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Prevalence of Beta-Papilloma virus in early onset squamous cell skin cancer

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Background and objectives: Cutaneous squamous cell carcinoma (cSCC) are frequently diagnosed and account for most of the 410 deaths that occur due to non-melanoma skin cancer in Australia each year. Evidence is mounting that HPV also contributes to cSCC. The objective of this study was to test this hypothesis further by determining the betaPV distribution in Australian cSCC cases diagnosed at a relatively early age, using both related and population controls.

Methods: Cases (n=111), recruited from sequential records of a dermatopathology referral service in Sydney, had been diagnosed with cSCC of any stage <50 yr (<10% of cases in Australia are diagnosed <50yr). The cases nominated both sibling (n=53) and unrelated partner (n=66) controls. Eyebrow hair bulbs were collected for detection and genotyping of betaPV using the PM-PCR reverse hybridization assay (RHA) method.

Results: Results from half the sample show an overall carriage frequency of 76% in cases, 68% in sibling controls and 72% in unrelated controls (p=0.018). HPV types 5, 15, 23, 24, 38 were the most commonly recurring, each present in >25% of subjects. 14% of the subjects tested negative for each of the 25 papillomavirus types, with no association with case control status. Four or more types were present in 35% of cases, 16% of siblings and 31% of unrelated controls (p=0.336). Putatively high cancer risk types (5, 8, 15, 20, 24, 36, 38) were present in 62% of cases, 48% of sibling controls and 57% of unrelated controls; 17% of the cases and unrelated controls, but 2% of the siblings, carried three or more types from the high-risk group.

Conclusion: The data suggest that control siblings were less likely than case siblings to exhibit long-term carriage of (multiple) betaPV types. However, there is no evidence at this stage for association of betaPV with early-onset cSCC, especially when cases were compared with unrelated controls.

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Molecular profiling of cancer stem cells: New avenue to cure cancer

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Calls (CSCs) are mainly found in tumors at very low concentration and have the similar properties of normal stem Cells. These cells have a property of self renewal & differentiation. However, the renewed cells are found to be tumorogenic in nature. These cells are also very much resistant to chemotherapies and radiotherapies. This may be the major mechanism involved in the relapse & metastasis of tumor cells at different site of human body. However, the origins of CSCs are not yet known clearly. Recently our study on Breast Cancer Stem Cells have shown that CSCs are very few in number and there is a probability of transferring cancer phenotype of these cells to neighboring normal cells to make normal adjacent cells to cancer cells. This results into formation of metastatic tumor which is resistant to general chemo or radiotherapies. So there is a need to establish a method to isolate these CSCs from the tumor of the patient by growing them in culture or any other fractionation method by using specific cancer antibodies. The isolated CSCs are then profiled by using several Molecular Markers involved in Cancer development. Differential expressions of these markers can be used as biomarkers to target CSCs present in patient tumor or in blood circulation during metastasis. Advancement in nanotechnology will help in delivering this specific bio-molecule to target CSCs without harming normal stem cells. This will have a great advantage to overcome several side effects caused by the present therapies of cancers. The strategies of targeting CSCs can also be useful in early diagnosed cancer as well as the cancer with severe metastasis to distant organs. This presentation will overview various strategies so far established in development of targeted therapies of cancers by eradicating cancer stem cells.

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