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Talk title: Caloric restriction and cardiovascular Protection

Calorie restriction (CR), is a strategy proven to extend healthy and maximum life span in rodents and primates. Recent evidence shows that adult-onset moderate CR delays the onset of age-associated cardiovascular disease and promotes survival in a primate species.

Cardiovascular diseases are the leading cause of death and illness in developed societies. Most present knowledge on the effects of CR is effective to lose weight in humans, which has been associated with significant improvement in the cardiovascular risk factor profile, including total body fat, total cholesterol, serum triglyceride. Based on a range of risk factors, it appears that long-term CR has a powerful protective effect against atherosclerosis, which constitutes the single most important contributor to cardiovascular diseases. In addition, CR may directly confer vasoprotection in humans through the following mechanisms, including increases bioavailability of NO and improves endothelial function; decreases oxidative stress and attenuates inflammatory processes in the vasculature; retards senescence of vascular cells.

Accumulating evidence reveals that aging is a major risk factor for cardiovascular disease. CR is always regarded as a process of limiting caloric intake with the intention of slowing down aging. The beneficial effects of low caloric intake are mediated by the sirtuin family member Sir2, a conserved nicotinamide adenine dinucleotide (NAD⁺)-dependent protein deacetylase that plays a role in the extension of life span and chromatin remodeling associated with gene silencing. SIRT1, the best characterized mammalian sirtuin, plays a protective role in diabetes mellitus by participating in glucose metabolism, increasing insulin secretion and increasing insulin sensitivity. SIRT1 mediates the effects of CR on endothelium-dependent vasomotor tone. We have shown that transgenic mice that overexpress SIRT1 in the vascular endothelium have better endothelium-dependent vasodilation and fewer atherosclerotic lesions when fed a high-fat diet. The recent studies from our lab have also demonstrated that SIRT1 prevents the hyperglycemia-induced endothelial damages. In addition, SIRT1 acts as a modulator of neointima formation following vascular injury. Moreover, in Macrophages, SIRT1 inhibits AP-1 transcriptional activity and COX-2 expression leading to amelioration of macrophage function and may be a mediator of CR-induced macrophage regulation. These results highlight the important roles of SIRT1 in mediating the benefits of CR and improving cardiovascular health. We summarized the beneficial effects of CR in cardiovascular system and proposed that SIRT1 maybe a mediator of CR and can become a novel therapeutic target for age-related cardiovascular diseases.

Biography

Dr. De-Pei Liu is currently a professor of National Laboratory of Medical Molecular Biology, Institute of Basic Medical Sciences, Chinese Academy of Medical Sciences (CAMS) & Peking Union Medical College (PUMC). Dr. Liu graduated with a Ph.D. from CAMS & PUMC in 1986. He completed his Postdoctoral Fellowship in molecular biology at University of California, San Francisco (UCSF), and was promoted to be a Professor of CAMS & PUMC in 1992. Dr. Liu's research expertise is molecular mechanisms of cardiovascular diseases, gene regulation and gene therapy. He has published more than 110 original research articles and invited reviews. His work has been cited more than 1500 times. Throughout his academic career, Dr. Liu has received numerous awards including three items of awards of Advance of Science and Technology, the Ministry of Health, P.R.China and one item of award of the National Natural Sciences Foundation. He is also serving as a member of Chinese Academy of Engineering (CAE), member of Institute of Medicine (IOM) of the National Academies and member of Third World Academy of Sciences (TWAS).

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