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Default Mode Network in Post-stroke Depression

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Post-stroke depression (PSD) is the most serious emotional disorder following stroke, affecting one-third of all stroke survivors. Depression is associated with poor short-term recovery and long-term rehabilitation outcomes, excess disability, cognitive impairment, and mortality in stroke survivors. The neuroanatomical model of PSD remains unclear despite decade's research. A new model proposes that grey and white matter lesions/ischemia in PSD disrupts the brain's affective regulation network, leading to hyperactivation of the limbic system and subsequent depressive symptoms. The default mode network (DMN) is such a network that comprises brain regions evolving in emotion regulation: ventromedial prefrontal cortex, ventral anterior cingulate cortex, the posterior cingulate, precuneus, inferior lateral parietal lobes, and parts of the temporal lobe. These brain regions present synchronic activation when the individual is in a state of wakeful rest and deactivation when attentive to the outside world. Thus the temporal correlation between the BOLD signal in DMN brain regions (measured by functional connectivity magnetic resonance imaging, FCMRI) at rest is thought to reflect important interrelationships among their structures with related functions.

The DMN theory of depression is proposed by Northoff et al. in 2011. According to the theory, depressive phenomenology is associated with specific subcortico-cortical systems, and neural hyperactivity during rest is one of the endophenotype for unipolar mood disorder.

Many evidences support this theory. For instance, abnormal resting DMN FC contributes to the patho-physiology of mid-life and late-life depression. Increased FC in anterior regions but reduced FC in posterior regions of the DMN has been found in mid-life depression. DMN hyperactivity is related to various depressive symptoms and psychopathology, including negative rumination, overgeneral autobiographical memory, negativity bias, sustained negative mood, and hopelessness.

FC abnormalities happen in stroke. Local structural damage due to stroke can result in dysfunction in remote regions connected to the lesion. It is plausible that grey and white matter lesions/ischemia in PSD disrupts the brain mechanisms of affective regulation, thus leading to limbic hyperactivation and subsequent depressive symptoms. In fact, in one study, yet so far, DMN FC at 10-days poststroke has been associated with depressive symptoms in 24 stroke survivors at 3-month follow up.

PSD is a complex disorder, and a host of imaging and non-imaging factors contribute to its development. This may partly account for the contrary findings yielding by substantial studies about the relationship between lesion location and PSD. The study on DMN FC have shed light on underlie neuroanatomical mechanisms of PSD. Study on brain network functional connectivity including DMN FC in PSD is in incipient stage and plenty of work is needed to clarify the role of brain functional connectivity in development, treatment response and outcome of PSD.

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