ectoderm and mesoderm cells except in the area of the basement membrane. The cytoplasm of both types of cells is lacking organelles where these cells are in close proximity to one another in the area of the basement membrane (Figure 2). The region is notable for the absence of ribosomes. Associated with these areas are mesoderm cells which have compressed the basement membrane against the ectoderm cells (Figure 3).

Similar observations were also made in the stage-5 in ovo specimens. Cytoplasmic changes were evident in the region where the band of nuclei appears by light microscopy. Mesoderm cells have compressed the basement membrane against the ectoderm layer and ribosome free areas are present in this region.

These observations suggest that primary neural induction in the chick embryo is associated with cellular communication between ectoderm and mesoderm cells. Furthermore, the cytoplasmic changes in the mesoderm emphasize the close interaction between ectoderm and mesoderm during induction.

Résumé. Exposé d'une étude ultrastructurale de l'induction neurale primaire chez l'embryon de poulet. Entre l'étape 4 et l'étape-5, les cellules mésodermiques compriment la membrane basale contre les cellules ectodermiques. Le cytoplasme des cellules mésodermiques et ectodermiques se libère d'organelles, spécialement de ribosomes, dans la région adjacente à la membrane basale. Ce fait suggère l'existence d'une communication cellulaire entre les cellules ectodermiques et mésodermiques, et ce processus peut être responsable de l'induction neurale primaire.

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EPSTEIN-BARR Virus-Binding Receptor on the Surface of Chronic Lymphocytic Leukaemic Lymphocytes

The nonpermissive or partially permissive interaction of the EPSTEIN-BARR virus (EBV) with the lymphoid cells has been clearly demonstrated, but other cells susceptible to the viral replication have not yet been found, and so the question arises whether the T or the B lymphocytes, or both, are susceptible to the EBV infection. JONDAL and KLEIN[1] have studied this problem and found that the B lymphocytes originating from healthy donors were the target cells and all EBV-carrying continuous human lymphoblastoid cell lines had receptors characteristic for B lymphocytes (the presence of surface-bound immunoglobulin molecules). In contrast, established cell lines of known T cell origin did not carry the EBV genome. These facts clearly indicate that the EBV is a B cell tropic virus. Experimental results show the occurence of B cell proliferation in chronic lymphocytic leukaemia (CLL) or, in other words, the CLL lymphocytes have characteristic surface markers of B cells[2,3]. For this reason it is of interest to study the susceptibility of CLL lymphocytes for the EBV infection, and it seems especially important to establish the presence or absence an EBV-receptor on the surface of CLL lymphocytes. In our experiments we used the special rosette test described by JONDAL and KLEIN, with only a slight modification. The virus receptors on the surface of the sensitive lymphocytes combine with the viral envelope materials accumulated in the membranes of membrane antigen positive cells. This finding indicates that the susceptible cells have to bind to the EBV-producing cells in the same way in which the EBV acts on the target cells. P3HR-1 is one of the best virus-producing lines, containing 'whole ring' membrane antigen positive cells.

Materials and methods. Purified CLL lymphocytes were mixed with EBV-producing P3HR-1 cells originating from Burkitt lymphoma at a ratio of 20 to 1. The cells were suspended in 0.3 ml PBS and incubated at 4 °C for 1 h. Then the suspension was dropped into slides, air dried, fixed in cold acetone-methanol (1:1) and stained with FITC conjugated anti-EBV-VCA-MA (virus capsid antigen and membrane antigen) immunsera. It has been shown previously that all VCA positive cells are also MA positive in EBV carrier cultures[4]. Samples were taken

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Binding of CLL lymphocytes to EB virus producing cells in the P3HR-1 line

CLL cases	Presence of surface bound immunoglobulins on the CLL cells[a]	VCA positive cells (%)	Rosette forming cells (%)	VCA positive cells forming rosettes (%)	VCA negative cells forming rosettes (%)
1	positive	2.5	2.0	90	0
2	positive	2.5	2.0	90	0
3	positive	3.0	3.0	95	0
4	positive	1.5	1.5	90	0
5	positive	3.0	3.0	90	0
6	positive	2.5	2.0	80	0

[a] Detected by membrane immunofluorescence with FITC labelled anti human IgM conjugate (Hyland).

from the cell suspension and the number of the rosetta-forming P3HR-1 cells were counted in Bürker chamber (only cells surrounded by complete lymphocyte rings were considered rosette-forming cells).

Results and discussion. It was found (Table) that the percentage of the EBV-VCA antigen positive and rosette-forming P3HR-1 cells was the same. Subsequent investigations by immunofluorescence showed that only VCA antigen positive P3HR-1 cells were surrounded by CLL lymphocytes, while VCA antigen negative cells did not form rosettes. This receptor was present on the surface of the CLL lymphocytes after one week of cultivation in vitro, and the presence of 10 µg/ml puromycin HCl for 2 days had no detectable effect on it. Our findings clearly show the existence of a rather stable EBV receptor on the surface of CLL lymphocytes.

The results of some authors and our own unpublished observations indicate that the CLL lymphocytes behave in the same way as the B cells[3]. Thus our findings seem to support the results of JONDAL and KLEIN, showing the B cell tropic characteristic of EBV. Our earlier studies demonstrated that the EBV induced cellular DNA synthesis and transformed the CLL lymphocytes, showing their increased susceptibility to EBV infection as compared with that of healthy lymphocytes. This in vitro observed, and quite remarkable, susceptibility of the CLL lymphocytes to EBV-infection (the presence of EBV receptors and increased stimulation of cellular DNA synthesis) seems to be a characteristic marker for these cells.

Zusammenfassung. Nachweis, dass Lymphocyten von chronisch lymphatischen Leukämien an der Zelloberfläche einen relativ stabilen Rezeptor für EPSTEIN-BARR-Virus enthalten und daher als B-Lymphocyten zu charakterisieren sind.

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Demonstration of Mast Cells in the Albino Rat Brain

It has been postulated that histamine is one of the physiologically acting neurotransmitter substances in the brain, and the regional distribution of histamine in the brain of different mammalian species has been studied and discussed by several authors[1–3]. In most parts of the organism, however, the histamine is produced and stored in mast cells which contain large amounts of this amine in cytoplasmic granules[4,5], and in some species mast cells has been observed in the thalamus region: hamster and hedgehog[6–9] and in the area postrema of several mammalian species[8,10]. Therefore, it seems reasonable to assume that at least some of the histamine in the brain could be located in mast cells, and that this cell might be a normal element in the brain of many mammalian species.

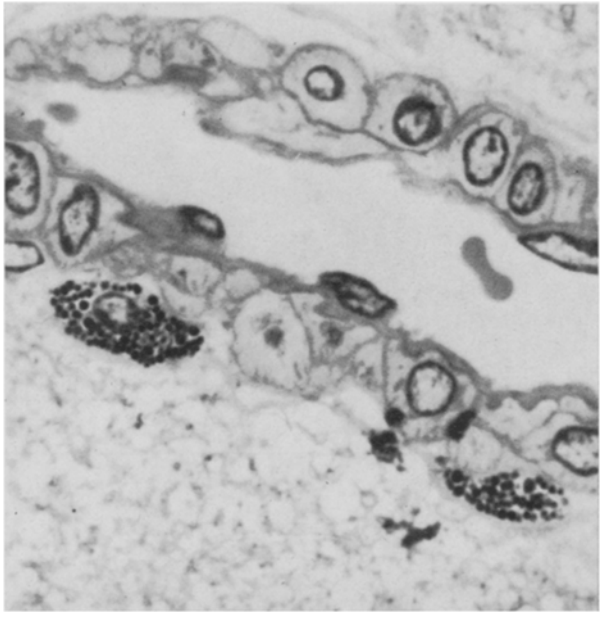

Fig. 1. Light micrograph of semi-thin epon section showing perivascular mast cells in the thalamus of the adult rat brain. Section stained with toluidine blue. ×1600.

In the present study, laboratory rats of the Møll Wistar strain were used. The brains were removed from the decapitated animals and fixated in 4% neutral formaldehyde (buffered with Ca-acetate) for paraffin-embedding, or in 4% glutaraldehyde (in Sørensen phosphate buffer, pH 7.0) and postfixed in 1% osmiumtetroxyde for epon embedding. Two parallel frontal section series were made of each paraffin embedded brain. The sections of 10 µm were stained in 0.2% toluidine blue at pH 3.7 (buffered with McIllvaine-Lillie's citrate/phosphate buffer) or in astra blue according to the method of BLOOM and KELLY[11]. Every 10th section was studied, and the number of mast cells in an actual section was obtained by dividing the cell countings with 1.9 to compensate for the possibility of observing mast cells from neighbouring sections. (The mast cells in tissues are mostly elongate, and the diameter

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