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Carbon monoxide: A bactericidal molecule

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Carbon monoxide (CO), once perceived to be a detrimental metabolite of heme degradation is now considered by many to be a key modulator of inflammation and immunity. In recent years, CO has been ascribed an additional novel role as a 'bactericidal agent' by harnessing and modulating the function of the macrophage. We previously demonstrated that exogenous administration of CO at 250 ppm accelerated the clearance of bacteria by cultured peritoneal macrophages that involved TLR-4 expression. We have now expanded on these observations further to determine the role of CO in macrophage-mediated bacterial killing. *In vivo*, mice injected intra-peritoneally with purified *Enterococci faecalis (EF)* and administered exogenous CO 1 hr post-infection, all survived long up to 72 hr while air-treated controls died by 48 hr. Similarly *in vitro*, administration of exogenous CO for 4 hr to ¬post-infected cultures of peritoneal macrophages augmented the macrophage killing machinery when compared to air in an agar plate assay; 8.3 x10 CFU/ml compared to 5.83 x 10^3 CFU/ml respectively. In this presentation, we will discuss the possible mechanism by which CO behaves as a therapeutic bactericidal agent.

Biography

B. Y. Chin spent her postdoctoral fellowship in Molecular Toxicology at US Department of Energy at Pacific Northwest National Laboratory in Richland WA. Currently, she is at Beth Israel Deaconess Medical Center/Harvard School of Medicine in Boston MA in the Department of Surgery. She is also a member of the teaching faculty of the 1st year medical curriculum anatomy histology and physiology at Harvard Medical School. She has published over 30 peer review articles and serves on the editorial board of World Journal of Pharmacology and as a guest editor in Current Chemical Biology.

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