Neurobiological changes resulting from psychotherapy

Psychology



The effects of psychotherapy and the tools related to its effect have typically been investigated by measuring changes in symptoms, psychological abilities, personality, and social functioning. Many psychiatrists presumed that psychotherapy treated psychological based disorders and pharmacology treated biological based disorders. However, with the introduction of neuroimaging procedures the ability to examine the biological effects of psychotherapy has become attainable. Neuroimaging has allowed the effectiveness of psychotherapy to be documented, tracked, and further developed for application purposes. Beyond its practical uses integrating neurobiology with psychotherapy permits linking specific mental functions with specific brain structures and functions. The advances in non-invasive brain imaging techniques has increased the number of studies investigating cognitive functioning, emotional experience, placebo effects, and psychotherapeutic related changes. Positron emission tomography (PET) measures the metabolic activity within different regions and functional magnetic resonance imaging (fMRI) measures blood oxygenation of vessels within the surrounding neural tissues. By comparing brain metabolic activity in individuals with and without psychiatric disorders, it is possible to identify how the functional neural circuitry is possible (Jokic-Begic, 2010). During the 1970s, the adult brain was considered to be fixed and organized; each brain region dominated particular functions. Research has shown in the last 30 years that growth and variation in the brain are not only determined by genetics, but also environment interaction. The existence of a gene does not mean that it is active. There are many variables that determine whether a gene will express itself and its psychological impact. For example,

predisposition to post-traumatic stress disorder (PTSD) is related to whether certain stress genes are active or inactive during a traumatic event. A person whose genotype has variations of the stress related gene FKBP5 are more likely to develop PTSD following a traumatic event than a person that does not carry these variations (Feinstein & Church, 2010). The Center of Disease Control and Prevention (CDC) conducted a study that identified 1, 058 genes involved in networks between the nervous, endocrine, and immune systems and whose expression can be assessed with clinical test. Inflated limbic system responses to harmless stimuli, misrepresentations in learning and memory, imbalances between sympathetic and parasympathetic nervous system activity, elevated levels of cortisol and other stress hormones, and impaired immune functioning are biological markers that depend upon gene expression. In order for psychotherapy to be successful, genes must be modified to create positive impacts in these areas of biological functioning (Feinstein & Church, 2010). Most stress-related hormonal responses occur in the hypothalamus. The prefrontal cortex, hippocampus, and amygdala monitor the hypothalamus which links these brain structures to a person's overall perception of stress. When a person is experiencing a threat, the amygdala is activated stimulating the flight-or-flight response controlled by the hypothalamus. The hippocampus and prefrontal cortex have opposite effects on the hypothalamus; both structures inhibit the sympathetic nervous system reducing anxious behavior. If the homeostatic balance between the parasympathetic nervous system (PNS) and sympathetic nervous system (SNS) are not maintained the ability for the individual to manage threats effectively and avoid clinical levels of anxiety and depression are lessened.

Prolonged stress suppresses the PNS and increases activity in the SNS. High sympathetic/ low parasympathetic ratios are linked to psychological and physiological disorders (Sharpley, 2010). Dramatic elevations of cortisol in the bloodstream are a major contributor to the efficiency and balance of prefrontal cortex, hippocampus, and prefrontal cortex. Cortisol is a steroid hormone produced by the adrenal cortex. Nearly all cells carry receptor sites for cortisol and influence homeostatic regulation and anti-inflammatory mechanisms. Elevated cortisol levels during periods of acute stress causes increased energy, immunity, memory capabilities, and pain tolerance. However, during periods of prolonged stress impaired cognitive performance, increased blood pressure, decreased bone density, lowered immunity, suppressed thyroid function, and compromised muscle tissue occur (Feinstein & Church, 2010). During stressful situations, the prefrontal cortex is dominated by the amygdala; the amygdala activates stress pathways in the hypothalamus and brainstem increasing the levels of norepinephrine and dopamine. With the increase of these hormones, fear conditioned circuits are reinforced which heighten emotion-based decision making. Psychotherapy has been known to reduce stress which, in turn, may stimulate the expression of genes that regulate the adrenal cortex (Sharpley, 2010). From the neurobiological perspective, different brain regions are responsible for emotion regulation. Generation and regulation involves bottom-up and top-down processing. Bottom-up processing is automatic, effortless, and implicit. Research studies evaluating emotion have associated bottom-up processing with the amygdala through the use of fMRI. Bottom-up processing is characterized by relevant stimuli, situational cues, and their

schematic associations. Research shows that refocusing, explicit evaluation, and cognitive processing of emotional experiences indicate a stronger amygdalous response, emotional reaction, and physical changes. Top-down processing is slow, thoughtful, and explicit. Top-down processing is guided by rule-based knowledge when processing information. The orbitofrontal cortex, ventromedial prefrontal cortex, and anterior cingulate cortex are the brain regions associated with top-down processing. Neuroscience indicates that top down regulation leads to reduced emotional response and regulation of negative emotions. Decreased activity in the amygdalohippocampol subcortical region (bottom-up processing) and increased activation in the frontal cortical regions (top-down processing) has been shown as a result of CBT (Fuchs, 2004). Research results related to the two types of processing contribute to the understanding of why psychotherapy is more successful than pharmacotherapy. Pharmacotherapy stimulates subcortical transmitter metabolism which alters the bottom-up regulation and changes. Conversely, psychotherapy alters a patient's essential personal patterns and attitudes which occur through top-down regulation. Drug treatment affects the limbic subcortical regions whereas CBT influence the medial frontal and cingulate cortex; this explains why CBT patients have a lower relapse rate, whether or not combined with medications. Without the changes in top-down regulation, there can be no permanent changes in behavior patterns (Jokic-Begic, 2010). The primary assumption of cognitive behavioral therapy (CBT) is that people learn throughout their entire lifespan. Because functional and dysfunctional behavior is learned, every behavior learned can be unlearned and replaced with one more functional. Patients of CBT learn and implement new

knowledge and skills. The new tools that the patient develops will allow them to perceive and adjust their thoughts, behaviors, and emotional states. Techniques used during CBT include: refocusing, cognitive reconstruction, problem-solving, and other techniques that that improve unpleasant emotional states (Jokic-Begic, 2010). In 1992, researchers performed neuroimaging analysis of neural effects of CBT on obsessive-compulsive disorder (OCD). Their findings were confirmed and improved 4 years later by another research team. In 2003, research by Japanese researchers also confirmed the previous research conducted. The treatment consisted of 4 stages. The first stage involved the patient relabeling disturbing thoughts and impulses as symptoms of the disorder. In the second stage, the patient reattributed the troublesome and stubborn symptoms to false messages coming from a dysfunctional brain. The third stage requires the refocusing of behavioral responses. The fourth stage encourages the patient to revalue the disturbing thoughts and impulses with much less importance. With these techniques, a defined difference between self-experience and OCD symptoms occur increasing the patient's feelings of control. Approximately, 80% of clients experienced a reduction in symptoms. PET scans before treatment showed significant brain activity in the right hemisphere between and where significantly reduced after therapy (Ventura et al. 2009). A study in 2004 examined the effectiveness of CBT on depression using neuroimaging. During therapy sessions, patients were taught a variety of behavioral and cognitive strategies that oppose negative mood and automatic reactions to negativity. Patients were asked to increase their attendance of enjoyable events. Patients were asked to document automatic

negative thoughts. Patients were given the Hamilton Depression Rating Scale (HAM-D) and PET scans before and after therapy. All patients exhibited a substantial reduction of depression symptoms according to the HAM-D. An increase in metabolic activity within the hippocampus/parahippocampal gyrus and dorsal cingulate gyrus were found in pre-treatment versus posttreatment CBT. A decrease in the dorsolateral and ventrolateral prefrontal regions, orbital frontal regions, posterior cingulate, inferior parietal regions, and inferior temporal regions were found. Similar results were confirmed in 2008 by researchers using fMRI (Ventura et al, 2009). Neuroimaging may allow patients to be grouped by particular biological factors that cause the disease. For example, it is likely that depression has several origins that may be able to be explored clinically. Therapy may be suggested on the functional characteristics of the brain as affected by the illness rather than a diagnosis. Neuroimaging of brain activity during disorder related and unrelated task in a standardized way will provide information on how a patient will process and react to stimuli presented in psychotherapy or pharmacological treatment (Kandel et al, 2005). The relationship between neuroscience and psychotherapy seems promising but there are some limitations. Neuroimaging does not show the real brain in action but scientific constructs. It is not certain that clinically important observable occurrence resemble the color images shown through neuroimaging. Also, the static view of the brain produced does not show the dynamic mechanisms of the brain. It is hard to imagine the brain as constant object because there is continuous interaction with the organism and its environment (Kandel et al, 2005). From the research conducted, it is evident that psychotherapy can

result in measurable changes in the brain. Even though, there are some limitations with advancement in technology and updated information on disorders the two fields will continue to connect and fill in the gaps. The postpsychotherapy changes that are detected can be intensively systematically explored. New biological insights into predispositions may provide opportunities for preventative therapy. With the continuous development of neuroscience, time can be devoted to individualized therapeutic choice, patient's progress, and recovery and relapse prediction. References Feinstein, D & Church, D. (2010). Modulating gene expression through psychotherapy: The contribution of noninvasive somatic interventions. Review of General Psychology, 14, 283-295. Doi: 10. 1037/a0021252 Fuchs, T. (2004). Neurobiology and psychotherapy: An emerging dialogue. Curr Opin Psychiatry, 17, 479-485. Jokic-Begic, N. (2010). Cognitive-behavioral therapy and neuroscience: Towards Closer Intergration. Psychological Topics, 2, 235-254. Kandel, E, Polan, H, Pittenger, C, Phil, M, & Etkin, A. (2005). Toward a neurobiology of psychotherapy: Basic science and clinical application. I Neuropsychiatry Clinical Neuroscience, 17, 145-158. Sharpley, C. (2010). A review of the neurobiological effects of psychotherapy for depression. Psychotherapy Theory, Research, Practice and Training, 47, 603-615. Ventura, P, Figueira, I, Mari, J, Oliveira, L, & Ribeiro, P. (2009). Does coginitive behavioral therapy change the brain? A systematic review of neuroimaging in anxiety disorders. I Neuropsychiatry Clinical Neuroscience, 21.