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What is the evidence from functional magnetic resonance imaging (fMRI) studies on schizophrenia?

Moderate quality evidence suggests decreased local organisation and small-worldness (balance of local organisation and global integration) in people with schizophrenia compared to controls. There was reduced connectivity within the default network (self-related thought), the affective network (emotion processing), the ventral attention network (processing of salience), the thalamus network (gating information) and the somatosensory network (sensory and auditory perception). There was reduced connectivity between the ventral attention network and the thalamus network, the ventral attention network and the default network, the ventral attention network and the frontoparietal network (external goal-directed regulation), the frontoparietal network and the thalamus network, and the frontoparietal network and the default network. There was increased connectivity between the affective network and the ventral attention network.

During executive functioning and working memory tasks, there was decreased activation in the frontal lobe, including the dorsolateral prefrontal cortex, and in neocortical regions, including the parietal and occipital cortices and bilateral caudate, fusiform gyrus, and cerebellum, and in subcortical regions, including the right putamen, hippocampus and left mediodorsal thalamus. Moderate to low quality evidence suggests significant increases in functional activation in the anterior cingulate cortex, temporal lobe, parietal cortex, lingual gyri, insula and the amygdala. During cognitive control tasks, there was decreased activation in the bilateral anterior cingulate/paracingulate gyrus, left inferior parietal gyrus, right middle/inferior frontal gyrus, bilateral middle frontal gyrus, right thalamus, and left cerebellum. There was increased activation in the right middle occipital and bilateral precentral gyri. During timing tasks, there was decreased activation in the bilateral caudate nuclei, left middle occipital gyrus, right inferior occipital gyrus, bilateral supplementary motor area, and right putamen. There was increased activation during timing tasks in bilateral superior parietal gyri, right inferior frontal gyrus, and right middle temporal gyrus. During memory encoding tasks, there was decreased activation in the medial frontal gyri and the hippocampus. During memory retrieval tasks, decreased activation was seen in the medial and inferior frontal gyri, the cerebellum, hippocampus, and the fusiform gyrus, with increases in the anterior cingulate cortex and the medial temporal gyrus. During episodic memory encoding, there was decreased activation in the right superior frontal gyrus, bilateral inferior frontal gyri, right inferior parietal gyrus, right lingual gyrus, left hippocampus, and right posterior cingulate. There was increased activation in the left precentral gyrus, left middle temporal gyrus, left post-central gyrus, left cingulate and left parahippocampal gyrus. During episodic memory retrieval, there was decreased activation in the left inferior frontal gyrus, left middle frontal gyrus, right cuneus, right cingulate gyrus, bilateral thalamus, and bilateral cerebellum. There was increased activation in the left precentral gyrus, right middle frontal gyrus, right thalamus and right parahippocampal gyrus. During emotion processing tasks, there was decreased activation in the parahippocampus, superior frontal gyrus, middle occipital gyrus, fusiform gyrus, lentiform nucleus, and thalamus. There was increased activation in left amygdala, left hippocampus, left medial frontal region, left cuneus, and bilateral parietal cortex. During explicit threat processing, there was decreased activation in the inferior frontal gyrus, right cerebellum lobule VI, left fusiform gyrus, and thalamus, and increased activation in the medial prefrontal gyrus to superior prefrontal gyrus. During implicit threat processing, there was decreased activation in bilateral amygdala extending into putamen, hippocampus and parahippocampal gyrus, and fusiform gyrus extending into the cerebellum lobule IV/VI. During theory of mind tasks, there was decreased activation in the left middle temporal gyrus, medial prefrontal cortex (frontal medial and paracingulate), right premotor cortex (central opercular, postcentral, precentral), medial occipitoparietal, right lingual gyrus, left orbitofrontal cortex, left lateral occipitotemporal, and left cingulate gyrus. There was increased activation in the left inferior parietal cortex and right inferior parietal cortex. During empathy tasks, there was decreased activation the right inferior frontal gyrus. During inhibition tasks, there was decreased activation in the anterior and middle cingulate cortex, and increased activation in parietal and occipital regions. There was also decreased activation in the basal ganglia and inferior frontal cortex, and increased activation in the superior temporal gyrus during inhibition tasks. During attention tasks, there was decreased activation in the anterior and middle cingulate cortex and the basal ganglia, and increased activation in the left supramarginal gyrus. During linguistic tasks (mostly semantic reading), there was decreased activation in the lateral temporal regions and left putamen, and increased activation in bilateral frontal cortex and left putamen. During reward stimuli tasks, there was decreased activation in the right ventral striatum.

During auditory hallucinations, there was increased activation in Broca's area of the temporal lobe, insula, hippocampus, left parietal operculum, left and right postcentral gyrus, and left inferior frontal gyrus, and decreased activation of Broca's area, the left middle temporal gyrus, left premotor cortex, anterior cingulate cortex, and left superior temporal gyrus. In people with schizophrenia and formal thought disorder, moderate quality evidence found functional alterations (hyperactivation or hypoactivation) in the left superior and middle temporal gyrus.

For more information see the technical table



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NeuRA
(Neuroscience Research Australia) is one of the largest independent medical and clinical research institutes in Australia and an international leader in neurological research.

Diseases of the brain and nervous system pose the greatest health, economic and social burden of any disease group because they are chronic, debilitating and have no known cures.

Medical research is the cornerstone of efforts to advance the health and wellbeing of families and the community. Our dedicated scientists are focussed on transforming their research into significant and practical benefits for all patients.

While we hope you find this information useful, it is always important to discuss any questions about schizophrenia or its treatment with your doctor or other health care provider.

HOW YOUR SUPPORT HELPS

We are able to make significant advances due to the generosity of countless people. Your donation allows us to continue to work towards transforming lives. For information on how you can support our research, phone **1300 888 019** or make a secure donation at neura.edu.au/donate/schizophrenia.

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