Supporting Information

Luu and Palczewski 10.1073/pnas.1721033115

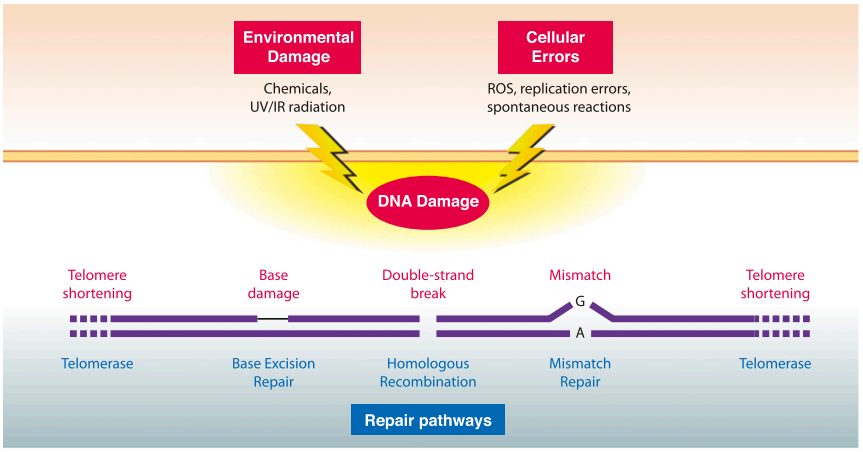

Fig. S1. Genomic instability. Environmental damage (exogenous) and cellular errors (endogenous) converge to form various types of DNA lesions over time, such as acquired somatic mutation (base damage), double-strand break, mismatch pairing, and telomere shortening. These lesions are repaired by appropriate somatic maintenance processes in a normal, healthy cell. However, these repair processes decline with age. ROS, reactive oxygen species.

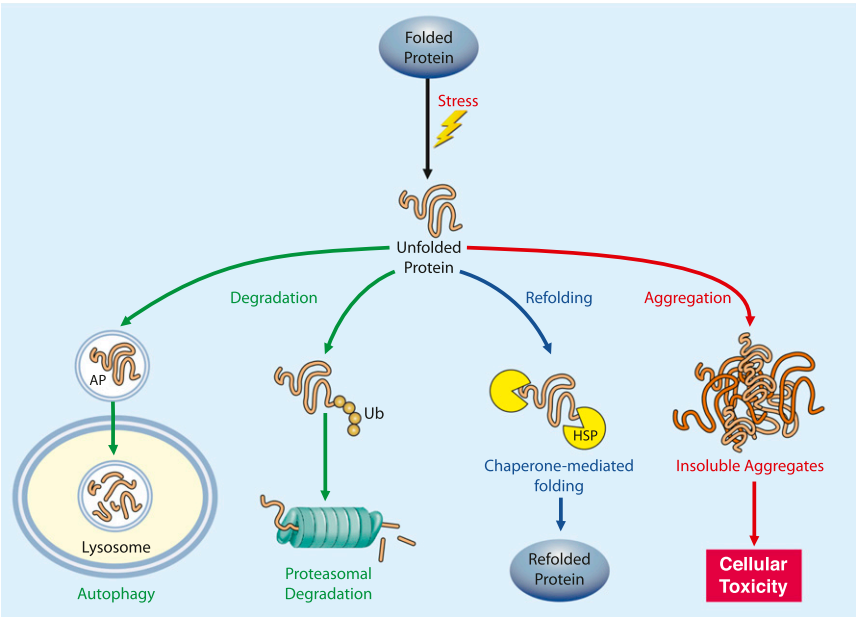

Fig. S2. Aggravating factors and ameliorating mechanisms of proteostasis. Various environmental stressors, such as oxidation, UV radiation, and pollution, disrupt proper protein folding. If unfolded cellular proteins are not refolded by chaperones or degraded by either autophagy or proteasomes, they form insoluble aggregates, causing impairments in cellular function. Increased abundance of cellular protein aggregates is a central hallmark of aging. AP, autophagosome; HSP, heat shock protein; Ub, ubiquitin.