NAC treatment of M. avium infected A-549 and MH-S resulted in a significant reduction of mycobacterial load (p<0.009 and p<0.012) (Fig. 1 C). In vivo, NAC treatment resulted in a significant reduction of mycobacterial load in the lungs of M. avium infected mice (p<0.007)(Fig.3 B). When in combination with clarithromycin, we also had an additional reduction (vs. clarithromycin monotherapy; p<0.001) (Fig.3 B). mRNA expression levels of Mouse beta defensin 3 significantly increased when treated with NAC and clarithromycin combination therapy (Fig. 3 C). Otherwise, there were no significant changes in mRNA expression of cytokines nor other antimicrobial peptides, e.g. cathelicidin antimicrobial peptide, other Mouse beta defensins (1, 2, and 4) and other antimicrobial peptides which can be analyzed with the previously mentioned PCR array kit.

This study demonstrated a potent anti-mycobacterial effect of NAC against M. avium. Interestingly in our study, expression level of HBD-2 increased in NAC containing therapy group. HBD-2 is a cationic antimicrobial peptide that exhibits a wide range of antimicrobial activity against viruses, bacteria, and fungi. Its gene expression has been identified in various human epithelia including lungs and trachea (4). In M. tuberculosis infection, HBD-2 has been shown to control bacterial growth and has chemotactic activity (5). Thus, increase in expression level of HBD-2 may be one of the possibility on how NAC is involved in anti-mycobacterial effects on M. avium.

NAC exhibits potent anti-mycobacterial effects and may limit M. avium infection. In addition with clarithromycin, it showed additive effect in reduction of mycobacterial loads. As NAC is already widely used in clinical medicine, it may be an additional option in treating M. avium infected patients in future, along with its classical drug regimens containing clarithromycin.