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Anti-myocardial ischaemic effect of pseudo-ginsenoside GQ injection against beta1-adrenoceptor (β1-AR) in rat cardiac myocytes (H9c2)

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Statement of the Problem: Myocardial ischemia (MI) is a prevalent complicated heart disorder worldwide. It is a clinically common pathophysiological phenomenon which characterized by reduced blood supply to the heart, resulting in nutrients depletion and causing hypoxia. Although there are many precious researches on MI injury, it is perennial among the leading causes of morbidity and mortality in humans in the industrialized countries. Therefore, research of an efficient agent for MI injury remains an important issue. Pseudo-ginsenoside GQ (PGQ) injection (1.1 class chemicals), firstly developed by our lab., is based on PGQ obtained by modifying 20(S)-ginsenoside Rg3 with the biological degradation and the chiral semi-synthesis method, etc., which was approved by the Chinese State Food and Drug Administration for the treatment of myocardial ischemia and phase I trial was carried on to evaluate the safety of PGQ injection.

Methodology & Theoretical Orientation: The effects of PGQ injection on the cardiovascular physiology of MI injury rats were examined by measuring various electrocardiographic parameters. NBT staining method was utilized to measure the heart infarct size, and the levels of LDH, CK, SOD, AST, MDA and cTnT were measured to further evaluate and validate the protective function of PGQ injection during MI injury. Flow cytometry and western blot analysis were used to detect the β1-AR expression *in vitro*.

Findings: Both data and histopathological examination all demonstrated that PGQ injection could significantly improve the heart function and decrease infarct size. And the levels of CK, LDH, AST, MDA and cTnT were decreased and the activities of SOD were increased. The underlying mechanism was explored by flow cytometry and western blot, and results showed that the expression of β1-AR was decreased by PGQ injection.

Conclusion & Significance: This study provides the substantial evidence for the effect of PGQ injection on myocardial ischemia.

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Ide sequestration, release and ecology of the anticancer drug Taxol from fungal endophytes

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Taxol is produced by Taxus trees and their resident endophytic fungi. A mystery has been why these endophytes synthesize Taxol apparently redundantly. A defining feature of these trees is that they can propagate branches from long-lived buds that lie underneath the bark; branch emergence is accompanied by bark cracks, potential pathogen entry points. Here, we show that Taxol acts as a fungicide against wood decaying fungi (WDF) to which these long-lived trees are susceptible. Reducing endophytes in plants resulted in increased WDF growth. Endophytes sequestered Taxol in intracellular hydrophobic bodies (Hb), which prevented plant cytoxicity. Taxol-producing endophytes with these Hb localized to vascular rays within wood, but hyperaccumulated where the rays intersected branch points and associated air pockets. Chloromethane, a chemical released by WDF, along with chitin or WDF, induced Hb release from endophytes. Hb was released from endophytes by exocytosis; chloromethane induced exocytosis genes. Combined, Taxol-producing endophytes contribute to the survival of their host by protecting their nutrient-rich vascular system and branch points against systemic fungal pathogen invasion.

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