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Subcommissural material and ventricular area changes in cirrhosis

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Hepatic encephalopathy (HE) is a neuropsychiatric complication of both acute and chronic liver failure. Whether brain structures with strategic positions in the interface of blood-brain barriers such as the circumventricular organs (CVO) play a role in HE is not yet established. Among the CVO, the subcommissural organ (SCO) secretes a glycoprotein called Reissner's fiber (RF) which condenses and forms an ever-growing thread-like structure into the cerebrospinal fluid (CSF). Here, we describe RF material within the SCO and its serotonin (5-hydroxytryptamine; 5-HT)-ergic innervation in a model of minimal HE following bile duct ligation (BDL) in rats using immunohistochemistry with antibodies against RF and 5-HT. 4 weeks after BDL surgery, we observed a significant rise of RF immunoreactivity in SCO areas compared with sham-operated controls. Moreover, we saw significant RF-immunoreactive materials within the ependyma and inside the parenchyma of the ventricular borders in BDL rats, but not in sham-operated controls. Increased RF material in cirrhotic rats is probably related to reduce 5-HT innervation found in BDL rats compared to sham-operated controls. Reduced 5-HT innervation of ependymal structures reflects probably a general 5-HT system deficit in BDL rats where 5-HT-immunoreactive neurons were significantly reduced within the nucleus of origin at the dorsal raphe nucleus of BDL rats compared with shams. Reduced 5-HT innervations of these ependymal structures in brain of BDL rats are the consequences of brain accumulation of neurotoxins such as ammonia and cytokines. We suggest that increased RF material may have neuroprotective, neurotransmitter recycling, and/or water homeostasis consequences in BDL rats.

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Cathepsin D mediates apoptosis during anuran tail regression

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Apoptosis is a genetically regulated form of cell death which has a role in embryogenesis, ageing, and many diseases. Many existing treatments like anti-cancer treatments act through apoptosis. New treatments are also being developed to modify apoptosis and can be used to manage common diseases. Anuran tail regression is a prominent example of programmed cell death mediated by apoptosis. Anuran tail thus acts as an efficient model to study apoptosis, since tail resorption during anuran metamorphosis is exclusively mediated by apoptosis. The tail undergoes complete resorption during metamorphosis to adapt to terrestrial mode of life in the adult phase of life. Lysosomal enzymes play pivotal role in degrading the tail tissues where they mainly degrade the cellular debris taken up by macrophages after initial death of cells under the influence of thyroid hormone. However, recent studies on tropical anurans have showed that lysosomal cathepsins especially, cathepsin D acts much earlier in the cell death process rather than only clearing cellular debris. Immunohistochemical localization of cathepsin D showed that it localized to apoptotic epidermal and muscle cells. Besides, cathepsin D also localized to degenerating blood cells, spinal cord and notochordal cells. Melanocytes, a source of lysosomal enzymes, were found in large numbers near the degrading tail tissues. Thus, cathepsin D seems to cause the initial cell death of the tail tissues which is followed by clearing of the cellular debris later on by melanocytes. Further studies should focus on the downstream effectors activated by cathepsin D which cause the initial death of the tail tissues and the exact role of melanocytes in causing degradation of tail tissues.

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