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EVALUATING THE EXTENT OF POLLUTION-INDUCED ANTIBIOTIC RESISTANCE IN ENVIRONMENTAL BACTERIAL STRAINS

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ABSTRACT: Antibiotic resistance in pathogenic microorganisms is now recognized as a global crisis, as infectious disease morbidity and mortality is increasing worldwide. Much of this increase has been linked to the spread of multiple antibiotic resistance and the inability to treat diseases caused by resistant organisms. Widespread use of antibiotics added as supplements in livestock and poultry feeds, plus indiscriminant use by the medical profession are usually assumed to be the sources of antibiotic resistance. Recent microarray, quantitative PCR (qPCR) and minimum inhibitory concentration (MIC) studies conducted in our laboratory, however, has shown that a variety of environmental contaminants induce an antibiotic resistant phenotype in an opportunistic human pathogen, Pseudomonas aeruginosa, via the induction of multi-drug efflux pumps, especially the MexAB-OprM system. The implications of such a phenotype are enormous because the types of antibiotics and the levels of resistance that occur in the induced phenotype are substantial enough to have significant impacts on public and livestock health, and on environmental monitoring practices used to detect pathogens. Currently, additional MIC assays are being conducted to determine the extent to which this phenotype occurs in other, common freshwater and soil environmental isolates. All MIC experiments are conducted using a common organic contaminant, pentachlorophenol (PCP), which has been shown to induce the phenotype. The bacterial strains that exhibit the PCP-induced antibiotic resistance phenotype are also being evaluated for their ability to induce the phenotype when exposed to common agricultural pesticides, industrial chemicals, and urban contaminants. We will present the results from induction assays of the contaminant-induced antibiotic resistance phenotype on various strains of bacteria, including MIC experiments and qPCR results for MexAB-OprM. The results from this study will indicate the degree to which non-antibiotic micropollutants may be contributing to the proliferation of antibiotic resistance.

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