中文題目：類肺炎性胸腔積液中脂磷壁酸促進第一型胞漿素原活化抑制劑的表現之機制探討

英文題目：Lipoteichoic Acid Upregulates Plasminogen Activator Inhibitor-1 Expression in Parapneumonic effusion

作　　者：李凱靈[1], 黎書亮[1], 鍾啟禮[2]
服務單位：台北醫學大學附設醫院胸腔內科[1], 台北醫學大學醫學院呼吸治療學系[2]

Background: Parapneumonic effusion (PPE) is commonly caused by Gram-positive bacteria (GPB) and often presents with pleural fibrinous loculation and fibrosis, characterized by overproduction of plasminogen activator inhibitor-1 (PAI-1) in pleural mesothelial cells (PMCs). Lipoteichoic acid (LTA), a surface adhesion molecule of GPB, binds to the pleural mesothelium and trigger pleural inflammatory responses. However, the effects of LTA on PAI-1 expression in human PMCs and the underlying mechanisms remain unclear.

Objective: The present study aims to elucidate the effects of LTA on PAI-1 expression in human PMCs and the underlying mechanisms.

Methods: Thirty consecutive patients with parapneumonic effusion were divided into culture negative (CN, n = 11), Gram negative bacteria (GNB, n = 7) and GPB (n = 12) PPE groups based on pleural fluid bacteriology, and the effusion PAI-1 levels were measured. In addition, MeT-5A human PMCs were treated with LTA and the expression of PAI-1 protein and mRNA, activation of associated signal pathways, and PAI-1 promoter activity were assayed.

Results: Pleural PAI-1 levels were significantly different between all the three groups ($p<0.001$) and the median levels from highest to lowest were: GPB (160.5 ng/ml), GNB (117.0 ng/ml) and CN (58.0 ng/ml)

Conclusions: Culture positive PPE, especially that cause by GPB, has significantly higher level of PAI-1 than culture negative PPE.LTA can upregulates PAI-1 expression, through activating the TLR2/JNK/AP-1 pathway in human PMCs. Better understanding of the regulation of PAI-1 expression in PMCs by LTA may provide potential therapies for infectious pleural effusion.