

Neuroimaging in Psychiatry

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KEYWORDS

- Biological psychiatry • Magnetic resonance imaging
- Radionuclide imaging • Tomography
- Substance related disorders • Anxiety disorders
- Mood disorders • Cognitive disorders
- Psychotic disorders

Psychiatry, the last phenomenologic medical specialty, is quickly entering an era of pathophysiologic understanding. Neuroimaging has been fundamental to this revolution of knowledge. It is not within the scope or purpose of this article to provide an exhaustive review of the data, rather, to give readers an overview of the major findings and areas of promise in psychiatric neuroimaging.

This article is organized by diagnostic categories: addictive disorders, anxiety disorders, cognitive disorders, mood disorders, and psychotic disorders. The category of miscellaneous has been added for those disorders that seem to defy classification or cases where there is only a limited amount of data to present. Within each section, structural and functional neuroimaging data are laid out. To the extent possible or reasonable, an attempt is made to extrapolate from this data an understanding of how the brain works.

ADDICTIVE DISORDERS

Neuroimaging in addictive disorders requires other considerations besides the usual structural versus functional issues. There are changes that are considered an inherent part of the disease process (trait markers)—those that correlate to the different stages of the addictive process, such as intoxication, tolerance, withdrawal, and craving (state markers), and those that are sequelae of abuse. The most common comorbidity for someone who has an addiction is another addiction, thus increasing the risk for confounders or, at the least, adding further categories to be explored.¹ There now are considerable data supporting the inclusion of several types of impulse control problems as behavioral addictions, as is the case with excessive gambling, Internet

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use, shopping, and sex. In addition, certain substances of abuse can be considered representatives of their class, such as alcohol with sedatives and cocaine or nicotine with stimulants. This section reviews only the data pertaining to alcohol, behavioral discontrol, and cocaine.

It has been known for some time that alcohol-dependent patients have smaller brain volumes.² Magnetic resonance spectrography has been used to show that before brain volume changes occur in young alcohol-dependent subjects, there are significant alterations in cerebral metabolites.³ Specifically, lower choline and creatine levels are shown in the cingulate and an increased ratio of glutamate to creatine.

Using T1-weighted MRI, researchers have shown smaller brain volumes in family history–positive patients who had alcohol dependence regardless of whether or not they had early-onset or late-onset disease.⁴ This suggests smaller premorbid brain growth but does not answer whether or not this is the result of the in utero environment or an actual trait marker. Other researchers have used CT to show significant cortical and cerebellar atrophy in alcohol-dependent patients.⁵ Cerebellar atrophy is associated with daily ethanol consumption. Relationships with liver function, cytokines, nutritional status, and hormone levels, however, are poor.

By using deformation-based morphometric MRI, studies have demonstrated those patients able to maintain abstinence had significant tissue volume recovery in the frontal, parietal, and temporal lobes and in the thalamus, brainstem, corpus callosum, anterior cingulate, insula, and subcortical white matter.⁶ Findings for light drinkers were less pronounced. Gray matter volume at baseline predicted volume gains during abstinence.

Among the behavioral addictions, pathologic gambling has been explored with functional imaging. At this time, the structural data on this subject are largely silent. The topic is nascent and likely will receive greater diagnostic clarity in a future edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR)*.⁷ Current studies involve normal subjects and risk-taking behavior.

The phenomenon of chasing losses came under study with functional MRI in healthy adults.⁸ Activity within the cingulate cortex was associated with chasing losses, not unexpected for an area that modulates incentive motivation and expectation of reward. Conversely, the decision to stop gambling was associated with increased activation in areas that involve anxiety and conflict monitoring and decreased activation of the cingulate. Functional MRI also has been used to show that the cingulate cortex and surrounding areas are involved not only in processing the perceived likelihood of an error but also the predicted magnitude of the consequences.⁹ Dysfunction in this area may serve in part to explain the irrational nature of the addictive process.

Cocaine dependence and abuse have been the source of extensive evaluation. Volumetric MRI has shown that children who had intrauterine cocaine exposure had lower mean cortical gray matter, total parenchymal volumes, and mean head circumference than comparison children.¹⁰ This problem was compounded by concomitant exposure to alcohol, tobacco, and marijuana with the greatest severity seen in those exposed to all four substances.

Perfusion functional MRI has been used to demonstrate reduced global cerebral blood flow in adolescents exposed to cocaine in utero.¹¹ This decrease was noted mainly in the posterior and inferior brain regions, including the occipital cortex and thalamus. After adjusting for global cerebral blood flow, areas with significantly increased relative cerebral blood flow were found, including the prefrontal, cingulate, insular, amygdala, and superior parietal cortex. It has been suggested that these relative increases in anterior and superior brain regions represent compensatory mechanisms.

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