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

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The Epstein-Barr virus (EBV) is detectable in Hodgkin/Reed-Sternberg (HRS) cells in a percentage of Hodgkin's disease (HD) tumours. However, the level of EBV-positivity in HD is variable and depends on several factors including age, sex and country of residence. In particular, EBV positive HD tumours appear to be less common in developed populations, with percentages of between 40-50% for North American and European cases, 57% for HD in China, but much higher in underprivileged populations. This study has utilised immunohistochemistry to examine the expression of p21 in Hodgkin/Reed-Sternberg (HRS) cells of HD specimens of histologically confirmed Hodgkin's disease (HD) and has subsequently mapped the distinct t(2;5)(p23;q35) translocation breakpoints in three ALCL cell lines. The t(2;5)(p23;q35) translocation fuses the nucleophosmin gene (NPM) on chromosome 5q35 to a neuronal protein kinase gene, anaplastic lymphoma kinase (ALK) on chromosome 2p23. The translocation is characteristic of a subgroup 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.