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PHARMACOLOGICAL AND NON-PHARMACOLOGICAL INTERVENTIONS FOR AGING

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Aging is a multifactorial process that leads to the loss of physiological functioning of an organism and thereby making it vulnerable to diseases. Therefore, slowing down the process of aging can delay the onset of many age related cerebral, cardiovascular diseases and metabolic syndromes. Understanding the potential mechanisms of how lifespan, age related processes, and cell senescence are controlled can help discover novel approaches to expand lifespan and improve the quality of life. Slowing down the process of aging will cause fewer diseases, improve better functioning of the brain and reduce pain associated with the development of age related alterations and diseases.

Aging is caused due to molecular changes hence it is possible to slow down aging and the onset of age related diseases by intervening in the molecular changes that take place. Over the years, studies have indicated that both pharmacological and non-pharmacological interventions can improve lifespan and reduce these diseases. Calorie restriction (CR), one of the most established interventions is responsible for the reversal of irregular gene expression, conserving chromatin stability, and delaying the onset of age related diseases, thereby prolonging the lifespan. Studies on CR have indicated a reduced incidence of cancer, cardiovascular diseases, brain atrophy, and diabetes in rhesus monkeys. Besides, regular physical exercise, reduced intake of saturated fats, alcohol, and sugar have shown increased lifespan by two or more years. Many studies have also cited the adaptation of a Mediterranean diet and its proven long list of health benefits. This includes longevity, improved cognition in older patients, prevention or slowing down of the onset of metabolic diseases, and osteoporosis. Antioxidants too have been shown to decrease oxidative stress and inflammation thus protecting DNA against damage and helping in DNA repair mechanisms. Certain studies also show, that reduction in insulin/insulin like growth factor 1 like signaling (IIS), mediated by the FOXO transcription factors, increases lifespan in nematodes, fruit flies, and mice models. Various studies involving the signaling of the mechanistic target of rapamycin complex 1(mTORC1) have emerged through the years. Studies centered on yeast and animal models have proven that mutations in mTORC1 improved the lifespan of the used models. These animal models have indicated the delayed onset of Parkinson's, delayed progression of Alzheimer's, and reduced incidence of tumorigenesis. Rapamycin studies also have demonstrated a decline in age related alterations in heart, liver, and adrenal glands in rapamycin treated mice. Stem cell exhaustion seen in the elderly has been improved after rapamycin treatment. However, the side effects of mTOR inhibitors remain a major concern. Nonetheless, the side effects were dose dependent therefore lowering the dosage reduced the observed side effects. This was proven by a clinical trial that used Everolimus at a low dosage for the elderly which displayed reduced side effects.

In conclusion, we found out that the most perspective way to prevent or slow down aging is by dietary restrictions, proper lifestyle, and inhibition of various pathways, particularly the mTOR pathway. Together with therapies to boost stem cell rejuvenation, DNA repair mechanisms, and inhibitors of mTOR, IIS, and AMPK it will be possible to slow down aging, its related processes, and associated diseases. The introduction of longevity drugs to inhibit these pathways can be promising prospects to help slow down aging and its related processes as it is not only scientifically but socio-economically important.