Long-Term Administration Green Tea Catechins Effect on Aging-Related Cardiac Diastolic Dysfunction & Troponin I Decline

New publication Genes & Diseases - long-term administration of green tea catechins effectson aging-related cardiac diastolic dysfunction & decline of troponin I

CHONGQING, CHINA, December 20, 2024 /EINPresswire.com/ -- A new publication from Genes & Diseases; DOI 10.1016/j.gendis.2024.101284, discusses the effect of long-term administration of green tea catechins on aging-related cardiac diastolic dysfunction and decline of troponin I.

Aging is an independent risk factor for cardiovascular diseases. Cardiac diastolic dysfunction (CDD), ultimately leading to heart failure with preserved ejection fraction (HFpEF), is prevalent among older individuals. Although therapeutics have made great progress, preventive strategies remain unmet medical needs.

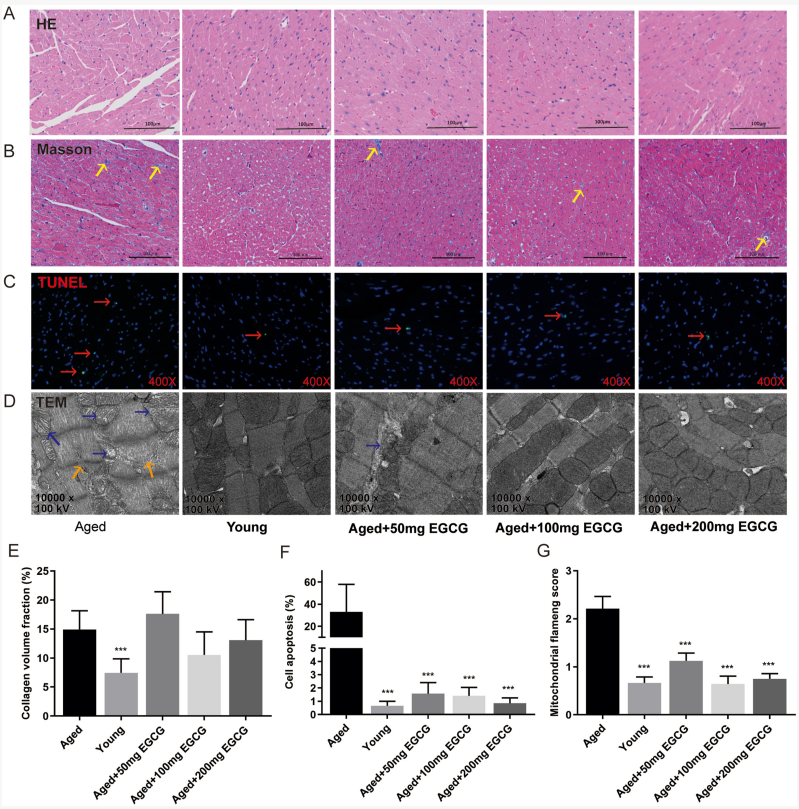

Fig 1: Long-term EGCG intake alleviates aging-related cardiomyocyte apoptosis and mitochondrial damage. (A, B) Representative histology of heart tissue sections stained with hematoxylin-eosin stain and Masson's trichrome stain (yellow arrows collagen fibr

Green tea catechins have been shown to be effective in improving aging-related cardiovascular and cerebral disorders in animal models and patients. However, little attention has been paid to whether long-term administration of epigallocatechin gallate (EGCG), the major bioactive ingredient of green tea catechins, could prevent the onset and progression of CDD.

In this study, 12-month-old female mice were orally administered 50, 100 and 200 mg EGCG mixed with drinking water for 6 months. Aged mice (18 months old) exhibited the major features of HFpEF, including CDD with pEF, cardiac fibrosis, increased cardiomyocyte apoptosis, and

mitochondrial damages, as well as elevated A/B-type natriuretic peptide. Cardiac troponin I (cTnI) expression was also reduced. Long-term administration of 100 or 200 mg EGCG prevented aging-related CDD and exercise capacity decline, along with alleviating myocardial apoptosis and mitochondria damage. The transcription and protein expression of cTnI were increased, which might be achieved by inhibiting the expression and activity of histone deacetylase 1 (HDAC1), and reducing its binding level near cTnI's promoter, thereby elevating acetylated histone 3 (AcH3) and acetylated lysine 9 on histone H3 (AcH3K9) in the aged mice.

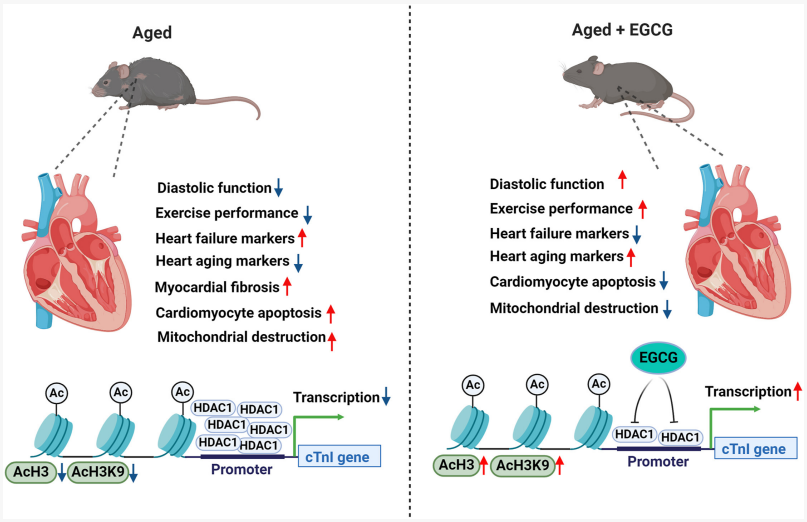

Fig 2: The schematic illustrating that long-term EGCG administration prevents CDD and cTnI decline in aging hearts. The scheme depicts the role of physiological aging in the pathophysiology of CDD. The hallmarks of HFpEF such as CDD, reduced exercise perf

This article reports a novel insight that long-term administration of EGCG is a potentially effective strategy in preventing aging-related CDD and cTnI expression decline.

Keywords: Aging, Cardiac diastolic dysfunction, Cardiac troponin I, Epigallocatechin gallate, Histone deacetylase 1

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