**Introduction**

Diarrheagenic *E. coli* and *Shigella* spp. are responsible for a substantial burden of diarrheal disease in Bangladesh. In Mirzapur, for example, the two are amongst the etiologic agents most responsible for diarrhea in children. The existing paradigm for enteric disease transmission is that the environment acts as a reservoir. There is limited to no emphasis on the potential role of the environment to amplify enteric diseases. In this study, we investigated whether or not the soil reservoir can aid amplification of diarrheagenic *E. coli*.

**Relevance:**

If soil can act to amplify diarrheagenic *E. coli*, then efforts to reduce diarrheal disease need to place greater emphasis on environmental hygiene in combination with existing emphasis on water and sanitation infrastructure provision.

**Hypotheses:**

1. Households practicing domestic animal husbandry contain more, and different, strains of *E. coli* than households without domestic animals.
2. *E. coli* in the soil are phenotypically and genetically distinct from enteric *E. coli* in human and animal feces.
3. *E. coli* in the soil are capable of persisting and growing in the soil better than enteric *E. coli* due to adaptation to the soil environment.
4. The soil environment contains a gene pool that provides opportunities for virulence and/or environmental persistence gene transfer.

**Methods**

1. Collected soil and fecal samples from 52 households (26 with, and 26 without, ruminants).
2. Isolated and assessed phenotypic and genotypic characteristics of *E. coli* in samples.
3. Assessed growth rates, and factors influencing growth rates of *E. coli* in microcosm studies.
4. Developed microcosm conditions under which *E. coli* strains (including pathogenic strains) are capable of long term persistence and/or growth.
5. Compared microcosm conditions developed in 4) to observed conditions in household environments to identify likelihood that pathogenic *E. coli* thrive in the environment.

**Results**

In 2010, researchers went to Tanzania to investigate contamination of women’s hands. It was demonstrated that women’s hands were contaminated with an average 3.5 log CFU *E. coli* per two hands, respectively. Furthermore, we found that household activities (e.g., sweeping, preparing food, cleaning dishes) readily and rapidly contaminate hands with *E. coli* only a few minutes after hand washing. These findings indicate that the source of fecal bacteria is likely the environmental reservoirs commonly found throughout the home, including household utensils, market produce, floors, and surfaces.

Follow up study found substantially higher concentrations of *E. coli* on household items (e.g., cups, plates, wash basins, toys, brooms) than are typically observed in industrialized regions like the United States and European Union. Similarly, soil samples from the household plot contained on average 2.1 log CFU *E. coli* per gram. Interestingly, *E. coli* and enteroaggregative concentrations measured in soils collected from inside the home, (including the location where food is prepared) were higher than concentrations in soils from inside or near the latrine.

**Conclusions**

If *E. coli* are naturalized to the soil environment in Bangladesh, their presence may contribute to on going transmission of pathogenic *E. coli*. Our work in Tanzania demonstrated the presence of virulence genes for five common diarrheagenic *E. coli* in soils. Though it is likely that the virulence genes detected in Tanzania were carried by enteric *E. coli*, it is also possible that the genes were detected in other microbiota, and/or that the genes were passed from enteric and environmental strains through horizontal gene transfer. Gene transfer between soil microbiota is a well-recognized phenomena, as is gene transfer of *E. coli* virulence genes. If horizontal gene transfer is possible between environmental *E. coli*, enteric *E. coli*, and/or other microbiota, then environmental *E. coli* pose a risk of becoming pathogenic if enteric *E. coli* are introduced. Evidence of the exchange of genetic material between environmental and enteric *E. coli* in the soil environment would dramatically impact the current paradigm concerning environmentally-mediated enteric disease transmission by suggesting that the environment can amplify disease transmission.

**References**


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