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The CDC classifies *Neisseria gonorrhoeae*, the causative agent of gonorrhea, as an urgent public health threat due to its increased drug resistance. Out of the 1.6 million new gonococcal infections monitored by the CDC, over fifty percent are resistant to at least one antibiotic. Given a single dose of ceftriaxone is the only recommended treatment for gonorrhea and *N. gonorrhoeae* ceftriaxone resistance prevalence is on the rise, there is increasing concern that gonorrhea will become untreatable in the near future. Therefore, new antibiotics must be developed against this bacterium. Our laboratory has discovered that resazomycins exhibit bactericidal activity against *N. gonorrhoeae*. While the mode of action of these compounds has yet to be determined, we have shown the antimicrobial activity of resazomycins is reduced against *N. gonorrhoeae* in 2% oxygen compared to 20% oxygen. Moreover, the addition of antioxidants at 20% oxygen to scavenge and neutralize excess reactive oxygen species (ROS) also limited the activity of resazomycins against *N. gonorrhoeae*. Together, these data suggest ROS-mediated cellular damage contributes to the antimicrobial activity of resazomycins. Therefore, we are working to generate a mutant of the master regulator of the oxidative stress response in *N. gonorrhoeae*, OxyR. We will then evaluate the sensitivity of the *oxyR* mutant to different resazomycins using agar dilution and time kill assays. Determining whether *oxyR* plays a role in resazomycin susceptibility will elucidate whether oxidative stress contributes to the bactericidal activity of these antibiotics.