Supplemental Table 3.

Name	Abbreviation	Sample	Change	Group	Excitatory / Inhibitory	Receptor	Notes
D-Alanine	Ala	HPC	Increase	CFC	Inhibitory	Glycine receptor	Has been shown to increase folllowing anti-depressant treatment.
D-Serine	Ser	PFC	Decrease	CFC	Excitatory	NDMA receptors and glycine receptors	Coagonist at the glycine site of NDMA receptors.
Gamma-aminobuytric acid	GABA	HPC	Increase	CFC	Inhibitory	GABAA receptors and GABAA-p receptors	Low GABA levels have been linked to depression.
Glycine	Gly	PFC	Decrease	CFC	Inhibitory / Excitatory	NDMA receptors and glycine receptors	Often released with GABA.
L-Glutamic acid	Glu	HPC	Increase	CFC	Excitatory	NDMA receptors and AMPA receptors	Precursor to GABA as well.
L-Tyrosine	Tyr	PFC	Decrease	CFC	Excitatory	CaSR	Precursor to neurotransmitters such as tyrosine, dopamine, norephinephrine, and epinephrine.
O-Phosphoethanolamine*	PE	HPC	Increase	CFC	-	-	Shows strong structural similarity to GABA. Co-released with taurine.
Phenylalanine	Phe	PFC	Decrease	CFC	Excitatory	CaSR	Precursor to neurotransmitters such as tyrosine, dopamine, norephinephrine, and epinephrine.
Taurine	Tau	HPC	Increase	CFC and context exposure	Inhibitory	Glycine receptor	Structural resemblance with GABA.
5-Hydroxy-L-tryptophan	5-HTP	Plasma	Increase	CFC	-	-	Immediate precursor to serotonin (5-HT), which elevates mood.
D-Serine	Ser	Plasma	Decrease	CFC	Excitatory	NDMA receptors and glycine receptors	Enhances glutamatergic signalling via NMDARs.
Gamma-aminobuytric acid	GABA	Plasma	Decrease	CFC	Inhibitory	GABAA receptors and GABAA-p receptors	Homeostatic balance of GABAergic inhibition is essential for controlling exitatory neurotransmission.
L-Glutamic acid	Glu	Plasma	Decrease	CFC	Excitatory	NDMA receptors and AMPA receptors	Precursor to glutamine, which stabilizes the immune system in times of stress.
N-Acetyl-L-tyrosine	NALT	Plasma	Decrease	CFC	-	-	Precursor to tyrosine, which converts to norepinephrine (NE) and dopamine (DA), enhancers of mood.
O-Phosphoethanolamine*	PE	Plasma	Increase	CFC	-	-	Inhibits mitochondrial dysfunction, suggested to be involved in the pathophysiology of affective illnesses.

*PE has not been shown to be a potent neurotransmitter yet, but has structural similiarity to GABA.