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Telomere dynamics in aging

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Preservation of basic genomic stability is hallmark of the survival of a living cell. Nuclear eukar-yotic genome constitutes a specific chromosome number and their defined morphology. Telo-meres, the physical ends of the linear chromosomes protect their morphology against degradation and through preventing fusion with other chromosomes or chromosome segments. These protec-tive caps comprise of a specific repetitive DNA sequence which does not code for any protein. Shortening of telomere length occurs with the dividing human cells while the telomerase enzyme complex adds the telomeric repeat de novo, thereby assuring the genomic stability. Enormous volume of research has established an association between the shortening of telomere length, and human diseases and aging. Decreased activity of telomerase and resultant shortening of telomere lengths in aged human being and in a few animal, models have led to infer telomere length as a biomarker of aging. Such studies have, however, not been able to conclude whether telomere shortening is the cause of aging or merely a consequence. Present review explores the mechanistic aspects of telomere biology.

Speaker Biography

Asefa K Ansari did her PhD in 1984 from University of Reading, England. At present, she is settled in Chicago, USA and serving as a Director of Basic Sciences at North West Suburban Col-lege. She has thirty-seven research publications in National and International Journals and thirty-five years of teaching and research experience. Basically, she is an Entomologist. But recently, she has developed a passion for Human Genetics.

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