

Table 1. Findings from neuroimaging studies that used activation paradigms to study the effects of psychodynamic psychotherapy

First author (year)	Clinical condition (n)	Neuroimaging method and activation paradigm	Baseline differences relative to healthy controls
Buchheim (2012)	Depression (16) HC (17)	fMRI (metabolic activity) Emotional stimuli: attachment-related scenes	Patients showed overactivation in regions of amygdala/hippocampus, dorsal prefrontal areas, and ventral cingulate region (BA25)
De Greck (2011)	Somatoform Disorder (15) HC (20)	fMRI Cognitive reward paradigms: anticipation of reward versus anticipation of no reward	Patients showed no differentiation in metabolic activity between paradigms. Controls had increased activity with anticipation of reward in bilateral ventral striatum, frontal cingulate cortex, thalamus, and left posterior midbrain
Beutel (2010)	Panic Disorder (9) HC (18)	fMRI Emotional paradigm: words with negative, neutral and positive valence	Patients showed a) decreased metabolic activity in the ventral and orbital prefrontal regions b) increased activity in temporal regions
Lai (2007)	Borderline Personality Disorder (2) HC (5)	SPET (regional perfusion rates) a) baseline: quiet and stress conditions b) post-treatment: stress condition	Quiet condition: a) Patients showed hyperperfusion rates in frontal and limbic areas Stress condition (5 min duration video stimulus with psychologically violent scenes) a) Patients showed hyperperfusion rates in limbic, temporal, parietal and occipital areas  b) HC showed no changes in regional brain perfusion rates

Table 2. Findings from neuroimaging studies of synaptic neurotransmission before and after psychodynamic psychotherapy

First author (year)	Clinical condition (n)	Neuroimaging (measure)	Baseline differences relative to healthy controls	Post-treatment changes relative to baseline
Hirvonen (2011)	Depression (22): -Treated with PP (8) -or with Fluoxetine (14) No HC	PET (D2/3 binding)		PP Group: No changes in dopamine in striatum and thalamus. Fluoxetine Group: Increased D
Lehto (2008)	Atypical depression (8) Typical depression (11) No HC	SPECT (SERT & DAT)	No baseline difference in striatal DAT and midbrain SERT between the two clinical groups	Midbrain SERT increased in the no change was observed in pe
Saarinen (2005)	Depression (1) HC (10)	SPECT (SERT)	Patient has decreased midbrain SERT (-2.5 SD)	Patient midbrain SERT normal

Tolmunen (2004)	Case 1: Hypomania and dysthymia Depression (6) HC (10)	SPECT (SERT & DAT)	Case 1: elevated midbrain SERT (+2 SD); elevated striatal DAT Patients with depression: decreased midbrain SERT	Case 1: decreased SERT in midbrain (both towards normalization) Depressed group showed increased SERT (towards normalization)
Viinamaki (1998)	Borderline Personality Disorder -Case 1: and mild depression -Case 2: and severe depression HC (10)	SPECT (SERT & DAT)	Case 1: decreased SERT (-2 SD) in medial prefrontal cortex, midbrain, thalamus; normal striatal DAT Case 2: decreased SERT in medial prefrontal cortex; normal striatal DAT	Case 1: SERT increased (normalization) in medial prefrontal cortex, midbrain and thalamus; no change in striatal DAT Case 2: no changes (and did not normalize)

Table 3. Direction of differences in synaptic neurotransmission

Brain region	Clinical condition	Baseline, (N across studies)	Post-treatment, (N across studies)
Midbrain	Depression	↓ SERT (9) 17-19	↑ SERT (16) 18
	Hypomania	↑ SERT (1) 18	↓ SERT (1) 18
Thalamus	Depression	↓ SERT (1) 19	↑ SERT (1) 19
			= D2/3 binding (8) 12
			↓ D2/3 binding (14) (after Fluoxetine)
Medial prefrontal	Depression	↓ SERT (2) 19	↑ SERT (2) 19
Striatum	Depression	= DAT (8) 18,19	= DAT (21) 19
			= D2/3 binding (8)
	Hypomania	↑ DAT (1) 18	↓ DAT (1) 18

DAT: dopamine transporter binding, SERT: serotonin transporter binding.

1 = Hirvonen 2011; 3 = Saarinen 2005; 4 = Lehto 2008; 5 = Tolmunen 2004; 6 = Viinamaki 1998 [SN](#)

References still need fixing