Abstract: P504

Bakuchiol attenuates myocardial ischemia reperfusion injury by maintaining mitochondrial function: the role of silent information regulator 3

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Topic(s):
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Background
Ischemia reperfusion (IR) injury (IRI) is associated with poor prognoses in the settings of both cardiac surgery and ischemic heart disease and causes mitochondrial oxidative stress and cell death. Silent information regulator 3 (SIRT3), a member of the histone deacetylase family, exerts anti-IRI effects. Bakuchiol (BAK), an analog of resveratrol and a monoterpene phenol isolated from the seeds of Psoralea corylifolia (Leguminosae), protects tissues from injury.

Method and results
This study was designed to investigate the protective effects of BAK treatment in the setting of myocardial IRI and to elucidate the potential mechanism of those effects. Prior to induction of IR, isolated rat hearts or cardiomyocytes were exposed to BAK in either the absence or presence of the SIRT3 siRNA. BAK exerted cardioprotective effects, as evidenced by the improvements noted in cardiac function following ischemia, attenuated myocardial apoptosis, and changes in several biochemical parameters (including increases in the level of the anti-apoptotic protein Bcl2, decreases in the level of the pro-apoptotic protein Bax, and decreases in the cleaved Caspase 3 level). However, SIRT3 siRNA blocked BAK-induced cardioprotection by inhibiting SIRT3 signaling. Additionally, BAK significantly increased the activities of mitochondrial succinate dehydrogenase, cytochrome c oxidase, and mitochondrial superoxide dismutase and decreased the production of malondialdehyde. These findings suggested that BAK significantly attenuated IR-induced mitochondrial oxidative damage. However, SIRT3 siRNA abolished BAK-dependent mitochondrial function.

Conclusion
In summary, our results demonstrate that BAK treatment attenuates IRI by attenuating IR-induced mitochondrial oxidative damage via the activation of SIRT3/PGC-1a signaling.
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