# Sensing the Toxic Metals Cu and Cd With Bacterial Biosensors

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## Abstract

Cells of a soil-borne pseudomonad respond differentially to the toxic metals Cu and Cd. Increasing concentrations of Cu induce cell death, whereas Cd initially causes stasis of growth from which cells recover if the metal is present at sufficiently low concentrations. Modeling of speciation with citrate and phosphate as potential ligands show differences in complexation between Cd and Cu. Cd phosphate was demonstrated not to elicit growth cessation in cells indicating that it was not bioavailable. Thus, the environment of the ion has a major effect. We have used promoter fusions of a reporter gene cassette encoding a luciferase and genes involved in oxidative stress to determine the threshold concentration at which cells show a response. Logarithmic cells respond between 0.01 and 0.1 mg Cu/L, whereas stationary phase cells require 10 mg Cu/L for the same decline in luciferase activity. Thus, age of cells is important in constructing a biosensor. For Cd, concentrations of 10 mg/L were needed to decrease luciferase activity in log phase cells and 100 mg/L had no effect in stationary phase. However, stationary phase cells of a catalase A isozyme reporter showed increased activities in time-dependent manner at 10 mg/L. An increase in this catalase isozyme and a second catalase isozyme was confirmed in wild type stationary phase cells at 2 h of treatment. Three proteins, shown by a proteomics approach as being upregulated in Cd-treated cells, are identified as proteins that respond to oxidative stress in the cell. Proteomics of Cu-treated cell extracts identified three cell membrane proteins amongst those up-regulated. None of the 15 proteins identified that increased in level after metal treatment correspond to those predicted to be metal-sensitive in publications. Thus, our knowledge of how Cu or Cd interact with cells is not complete.