The most severe diseases correlated with infection by the gastric pathogen *Helicobacter pylori* are attributed to the long-term persistence of the bacterium. This persistence is in turn supported via the bacterium's stringent capacity to combat host-induced stress. Persistent pathogens like the *Helicobacters* must withstand oxidative damage to macromolecules, and this damage in proteins oftentimes occurs at the electron-rich sulfur atom of cysteine and methionine residues. Methionine sulfoxides (Met-SO) can be reductively repaired back to Met-S by the peptide repair enzyme methionine sulfoxide reductase (Msr), an enzyme present in most organisms. Two Met-rich and abundant repair target proteins in *H. pylori* are catalase (dissipates hydrogen peroxide) and urease (produces ammonium); the catalytic activity of these two enzymes is already known to play key roles in pathogen survival. By use of tandem mass spectral analysis, the repaired Mets in these proteins were identified. Site specific mutant versions of the pure enzymes, whereby residues at the active site of the abundant enzymes are replaced (to abolish catalytic activity, but all Mets are retained) and phenotypic analyses of the corresponding mutant strains of the bacterium (at these same sites) reveal that the MetSO/MetS repair cycle can quench/dissipate the harmful neutrophil-produced oxidant hypochlorous acid; this oxidant quenching is shown to significantly aid bacterial survival. Both enzymes--catalase and urease--are playing a previously unrecognized antioxidant role that is independent of their well-studied catalytic activity. Catalase from all organisms, from bacteria to rats and to humans, has similar structure and surface Met content, so the newly identified roles may be broadly applicable to stress survival biology.