

Parvalbumin, calbindin, and calretinin mark distinct pathways during development of monkey dorsal lateral geniculate nucleus

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Abstract

Immunocytochemical labeling was applied to follow the developmental changes in the calcium-binding proteins parvalbumin (PV), calbindin D28k (CaB), and calretinin (CaR) during fetal and infant development of Macaca monkey dorsal lateral geniculate nucleus (LGN). For all three proteins, LGN cell body and retinal ganglion cell (RGC) axon labeling patterns changed temporally and spatially over development, and many of these were LGN laminar specific. CaR+ and CaB+ cells were present at the youngest age studied, fetal day 55 (F55). After lamination of the LGN occurred between F90 and F115, CaR+ and CaB+ neurons were specific markers for the S, intercalated, and interlaminar layers. Double label immunocytochemistry showed that all CaR+ cells contained CaB, and none contained GABA. CaR+ cell bodies decreased in number soon after birth so that adult LGN contained only a very small number of CaR+ cells. These patterns and cell counts indicated that a downregulation of CaR had occurred in the CaB+ population. Although CaB+ cell density in S and interlaminar zones declined in the adult, cell counts indicated that this is due to dilution of a stable population into a much larger nucleus during development. PV+ cells appeared at F85 only within the putative magnocellular (M) and parvocellular (P) layers, and PV remained a marker for these layers throughout development. Fetal PV cells also contained GABA, indicating that they were LGN interneurons. After birth, GABA-/PV+ cell numbers increased dramatically throughout the whole nucleus so that by the end of the first year, P and M layers were filled with PV+ cells. Their number and size indicated that these were the LGN projection neurons. Beginning at F66, bundles of PV+ axons occupied the anterior-middle LGN and filled the optic tract. Up to F101, PV+ synaptic terminals were restricted to P layers, but after F132 labeling in M layers was heavier than in P layers. Axonal labeling for CaR began at F125. Prenatally CaR+ terminals were present mainly in P layers, whereas by postnatal 9 weeks labeling in M layers much exceeded P layers. Axonal labeling for CaB was present at F132, but CaB+ terminals were observed only after birth with labeling always heavier in M than P layers. By postnatal 9 weeks, PV, CaR, and CaB were colocalized in the same axons and terminals. These experiments indicated that during development and in the adult LGN, both CaR and CaB were markers for the LGN neurons in the S and intercalated pathway. CaR was present transiently while CaB persisted into adulthood. PV was a M and P layer marker first for interneurons and later for projection cells. The complex temporal

developmental patterns found in this study suggested that viewing PV, CaB, and CaR simply as calcium-buffering proteins severely underestimates their functional roles during visual system maturation. © 1996 John Wiley & Sons, Inc.