ROLE OF THE CEREBELLAR INNER CORTICAL LAYER IN THE ONSET OF FISSURATION IN THE AVIAN EMBRYO

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During the development of the cerebellum, mechanisms as cellular proliferation in the cortex or between the cortex and the white matter (Mares and Lodin, 1970 a, b), piamater and cortex relation (Sievers et al, 1987), genetic factors (Wahlsten and Andison, 1991) and changes in the deepest part of the cerebellar cortex (Goffinet, 1983, Peña-Melían, 1986) have been hypothesised as having a role in fissuration. However, the intime mechanism of this process still remains unclear.

It is well known that during development of the cerebellum in chick (Feirabend, 1990) and cetacea (Korneliussen, 1967) there is a period when four clusters of first order Purkinje cells (PC) are observed in the inner cortical cell layer (ICCL). Each PC cluster relates with each of the four cerebellar nuclei, although the role of these clusters in the ICCL has not yet been well established. The aim of this work has been to find a relationship between the clusters of first order PC and the appearance of primary fissures (prima, praepiramidalis, secunda and sulcus uvularis-1) in the cerebellum of the avian embryo.

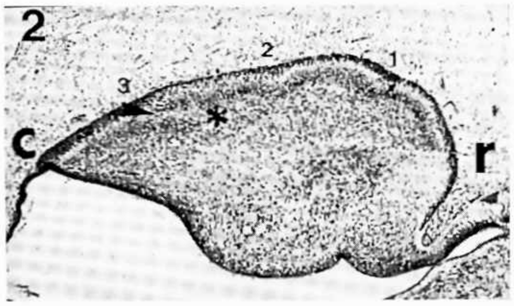

Figure 2: Parasagital section through the cerebellum of a 35+ HH chick embryo. Prima (1) fissure and the anlage of the praepiramidalis (2), secunda (arrow) and sulcus uvularis-*1* (3) fissures are observed. Asterisc indicates one gap in the ICCL. r: rostral. c: caudal. x10..

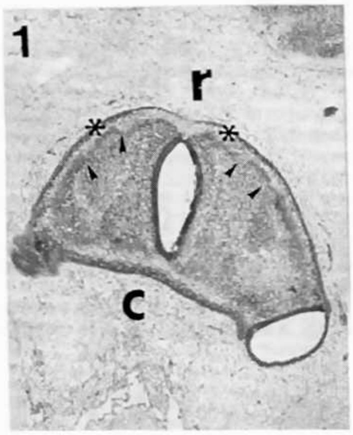

Figure 1: Transversal section through the cerebellum of a 35 HH. chick embryo. Fragmentation in the ICCL is observed (arrowheads) as well as overlaping of the fragments. r: rostral. c: caudal. *: Cluster of primary order PC. x10.

34 HH (Hamburger and Hamilton, 1951) to 37 HH chick embryos and 23 Z (Zacchei, 1961) to 26 Z quail embryos were fixed in 4 % paraformaldehyde and embedded in wax. Transversal and sagital sections were stained with Hematoxilyne-Eosyne or Bodian, dehydrated and mounted in Eukytt. Some of the sections were drawn in light chamber for reconstructions.

The cerebellar surface of 34 HH chick embryos does not show any fissures. However, ICCL is divided in several plaques (usually four) consisting of primary order PC clusters. These clusters are very close to each other, sometimes overlaping, as in the cerebellar top (Fig. 1). When fissuration starts, at 35+ HH in the avian embryo (Peña-Melián, 1986), the gaps between the ICCL plaques underly each fissure, and convergence of both two plaques is seen (Fig. 2). Once the fissures are established (36 HH in chick embryo) the cerebellum has one dorsal fissurated zone and one ventral not fissurated zone. The ICCL is a continuum in the fissurated zone, while in the not fissurated one the previously observed fissurated pattern is seen (Figs. 3 and 4). If the gaps amongst plaques are projected towards the dorsal surface, they coincide with the fissures (Fig. 4), which suggests that the appearance of the first fissures (prima, praepiramidalis, secunda and sulcus uvularis-1) occur over the space between the ICCL plaques, although they fusse afterwards. Our observations suggest that initial fissuration

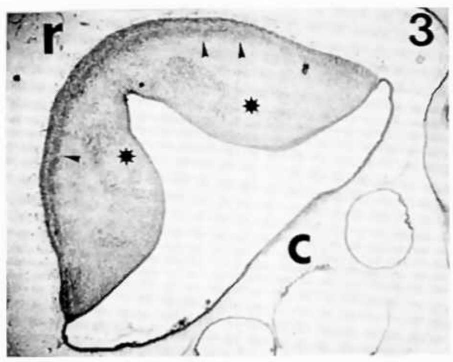

Figure 3: Ventral not fissurated part of the cerebellum of a 36 HH chick embryo. ICCL is divided in plaques by small gaps (arrowheads). *: Underlying nuclei. r: rostral. c: caudal. x10.

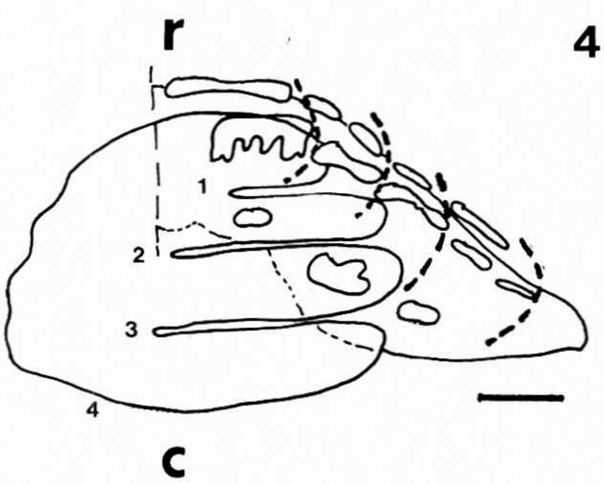

Figure 4: Overlaping drawings performed with light chamber belonging to the cerebellum of a 36+ HH chick embryo. Fissurated and not fissurated parts are overlaped. Gaps in the ICCL coincide with fissures (dotted line). 1: Prima fissure. 2: Praepiramidalis fissure. 3: Secunda fissure. 4: Sulcus uvularis-*1*. Bar: 250 micromeres.

could be due to an expansive process of the cerebellum, where cortex would be composed by more dense zones (primary order PC clusters) projecting centrifugally to form the folia, and less dense zones (spaces amongst clusters), less resistent, therefore allowing the surface to bend at such level to form the fissures. We are now studying rat and mouse cerebellum in order to know if this mechanism can be observed in mammalian embryos as well. Subsequent fissures are also being studied.

References

Feirabend, H.K.P. (1990). Development of longitudinal patterns in the cerebellum of the chicken (*Gallus domesticus*): a cytoarchitectural study on the genesis of cerebellar modules. European Journal of Morphology. 28 (2-4): 169-223.
Goffinet, A.M. (1983). The embryonic development of the cerebellum in normal and reeler mutant mice. Anatomy and Embryology. 168:73-86.
Hamburger, V., Hamilton, H.L. (1951). Series of normal stages in the development of the chick embryo. J. Morphol. 88: 49-92.
Korneliussen, H. K. (1967). Cerebellar corticogenesis in cetacea, with special reference to regional variations. J. Hirnforsch. 9: 151-185.
Mares, V., Lodin, Z., Srajer, J. (1970). The cellular kinetics of the developing mouse cerebellum. I.- The generation cycle, growth fraction and rate of proliferation of the external granular layer. Brain Res. 23: 323-342.
Mares, V., Lodin, Z. (1970). The cellular kinetiks of the developing mouse cerebellum. II.- The function of the external granular layer in the process of gyrification. Brain Res. 23: 343-352.
Peña-Melián, A.L., Puerta Fonollá, J., Gil-Loyzaga, P. (1986). The ontogeny of the cerebellar fissures in the chick embryo. Anatomy and embryology. 175: 119-128.
Sievers, J., Hartmann , D., Gude, S., Pehlemannn, F.W., Berry H. (1987). Influences of meningeal cells on the development of the brain. Nato Asi Series. 45: 171-188.
Wahlsten, D., Andison, M. (1991). Patterns of cerebellar foliation in recombinant inbred mice. Brain Res. 557: 184-189.
Zacchei, A.M. (1961). Lo sviluppo embrionale della quaglia giaponese (Coturnix coturnix japonica). Archivio di Anatomia. LXVII: 36-62.