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# Mechanism of obliteration of Sylvius aqueduct in the H-Tx rat

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## Background

There is strong evidence associating a dysfunction of the subcommissural organ (SCO) with the pathogenesis of fetal onset hydrocephalus. In the HTx rat, obliteration of Sylvius (SA) and dilatation of the lateral ventricles start to occur at around E18. This rat animal model of fetal onset hydrocephalus has been the subject of numerous investigations. However, the mechanism and sequence of neuropathological events leading to SA obliteration are not known. The aim of the present investigation is to clarify the role actually played by the SCO in the obliteration of SA.

## Materials and methods

The brain of normal and hydrocephalic E15, E16, E17, E18, E19, E20, E21, PN1, PN3, PN5, PN7 and PN10 H-Tx rats was processed for: (1) immunocytochemistry using antibodies against (i) the secretory proteins of the SCO (AFRU), (ii) nestin and (iii) ciliated ependyma; (2) *Limax flavus* agglutinin (LFA; affinity = sialic acid) binding; (3) transmission and scanning electron microscopy.

## Results

Up to E18, all embryos from the same litter have a patent SA. However, some of these embryos, most likely corresponding to the mutants that will develop hydrocephalus, displayed an abnormal SCO. The cephalic third and the caudal third of the SCO were strongly immunoreactive with AFRU, anti-nestin and strongly bound LFA. The middle third of the SCO did not react with AFRU and anti-nesting and LFA binding was weak.

At E18 the middle, non-secretory, third of the SCO progressively fused with the opposing region of the ventral wall of SA, resulting in the SA obliteration detected from E19 on.

## Conclusion

1. In the rostro-caudal axis, the SCO is formed by three distinct zones whose differentiation would be controlled by different genes.

2. A malformation of one of these zones precedes the obliteration of SA.

3. Such a malformation is the primary cause of SA obliteration.

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