Author(s)	Imaging Modality	Participants	Aims	Results	Limitations/Future Directions
Araujo et al., 2014	MRI	25 BPD, 25 HC (F) Those with a current Axis I or II disorder were excluded.	To investigate whether structural atypicalities of the cortex are present in BPD patients.	Reduced unilateral thickness of the l. lat OFC, r. mid. front. gyrus, area of l. med. oFC and r. insula and increased area and thickness of the bilateral parietal gyri, r. postcentral gyrus thickness and area l. sup frontal gyrus in BPD compared to HC.	All patients taking at least one psychotropic agent. Small sample size. Not generalizable to males with disorder nor to typical BPD patient who will likely have several comorbid conditions. No clinical control.
Bertsch et al., 2013	fMRI	40 BPD, 41 HC (F) Those with IQ <85; pregnancy; endocrine or neurological disorders; use of any type of regular medication except contraceptives; lifetime diagnoses of schizophrenia, schizoaffective disorder, or bipolar disorder; and current alcohol or drug dependence were excluded.	To investigate effect of oxytocin on amygdala response during an emotion classification task.	Quicker initial gaze fixation to eyes of angry faces in BPD group and increased amygdala response to angry faces relative to HCs, hyper-reactivity dampened after oxytocin. Increased amygdala activity positively correlated with quicker disengagement from eyes of angry faces in BPD group.	No clinical control. Focused on amygdala, no whole brain changes reported. Limited sample size.

Brunner et al., 2010	MRI	20 BPD, 20 other MHD (mixed diagnoses) , 20 HCs adolescents (F)	To investigate structural changes in brain volume present in adolescent-onset BPD.	Reduced bilateral DLPFC and left OFC grey matter density in BPD compared to HCs. Decreased grey matter in right DLPFC in clinical controls compared to HCs. No significant grey matter alterations in BPD relative to clinical control. No intergroup differences in limbic system and WM structures.	Small sample size. Gender bias. Comorbid diagnoses may influence brain morphology. Larger cohort studies may allow for examination of symptomatic variability within groups.
Bungert et al., 2015	fMRI	20 BPD, 20 HC (F) Those with a lifetime history of psychotic disorder, current major depression, substance abuse or addiction, pregnancy, organic brain disease, a history of skull or brain damage, severe neurological illnesses, and currently using psychotropic medication were excluded.	Investigated experience of physical pain in BPD compared to controls following social inclusion/exclusion during a virtual ball-tossing game. Examined effect of rejection sensitivity on experience and neural processing of physical pain post inclusion/exclusion.	Social exclusion in ball tossing game led to hypersensitivity to physical pain in both groups (subjective measures) as well as increased AI and thalamic activation. Exclusion linked to additional posterior AI activation, inclusion linked to reduced amygdala activation in response to nociceptive stimuli in BPD group relative to HCs. Increasing rejection sensitivity related to less difference in	Within subjects so all patients experienced inclusion as well as exclusion, which may have dampened/enhanced the effects of each. Gender bias. Excluded major depression which may reduce generalisability to the average individual with BPD. Future studies may wish to use this paradigm on subgroups of BPD patients (i.e. high impulsivity, non-suicidal self-injury etc). Small sample size.

				amygdala and insula activation in response to pain after inclusion and exclusion.	
Carrasco et al., 2012	DTI	28 BPD (13M, 15F), 26 HCs Those with current major depression, substance dependence, life-time diagnosis of schizophrenia, bipolar disorder or organic mental disorders, and those using psychotropic medication in the two weeks prior to study were excluded.	To investigate microstructural damage to white matter tracts of PFC in a representative sample of BPD patients.	Decreased fractional anisotropy (FA) in genu and rostral corpus callosum, bilateral prefrontal white matter fasciculi and orbitofrontal white matter in BPD group compared to controls. No increased FA in relation to controls.	Small sample size. Some patients on long term medications which could have altered brain morphology. Decreased FA cannot be specifically linked to BPD as comorbid conditions, disorder severity and additional complications could influence white matter development. DTI more prone to artefacts than other modalities.
Chanen et al., 2008	MRI	20 BPD (15F, 5M), 20 (15F, 5M) HCs Those with schizophrenia or affective psychotic disorders, anorexia nervosa, current alcohol dependence, history of head injury, loss of consciousness for 10 min or more, seizures, thyroid	To investigate the hippocampal, orbitofrontal and amygdala volumes of teenagers with first-presentation BPD.	Right side OFC grey matter loss relative to controls, no significant differences in hippocampal and amygdala grey matter. Smaller amygdala bilaterally in males with BPD (sample size incredibly small). Correlations between right amygdala volume and symptoms (i.e. inappropriate	Longitudinal studies needed to observe whether or not hippocampal/amygdala deficits appear later in course of disorder Controversial diagnostic criteria for BPD in youth. Structural changes over time cannot be determined from this paper. Comorbidity may influence brain morphology. Large number of statistical analyses used

		disorder or other significant medical illness were excluded.		anger, externalisation/internalisation, impulsivity) in females.	increasing possibility of Type I error. Small sample size.
de Araujo Filho et al., 2014	MRI	25 BPD, 25 HCs (F) Those with any other psychiatric comorbidity at time of investigation were excluded.	To investigate differences between volumes of OFC in BPD and HC samples.	Reduced cortical thickness bilaterally in medial OFC, decreased curvature and depth of sulcus in right medial OFC and increased curvature of left in BPD compared to control.	High rates of past psychiatric comorbidity which could confound results. High levels of psychotropic use. Cannot generalise to males nor to those with comorbidities. Small sample size.
Depping et al., 2016	MRI	22 MDD, 17 BPD, 22 HCs (F) Those with medical/ neurological disorders, drug or alcohol abuse, a history of head trauma, lifetime or current comorbid Axis I and II disorders (for MDD), lifetime schizophrenia or bipolar disorder diagnosis or ADHD were exluded.	To investigate and compare the structural networks that are shared and distinct in MDD and BPD to healthy controls (MRI).	Reduced volume of bilateral frontostriatal network in MDD compared to BPD and HC. Reductions in medial/temporal frontal network (hippocampus, parahippocampus and amygdala) volumes in BPD relative to HCs and MDD. Structural pattern of lateral PFC and cingulate significantly related to depressive symptoms in MDD and BPD	Limited sample size across groups. Differential therapeutic measures taken across clinical groups. Small sample size.
Domsalla et al., 2014	fMRI	20 BPD and 20 HCs (F). Those with a lifetime history of psychotic or bipolar I disorder,	To develop an understanding of rejection sensitivity in BPD at the neural level using a	Both BPD and HCs felt excluded to similar degree in exclusion groups but BPD felt more excluded during inclusion and control than	Brain structures activated may not be exclusive to social-emotional processes, should add a non-social control condition to further examine (i.e. tossing and catching)

		current major depressive episode, current substance abuse or addiction, pregnancy, organic brain disease, psychotropic medication use skull/brain damage, or severe neurological illness were excluded.	virtual ball-tossing paradigm.	HCs; more dissociative symptoms in BPD across conditions compared to HC. Greater activation of medial PFC and dACC in BPD group across conditions. Cortical activation differences: Greater activation in precuneus, dlPFC, insula and mPFC in BPD brain during control task (equal tosses between participants), greater dlPFC activation in BPD during exclusion conditions (other activation comparisons failed to reach statistical sig).	No clinical control group, findings may not be specific to BPD. Some differences in activation reported at 10% significance level. Small sample size.
Hazlett et al., 2012	fMRI	33 BPD (20F, 13M) 28 SPD (12F, 16M) 32 HC (20F, 12M) Those with a history of psychotic disorder, bipolar I affective disorder, or current MDD, medical/ neurological illness, head injury, substance dependence/abuse (in past 6 months) and those using psychotropic	To investigate differences in amygdala response to neutral and affect-inducing (positive/negative in valence) stimuli in BPD, schizotypal personality disorder and HC groups.	Greater amygdala activation in BPD compared to SPD and HCs to affect-inducing stimuli (no group difference for neutral stimuli), increased time to return to baseline activation levels in BPD group relative to HCs and SPD. Greater amygdala response to repeated pictures in BPD suggesting impaired amygdala habituation relative to SPD and HCs.	Several BPD patients had history of antidepressant, neuroleptic and benzodiazepine use which could confound results. Limited sample size.

		medication were excluded.			
Herbort et al., 2016	fMRI	21 BPD, 23 HCs (F) Those with a history of schizophrenia, bipolar disorder, schizoaffective disorder, lifetime diagnosis of adult ADHD, substance abuse and currently using psychotropic medication were excluded.	To examine the relationship between striatal responses to rewards/losses and impulsivity in those with BPD.	Reduced activity in nucleus accumbens and ventral striatum in response to reward and loss predicting cues compared to neutral cues in BPD group. Negative correlation between ventral striatum loss anticipation cues and self-reported impulsivity scores in BPD, converse relationship observed in HCs. Positive correlation between striatal response to both losses/gains and impulsivity scores in BPD. Blunted neural response to reward/loss anticipation may lead individual to thrill-seek more to compensate?	Insufficient sample size to examine regions other than striatum. Used monetary rewards only, future research may want to investigate social reward/punishment. No clinical control group, muted striatal response to losses/gains apparent also in depression and bipolar II disorder (Ubl et al., 2015; Yip et al., 2015)
Jin et al., 2016	MRI	34 HCs (15M, 19F), 34 BPD (17F, 17F) Those with past or current diagnosis of schizophrenia, paranoid disorder, schizoaffective	To investigate grey matter differences across experimental groups and its relationship to childhood trauma and attachment	Increased bilateral volume of middle cingulate cortex, posterior cingulate cortex and precuneus compared to HCs, no other significant differences in grey matter concentration.	Used lower accuracy widespread voxel-by-voxel univariate analyses. Not generalisable to patients with comorbid conditions.

		disorder, bipolar disorder, physical disorder with psychiatric consequence (e.g., hypothyroidism, seizure disorder, brain injury) were excluded.	styles.	Childhood trauma not correlated with grey matter volume across groups. HCs with more insecure attachments had less grey matter in precuneus, MCC and middle occipital gyrus but no negative correlations between insecure attachment and volume in BPD group.	
Kimmel et al., 2016	MRI	Meta-analyses: 256 BPD and 272 HCs	To investigate age-related neural changes in BPD.	Greater r. sup. motor area volume in BPD relative to controls. Smaller grey matter volume in r. sup./midd. temp gyri, inferior frontal gyrus pars opercularis, left hippocampus compared to controls. Left superior parietal-occipital volumes increase with age in BPD (younger patients show reduced parieto-occipital volumes). Right amygdala volume decreases with age. Grey matter deficits in limbic areas ostensibly worsen with age in BPD.	Meta-analyses examine summarised, compiled data rather than raw, experimental data. Voxel-based morphometry may not be powerful enough to accurately detect differences in very small structures such as hippocampus and amygdalae. Publication bias (unpublished data not included).

				Psychotropic medications not correlated with regional grey matter volume differences between BPD and HC groups.	
King-Casas et al., 2008	fMRI	55 BPD (37F, 1M) 38 HCs (51F, 4M)	To investigate cooperation in BPD using an economic exchange game.	Behavioural: BPD less able to maintain co-operation and repair broken co-operation Neurological: positive association between in AI activity and input/output responses (value of monetary offers received/money offered as repayment to partner respectively) AI activity only related to output (money repaid to partner) and not input (relationship independent of medication status). Indicates BPD have low expectations of others such that low offers not seen as violation of social norms Investments levels lower for pairs with BPD player than for HC vs HC (indicator of untrustworthiness/non-cooperation) Lower levels of self-reported	Economic exchange games are not an exact replica of real-world social interaction. Monetary element may compel subjects to behave more antisocially than normal (in a bid to maximise earnings). Lack of clinical control – difficult to attribute mode of gameplay specifically to BPD.

				trust in BPD compared to control	
Kluetsch et al., 2012	fMRI	25 BPD and 22 HCs (F) Those with a history of head trauma, chronic pain, serious medical/neurological illness, current MDD, alcohol or substance abuse/dependence, lifetime diagnosis of bipolar disorder and schizophrenia, and pain disorders, and those taking medication within two weeks of scan.	Evaluate connectivity of the default mode network (DMN; comprising the mPFC, PCC including the precuneus, inferior parietal lobule, lateral temporal cortex, and hippocampal formation) with respect to nociceptive or neutral stimuli in BPD and HC individuals.	L. retrosplenial cortex and l. sup. front. gyrus less integrated into DMN in BPD Lower DMN response to nociceptive stimuli was associated with greater symptom severity in those with BPD. L. DLPFC less connected to pCC seed region during painful vs neutral stimuli in BPD.	No non-NSSI control group – is the effect specific to BPD or BPD with self-injurious behaviour? Higher temperature used for BPD condition due to greater reported tolerance to pain, so group differences may be due to stimulus intensity as opposed to brain connectivity. Comorbid conditions may have confounded results. Further research should look at DMN connectivity in response to social/autobiographical stimuli (group differences here mediated by appraisal of stimuli as more or less self relevant/aversive).
Koenigsberg et al., 2009	fMRI	18 BPD (10F, 8M), 16 HC (9F, 7M) Those with bipolar I disorder, schizophrenia, schizoaffective disorder, substance dependence, organic mental syndromes, or substance abuse disorder in past 6 months were excluded	To understand affective instability in BPD through psychological distancing from aversive stimuli.	Distancing vs looking at negative stimuli caused increased activation of DLPFC, IPS areas, ventrolateral prefrontal cortex, and posterior cingulate/precuneus regions in both groups (both groups reported less negative affect when distancing). When viewing negative stimuli, BPD show greater activation sup. temporal gyrus,	Very small sample size so little statistical power. BPD sample also met criteria for PTSD, GAD and other Axis II disorders so results may not be exclusive to BPD. No way of definitively tested that “distancing” took place. Future studies should look at other reappraisal strategies (reinterpretation etc).

		as were those taking psychotropic medication within two weeks of scan.		PCC, ACC, and cerebellum vs the HC. Increased amygdala activation in BPD relative to baseline during distancing vs looking. Less activation in DACC and IPS, less amygdala deactivation, greater sup. temp sulcus and sup. front. gyrus in BPD during distancing relative to looking.	
Krause-Utz et al., 2014	fMRI	20 BPD, 17 HC (F) Those with current MDD, lifetime psychotic disorder, bipolar affective disorder, mental retardation, developmental disorder, and in suicidal crisis were excluded.	To investigate resting state functional connectivity in ROIs (frontolimbic regions).	BPD showed evidence of increased amygdala-insula (as well as oFC and putamen) resting state functional connectivity (RSFC). Stronger functional connectivity between ACC and dmPFC in BPD, whereas HCs showed diminished connectivity. Decreased RSFC in between left vACC and V1, lingual gyrus and cuneus in BPD compared to controls.	Large proportion of BPD subjects reported trauma in past, associations may be linked to trauma as oppose to condition. BOLD responses of amygdala can be confounded by physiological factors such as venous drainage. Small sample size.
Kreisel et al., 2015	MRI	39 BPD (33F, 6M) and 39 HC (33F, 6M)	To investigate whether there are differences in	Hippocampal volumes did not differ across groups.	History of psychotropic use in sample – possible confounder.

		Those with current/previous medical conditions (e.g., stroke, ischemic heart disease), history of anorexia, schizophrenia, schizoaffective disorder, major depressive episodes with psychotic symptoms, or substance abuse within the 6 months were excluded.	hippocampal grey matter volume between HCs and BPD groups.	Exploratory analyses revealed that comorbid PTSD with BPD gave rise to smaller hippocampi (head and body) than BPD without PTSD. Those with >7 DSM-IV BPD criteria showed reduced volume of head of hippocampus than those with fewer symptoms.	Several comorbid conditions in patient past including anorexia and bulimia (starvation leads to general cortical shrinkage). Further studies should look to examine whether numbers of symptom criteria fulfilled affects morphology of other structures.
Lischke, Herpertz, Berger, Domes, & Gamer, 2017	fMRI	51 BPD, 48 HCs (F) Those with schizoaffective disorder, schizophrenia or intellectual disability or taking regular medication within past 8 weeks of the scan.	To examine paralimbic activity to emotional and neutral scenes after intranasal administration of oxytocin.	OT decreased amygdala reactivity in BPD but increased reactivity in HCs. Greater baseline paralimbic activation in BPD (after placebo administration) Negative correlation between amygdala activity and gaze behaviour (greater amygdala activity implied less looking at emotive stimuli). No abnormal activity in PFC, nor atypical connectivity between paralimbic and prefrontal regions in BPD.	Did not report symptom profile nor comorbid conditions of sample thus generalisability is questionable.

				OT regulated atypical relationship between amygdala activity and gaze behaviour across scenes in BPD group, irrespective of the valence.	
Maier-Hein et al., 2014	DTI	20 BPD adolescents, 20 HCs and 20 clinical control (mixed diagnoses) (F) Those with lifetime diagnosis of SCZ, schizoaffective disorder, bipolar disorder, pervasive developmental disorder, alcohol/drug dependence, significant neurological disease, BMI of 16.0 or lower, and IQ <85 were excluded.	To highlight BPD-specific white matter changes in adolescents	Decreased FA in fornix in BPD group compared to CC as well as white matter alterations in thalamus-hippocampus connecting tracts. No changes in FA of inferior frontal WM.	High levels of psychotropics usage in samples. Comorbid disorders such as PTSD present in BPD group so BPD-specific changes difficult to specify. Small sample size. Tractography still lacks experiential validation. Cannot generalise to male patients.
Mensebach et al., 2009	fMRI	18 BPD, 18 HCs (F) Those with infectious diseases, anorexia, SCZ, schizoaffective disorders, and MDD with psychotic symptoms, alcohol or drug dependence were	To assess whether deficits in episodic/semantic memory are present in BPD through free-recall and verbal fluency tasks.	No differences in performance across groups, both groups showed activation of bilateral frontal, temporal and limbic neocortical areas during episodic and left lateral frontal and temporal, bilateral medial frontal and left parietal neocortical regions during	Small sample size. High rates of comorbid disorders, namely PTSD, depressive and panic disorders - result cannot be exclusively due to BPD. All patients treated by DBT and psychotropics – possible confounder.

		excluded.		semantic memory Neurally, during episodic task, BPD showed increased activation of bilateral PCC, left mid, sup temp gyri, r. inf. front. gyrus and r. ang. gyrus. During semantic memory task, BPD showed increased activation of PCC, r. fusiform gyrus, l. ACC and l. postcentral gyrus Those with BPD need to recruit additional brain structures to carry out tasks which are less neurally taxing for controls. Post hoc analyses showed no differences in performance for BPD-PTSD subjects compared to BPD.	Future research should use larger cohorts to allow for subgroup analysis by comorbidity.
Morandotti et al., 2013	MRI	18 BPD, 19 HC (F) Those with current or lifetime personality disorders, schizophrenia, schizoaffective disorder, bipolar or a	To investigate grey matter volume of vPFC in BPD and its relation to child abuse.	Right VLPFC reduced in BPD with history of childhood abuse compared to non-abuse BPD (no other pairwise comparisons significant). Aggression self-report scores (as well as irritability and	Comorbid depressive disorders present – possible confounder. Incredibly small subsample sizes, future work should carry out larger cohort studies. Child abuse history measured in hindsight using patient self-report (memories subject to

		history of alcohol or substance abuse within the 6 months preceding the study were excluded.		negativism subscale scores) positively correlated with VLPFC volume in BPD with child abuse group. Self-reported irritability higher in abused subgroup than non-abused. Total intracranial did not differ between groups, no overall main effect of diagnosis on grey matter volume.	distortion) Child abuse in itself may relate to diminished PFC volume as no differences between BPD and HCs found.
Muller et al., 2015	MRI	34 BPD (20 F, 14M) 31 HC (16F, 15M) 17 BPD-Remission (F) Those with neurological disorders, current alcohol or drug abuse, SCZ, schizoaffective disorder, or bipolar disorder; severe medical illness, including heart problems; psychotropic medication were excluded.	To reveal neural activity underpinning distorted interoception and its relationship with emotional dysregulation in BPD.	Reduced amplitude in heart-rate evoked potentials in BPD compared to HC, BPD-remission lies between. Heart-rate evoked potentials negatively correlated with emotional dysregulation and positively correlated with AI and bilateral dACC volume. No sig relationship between HEP amplitude and amygdala/hippocampal volumes across groups and no sig. AI or ACC volume diff between both BPD groups and HC.	Only women comprise the BPD-R group so difficult to compare results from mixed group groups to same sex. Level of statistical significance taken as 0.01. No evidence as to what, if any, therapies contributed to remission status Exploratory analyses for smaller BPD-R subgroup were not corrected for multiple tests
New et al., 2013	DTI	38 BPD (14 adolescents, 24-adults)	To investigate development	Decreased bilateral FA in inf. long. fasciculus in BPD	More males in adult sample than adolescent samples – possible confounder

		32 HCs (13 adolescents, 19-adults) (mixed gender) Those with serious head injury or neurological disorder, schizphrenia, any other psychotic disorder, bipolar disorder I or pervasive developmental disorder, and those taking medication prior to scan were excluded.	changes in WM tracts in BPD from adolescence to adulthood.	adolescents relative to HC adolescents. Higher FA in HC adolescents compared to all other groups. Lower FA in BPD adolescents compared to HC adolescents in uncinated and occipitofrontal fasciculi (temp lobe WM tracts). No between group FA differences in adults.	MDD very prevalent in BPD samples, FA may be due in part to this. Future research should include clinical control to assess whether this is disorder specific. Adult BPD unmedicated but adolescents on variety of medications (ethical concern).
Nicol, Pope, Romaniuk, & Hall, 2015	fMRI	20 BPD (17F, 3M) 16 HC (14 F, 2M) Those with bipolar I disorder or SCZ, current alcohol/drug dependency, or any neurological illness.	Examine relationships between child abuse, psychotic symptoms and brain activation to fearful stimuli in BPD	Decreased activation of r. cuneus in BPD group Sig positive correlation between physical childhood abuse (as reported by CTQ) and activation of midbrain, pulvinar, cerebellum and med. front. gyrus in response to fearful vs neutral faces. No correlation between emotional abuse and activation. Positive correlation between midbrain activation and reported psychotic symptoms.	Upwards of 60% sample on antidepressants and/or neuroleptics. 85% of sample had comorbid conditions including depressive disorders and PTSD so difficult to determine whether activation pattern specific to BPD. Disproportionately female samples. Insufficient range of CTQ scores from HCs thus cannot conclude this pattern of activation is exclusive to BPD child abuse population relative to general child abuse population.

				No differences in activation between those treated with antipsychotics, antidepressants and other treatments.	
Niedtfeld et al., 2013	MRI	60 HC, 21 BPD-PTSD, 39 BPD (F) Those with severe medical or neurological illnesses, organic brain disease, mental retardation, medical history of skull- and/or brain-damage, pregnancy, left-handedness, pieces of metal in the body, claustrophobia, as well as those using psychotropic medication two weeks prior to the study we excluded.	To examine grey matter volume differences in BPD groups with and without comorbid PTSD relative to controls.	Smaller r. amygdala, r. hippocampus, fusiform, lingual and cingulate gyri observed in BPD relative to HC Comorbid PTSD with BPD linked to increased grey matter volume in sup. temp. gyrus and DLPFC. No subgroup differences in volume of hippocampi/amygdalae. BPD symptom severity predictor of amygdala and dorsal ACC volume (negative correlation) irrespective of comorbidity as well as smaller grey matter volume in cerebellum and fusiform gyrus.	No PTSD without BPD control group to determine whether changes specific to comorbid condition or PTSD alone. Non-PTSD group may have experienced trauma but was not considered “traumatic” enough to meet PTSD criteria. Lacked statistical corrections for multiple comparisons increasing probability of type I error.
Niedtfeld et al., 2017	fMRI	28 BPD-DBT, 15 BPD-C, 23 HC Those who are left-handed or have experienced traumatic brain injury, lifetime	To alter the neural processing of pain-mediated affect regulation post-DBT To determine the	Reduced activation of amygdala and altered left amygdala and dorsal ACC connectivity following nociceptive stimuli in BPD (inhibitory coupling), attenuation of this effect post	High percentages of BPD groups receiving pharmacotherapy – possible confounder. IQ was not controlled for. Low statistical power due to small sample size.

		SCZ or bipolar I disorder, mental or developmental disorders, substance dependence during the last year, current severe depressive episode, and those using benzodiazepines were excluded from partaking.	effect of DBT on hot/cold pain thresholds.	DBT treatment. No main effect of treatment on pain thresholds in BPD groups.	Did not assess inter-patient variability in picture evaluation and pain perception. Future studies should aim for a double blind RCT design. Gender of sample unspecified.
Ninomiya et al., 2018	DTI	35 BPD (11F, 24M), 50 HC (17F, 33M) Those with comorbid Axis I disorders, using medication, and those suffering from alcohol or drug abuse were excluded.	To examine the effect of borderline personality disorder on white matter tract integrity.	BPD reported higher levels of anxiety, emotional abuse/neglect, self-denial, anger-hostility and depression-dejection Lower axial diffusivity in BPD cingulum, inf. fronto-occipital fasciculus and inf. long. Fasciculus AD of cingulum positively correlated with depression in BPD Physical neglect negatively correlated with AD of inf. fronto-occipital fasciculus	Small sample size. Subjects were un=medicated without comorbid conditions thus not a very representative of the average individual with BPD.
O'Neill et al., 2013	MRI	20 BPD, 21 HC (F) Those suffering from substance dependency	To examine volumetric abnormalities in hippocampus (as	BPD group scored higher on both depression scales than HCs.	All patients treated with psychotropics in past/present Future research may wish to incorporate a

		and additional psychiatric disorders (aside from current/past comorbid medical conditions) were excluded.	well as its sub-regions), basal ganglia and ACC in BPD vs healthy conytols	No intergroup differences in total intracranial volume. Smaller bilateral hippocampal tails and l. head and body in BPD. Reductions in caudate and DLPFC of BPD group. No correlation between hippocampal volume and depression nor impulsivity scores in BPD group.	memory task to see whether hippocampal differences directly affect performance. Did not assess trauma levels in group (also known to affect size of hippocampus). Small sample size. Future studies may wish to use MRS to see how volumetric deficits influence neurometabolites in hippocampus (N-acetylaspartate (tNAA) and creatine (Cr)).
O'Neill et al., 2015	fMRI	19 HC, 17 BPD (F) Those with neurological disorders, severe medical illness, head injury, and alcohol or substance dependency were excluded (other clinical comorbidities were not reported).	To investigate between group differences in functional connectivity between emotional and ToM networks as well as in the default mode network (DMN).	Higher impulsivity, neuroticism, depression and lower extraversion in BPD group. Fewer ToM trials were reportedly understood by BPD. Decreased functional connectivity between subgenual ACC and l. sup. temp lobe, r. supramarginal parietal lobes and r. mid. CC in BPD during ToM task condition. Increased functional connectivity seen between	DMN data taken from 10s rest period within task, so not truly representative of resting state. BPD received fewer years of education than HCs thus understanding of jokes could have been impaired by this (not known if there is an interaction between years of education and ToM).

				precuneus and l. inf. front. lobe, l. precentral/mid. front., and l. mid. occipital/superior parietal lobes particularly during rest Psychotropic usage as co-variate did not influence data.	
Prossin, Love, Koeppe, Zubieta, & Silk, 2010	PET	18 BPD, 14 HC (F) Those with any concurrent axis I and III diagnoses (except for mood disorder); history of psychosis or head trauma; and current or recent (within 3 months) illicit substance use, abuse, or dependence were exlcluded	To investigate extent to which opioid system (Mu-opioid receptors) in BPD accounts for emotion dysregulation.	Significant effect of condition on PANAS scores and of diagnosis, with BPD patients reporting more sadness after vignette. BPD showed greater binding potential than HCs in neutral state in bilateral OFC, caudate, l. amygdala and nucleus accumbens, lower binding potential in pos. thalamus. Endogenous activation for HCs observed in l. ant. thalamus, l. medial thalamus, r. hippocampus during sadness. Endogenous system activation in l. pos. thalamus, l. OFC, l. ventral pallidum, l. amygdala and l. inf. temp. cortex during sadness state for BPD group. Greater endogenous opioid	Very small sample size. Difficult to know whether neutral task conditions were adhered to.

				system activation in BPD relative to comparison subjects during sadness in the pregenual ACC, left OFC, left ventral pallidum, left amygdala, and left ITC.	
Reitz et al., 2015	fMRI	21 BPD, 17 HC (F) Those experiencing a current episode of MDD, lifetime diagnosis of schizophrenia, bipolar, acute suicidal tendencies, major medical or neurological illness and those using psychotropic medication were excluded.	To investigate neural correlates of NSSI in BPD.	Decreased amygdala activity and regulation of functional connectivity with SFG after incision in BPD group Increase in amygdala activity for HCs over time after stress induction. HCs showed reduced amygdala-sup. front. gyrus connectivity in response to incision over sham, whereas amygdala- sup. front. gyrus connectivity increased in BPD group after incision. Steeper decline in aversive tension in BPD following incision vs sham compared to controls whereas HCs showed greater decrease in aversive tension following sham. Heart rate stayed higher in BPD after sham vs incision.	Incision was not inflicted by oneself so not truly representative of NSSI Cannot say incision directly affected stress during task as it was administered afterwards Looked at changes in ROIs, not activation produced by NSSI Cannot generalise to men nor to the average individual with BDP and depression or other comorbidities.
Richter et	MRI	20 HC, 20 BPD, 20	To investigate	No group differences in	Many concurrent disorders in both BPD and CC

al., 2014		clinical control (F) Those with schizophrenia, schizoaffective disorder, bipolar disorder, pervasive developmental disorder, alcohol/drug dependence, or neurological disease, a BMI<16 or IQ≤85 were excluded.	differences in brain volume between adolescent BPD patients relative to healthy and clinical controls.	cortical thickness. Smaller r.hippocampus, l. orbital inf. front. gyrus in BPD and CC compared to HC. Smaller l. hippocampus, r. amygdala, r. mid. front. gyrus, and r. sup temp gyrus in BPD compared to HC (other pairwise comparisons not significant for these areas). BPD and CC differed only in r. orbital front. gyrus and l. sup parietal gyrus volume.	groups such as mood disorders, anxiety and eating disorders thus difficult to attribute results to BPD alone. Several psychotropics used in BPD group.
Ruocco et al., 2016	fNIRS	31 BPD (F) Those with psychotic disorder, current substance dependence, illness that may impact brain function (e.g., significant head trauma) or an estimated IQ <80 were excluded.	To determine factors which may predict treatment response and attrition by examining neural activation during response inhibition. To ascertain whether activation in pFC pre-DBT was associated with either reductions in self-harm with treatment or	Activation in bilateral medial/inf. frontal gyri reduced during response inhibition prior to treatment. Activity increased in these regions 7mo post treatment. Completers showed less DLPFC activation during response inhibition than non-completers (showed higher activation in mPFC and r. inf. front. gyrus.	ROI study focusing only on the frontal cortices. Prelim. Study so cannot definitively conclude that fNIRS can predict treatment outcomes. Replications using larger cohorts and RCTs necessary to validate results – may lead to clinical measure of identification of at-risk groups and early self-harm intervention.

			treatment attrition.		
Salvador et al., 2016	Diffusion MRI	103 HC, 103 BPD (F) Those with brain trauma, neurological diseases, alcohol/ substance abuse or dependence in 6 months, current comorbid Axis I disorders or previous bipolar or psychotic disorder diagnosis.	To examine global brain connectivity (GBC) in BPD relative to HCs.	High resting state activity (fluctuations) found in the l. hippocampus and amygdala, increased functional connectivity of these regions with the anterior cingulated. White matter reductions of fractional anisotropy in corpus callosum (genu/body) but also involving part of the corona radiata, external capsule (including uncinate fasciculus and inf. fronto-occipital fasciculus), l. ant. limb of int. capsule in BPD. Greater global brain connectivity in BPD located in ant. cingulate, reduced GBC found in r. temp. lobe only, correlated with emotion regulation Reductions in global brain connectivity was not correlated with diagnosis as measured by (Diagnostic Int. for Borderlines)	Pharmacological treatment permitted. Correlations were not corrected for multiple tests. Diagnostic measures used did not explain severity of condition. Results cannot be generalised to males as sample consists solely of women. MRI sequences not powerful enough to detect changes in small brain structures.
Sato et al., 2012	MRI	25 BPD, 25 HC (F)	To explore how MRI can be used in	L. med. orbitofrontal, rostral ACC, PCC, middle temporal	Antidepressant, antipsychotic and mood stabilisers in use amongst the clinical

		Those with axis I and II (aside from BPD) disorders were excluded.	the clinical diagnoses of BPD.	cortices and r. parahippocampal areas contain most discriminative alterations compared to HCs (volumetric differences in grey matter). Above areas purported to have discriminant clinical value.	population. No clinical control so extent to which these changes are exclusive to BPD is debatable. Cannot generalise results to males .
Scherpiet et al., 2014	fMRI	18 BPD, 18 HC (F) Those with present or previous bipolar I disorder, schizophrenia, or schizoaffective disorder were excluded.	To examine how brain activity changes when anticipating stimuli of known or ambiguous valence in BPD vs HCs.	Observed reduced signal change in l. dACC and l. MCC in BPD vs HCs when anticipated negatively-valenced stimuli Increased activation in l. pregenual ACC, l. PCC and l. visual areas such as lingual gyrus in BPD compared to HC When valence of anticipated stimuli was ambiguous compared to neutral, BPD group showed less activation in l. MCC projecting into the med. and bilateral DLPFC and caused r.inf. front. gyrus within the VLPFC and insula activation. When anticipating negative stimuli relative to neutral, BPD	Occasional usage of cannabinoids, alcohol and current depressive episodes permitted in BPD groups. Small sample size.

				showed increased activation in r. vACC, med. front. gyrus, MPFC, r. lingual gyrus, cuneus, l. PCC	
Silvers et al., 2016	fMRI	46 attempters, 14 non-attempters (F) Those with past or present bipolar I or psychotic disorder were excluded.	To examine that which differentiates non-attempters from suicide attempters at a neural level during emotion regulation.	Both BPD groups experienced less negative affect when distancing compared to immersing. Aversive memories activated the lat. prefrontal, temp. (including the hippocampus and amygdala) and occipital cortex irrespective of diagnosis. Attempters recruited thalamus more than non-attempters, but non-attempters recruited occipital cortex more than attempters during recall. Greater activation of lat. OFC in attempters when both distancing and immersing compared to non-attempters whereas attempters showed diminished signal from the precuneus when distancing. Attempters who were successfully able to distance	Not generalisable to men. Participants simply instructed to recall aversive memories thus difficult to ensure whether or not this was adhered to. Clinical control group of attempters would have been beneficial to include. Comorbid depression present in BPD condition, no mention of other comorbidities (no demographics). Non-attempter group much smaller than attempter.

				themselves showed recruitment of precuneus akin to non-attempters.	
Soloff, Nutche, Goradia, & Diwadkar, 2008	MRI	34 BPD (22F, 12M), 30 HC (19F, 11M) Those with a past or current Axis I diagnosis of schizophrenia, delusional (paranoid) disorder, schizoaffective disorder, bipolar disorder or psychotic depression were excluded.	To assess structural brain changes associated with BPD relative to HCs.	Bilateral reductions grey matter reductions in ventral cingulate gyrus and med. temp. lobe (such as hippocampus, amygdala, parahippocampal gyrus, and uncus). Reductions unilaterally in right insula, l. sup. temp. gyrus in BPD. Increases in grey matter volume for BPD in r. med. front. gyri, r. parietal and precuneus, l. sup. front. and l. inf. parietal gyri, l. insula and l. putamen. Gender differences within the BPD group: women had reductions in the med. temp. lobe, including the amygdala; men had less grey matter in ACC compared to HCs. When partialling out depression scores, differences in ventral cingulate became non-significant but differences	Data can be confounded by Axis I comorbidities. Larger sample studies needed to control for gender, clinical characteristics, Axis I and Axis II co-morbidities.

				in med. temp. cortex remained.	
Soloff et al., 2012	MRI	68 BPD (16M, 52F) of whom 44 had attempted suicide. 52 HC (28M, 24F) Those with any past or current Axis I diagnosis of schizophrenia, delusional (paranoid) disorder, schizoaffective disorder, bipolar disorder, or psychotic depression, physical disorders of known psychiatric consequence and significantly reduced IQ were excluded.	To determine brain structures in BPD which differentiate attempters from non-attempters.	History of child abuse more prevalent in attempters than non-attempters. Smaller conc. of grey matter in l. insula in attempters relative to non-attempters. Greater grey matter volume of l. lingual and l. mid. temp. gyrus. Attempters with high lethality had diminished r. mid-sup. temp. gyrus, r. mid. inf. orbitofront. Gyrus, r. insular cortex, l. fusiform gyrus, l. lingual gyrus, r. parahippocampal gyrus in comparison to low lethality.	Global brain differences cannot be ascertained from ROI studies. Imbalanced gender proportions for BPD group. Comorbidities included MDD and PTSD. Structural brain changes may be an effect of consequences of suicide attempt (i.e coma etc) Results may indicate changes due to predisposition for suicidality irrespective of clinical diagnosis. ROI structural MRI studies do not imply functional impairment.
Takahashi, Chanen, Wood, Walterfang, et al., 2009	MRI	20 BPD (5M, 15F) 20 HC (5M, 15F) Those with schizophrenia spectrum disorders or affective psychoses, anorexia nervosa, or current alcohol dependence (≥ 2 months).	To examine region specific structural changes in first presentation BPD.	Shorter AI observed in BPD relative to controls, larger third ventricle, no differences in cavum septum pellucidum AI length did not differ between those with and without comorbid disorders. No significant effect of gender on midline structures.	Control sample significantly older than BPD. Small number of males could confound results. Future research needed to examine whether or not these differences limited to BPD (clinical control groups necessary).

				Exclusions of those with reported past substance addictions did not alter findings.	
Takahashi, Chanen, Wood, Yucel, et al., 2009	MRI	20 BPD (5M, 15F), 20 HC (5M, 15F) Those with schizophrenia spectrum disorders or affective psychoses, anorexia nervosa, or current alcohol dependence (≥ 2 months).	To examine region specific structural changes in first presentation BPD.	No significant difference between groups in volume of insular cortex. No correlation between insular volume and episodes of parasuicidality, trauma, or comorbid Axis I disorders. Negative correlation between insular volume and impulsivity. Bilateral reductions in AI as well as posterior insula volume in BPD pps with violent episodes in past six months compared to non-violent BPD. Exclusions of males and participants taking antidepressants did not alter findings.	Controls significant older than BPD groups. Small sample size. BPD more heterogenous in adolescents (diagnostic methods less coherent). Impulsivity measured by way of violent episodes, manifests itself in a variety of other ways.
van Eijk et al., 2015	fMRI	Sample 1 – 18 BPD, 18 HC (F) Sample 2 – 26 BPD, 25 HC (F)	To assess response inhibition and neural correlates in BPD (without	No significant differences in fMRI BOLD signal during response inhibition across groups for all three tasks.	Small sample size (reliability improved however by two samples). Response inhibition only one aspect of

		Those with a lifetime diagnosis of ADHD, schizophrenia or bipolar disorder, substance abuse within the last three years, or a current depressive episode in the BPD group were excluded.	ADHD) vs HC.	No significant group differences in activation of neural inhibitory network (including r. inf. front. gyrus, striatum, pre-supp. motor area), activated in both groups. Both samples differed significantly from control on self-reported impulsivity scales (UPPS and BIS-11), BPD more impulsive across all measures. Samples did not differ from controls in terms of reaction times nor commission error rate across all three tasks (no performance deficit).	impulsivity. Future work should clearly define area of impulsivity that is of interest.
Winter et al., 2017	fMRI	31 BPD-DBT, 15 BPD-C (without DBT treatment), 22 HCs (F) Left-handed subjects and those with traumatic brain injury, lifetime schizophrenia or bipolar I disorder diagnoses, mental or developmental disorders, substance	To investigate the notion that neural correlates of distraction in BPD can be altered through DBT.	BPD-DBT group showed decreased activity in right inf. parietal lobe/supramarginal gyrus during distraction with negative relative neutral stimuli, compared to HCs and BPD without treatment groups where this decrease correlated with reduction in self-reported symptom severity (DBT group greater reduction in severity than	BPD-DBT group received residential treatment compared to BPD-C group who had outpatient services. Adherence to DBT program regulations were not reported. Not generalisable to males. Small sample size of BPD-C group. Further research using another measure of

		dependence during the last year, drug consumption in the last 2 months, current diagnosis of a severe depressive episode, and benzodiazepine use were excluded.		BPD-C). Treatment responders shown less perigenual ACC activity when viewing negative over neutral stim (less sensitive to emotionality during distraction). Non-responders showed elevated activity in AI when viewing negative over neutral stimuli (not shown in DBT responders)	emotion regulation separate from distraction needed.
Zhou et al., 2017	MRI	30 BPD, 32 HC Those with past or current Axis I diagnosis (e.g., schizophrenia, delusional (paranoid) disorder, schizoaffective disorder or bipolar disorder were excluded.	To investigate the notion that those with BPD have reduced volume of the fronto-limbic cortices.	Greater instance of insecure attachment as well as emotional and physical neglect, emotional and physical abuse in BPD. Greater frontolimbic cortex asymmetry observed in BPD than HC: thinner cortices in l. ACC and less surface area and grey matter volume in l. AI of BPD groups. Asymmetry of ACC and AI positively correlated with attentional impulsivity.	Cross-sectional rather than longitudinal study. Future work should aim to confirm whether volumetric differences are congenital or acquired after illness onset. Study focused on ACC and AI which are two small regions in larger frontolimbic network of brain.