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Our group has so far reported the pathogenic role of cellular senescence and systemic insulin resistance in age-related cardiovascular-metabolic disorders including heart failure, atherosclerotic diseases, obesity and diabetes (*Nat Med 2009, Cell Metab2012, 2013, 2014, Nature Aging 2021, 2024, J Clin Invest 2010, JMCC 2015, 2019, Sci Rep 2019, 2021, 2022*). "Metabolic remodeling" is one of the keywords for aging research, and studies with brown adipose tissue showed maintaining the homeostasis of this organ as crucial to combat obesity or heart failure (*J Clin Invest 2014, Cell Rep 2018, iScience 2022, 2022, 2022, Sci Rep 2022, EMBO Journal 2024*).
Through these studies, I came to consider the mechanisms contributing for the synchronization of aging (sync-aging) as interesting. Now, we define "senometabolite" or "senoprotein" as circulating molecules having causal roles for the sync-aging. Based on several unpublished data, we are now trying to establish new concepts for diseases; One is Age-related fibrotic disorders (A-FiD), and another is Senometabolite Related Disorders (SRDs). Together with the suppression of sync-aging, we are also trying to establish a method to reverse aging with the senolytic approach (specific depletion of senescent cells) (Please also see; https://www.cv-aging.com).

Representative publications (as a first/corresponding author);
Nat Med 2009, Cell Metab2012, 2013, 2014, Nature Aging 2021, 2024, J Clin Invest2010, 2014, Cell Rep2018, Sci Rep2019, 2021, 2021, 2022, 2022, JMCC2015, 2016, 2019, iScience 2022, 2022, 2022, EMBO Journal 2024, Circ Res 2025 (in press).