세미나 초록

발표주제	Host-Pathogen Interaction : from Infectious Diseases to Therapeutic Platform
발표내용	Macrophages release iron into the bloodstream via a membrane-bound iron export protein, ferroportin (FPN). The hepatic iron-regulatory hormone hepcidin controls FPN internalization and degradation in response to bacterial infection. Salmonella typhimurium can invade macrophages and proliferate in the Salmonella-containing vacuole (SCV). Hepcidin is reported to increase the mortality of Salmonella-infected animals by increasing the bacterial load in macrophages. Here we assess the iron levels and find that hepcidin increases iron content in the cytosol but decreases it in the SCV through FPN on the SCV membrane. Loss-of-FPN from the SCV via the action of hepcidin impairs the generation of bactericidal reactive oxygen species (ROS) as the iron content decreases. We conclude that FPN is required to provide sufficient iron to the SCV, where iron serves as a cofactor for the generation of antimicrobial ROS rather than as a nutrient for Salmonella.