中文題目：Gelsolin (GSN) 透過 p38 訊息途徑和 GATA-4 轉錄因子活化誘發心肌細胞肥大和腦利鈉尿素蛋白表現

英文題目： Gelsolin (GSN) induces Cardiomyocyte hypertrophy and BNP expression via p38 signaling and GATA-4 transcriptional factor activation

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**Background:** Cardiomyocyte hypertrophy is an adaptive response of the heart to various types of stress. Gelsolin (GSN) is a member of the actin-binding proteins (ABPs), which regulate dynamic actin filament organization by severing and capping. Moreover, GSN also regulates cell morphology, differentiation, movement, and apoptosis.

**Methods:** In this study, we used H9c2 and H9c2-GSN stable clones, in an attempt to understand the mechanisms of GSN overexpression in cardiomyocytes.

**Results**: This data showed that the overexpression of GSN in H9c2 induced cardiac hypertrophy and increased the pathological hypertrophy markers atrial natriuretic peptide (ANP) brain natriuretic peptide (BNP). Furthermore, we found that E-cadherin expression decreased with the overexpression of GSN in H9c2, but ß-catenin expression increased. These data presume that the cytoskeleton is loose. Further, previous studies show that the mitogen-activated protein kinase (MAPK) pathway can induce cardiac hypertrophy. Our data showed that p-p38 expression increased with the overexpression of GSN in H9c2, and the transcription factor p-GATA4 expression also increased, suggesting that the overexpression of GSN in H9c2-induced cardiac hypertrophy seemed to be regulated by the p38/GATA4 pathway. Moreover, we used both the p38 inhibitor (SB203580) and GSN siRNA to confirm our conjecture.

**Conclusion**: We found that both of these factors significantly suppressed gelsolin-induced cardiac hypertrophy which through p38/GATA4 signaling pathway. Therefore, we predict that the gene silencing of GSN and/or the downstream blocking of GSN along the p38 pathway could be applied to ameliorate pathological cardiac hypertrophy in the future.

**Keywords**: Cardiac hypertrophy, Gelsolin (GSN), Cytoskeleton