


Etiopathophysiological role of the renin–angiotensin–aldosterone system in age-related muscular weakening: RAAS-independent beneficial role of ACE2 in muscle weakness

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Abstract

Aging is accompanied by major changes in body composition that can negatively affect functional status in older adults, including a progressive decrease in muscle mass, strength, and quality. The prevalence of sarcopenia has varied considerably, depending on the definition used and the population surveyed—a 2014 meta-analysis across several countries found estimates ranging from 1% to 29% for people aged 60 years or older, who live independently. The potentially relevant studies were retrieved from the ScienceDirect/Medline/PubMed/Public library of science/Mendeley/Springer link and Google Scholar. Multiple keywords were used for the literature search both alone and in combination. Some of the important keywords used for literature search were as follows: “Epidemiology of muscle weakness/muscle disorders,” “Pathogenesis of RAAS in muscle weakness,” “Role of Angiotensin 1–7/ACE-2/Mas R axis in muscle weakness,” and “Correction pathophysiology of muscle weakness via ACE2.” The renin–angiotensin system (RAAS), a major blood pressure regulatory system, is a candidate mediator that may promote aging-associated muscle weakness. Previously, studies explored the proof concept for RAAS inhibition as a therapeutic target. Furthermore, in RAAS, angiotensin II, and angiotensin-converting enzyme 2 (ACE2) have been reported to induce endoplasmic reticulum (ER) stress via glucose-regulated protein 78/eukaryotic translation initiation factor 2 α (eIF2 α)/activating transcription factor 4 (ATF4)/CHOP axis in the liver. In addition, other mitochondria and ER physical interactions contribute to skeletal muscle

consequences in muscle weakness. Thus, the study has been designed to investigate the RAAS-independent beneficial role of ACE2 in muscle weakness.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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PEER REVIEW

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DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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