotitis in the middle of a mumps epidemic). Therefore, until further notice, we recommend serologic testing to try to confirm suspected mumps.

The first (acute) serum specimen should be collected within 5 days of illness onset and tested for IgM

antibodies. If this test is negative, a second (convalescent) serum specimen for IgM antibodies should be collected 2–3 weeks after onset, because the rise of IgM titers has been slow in patients with confirmed cases of mumps—especially in vaccinated persons. The paired sera can also be used to detect a significant rise in IgG antibody levels.¹

CDC is currently offering to test, at no charge, serum from patients with unexplained parotitis for anti-mumps IgM and IgG antibody, as long as you're willing to ship the specimen to them and fill out the requisite form (<http://www.cdc.gov/nip/diseases/mumps/lab-form-5034.pdf>). Serologic testing is available at commercial labs as well, and results may be available more quickly.

Negative laboratory tests, especially in vaccinated persons, are not sensitive enough to rule out mumps reliably.

So—remain alert for possible mumps, test suspected cases with serology, and report them to the local health department.

For more information, refer to www.oregon.gov/DHS/ph/acd/diseases/mumps/mumps.shtml.

REFERENCE

1. CDC. *MMWR* 2006; 55:1152–3. Brief report: Mumps activity—United States, January 1–October 7, 2006.