

Abstract

Naked mole rat and blind mole rat are useful model organisms for human age-associated diseases studies. Unlike human, their long lifespan is not accompanied by physical health impairment. In both species, the genes involved in aging process or carcinogenesis are under positive selection or their regulation differs from the regulatory pattern known in other rodents or human. Some genes are present in higher number of copies, missing or entirely new and not observed in other organisms. In naked mole rat, the degenerative development is reduced by elevated level of proteins which prevent amyloid β aggregation and contribute to oxidative damage tolerance. Their healthy aging is also caused by effective elimination of damaged proteins, natural caloric restriction or angiogenesis enhancement. High level of α -2-macroglobulin in blood, which is able to inhibit signal pathways required for tumor growth and malignancy, as well as the early contact inhibition repress tumorigenesis in naked mole rat. Many different mechanisms are involved in prolonged lifespan in both naked and blind mole rat species. The aim of this thesis is to present the most important genome and proteome differences contributing to their long lifespan.

Key words: naked mole rat, blind mole rat, tumorigenesis resistance, senescence, aging, age-associated diseases