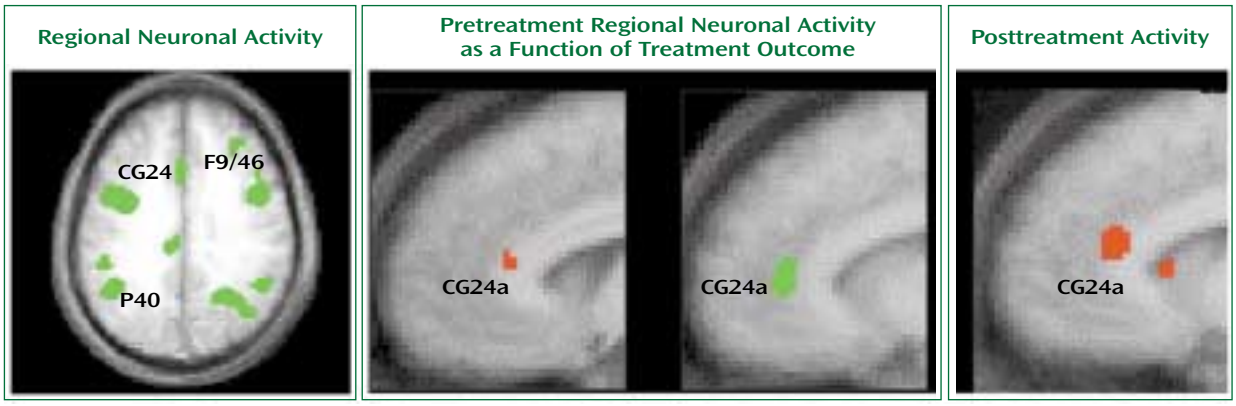
Depression, II



Regions of hypometabolism are shown in green; regions of greater glucose metabolic activity are shown in red. The image on the far left indicates regions of hypometabolism in depressed patients relative to healthy comparison subjects. The other three images depict pretreatment glucose metabolic activity in the rostral cingulate cortex (CG24a) in depressed subjects (relative to healthy subjects) who eventually (from left to right) responded to treatment, did not respond to treatment, and in the state of full remission.

Localization of Pathophysiology

In depression, neuronal activity patterns change in several regions in the human brain. These changes mark the regions that are putatively involved in the expression of the symptoms of depression or the maintenance of the mood disorder. The image on the far left shows some of these regions and is a comparison of neuronal activity (measured by using positron emission tomography with fluro-deoxyglucose) between volunteers with unipolar depression and healthy comparison subjects. The regions of hypometabolism in the depressed patients include the dorsal and ventral prefrontal cortex (F9/46), the inferior parietal region (P40), and the anterior cingulate cortex (CG24) as well as the anterior insula and posterior cingulate (not shown). There is an overlap of abnormalities in these regions in persons with unipolar depression and those with depression and Parkinson's disease, suggesting that the mood change itself, not the overall diagnosis, accounts for the change in activity patterns. Since mental functions subserved by these areas are adversely affected in actively depressed persons (e.g., seen in altered cognitive performance), these observations hold face validity in addition to their experimental demonstration.

Beyond these findings, we were interested in identifying brain regions that would vary with treatment response and eventual depression remission. Thus, we analyzed pretreatment regional neuronal activity as a function of treatment outcome. As displayed in the other three images, a discrete area in the rostral cingulate cortex (CG24a) predicted eventual treatment response. In those who responded, glucose metabolic activity in this area was high; in nonresponders, it was low, measured prospectively. In persons in full remission, increased activity in this region persisted, suggesting a central role for this region in relation to the normalization of cortical and paralimbic dysfunction that accompanies recovery from depression. The presence of this metabolic signature may prove useful in identifying those at risk for a nonresponding disease course.

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