The seroepidemiology of *Cryptosporidium parvum*: exposure is probably far more frequent than previously thought

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Cryptosporidium parvum (CP), an emerging waterborne pathogen, kills malnourished children and people with AIDS, and causes large diarrhoea outbreaks. Changing water use and climate patterns will alter its incidence. Published seropositivity rates to oocyst (transmission stage) antigens in older children and adults include: 17% Virginia, USA; 58% Oklahoma, USA; 65% Peru/Venezuela; 95% Fortaleza, Brazil. This has been conventionally interpreted as reflecting lifetime exposure. Much lower (5–20%) childhood serological rates have been reported, even though clinical cryptosporidiosis is most common in children. We assessed the assumption that anti-oocyst antibodies (AOA) accurately reflect lifetime exposure by reviewing 265 papers, dating from 1966 to the present, that directly or tangentially deal with CP serological responses.

We found a remarkable congruence of animal and human data documenting that AOA responses are transient (generally ±3 months), and not permanent. No studies have shown persistence of human AOA without known or presumed re-exposure. Tissue stage antibodies (TSA), which unlike AOA can only arise after infection and not simply oocyst exposure, appear to be more persistent (±1 year), and more common in childhood than AOA, though the number of studies is small. We conclude:

- The most scientifically conservative view is that AOA best reflects recent and not lifetime exposure, profoundly increasing (two orders of magnitude) estimates of lifetime exposure.
- Conventional AOA testing in adults may be most useful in detecting short-term or recent changes in oocyst exposure as water supply/climate changes occur.
- Future population-based studies should assess both AOA and TSA, so as to differentiate oocyst exposure from infection.

Birth outcomes and drinking water contamination

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Recent evidence linking adverse birth outcomes and chemical contaminants in public drinking water systems have been evaluated. A New Jersey study found associations between chlorinated disinfection by-products and neural tube defects (NTDs), oral cleft defects, cardiac defects, and small for gestational age (SGA). A follow-up to this

study focusing on NTDs has also found an association with chlorination disinfection by-products. A study in Iowa found an association between chloroform and SGA. Chemical contaminants in public drinking water systems, such as chlorinated and aromatic solvents, herbicides, and nitrates have also been studied. The New Jersey study found associations between NTDs and trichloroethylene (TCE), carbon tetrachloride, the dichloroethylenes, and benzene. Oral clefts were associated with perchloroethylene, TCE, carbon tetrachloride and the dichloroethylenes. In a study at Woburn, Massachusetts, TCE was linked to excess NTDs and cleft lip. A study in Tucson, Arizona, linked TCE with cardiac defects. An Iowa study of triazine contaminated drinking water found excesses of SGA, oral clefts, cardiac defects, and limb reductions. Two studies of nitrates in drinking water have found excesses of central nervous system defects. The presentation will discuss the difficulties of conducting studies of drinking water contamination and birth outcomes and the limitations of the studies conducted so far.

Disinfection by-products and bladder and colorectal cancer: a quantitative analysis of published results from interview studies

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Since the formation of chlorination by-products (CBPs) was first recognized, many studies have investigated possible associations between exposure to CBPs and cancer. Many studies have reported elevated relative risks for specific cancer sites, but the degree to which these associations reflect the role of chance or bias remains controversial.

This study examined the association between exposure to CBPs and both bladder and colorectal cancer using interview studies published up to the end of 1997. The general population risk associated with exposure to chlorinated surface water was estimated for each study together with a dose-response relationship based on combination of time and concentration as THM ppm–years of exposure. These results were pooled to generate an overall estimate of risk using both fixed effects and random effect models. The dose-response data were combined using a generalized additive model (GAM). The influence of study characteristics on results was examined and the effect modification by sex and smoking was considered.

For bladder cancer, these results were highly consistent with a pooled relative risk of 1.31 (95% CI: 1.19, 1.45). These risks increase monotonically and the dose-response curve is concave upwards. The results for colorectal cancer were less consistent. Some studies reported elevated risks for colon cancer and others reported elevated risks for rectal cancer. The nature of this risk appears to be modified by gender and by water source characteristics.