Title: The ependymal detach due to neuraminidase provokes hydrocephalus

Authors: María del Mar Fernández-Arjona(\*), Pablo Granados-Durán, María del Carmen Gómez-Roldán, María D López-Ávalos, Jesús M Grondona, Manuel Cifuentes, Margarita Pérez-Martín, Pedro Fernández-Llebrez

Mailing adress: Department of Cell Biology, Genetic and Physiology, IMABIS, University of Málaga, 20971 Spain.

(\*) Phone: 34 9 52 132006 Fax: 34 9 52 132000. Email: marfernandez@uma.es

Background: In many cases there seems to exist a tight relationship between the integrity of the ependymal barrier and the onset of hydrocephalus (see 1). We wanted to investigate this by means of a repetitive model that could be easily raised in the laboratory from normal rats.

Material and methods: We observed that the, easily available, enzyme neuraminidase from *Clostridum perfringens* destroys the ependyma (2). So we proved different amounts of the drug at different times and explored by means of morphological techniques, at structural and ultra-structural levels, the events occurring after the intracerebroventricular administration of the drug into the normal rat brain

Results: The administration provoked the detachment of a great part of the ciliated cubic ependyma but not other types of ependyma, particularly those attached by tight junctions, as is the notable case of the choroid plexus. The effect is dose-dependent and led to hydrocephalus when the detachment is massive and affects the brain aqueduct. The absence of ependyma marks a zone of great permeability where the immuno-responsible cells proceeding from the vessels, mainly from those venules located in the choroid plexus, easily penetrated the brain parenchyma. With time, these zones are covered by impermeable astrocytic glial scars (3, 4). We think that this could represent a good model for study the relationships between the ependymal detach and the onset of hydrocephalus.

Conclusions: The microbial neuraminidase broke the intercellular junctions of the ependymal line, non sealed by tight junctions. The dissapearance of this barrier led to an increased permeability or/and a stenosis of the cerebral acueduct that in turn provokes hydrocephalus.

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No conflict of interest to declare