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How Does Psychotherapy Change The Brain: What Neuroimaging Has Taught Us About Psychotherapy

In an era of renewed focus on mindbrain relationships, we're closer to answering this question than ever before. The past few years have seen dramatic growth in studies using functional neuroimaging to investigate the effects of psychotherapy on brain function. These studies have suggested that psychotherapy causes plausible changes in brain activity, and that these changes overlap partially – but not completely – with comparable medication interventions.

The psychotherapy interventions most frequently studied with imaging thus far have been behavioral therapy (BT) or cognitive-behavioral therapy (CBT). Baxter and colleagues at UCLA studied 18 patients who underwent a 10-week course of BT for obsessive compulsive disorder (OCD). Similar to patients who had responded favorably to treatment with fluoxetine, patients who received BT exhibited a reduction in metabolic activity in the caudate nucleus. Moreover, a correlation was found between the extent of metabolic change in the caudate and the degree of clinical improvement. Given the established role of the caudate in OCD, these findings were not surprising.

In a related study, Brody and associates found that pre-treatment metabolism in the left orbitofrontal cortex (which may act to extinguish habitual, compulsive responses) positively correlated with response to BT in 18 patients with OCD. Interestingly, among a comparison group of patients who received fluoxetine, a significant negative correlation was found between clinical response and left orbitofrontal metabolism. These findings suggest that pre-treatment imaging might ultimately be useful to predict whether certain patients will respond better to pharmacotherapy or CBT.

The use of CBT as a treatment for phobic disorders has also been found to influence brain function. Of particular interest in these studies is the effect of treatment on activity in the limbic system, which has long been associated with the neurobiology of fear. Furmark and colleagues found that CBT for social phobia induced significant reductions of activity in the amygdala, hippocampus, and adjacent temporal cortex in 6 patients who were scanned during an anxiety-provoking performance paradigm. Similarly, Paquette and associates found that group CBT diminished activation in the parahippocampal gyrus and dorsolateral prefrontal cortex in 12 arachnophobic patients exposed to pictures of spiders. These investigators linked the abatement of the parahippocampal gyrus response to a dampening of contextual memory (which is believed to be mediated by this structure). They further suggested that the prefrontal cortex changes might reflect the restructuring of conscious cognitive defenses – in other words, after CBT, the prefrontal cortex may need to work "less hard" in the presence of a fear-inducing stimulus(e.g., to plan an escape from perceived danger).

Neuroimaging studies have also been used to examine the effects of psychotherapy on the brain in depressed patients. Remarkably, studies looking at two different therapy modalities, CBT and interpersonal therapy (IPT), have indicated quite similar patterns of change with respect to prefrontal cortical metabolism following therapy. In a recent study of 17 medication-free patients with depression, Goldapple and associates found that CBT reduced metabolism in multiple frontal regions, including the dorsolateral prefrontal cortex. Again, the authors suggested

that CBT alters the prefrontal cortex by allowing it to work “less hard” on active rethinking and reappraisal of emotional ideas, akin to diminishing rumination. Brody and colleagues obtained analogous results looking at IPT in 14 patients with depression, linking IPT with decreased metabolism in the prefrontal cortex bilaterally.

Why might cognitive and interpersonal therapies affect the brain in similar ways, despite significant differences in theoretical approach? A recent comparison of CBT and IPT, conducted by Stuart Ablon and his colleagues, suggested that despite the differing “brand names”, remarkable overlap in therapeutic process and technique occurred between these two modalities. It is tempting to relate this phenomenon to the idea that both CBT and IPT engage similar affective circuitry, as the neuroimaging studies have thus far suggested. However, to fully evaluate this idea would necessitate, at the least, several intermediate scans during treatment, and ideally, real-time imaging during actual therapy sessions. The logistical, technical, and financial limitations of PET and functional MRI scanning preclude this kind of work at present.

The potential use of next-generation, non-invasive neuroimaging tools (such as optical imaging, which uses nearinfrared light to measure cortical blood flow) would provide an ideal means to study the longitudinal effects of psychotherapy on brain function. Additional work in this area will further demystify and validate psychotherapy in the eyes of patients and clinicians alike. Functional neuroimaging also offers the possibility of improving clinical outcomes in two ways: first, by helping to inform treatment selection, and second, by providing an enhanced vocabulary for discussing psychological and therapeutic concepts central to psychotherapy. The added perspective of functional brain imaging, when used to its full potential, may thus strengthen the credibility and utility of a time-honored approach to psychiatric treatment.

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References listed below.

Ablon JS, Jones EE. Am J Psychiatry. 2002;159:775-83.

Baxter LR Jr, Schwartz JM, Bergman KS, et al. Arch Gen Psychiatry.1992;49:681-9.

Brody AL, Saxena S, Schwartz JM, et al. Psychiatry. Res 1998;84:1-.

Brody AL, Saxena S, Stoessel P, et al. Arch Gen Psychiatry. 2001;58:631-40.

Furmark T, Tillfors M, Marteinsdottir I, et al. Arch Gen Psychiatry. 2002;59:425-33.

Goldapple K, Segal Z, Garson C, et al. Arch Gen Psychiatry. 2004;61:34-41.

Paquette V, Levesque J, Mensour B, et al. Neuroimage. 2003;18:401-9.

Schwartz JM, Stoessel PW, Baxter LR Jr, et al. Arch Gen Psychiatry. 1996;53:109-13.