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**Activity:**

**Calbindin and calmodulin changes in the rat hippocampus and prefrontal cortex as a function of aging**

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According to the calcium hypothesis of aging, one of the reasons for cognitive decline with advanced age is altered regulation of intracellular calcium (Ca<sup>2+</sup>). Calcium binding proteins (CaBPs) are intracellular proteins that help regulate Ca<sup>2+</sup>, and their decline with advanced age may contribute to elevated Ca<sup>2+</sup> levels. Certain CaBPs, like calbindin (CB), may also provide neuroprotection by minimizing the ability of excess Ca<sup>2+</sup> to trigger cell death cascades. Both the medial prefrontal cortex (mPFC) and the hippocampus (hpc) are involved in a variety of behavioral tasks. The mPFC is involved in tasks that require modification of existing behaviors, such as extinction of conditioned fear, which we recently showed is impaired during normal aging (Kaczorowski, et al., 2008). Tasks that require the hpc, such as trace and contextual fear conditioning, are also impaired in aged rats (Moyer & Brown, 2006). In an attempt to determine whether changes in CaBPs may contribute to observed aging-related behavioral deficits, we have begun evaluating CaBPs in these two brain regions. In the current studies, CB and calmodulin (CaM) were studied in the hpc and mPFC from adult (3 mo.), middle-aged (15 mo.), and aged (28 mo.) rats using Western blot analysis. After dissecting out dorsal (dhpc) and ventral (vhpc) portions of hpc as well as the infralimbic (IL) and prelimbic (PL) subdivisions of mPFC, tissues were homogenized, normalized for total protein, subjected to SDS-PAGE, transferred to membranes, incubated with anti-CB and anti-CaM primary antibodies, and proteins were visualized using secondary antibodies. After development, the blots were analyzed using Image J. Within each brain region, the aged bands were compared to their corresponding adult control bands. Results suggest a decrease in CB and CaM protein expression in IL (aged CB: 23% decrease; aged CaM: 34% decrease) but only a decrease in CB expression in PL (aged CB: 22% decrease; aged CaM: 7% decrease). These region-specific changes in mPFC may contribute to our observed aging-related extinction deficits. Within the hpc, results suggest a selective decrease in CaM, but not CB protein expression in vhpc (aged CB: 11% decrease; aged CaM: 32% decrease) and a decrease in CB, but not CaM expression within dhpc (aged CB: 37% decrease; aged CaM: 1% decrease). These CaBP changes in hpc may be associated with age-related deficits in trace fear conditioning. These data suggest that targeting CaBPs may be a useful approach for developing novel therapeutics for protecting neurons against aging-related neurodegenerative disorders.

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