



MADRAS DIABETES RESEARCH FOUNDATION

ICMR CENTRE FOR ADVANCED RESEARCH ON DIABETES

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Signed Statement from the Applicant

This is to attest and certify that the research work under reference has not been given any award in the past.

While all the text books of medical endocrinology emphasize the occurrence of type 2 diabetes in the 4th decade (and onwards) of an individual, I used to wonder watching the prevalence rates of type 2 diabetes advancing a decade in individuals worldwide, particularly in South Asians and migrant Indians. Ever since I returned from USA after my post-doctoral training (1991-95), and when I realized the limitations of much of the inflammatory and oxidative stress markers in the association and causality of the etiology of diabetes, there is a serendipity idea in my mind to measure telomere length as a robust biomarker as its increased shortening has been shown associated in certain disease-states. With the help of one of my doctoral students and the infrastructure support from the head of the institution (MADRAS DIABETES RESEARCH FOUNDATION), our study for the first time in the world literature, reported an association of telomere shortening in patients with type 2 diabetes (published in Diabetic Medicine 2005; citations=224). Our 2nd study, again for the first time reported an association of telomere shortening in prediabetic individuals as well as in diabetes patients with macroangiopathy (published in Atherosclerosis 2007; citations=210). Since this time, I not only following the school of thought 'accelerated aging in diabetes' but also extended my studies to unravel the central role of shortened telomeres in the transformation of 'senescence-associated secretory phenotype' (SASP) in diabetic-state that is linked to increased inflammation, oxidative stress, glycation and other cellular stress signals. Our studies got attention worldwide and received due endorsement by none other than the Noble Laureate, Dr.Elizabeth Blackburn (from the University of California) when her team has demonstrated that shortened telomere predicts risk of diabetes in American Indians (2013). Performing the state-of-the-art clinical research, my subsequent studies demonstrated the accelerated cellular senescence (biological ageing) in type 2 diabetes linked to alterations in a panel of miRNAs, LncRNAs (Human Genomics 2018; citations=177), HADCs as well as increased mitochondrial and ER stress. One of my studies unraveled elevated systemic levels of bisphenol-A (BPA) in patients with type 2 diabetes linked to accelerated senescence and shortened telomeres. Very recently, one of our studies also demonstrated increased telomere shortening in diabetic patients with depression that is very well correlated to altered levels of miR-128, BDNF, cortisol and poor glycemic control.

Most of my studies have clinical foresight and warrant 'targeting aging' so as to pave way for the development of novel therapeutic regimen of anti-ageing / senolytic agents. I attest that these biomarkers do have a role in lifestyle intervention studies. While I acknowledge the assistance by my doctoral students, colleagues, infrastructure support and encouragement by the head of the institution as well as financial assistance from various funding agencies, I attest that I have conceived the research topic, implemented it and majorly contributed to the publications and clinical translational outcomes and futuristic research and development avenues.

With Regards

 30/8/2024

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