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Differential permeability to horseradish peroxidase in affected and non-affected ventricular walls during postnatal development of normal and hydrocephalic *hyh* mice

Patricia Páez[1], Ruth Roales-Buján[1], Sara Rodríguez[2], Federico Bátiz[2], Antonio J Jiménez[1], Esteban M Rodríguez[2] and José-Manuel Pérez-Fígares*[1]

Address: [1]Departamento de Biología Celular, Genética y Fisiología, Universidad de Málaga, E-29071 Málaga, España and [2]Instituto de Histología y Patología, Universidad Austral de Chile, Valdivia, Chile

Email: José-Manuel Pérez-Fígares* - figares@uma.es

* Corresponding author

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Background

Hyh mutant mice suffer a congenital hydrocephalus triggered by ependyma denudation [1]. The ventricular surface in non hydrocephalic newborn mice is lined by the immature ependyma, which is characterized for being vimentin (-) and S100β (-), at variance in the adult animals the mature ependyma expresses vimentin and S100β [2]. On the other hand, in the hydrocephalic mice the ependyma begins to denudate on the 12th day of gestation, and at PN8 only some areas of lateral ventricle are still endowed with ependyma. In parallel, astroglia starts to cover the denuded surface forming a new cell layer, the glial scar, which lines the damaged ventricular surface. We have studied the permeability to horseradish peroxidase (HRP) of these four regions at the ventricular wills: mature ependyma, and denuded areas with or without glial scar.

Materials and methods

Control and hydrocephalic *hyh* mice (Jackson Lab., USA) at 3rd and 30th day of post-natal life were injected into a lateral ventricle with 3% HRP. 15 min after the injection the animals were sacrificed under anesthesia. HRP was detected by immunocitochemistry with specific antibodies. Inmunocitochemistry for PCNA (to label proliferating cells) and GFAP, S100β and vimentin was used

Results

In non-hydrocephalic mice the immature ependymal layer was impermeable to HRP, whereas the mature ependyma was permeable. In hydrocephalic animal the areas where the ependyma had detached and the glial scar had not yet form were permeable to HRP, diffusing through the parenchyma. The glial scar was recognized for being GFAP positive and surprisingly, vimentin positive. When this barrier was fully developed at PN-30, it was apparently impermeable. However, the presence in the neuropile of cells labelled with HRP might indicates that some HRP has passaged through the glial scar. In adult hydrocephalic animals, there are zones where the ependyma is not denuded. This ependyma and the neighbouring glial scar appear impermeable to HRP. However, the HRP labelling of subventricular structures in these levels suggest that some tracer has passed through.

Conclusion

The different permeability properties between mature and immature ependymal layers suggest that differences exist in cell adhesion features and permeability. In hydrocephalic mice, denudated areas devoid of glial scar are very permeable to HRP. Thus, ependymal denudation implies the loss of CSF-parenchyma barrier, which could influence the CNS development. In adult hidrocephalic mice there are ependimal patches that do not detach. This particular ependyma, as the glial layer lining the denuded area, prevents partially or completely the passage of HRP. The HRP labelling of subventricular structures in this two regions could be an indication that some HRP has passed through the non-detached ependyma and through the glial sheath by an as yet unknown mechanism. This suggests that these ependymal areas could correspond to an specific ependyma population that in the normal animal

would be a tight ependyma, and that such an ependyma would have the same barrier properties as those of the glial scar. What actually are these barrier properties are being further investigated in our laboratory.

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