**50. Tetramethylpyrazine Retards the Progression and Fibrogenesis of Endometriosis**

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Abstract

The development of more efficacious, non-hormonal therapeutics for endometriosis is still an unmet medical need begging to be fulfilled. Growing evidence indicates that endometriotic lesions are wounds undergoing repeated tissue injury and repair, and, as such, platelets play an important role in lesional progression. Tetramethylpyrazine (TMP), a compound derived from a herb that has been used for thousands of years to combat "blood stasis" in traditional Chinese medicine, is a prescription drug in China for the treatment of cerebrovascular disorders. We tested the hypothesis that TMP can decelerate lesional progression through arresting epithelial-mesenchymal transition (EMT), fibroblast-to-myofibroblast transdifferentiation (FMT), and fibrogenesis. We found in our in vitro experiments that TMP treatment suppresses platelet-induced EMT, FMT, cellular contractility, and collagen production in a concentration-dependent manner. We also showed that in a mouse model of endometriosis, treatment with TMP significantly reduced lesion weight and the extent of lesional fibrosis and improved hyperalgesia, mostly likely through the reduction of lesional aggregation of platelets and the lesional expression of markers of EMT, FMT, and fibrogenesis. In light of our results and in view of its excellent safety profiles, TMP appears to be a promising drug candidate for treating endometriosis.



Keywords: Endometriosis; Epithelial-mesenchymal transition; Fibroblast-to- myofibroblast transdifferentiation; Fibrogenesis; Mouse; Tetramethylpyrazine.