Abstract

Apolipoprotein B mRNA editing enzyme, catalytic polypeptide (APOBEC) are a family of evolutionarily conserved cytidine deaminases with the ability to bind and modify RNA and/or ssDNA. APOBEC1-4 have a number of functions in cells. Members of the APOBEC3 subfamily cause restriction of foreign nucleic acids, retrotransposons and viruses, including human papillomaviruses (HPV), and may contribute to the clearance of infection. Certain HPVs are referred to as oncogenic viruses because of their ability to induce immortalization and transformation of epithelial cells via E5, E6 and E7 oncoproteins. E6 and E7 can also induce transcription or inhibit degradation of some APOBEC3. This results in an increase in their levels in cells. APOBEC3 also act as cellular mutators, as they can catalyze deaminations on transiently produced ssDNA during replication or transcription. Deregulation of APOBEC3 caused by oncoproteins may contribute to mutagenesis. This bachelor thesis focuses on APOBEC proteins, their activation and function during HPV-induced carcinogenesis, and in particular the extent and consequences of APOBEC3 mutations.

Keywords: APOBEC, mutagenesis, papillomavirus, oncoproteins, carcinogenesis