



Interactions of Quorum Sensing Bacteria with Protozoan Parasites in Freshwater Ecosystems

Liang Chen *

Department of Bacteriology, Eastern China Medical University, Nanjing, China

DESCRIPTION

Freshwater systems host complex webs of microbial life. Bacteria often communicate through chemical signals in quorum sensing networks, altering gene expression in response to cell density. These networks regulate factors like toxin production, motility, pigment synthesis, and biofilm formation. Protozoan parasites co-exist in such settings, and their survival, reproduction, or infective capacity may be influenced by bacterial signaling. This article examines what is known about how quorum sensing affects protozoan parasitism, and what this means for ecosystem health.

Bacterial quorum sensing involves autoinducer molecules such as acyl homoserine lactones in gram-negative bacteria, oligopeptides in gram-positive bacteria, and autoinducer-2 as a more universal signal. When concentrations reach threshold, bacteria alter behaviours collectively. For instance, they may produce metabolic byproducts or toxins which could damage protozoan membranes or interfere with parasite attachment to host tissues. In one study, bacterial populations with active quorum sensing generated compounds that inhibited *Giardia lamblia* encystment. By contrast, mutant strains defective in autoinducer production allowed parasite stages to proceed more rapidly.

Protozoan parasites such as *Entamoeba*, *Trichomonas*, *Blastocystis*, and *Toxoplasma* can encounter bacterial biofilms rich in quorum sensing activity. Within biofilms, autoinducer concentration is high; bacteria may produce lytic enzymes or reactive oxygen species when densely packed. Protozoa swimming in or feeding near biofilms may suffer reduced viability. On the other hand, some protozoa exploit bacterial metabolites—for example, bacterial-derived vitamins or coenzymes as nutritional supplements that support protozoan growth.

Temperature, nutrient concentration, and presence of pollutants modify quorum sensing efficacy. Elevated organic carbon enables

enables faster bacterial growth, leading to more rapid build-up of signal molecules. Warm water permits greater diffusion yet also faster metabolic turnover. Pollutants like heavy metals or certain pesticides diminish quorum sensing by interfering with signal receptors or degrading signal molecules. In contaminated freshwater, protozoa may evade bacterial inhibition due to suppression of bacterial communication.

Field investigations in ponds and lakes where eutrophication has occurred record shifts in community composition. Bacteria capable of strong quorum sensing become more common, while susceptible protozoan parasite infections decline among small invertebrates. Laboratory microcosm work confirms that bacterial populations with intact signaling pathways can reduce prevalence of *Acanthamoeba* cysts in water over time relative to non-signaling controls. Another approach is designing compounds that mimic autoinducers, stimulating bacteria to produce anti-parasitic compounds. Care must be taken to ensure that such additives do not themselves disrupt other components of aquatic ecology.

CONCLUSION

Parasitic disease burden in animals that drink from such waters may alter with these dynamics. For instance, livestock exposed to water containing protozoan parasites may receive lower infective doses when bacterial signaling is high and suppressive. Conversely, if bacterial quorum sensing is perturbed for instance by chemical runoff, antibiotic pollution, or extreme temperature shifts protozoan parasites may multiply unchecked, raising risk. Applications may include development of microbial additives to water systems. Introducing or enhancing populations of bacteria with effective quorum signaling could help control parasite stages. Another approach is designing compounds that mimic autoinducers, stimulating bacteria to produce anti-parasitic compounds. Care must be taken to ensure that such additives do not themselves disrupt other components of aquatic ecology.

Correspondence to: Liang Chen, Department of Bacteriology, Eastern China Medical University, Nanjing, China, E-mail: liang.chen@ecmu.edu.cn

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