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Drinking Water Engineering and Science Discussions

Interactive comment on "Application of DVC-FISH method in tracking *Escherichia coli* in drinking water distribution networks" *by* L. Mezule et al.

F. Hammes (Referee)

Frederik.Hammes@eawag.ch

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GENERAL COMMENTS

The authors present an interesting study that demonstrates the application of the DVC-FISH method to real drinking water network samples and which clearly highlights some of the shortcomings of conventional detection methods. This is relevant, as conventional cultivation-based E. coli detection is used worldwide to monitor the hygienic quality of drinking water.

However, this "improved" detection approach also brings new challenges with respect to data interpretation. Based on findings like these, one needs to ask critical questions such as: (1) are cultivation based E. coli data meaningful at all? (2) Are current EU

drinking water monitoring guidelines appropriate? (3) Are alternative detection methods useful to detect/predict outbreaks of drinking water related disease?

It is my opinion that some of these critical questions can be acknowledged and addressed more in the discussion of the data, and this is pointed out in the specific comments below.

SPECIFIC COMMENTS

P521, L15 and elsewhere: The authors state that no waterborne outbreak of disease has been reported in Riga in the last 10 years. Based on this statement, and their data clearly showing viable and sometimes cultivable E. coli in the Riga drinking water (Table 1 & P522, L15-23), the following questions: 1. Does this mean that E coli is not a meaningful parameter for detecting/predicting outbreaks of waterborne disease? 2. Alternatively, does this mean that molecular methods (which detect much more E. coli than conventional methods) show organisms that are not relatable to disease (e.g., organisms that have lost their pathogenicity or their ability to divide sufficiently to cause disease)?

P523, L1-7: Is it possible to distinguish between E. coli accumulation and actual growth in the network? The authors analysed rather young biofilms (2 weeks old). Is there any indication that older biofilms would harbour either (a) more E. coli (due to accumulation and/or growth) or (b) less E. coli (due to increased competition and predation)?

P523, L8-15: More E. coli was found further away from the treatment plant and several explanations for that are suggested. Is there any evidence to suggest that the higher numbers are due to growth of E. coli in the network? Is an alternative explanation that a longer distance in the network increases the risk/possibility of pipe-failure and leakage and thus external contamination, which would also explain increased numbers?

P523, L15-17: This comment is very similar to the first comment above. Although these are indeed theoretical risks, the fact that no outbreaks have been reported in

the network under investigation seems to suggest that a correlation between these organisms and disease is not evident.

P523, L19: It seems that this statement/paragraph requires a reference.

P523, L26-30: A positive correlation between HPC and total counts are reported and interpreted. However, it would be useful to place this in perspective: only about 1% of the total bacteria were cultivable – a number fairly typical for drinking water biofilms. It is not clear how this directly relate to "the formation of more favourable conditions for colonisation and growth"?

P524, L10-11: The implication of this sentence seems to be that one should expect denser biofilms which would explain the higher cell concentrations in the water (L5-6). Although this seems logic, it contradicts the statement in L4-5 that "no correlation between TBC in biofilm and water was observed". Can it be that two week old biofilms measured in this study are simply not representative of the actual biofilm situation in the network, to which the water is exposed continuously?

P524, L12-15: Although this is indeed similar to the data of Delahaye to some extent, it contradicts other data e.g. our from group (Hammes et al. 2010, Water Research) which suggest that a correlation between TBC and ATP should be expected. Can this be a result of chlorine disinfection that affects viability (thus ATP) but not TBC? Is there a logic explanation for the high variability in ATP values?

TECHNICAL CORRECTIONS

1. Please add scale bars on Figure 3

Interactive comment on Drink. Water Eng. Sci. Discuss., 5, 515, 2012.