Editorial

Quality Check in Oral Cell Lines: The Need for Molecular Characterization

Oral cell lines have provided valuable insights into the various molecular pathways in oral carcinogenesis. Several landmark studies in oral oncology have utilized commercially available normal, dysplastic and cancer cell lines to decode the genetic alterations leading to the development of oral cancer. Most of these studies have shown a significant degree of variation in their mutation landscapes. These variations were thought to represent the heterogeneity of oral cancer.[1] But in a recent study, Dickman et al have shown that normal and dysplastic cell lines carry specific genetic alterations within the parent cell line, thus questioning the authenticity of several published mutation profiles. These genetic alterations in the commercial cell lines have been attributed to several factors, the most common being immortalization. Normal and dysplastic cell lines unlike cancer cell lines attain senescence following limited number of replication. Immortalization of the normal and dysplastic cell lines would aid the researcher in maintaining a viable population of cells for further studies. Ideally, the immortalized cell line must possess potential for indefinite replication and must retain the genetic makeup of its parent cell line.[2]

The most commonly employed technique for immortalization is by deactivation the tumor suppressor genes, such as p53 and K-Ras.[3] This is achieved by subjecting the cell lines to viral oncogenes simion virus 40T [(SV40T)[4] or E6 and E7].[5,6] Micro RNAs have also been used to silence multiple tumor suppressor genes and cell cycle inhibitors enabling prolonged cell replication.[7] Addition of telomerase reverse transcriptase (hTERT) immortalizes the cell line by preventing telomere loss.[8,9] Immortalization through any of the above mentioned techniques possess a risk of additional mutations.[1,2] Thus, utilizing normal/dysplastic cell lines which are not characterized postimmortalization could pollute the final study data.

To conclude, researchers must insist on the manufacturers to provide the complete molecular characterization data of the cell line. Further studies on normal, dysplastic and cancer cell lines must analyze the molecular profile of the parent cell line for comparison with the resulting mutation profile of the study. Thus, molecular characterization of cell lines would enable the researcher to filter the mutations inherent in the parent cell line from the mutation resulting from the study. This in turn would enable the researchers to decode a relatively accurate mutation landscape for oral carcinogenesis.

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