

PAIN AND DEPRESSION – COMMON PSYCHONEUROIMMUNOLOGICAL PATHOMECHANISMS

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The association with pain and depression is long known and demonstrated in large cohorts to involve about 60-80% of patients with severe depression. However, the relevant pathophysiological link appears not well known.

A review about available ideas and findings with regard to the association of pain and depression is given including an evaluation about the plausibility to explain the anatomical distribution of symptoms, based on both, clinical and experimental, approaches.

Pain can relate to stress, in this case likely explained by a body brain connection in between the autonomic and the endocrine system. To differentiate central from peripheral pain is very difficult as finally a central involvement is always needed. Pain in experimental autoimmune encephalitis (and in Multiple Sclerosis) is very frequent but its pathogenesis is poorly understood. Mast cell degranulation can activate various pain pathways of pain centres in the cervical or lumbar

spinal cord and explain the localization of widespread tactile pain hypersensitivity. CSF was in one hypothesis put forward to play an underestimated role as a pathogenic link in the framework of the volume transmission mode involving extracellular CNS fluid and CSF. CSF signalling may even extend along the peripheral CSF outflow pathway into peripheral tissues. Low level neuroinflammation likely is involved in the pathogenesis of a subgroup of depression, but it is not clear whether these cases correlate with the subgroup suffering from chronic pain.

The parallelism of pain and depression in large subgroups is compatible with various pathogenetic models but especially with a neuroimmune-inflammatory framework.

Bechter K., *The peripheral cerebrospinal fluid outflow pathway - physiology and pathophysiology of CSF recirculation: a review and hypothesis*. Neurol Psychiatry Brain Res, 2011. 17(3): p. 51-66