

Please read this bit first

The HPCSA and the Med Tech Society have confirmed that this clinical case study, plus your routine review of your EQA reports from Thistle QA, should be documented as a “Journal Club” activity. This means that you must record those attending for CEU purposes. Thistle will **not** issue a certificate to cover these activities, nor send out “correct” answers to the CEU questions at the end of this case study.

The Thistle QA CEU No is: **MT00025**.

Each attendee should claim **THREE** CEU points for completing this Quality Control Journal Club exercise, and retain a copy of the relevant Thistle QA Participation Certificate as proof of registration on a Thistle QA EQA.

Cycle 24 – Organism 10:

Shigella boydii

In the 1950's *Shigella* was accepted as a genus and subgrouped into four species: *S. dysenteriae*, *S. flexneri*, *S. boydii*, and *S. sonnei* (also referred to as subgroups A-D).

S. boydii inhabits the intestine and rectum of humans and other primates. It can survive in faeces and soil and/or food/water contaminated with faecal matter. For example, in Guadalajara, Mexico, *Salmonella* and *Shigella* species were found in freshly squeezed orange juice, oranges, and wiping cloths found in public markets and street booths. *S. boydii* was specifically found in the oranges and wiping cloths. This may indicate poor sanitary methods of food processing which led to raw sewage exposure

Shigella bacteria are thought to be derived from different strains of *Escherichia coli*. *S. boydii* is the most genetically divergent and some serotypes seem to be more closely related to other species. *S. boydii* type 13, for example, shares sequence similarities with *Vibrio cholerae* for the genes encoding the O antigen, the polysaccharide part of the lipopolysaccharide (LPS), and therefore these may be more closely related

Shigella's structural characteristics follow that of Gram-negative bacteria. Most research would agree that *Shigella* are non-motile but some evidence suggests that they do in fact have flagella, although motility is not necessary for infection of intestine. The flagella tend to be on one pole of the cell and about 10 microns in length and 12-14nm in diameter. The genes coding for the flagella in *S. dysenteriae*, *S. flexneri*, *S. boydii* and *S. sonnei* were found to be different and confirm the genetic diversity among the species.

S. boydii, when found in the intestine, go through anaerobic metabolic pathways but can survive outside of the body due to its ability to utilize aerobic pathways. More specifically, *S. boydii*, typically does not have oxidase enzymes but rather catalase enzymes (catalyzes the reduction of H₂O₂ to H₂O). Methyl red testing is positive, meaning that the bacteria uses a mixed acid fermentation pathway. Voges–Proskauer and Simmons' citrate reactions are negative, meaning that this organism does not utilize the butylene glycol pathway or produce acetoin. Lysine decarboxylase, arginine dihydrolase and ornithine decarboxylase are not present. *S. boydii* does not produce H₂S, does not hydrolyze urea and does not grow in KCN broth. Carbohydrates are usually fermented and these include glucose (in the absence of gas production), D-mannitol, arabinose, trehalose and mannose.

Shigella bacteria cause diarrhea and shigellosis (bacillary dysentery) through oral-faecal transmission. *Shigella* is a highly infective agent able to infect a host with less than 20 cells with

an onset of about 12-48 hours, in favorable conditions. Once ingested, the *Shigella* makes its way through the gastrointestinal tract until it reaches the epithelial cells of the intestinal mucosa, there it infects, causing irritation, inflammation and necrosis (swelling and breaking of infected cells, which spreads infection). General symptoms include stomach cramps, high fever, mucus in feces, and bloody diarrhea due to ulceration of intestinal lining and rectum.

In most cases these symptoms are mild and resolve in about a week but other cases can become severe enough to be fatal without proper medical care. The elderly, very young and those weakened by disease are much more sensitive to the bacteria. In very young children very high fever may also be accompanied with seizures. Usually *Shigella* bacteria are found in areas of poor sanitation. Food washed with contaminated water or not cleaned properly may also be a target. In 1998 an outbreak of Shigellosis occurred in Chicago due to *Shigella boydii* type 18 found on the cilantro and parsley in bean salad.

At the National *Shigella* Reference Centre in Israel between 2000 and 2004, 5,616 *Shigella* isolates were tested for resistance to certain antibacterial products. In one strain of *Shigella boydii* 2 and in two strains each of *Shigella flexneri* 2a, *S. flexneri* 6, and *Shigella sonnei* there was discovered resistance to ceftriaxone. All of these strains were found to be producers of extended-spectrum beta-lactamase (ESBL) and also very sensitive to tazobactam which inhibits the bacterial beta-lactamases. More research is needed to determine the rate of resistance to antibiotics and rate of transfer of resistant genes.

At the French National Reference Centre for *Escherichia coli* and *Shigella*, from 2000 to 2004, 4,198 *Shigella* isolates were obtained. 180 of those isolates, taken from patients infected with diarrhea and dysentery, did not respond to any available antisera (serum containing antibodies) that had been used on all known *S. boydii* serotypes. All of the isolates shared characteristics of *S. boydii* serotypes except that their rRNA genes were unique from all others. A new antiserum was manufactured and destroyed the new serotype *S. boydii* 20. 91 of the 180 patients had travelled internationally while infected therefore further studies must be done in order to determine the epidemiology of this serogroup and the its potentially widespread threat.

CPD Questions:

1. Discuss the “connection” between *Shigella* species and *E. coli*.
 2. How many cells of *Shigella* species are needed to infect a human host? How does this compare to other “faecal-oral” transmitted infections?
 3. What is the connection between ESBL producers and the laboratory evaluation of antibiotic sensitivity?
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