Early neural death in the neuroretina: Extent, regulation and possible functional role

Summary: Orchestrated proliferation, differentiation, and cell death contribute to the generation of the complex cytoarchitecture of the central nervous system, including that of the neuroretina. We have characterized in detail a little studied, early phase of programmed cell death affecting proliferating neuroepithelial cells and recently born neurons, in particular retinal ganglion cells. The magnitude of early neural cell death exceeded that of neuronal differentiation and, thus, the elucidation of its functional role may reveal novel aspects on neural development.

The dramatic increase in cell death found in the developing nervous system, but not in other embryonic tissues, of mice with deficient double-strand break (DSB) DNA repair may point out an intriguing possibility. These observations indicate that DSBs are generated during neural development and lead to cell death when the repairing process is defective. DSBs are repaired by homologous recombination or non-homologous end-joining (NHEJ). NHEJ is active in both cycling and post-mitotic cells. NHEJ is error-prone and might provide a molecular mechanism for neuronal diversification and somatic mosaicism. These alternatives will be discussed in relation to our observations.

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