<table>
<thead>
<tr>
<th>Title</th>
<th>Nuclear and cell membrane surface area alteration in hexamethylene bisacetamide (HMBA) induced human colonic carcinoma cell line (LoVo)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author(s)</td>
<td>Lau, TYH; Liao, X; Shum, DKY; Cheung, A; Tipoe, GL</td>
</tr>
<tr>
<td>Citation</td>
<td>The 177th Meeting of the Pathological Society of Great Britain and Ireland, Leicester, UK., 1–3 July 1998. In Journal of Pathology, 1998, v. 186 n. 1, p. 19A</td>
</tr>
<tr>
<td>Issued Date</td>
<td>1998</td>
</tr>
<tr>
<td>URL</td>
<td><a href="http://hdl.handle.net/10722/95768">http://hdl.handle.net/10722/95768</a></td>
</tr>
</tbody>
</table>
NUCLEAR AND CELL MEMBRANE SURFACE AREA ALTERATION IN HEXAMETHYLENE BISACETAMIDE (HMBA) INDUCED HUMAN COLONIC CARCINOMA CELL LINE (LOVO)


The aim of the present study was to determine the effect of HMBA on the morphology of the nuclear and cell membrane of LOVO (CCL-229) using electron microscopy and stereological techniques. Two mM and 4mM of HMBA were included in the culture medium for a period of 7 days and then for a further 3 days HMBA-containing medium was omitted. Control flaskas were never exposed to HMBA. Cell numbers were counted on days 0, 3, 5, 7, and 10 using a haemocytometer and cells were processed for electron microscopy. Inhibition of the growth of CCL-229 cells was produced by 2mM and 4mM HMBA treatment during the first 7 days of culture but growth resumed after HMBA was withdrawn on day 7 and there was an increase in cell number until day 10. The nuclear to cytoplasmic ratio (N/C) and surface-to-volume ratio (S/V) of the nuclear and plasma membranes were estimated using simple point-counting techniques. The nuclear volume (VN) was estimated from point-sampled intercepts. From these data, cytoplasmic volume (VC), cell surface area of plasma membrane (SMCCELL) and nuclear membrane (SNCCELL) were calculated. Statistical analyses revealed significant increase in VC, VN and VCELL with a decrease in N/C in HMBA-treated group on day 7 when compared with control, but no significant alterations in VSM. The values of VCELL and NC were maintained up to day 10 despite the absence of HMBA. The SNCCELL significantly decreased in 4mM HMBA-treated cells on day 7 and 2mM HMBA-treated group on day 7 when compared with the control. The SMCELL also decreased in both HMBA-treated group on day 1 and in the 2mM HMBA-treated group on day 3 when compared with the control. Generally, SMCCELL progressively decreased in the control group from day 1 to day 10 and had lower values when compared with both HMBA-treated groups. The SMCCELL significantly increased in cells treated with 2mM and 4mM HMBA on day 7 and persisted till day 10 when compared with the control group. The SMCCELL increased in the 4mM HMBA-treated group on day 7 when compared with the controls. We conclude that HMBA suppresses cell proliferation and induces an increase in cell volume and surface area of plasma membrane in the later stages of the culture period.

Supported by the Committee on Research and Conference Grants, The University of Hong Kong.

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


1School of Health Sciences, University of Wolverhampton, Wolverhampton, WV1 1JD, 2Centre of Molecular Biology and Medicine, Palacay University, Olomouc, Czech Republic, *Masaryk Memorial Cancer Institute, Brno Czech Republic, 3Department of Histopathology, New Cross Hospital, Wolverhampton, WV10 0QP, 4Department of Histopathology, Russells Hall Hospital, Dudley, West Midlands, DY1 2HG, 5West Midlands Cancer Intelligence Unit, The Public Health Building, The University of Birmingham, Birmingham, B15 2TT, 6Department of Histopathology, Birmingham Heartlands Hospital, Bordesley Green East, Birmingham, B9 5SS, and 7Institute for Cancer Studies, University of Birmingham, Edgbaston, Birmingham, B15 2TT.

Is expression of p21WAF1/CIP1 related to EBV status in Hodgkin's disease?


1School of Health Sciences, University of Wolverhampton, Wolverhampton, WV1 1JD, 2Centre of Molecular Biology and Medicine, Palacay University, Olomouc, Czech Republic, *Masaryk Memorial Cancer Institute, Brno Czech Republic, 3Department of Histopathology, New Cross Hospital, Wolverhampton, WV10 0QP, 4Department of Histopathology, Russells Hall Hospital, Dudley, West Midlands, DY1 2HG, 5West Midlands Cancer Intelligence Unit, The Public Health Building, The University of Birmingham, Birmingham, B15 2TT, 6Department of Histopathology, Birmingham Heartlands Hospital, Bordesley Green East, Birmingham, B9 5SS, and 7Institute for Cancer Studies, University of Birmingham, Edgbaston, Birmingham, B15 2TT.

p21WAF1/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 LMP1. 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 stained strongly) was demonstrated in 6/18 EBV-positive cases but only in 7/45 EBV-negative tumours. All EBV positive cases showed 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.

This project was supported in part by DACR No. 2001/60497.

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


Department of Pathology, University of Leicester.

The (2;5)(p23;q35) translocation fuses the nucleophosmin gene (NPM1) 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 in 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.