

ROGERS, EMILY D.<sup>1</sup>, JULIE A. GIARDINA<sup>2</sup>, AND DENNIS C. HANEY<sup>3</sup>. <sup>1</sup>Erskine College, <sup>2</sup>Northwestern University, and <sup>3</sup>Furman University--Factors affecting mortality and zinc uptake in the gills of freshwater fish.

In 1985, zinc-saturated hydrochloric acid from a ruptured galvanizing plant retention pond entered the groundwater, contaminating a section of the Upper Enoree River near Travelers Rest, South Carolina. Zinc concentrations today range from 0.1 to greater than 9ppm, and there are no fish present at locations nearest to the contamination site. A field mortality study was conducted by placing *Notropis lutipinnis* at five sites along the Upper Enoree River. Lab mortality experiments were also carried out using dilutions of zinc-contaminated water from one of the field sites. In a second laboratory component of the mortality study, zinc was added to uncontaminated river water to approximate the concentrations of each of the graduated dilutions described above. When the results of the three experiments were compared, field mortality, even at low zinc concentrations, was found to be 67.8% higher than both lab groups. The second part of the study investigated binding interactions in the gills. Calcium and zinc share binding sites on the gills, and our research has suggested that the presence of calcium in zinc-contaminated waters may mitigate the toxic effects of zinc. *Nocomis leptocephalus* were exposed to varying zinc (0ppm, 0.2ppm, and 0.6ppm) and calcium (0ppm, 6ppm, and 12ppm) concentrations using a three by three design, with zinc and calcium binding to gill tissue quantified using ICP-AES. The results show that zinc binding when calcium was present was suppressed by at least a factor of two at all concentrations of exposure.