<table>
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<th><strong>Title</strong></th>
<th>Nuclear and cell membrane surface area alteration in hexamethylene bisacetamide (HMBA) induced human colonic carcinoma cell line (LoVo)</th>
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<td><strong>Author(s)</strong></td>
<td>Lau, TYH; Liao, X; Shum, DKY; Cheung, A; Tipoe, GL</td>
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Although these results are not statistically significant they suggest that EBV-and/or the lymphocyte depletion subtype were excluded from the analysis. Groups were 2.3 and 0.34. Similar findings were obtained when the lymphocyte predominant material deprivation), whereas only 56/97 EEV-negative patients had scores above zero. Median Townsend scores for EBV-positive and EEV-negative individual patients based on data from the 1991 OPCS Census. The demonstration of LMPI. EBV status determined in this way for each patient was supported by the Committee on Research and Conference Grants, The University of Hong Kong, Lady Ho Man Tin, to detect any translocations. We have cloned size suitable for direct sequencing. This has enabled the design of NPM and ALK exon and intron-specific primer pairs to amplify across the translocation breakpoint in archival fixed tissue biopsy material, to detect any translocations. In particular, EBV positive HD tumours appear to be higher in underprivileged populations, data supporting this assumption is lacking. This study has analysed 123 cases of histologically confirmed Hodgkin's disease from the West Midlands, UK, for the presence of EBV in HRS cells using both in situ hybridisation to detect the EBERs or by immunohistochemistry for LMPI. This has enabled the design of NPM and ALK exon and intron-specific primer pairs to amplify across the translocation breakpoint in archival fixed tissue biopsy material, to detect any translocations. We have cloned size suitable for direct sequencing. This has enabled the design of NPM and ALK exon and intron-specific primer pairs to amplify across the translocation breakpoint in archival fixed paraffin-embedded material. The resulting amplified fragments are of a size suitable for direct sequencing.

Is expression of p21/WAF1/CIP1 related to EBV status in Hodgkin's disease?

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p21/WAF1/CIP1 (p21) is a nuclear protein inhibitor of several cyclin-dependent kinases (cdks). It is a component of the quaternary complexes that control the cell cycle and includes cyclin D1, cdks, and the proliferating cell nuclear antigen (PCNA). This study has utilised immunohistochemistry to analyse the expression of p21 in Hodgkin-Reed Sternberg (HRS) cells of 63 cases of histologically confirmed Hodgkin's disease in relation to Epstein-Barr virus (EBV) status. EBV status was determined by in situ hybridisation for the detection of the EBERs or by immunohistochemistry for LMPI. p21 nuclear expression was demonstrated in the majority of cases but intensity of expression was variable. High level expression of p21 (50% HRS cells stained or >20% HRS cells strongly stained) was demonstrated in 6/18 EBV-positive cases but only in 7/45 EBV-negative tumours. All EBV positive cases showed loss of p21 staining in HRS cells. Four EBV-negative tumours did not express detectable levels of p21. An association between high level expression of p21 and decreased survival was found in EBV-negative patients. No such trend was noted for EBV-positive patients, which may reflect the relatively low numbers of patients in this group.

Effect of material deprivation on Epstein-Barr virus infection in Hodgkin's disease: preliminary analysis of a West Midlands population

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Mapping the (2;5)(p23;q35) Nucleosomlin-Anaplastic Lymphoma Kinase Breakpoints in Anaplastic Large Cell Lymphoma Cell Lines


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The (2;5)(p23;q35) translocation fuses the nucleosomlin gene (NPM) on chromosome 5q35 to a neuronal protein kinase gene, anaplastic lymphoma kinase (ALK) on chromosome 2p23. The translocation is characteristic of a sub-group of CD30+ anaplastic large-cell non-Hodgkin's lymphomas (ALCL). Previous studies have used long range amplification of genomic DNA, isolated from fresh tissue biopsy material, to detect any translocations. We have cloned and sequenced the wild type NPM and ALK introns and the Karpass 299 translocation intron and have subsequently mapped the distinct intron-specific translocation breakpoints in three ALCL cell lines (SU-DHL-1, SUP-M2 and Karpass 299) by sequence comparison. This has enabled the design of NPM and ALK exon and intron-specific primer pairs to amplify across the translocation breakpoint in archival fixed paraffin-embedded material. The resulting amplified fragments are of a size suitable for direct sequencing.