Tumour cells can adopt telomere maintenance mechanisms (TMMs) to avoid telomere shortening, an inevitable process due to successive cell divisions. In most tumour cells, telomere length (TL) is maintained by reactivation of telomerase, while a small part acquires immortality through the telomerase-independent alternative lengthening of telomeres (ALT) mechanism. A great amount of data was generated, and different TMMs were reported and explained in detail, benefiting from genome-scale studies of major importance. Gaspar et al. addressed seven different TMMs in tumour cells: mutations of the TERT promoter (TERTp), amplification of the genes TERT and TERC, polymorphic variants of the TERT gene and of its promoter, rearrangements of the TERT gene, epigenetic changes, ALT, and non-defined TMM (NDTMM). They gathered information from over fifty thousand patients reported in 288 papers in the last years. This wide data collection enabled us to portray, by organ/system and histotypes, the prevalence of TERTp mutations, TERT and TERC amplifications, and ALT in human tumours. Based on this information, they discussed the putative future clinical impact of the aforementioned mechanisms on the malignant transformation process in different setups, and provide insights for screening, prognosis, and patient management stratification [1)]

[1)]

Gaspar TB, Sá A, Lopes JM, Sobrinho-Simões M, Soares P, Vinagre J. Telomere Maintenance Mechanisms in Cancer. Genes (Basel). 2018 May 3;9(5):241. doi: 10.3390/genes9050241. PMID: 29751586; PMCID: PMC5977181.