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Mycobacterial Redox Heterogeneity & Antibiotic Resistance

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ABSTRACT: Approximately 30% of the global population is infected with *Mycobacterium tuberculosis* (*Mtb*). Persistence of *Mtb* in host phagocytes depends on its ability to resist oxidant-mediated antibacterial responses. Mycothiol (MSH) is the main antioxidant that provides an abundant source of reducing equivalent, which protects *Mtb* from oxidative stress encountered during infection. The majority of research into redox signaling in *Mtb* has relied on chemical analysis of MSH in whole cell extract, which creates oxidation artifacts and prohibits dynamic imaging of MSH redox state during infection. We have successfully developed a novel and noninvasive tool based on genetically encoded redox sensitive fluorescent probes to perform real-time measurement of mycothiol redox potential (E_{MSH}) in *Mtb* during infection. For the first time we reveal the E_{MSH} of virulent and avirulent mycobacterial strains, including drug-resistant clinical isolates. We used this technology and came to the surprising conclusion that within a single infected macrophage there is heterogeneity in the redox signature of individual *Mtb* bacilli. Importantly, we show that anti-TB drugs accelerate oxidative stress in *Mtb* within infected macrophages and redox heterogeneity can contribute to emergence of drug tolerant population. These findings have implications for mycobacterial persistence following treatment with anti-TB drugs.

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