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Transdiagnostic neuroimaging in psychiatry: A review

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ABSTRACT

Transdiagnostic approach has a long history in neuroimaging, predating its recent ascendance as a paradigm for new psychiatric nosology. Various psychiatric disorders have been compared for commonalities and differences in neuroanatomical features and activation patterns, with different aims and rationales. This review covers both structural and functional neuroimaging publications with direct comparison of different psychiatric disorders, including schizophrenia, bipolar disorder, major depressive disorder, autism spectrum disorder, obsessive-compulsive disorder, attention-deficit/hyperactivity disorder, conduct disorder, anorexia nervosa, and bulimia nervosa. Major findings are systematically presented along with specific rationales for each comparison.

1. Introduction

With the explosion of neurobiological research in the last several decades, there have been growing attempts to transcend the limitations of the established nosological systems that primarily rely on categorical classification of psychiatric disorders (Krueger and Eaton, 2015). A dimensional, or transdiagnostic approach is often cited as the more suitable basis for psychiatric classification, promising to erase the principal discrepancy between nosological categorization of psychopathology and dimensional understanding of personality structure, which, inter alia, is seen as impeding the reliable identification of diagnostic and prognostic biomarkers for clinical use (Venkatasubramanian and Keshavan, 2016). In psychiatric neuroimaging this approach translates into comparative analysis of related psychiatric disorders with the aim of uncovering the nature of their relatedness, overlapping and distinct mechanisms beneath their respective semiotics, and the practical utilization of neuroimaging data for correct diagnosis early in the course of illness, thus obviating the current reliance on longitudinal observation. This paper is intended to provide an overview of the current state of both structural and functional neuroimaging in direct comparison of major psychiatric disorders.

2. Multidiagnostic comparisons

The rationale often stated in publications using this relatively recent comparative approach is to identify a common foundation of "mental illness", however ephemeral its heuristic value may be. The earliest morphometric studies, comparing multiple diagnostic groups, reported no differences in ventricle-to-brain ratio in patients with unipolar and bipolar depression, schizophrenia, and healthy subjects (Risch et al., 1992) and greater-than-normal proportion of abnormal MRI scans in each of the patient groups (Lewine et al., 1995).

A comparative voxel-based morphometry study of 404 patients with posttraumatic stress disorder, psychosis, unipolar depression, and obsessive-compulsive disorder found that all diagnostic groups shared gray matter density increases in the putamen (as did their unaffected relatives), and these increases directly correlated with severity of their symptoms (Gong et al., 2018). A meta-analysis of 193 published voxel-based studies, searching for common substrate in mental illness, compared six diagnostic groups (schizophrenia, bipolar disorder, major depressive disorder, addiction disorders, obsessive-compulsive disorder, and anxiety disorders) and found common reductions in gray matter density in the dorsal anterior cingulate and bilateral insulae (Goodkind et al., 2015).

Several published reports conducted analogous multigroup comparisons of diffusion tensor imaging data in order to evaluate diagnostic commonalities in the white matter. In comparative assessment of 102 children with obsessive-compulsive disorder, early-onset schizophrenia and bipolar disorder, White et al. (2015) found no differences in the numbers of white matter potholes (cluster areas of decreased fractional anisotropy) in children with OCD, but global deficits in both schizophrenia and bipolar disorder. Another diffusion tensor imaging comparison of 200 children and adolescents with attention-deficit/hyperactivity, autism spectrum and obsessive-compulsive disorders found that compared to typically developing children all patient groups

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shared fractional anisotropy decreases only in the left splenium of the corpus callosum (Ameis et al., 2016). In the largest such comparative study to date, Chang et al. (2018) used both diffusion tensor imaging and voxel-based morphometry to evaluate 485 medication-naïve subjects with schizophrenia, bipolar disorder, major depressive disorder as well as healthy subjects, and found that all white matter changes were shared by patients with schizophrenia and bipolar disorder, but not MDD, encompassing widespread fractional anisotropy reductions in the association, callosal, thalamocortical, and cerebellar fasciculi and tracts. Shared reductions in gray matter density in all three patient groups, however, were widespread, involving significant clusters in the dorsolateral prefrontal, orbitofrontal, cingulate, temporopolar, insular, angular, and parahippocampal cortex as well as the cuneus. In the authors' estimation, out of all significant gray matter differences between 4 compared groups, 87.9% were represented by overlapping decreases in three patient groups in the right temporal pole, bilateral orbitofrontal and dorsolateral cortex, insula, amygdala, cingulate and parahippocampal gyri.

3. Bipolar disorder and schizophrenia

Arguably the most thoroughly studied transdiagnostic comparison to date and more than any other based on historical circumstances of the very development and evolution of psychiatric nosology, comparison of patients with two major psychotic disorders can be readily traced back to the Kraepelinian dichotomy and its modern discontents (Jablensky, 2010; Craddock and Owen, 2010; d'Albis and Houenou, 2015).

3.1. Early studies

In view of the above, it is not surprising that the very first neuroimaging studies with transdiagnostic comparisons focused on patients with bipolar disorder and schizophrenia. A series of comparative studies using computerized tomography reported increased incidence of shrinkage of the cerebellar vermis in both patients with schizophrenia and bipolar disorder (Heath et al., 1979; Lippman et al., 1982), only in patients with mania and not schizophrenia (Nasrallah et al., 1981, 1982) or in neither patient group (Yates et al., 1987); ventricular enlargement only in patients with schizophrenia (Johnstone et al., 1981; Weinberger et al., 1982; progressive in a longitudinal study, Woods et al., 1990) or in both patient groups (Pearlson and Veroff, 1981; Nasrallah et al., 1982); overall reduction in brain volume only in patients with schizophrenia (Nasrallah et al., 1982), and no intergroup differences either in ventricle-to-brain ratio or total cerebral volume (Rieder et al., 1983). The first functional neuroimaging of these patient groups used ^{18}F-fluorodeoxyglucose positron emission tomography and described diminished anteroposterior gradient in cortical glucose metabolism in both patients with schizophrenia and bipolar disorder (Buchsbaum et al., 1984). The earliest of the MRI studies began to appear at the close of the first decade of psychiatric neuroimaging, generally employing total volume or crude regional comparisons. Accordingly, many of these studies yielded negative or somewhat inconsistent results. No between-group differences were reported in the corpus callosum (Hauser et al., 1989), in first-episode adolescent patients in total ventricular volume (Friedman et al., 1999) or thalamus (Dasari et al., 1999), and in ventricle-to-brain ratio (Risch et al., 1992). Temporal lobe showed differences only in lateralization (Johnstone et al., 1989), smaller volumes in the right hemisphere in male patients with schizophrenia compared to bipolar disorder or healthy subjects (Rossi et al., 1991), and no differences in the temporal poles (Kasai et al., 2003a), whereas temporal horns were reported enlarged in both patient groups (Roy et al., 1998). Reduced total cortical volumes were found in patients with schizophrenia in comparison to those with bipolar disorder (Harvey et al., 1994; Zipursky et al., 1997). The first application of fractal analysis for evaluation of differences in complexity (fractal dimension) of the boundary between the cortex and white matter reported the greatest complexity in bipolar disorder, followed by healthy subjects and the lowest fractal dimension in schizophrenia (Bullmore et al., 1994).

3.2. Structural neuroimaging

3.2.1. Cortical thickness and gyrification

Several studies compared cortical thickness and gyrification patterns between patients with bipolar disorder and schizophrenia, generally utilizing the open source FreeSurfer software package. Some of these reported widespread reductions in cortical thickness in patients with schizophrenia compared to those with bipolar disorder (Rimol et al., 2010, 2012; Godwin et al., 2018), others found more localized reductions in patients with schizophrenia in the medial frontal cortex (Doan et al., 2017), left rostral middle frontal and right inferior temporal gyri (Knöchel et al., 2016), and left planum temporale (Ratnanather et al., 2014), reduced laterality of the planum temporale in schizophrenia (Ratnanather et al., 2013) and increased sulcal width across lobes in adolescents with first-episode schizophrenia (Janssen et al., 2014). Compared to healthy subjects, both patients with schizophrenia and bipolar disorder showed similar cortical thinning in the pars opercularis of the inferior frontal gyrus and anteroposterior cingulate (Knöchel et al., 2016), hippocampus (Rimol et al., 2010), lateral occipital/early retinotopic cortex (individually localized by fMRI, Reavis et al., 2017b), and in the frontal cortex in first-episode adolescent patients (Janssen et al., 2014). On the other hand, Palaniyappan and Liddle (2014) found only a small overlap (25%) in gyrification changes between schizophrenia and bipolar disorder in comparison to healthy subjects. Higher gyrification in dorsal anterior/subgenual cingulate cortex was reported in patients with psychotic bipolar disorder than in schizophrenia patients and healthy subjects (Nenadic et al., 2015a), whereas another report found lower prefrontal gyrification in both patient groups (McIntosh et al., 2009). No study to date has yielded a region with lower cortical thickness in subjects with bipolar disorder than in patients with schizophrenia.

3.2.2. Voxel-based morphometry

Automated voxel-based morphometry tended to produce more between-group differences than less ubiquitous traced region-of-interest approaches. The vast majority of these studies found lower gray matter density in patients with schizophrenia than in those with bipolar disorder in widespread cerebral regions (see Maggioni et al., 2016 for an earlier review)—dorsolateral and medial prefrontal as well as precentral cortex (Molina et al., 2011; Nenadic et al., 2015b), thalamus (Molina et al., 2011; Nenadic et al., 2015b), frontotemporal cortex (Ivleva et al., 2012, 2013), hippocampus (Brown et al., 2011; Nenadic et al., 2015b), and amygdala (Brown et al., 2011), right posterior cingulate (where bipolar patients had even abnormally high density, De Castro-Manglano et al., 2011b), anterior cingulate (Yüksel et al., 2012; Doan et al., 2017), and putamen (Brown et al., 2011; Molina et al., 2011). Conflicting results have been obtained for the cerebellum and insula, where lower gray matter density was found in patients with schizophrenia by some researchers (Molina et al., 2011; Nenadic et al., 2015b; Amann et al., 2016; Doan et al., 2017) and in patients with bipolar disorder by others (De Castro-Manglano et al., 2011b). Several studies reported similar decreases in gray matter density in both patient groups in overlapping clusters in the superior temporal gyrus and inferior parietal lobule (Cui et al., 2011b; Chang et al., 2018), orbital cortex (Chang et al., 2018), posterior thalamus (Mamah et al., 2016), and throughout the neocortex (Ivleva et al., 2013). In line with the majority of these reports, a large study by Ivleva et al. (2017) concluded that both schizophrenia and bipolar disorder display overlapping endophenotypes in regional gray matter reductions, but they are more widespread in the former.

The largest voxel-based morphometry (with region-of-interest

follow-up analyses) study to date was conducted by Maggioni et al. (2017) using fused databases in Germany, Italy and Spain (a total of 802 subjects, including 243 with schizophrenia, 176 with bipolar disorder, and 383 healthy comparison subjects). This landmark study confirmed mostly overlapping gray matter reductions in both patient groups, so that regions affected in bipolar patients were also included into clusters affected in schizophrenia, with confined gray matter density changes in bipolar disorder and more widespread changes in schizophrenia. The authors found significant differences between two patient groups only in the right insula and its operculum and in the left thalamus.

3.2.3. Region-of-interest analyses

These approaches predate voxel-based morphometry, concentrate on more specific regional hypotheses with fewer statistical comparisons, and assess traced regional volumes rather than voxel density in template-registered images. As such, they may be more sensitive to volumetric differences in anatomical landmarks but are less suitable for assessment of differential patterns between comparison groups and of more confined clusters of volumetric differences. The methods therefore complement each other and region-of-interest approaches are often used for confirmatory follow-up analyses.

Smaller volumes in patients with schizophrenia than in those with bipolar disorder were found in the frontal, parietal (Zipursky et al., 1997), and temporal lobes (Rossi et al., 1991), insula (Kasai et al., 2003a), cerebellum (Laidi et al., 2015), amygdala (Altshuler et al., 2000; Mahon et al., 2012, 2015; but no differences in Mamah et al., 2010), pineal gland (Findikli et al., 2015), and hippocampus (Altschuler et al., 2000; Mamah et al., 2010; Radonić et al., 2011; Knöchel et al., 2014; Arnold et al., 2015), particularly its presubiculum and subiculum (Haukvik et al., 2015), entorhinal and parahippocampal cortex (Mathew et al., 2014). Smaller volumes in patients with nonpsychotic bipolar disorder than schizophrenia were reported in the caudate and globus pallidus (Womer et al., 2014), while no intergroup differences were found in the cingulate (Koo et al., 2008), thalamus (Womer et al., 2014; Skåtun et al., 2018), habenula (Schafer et al., 2018), and nucleus accumbens (Womer et al., 2014), or corpus callosum (Hauser et al., 1989; Walterfang et al., 2009).

3.2.4. Longitudinal studies

Longitudinal studies comparing patients with schizophrenia and bipolar disorder aim to uncover differences in trajectory of volumetric changes in each disorder over time. These differential trajectories may reflect developmental and maturational differences at early stages of their respective pathogeneses or differences in neurodegeneration and aging at later stages. This, along with the variation in follow-up scanning periods, renders longitudinal neuroimaging studies notoriously difficult to compare. Indeed, accelerated brain aging (as determined by the brain age gap estimation morphometric parameter in cross-sectional design) was reported in a direct comparison study in schizophrenia but not in bipolar disorder (Nenadić et al., 2017), or may at least be more pronounced in schizophrenia (Koutsouleris et al., 2014). Moreover, it has been generally postulated for schizophrenia rather than affective disorders (Kirpatrick et al., 2008; Kochunov et al., 2013), and supported in schizophrenia by some longitudinal studies (Mitelman et al., 2009; Schnack et al., 2016).

A series of 1.5-year follow-up reports from the same research group observed that transverse temporal gyrus, which was smaller in volume in patients with schizophrenia than with bipolar disorder at first hospitalization, continued to decrease in volume over time only in the former (Kasai et al., 2003b; Salisbury et al., 2007), along with longitudinal volume reductions in the planum temporale (Kasai et al., 2003b), other frontotemporal regions and concomitant enlargement of the lateral ventricles (Nakamura et al., 2007). A cingulate gyrus analysis over a 1.5-year period found diffuse volume loss over time in patients with first-episode schizophrenia compared to localized subgenual loss in patients with bipolar disorder (Koo et al., 2008). In the same vein, a smaller 2-year follow-up study found more circumscribed volume loss only in the anterior cingulate gyrus in patients with bipolar disorder, compared to more widespread changes in patients with schizophrenia (Farrow et al., 2005). Another early comparison of patients with schizophrenia and bipolar disorder over a 1.5-year follow-up period found no differential volume changes over time (Dickey et al., 2004).

Most of the longitudinal studies that followed confirmed more widespread volume reductions over time in schizophrenia than in bipolar disorder (see Liberg et al., 2016 for an earlier review)—in the interthalamic adhesion (over 13-month follow-up, Trzesniak et al., 2012), frontal lobe (2-year follow-up, Arango et al., 2012), frontal pole, superior, middle and inferior frontal gyri (1.5-year follow-up, Ohtani et al., 2018), insula (1.5-year follow-up, Lee et al., 2016), left precentral/postcentral cluster (2-year follow-up in first-onset adolescents, Castro-Fornieles et al., 2018). In this last, early-onset study, both schizophrenia and bipolar patients showed gray matter decreases over time in comparison to healthy subjects, but they were more widespread in schizophrenia; both patient groups also showed lesser expansion in white matter over time in comparison to healthy controls, more prominent in patients with schizophrenia (Castro-Fornieles et al., 2018).

Only a single contrasting study thus far reported longitudinal density reductions across cerebral lobes, thalamus, and cerebellum in first-episode patients with bipolar disorder and healthy subjects, but not in patients with schizophrenia (De Castro-Manglano et al., 2011a).

3.3. Diffusion tensor imaging

The first application of diffusion tensor imaging in direct comparison of patients with bipolar disorder and schizophrenia (McIntosh et al., 2008a), precisely a decade after its first use in psychiatry (Buchsbaum et al., 1998), found no between-group differences in integrity of the uncinate fasciculus and thalamic radiations (reviewed in O'Donoghue et al., 2017). Similarly, negative results have been obtained by a range of authors across the white matter (Cui et al., 2011a; Sui et al., 2011; Anticevic et al., 2015; Kumar et al., 2015; Ho et al., 2017; Squarchina et al., 2017; Chang et al., 2018) and in specific regions assessed (the anterior limb of the internal capsule, anterior thalamic radiations, uncinate fasciculus, Sussman et al., 2009; corpus callosum, Li et al., 2014 and Nenadić et al., 2017), some of which found that both patient groups had lower fractional anisotropy compared to healthy subjects (widespread decreases, Cui et al., 2011a and Chang et al., 2018; in the anterior thalamic radiation and uncinate fasciculus, Sui et al., 2011; corpus callosum, Li et al., 2014; ventral anterior cingulate, Anticevic et al., 2015; frontotemporal and callosal networks, Squarchina et al., 2017). Several authors suggested that overlapping decreases in white matter integrity in both schizophrenia and bipolar disorder may underlie their shared psychotic symptomatology (Anticevic et al., 2015; Squarchina et al., 2017).

Two studies reported significantly lower fractional anisotropy in patients with schizophrenia than bipolar disorder—in the superior temporal, parahippocampal and superior/middle occipital white matter (Anderson et al., 2013) and in the right inferior longitudinal and inferior fronto-occipital fasciculi (Tønnesen et al., 2018). In contrast, Lu et al. (2011) found lower fractional anisotropy in the cingulum, internal capsule, posterior corpus callosum, tapetum, and occipital white matter in untreated first-episode patients with bipolar disorder than schizophrenia. Ho et al. (2017) reported more pronounced lateralization abnormalities in bipolar disorder than schizophrenia in the cerebral peduncle and posterior limb of the internal capsule.

Morphometric approaches to white matter comparison between patients with schizophrenia and bipolar disorder have been scarce and reported no intergroup differences in the corpus callosum (Hauser et al., 1989; Walterfang et al., 2009), reduced callosal volumes in patients with bipolar disorder in comparison to schizophrenia and healthy

subjects (Tréhout et al., 2017), or overall decreased white matter density in both patient groups (Doan et al., 2017).

3.4. Functional imaging

The earliest positron emission tomography study comparing patients with schizophrenia and bipolar disorder detected flattened normal anteroposterior gradient in cortical glucose metabolism in both patient groups, primarily driven by absolute increases in posterior cortex rather than decreased rates in the frontal lobe (Buchsbaum et al., 1984). Another ^{18}F-fluorodeoxyglucose PET study comparing metabolic rates across several distributed cerebral networks found more widespread abnormalities in patients with schizophrenia, followed by acutely manic patients, with lowest abnormalities in depressed participants (Al-Mousawi et al., 1996). No differences in age-associated decline in D_2 dopamine receptors, as assessed with N-[^{11}C]methylspiperone PET, were found between patients with schizophrenia, bipolar disorder and healthy subjects (Wong et al., 1997). Similar reductions in regional cerebral blood flow in the left superior temporal gyrus during word generation (verbal fluency task) were found in asymptomatic patients with schizophrenia and bipolar disorder (Dye et al., 1999). These patient groups also showed comparable reductions in vesicular monoamine transporter concentrations in the ventral brainstem but lower radioligand binding potentials in schizophrenia than asymptomatic bipolar disorder were detected in the thalamus (Zubieta et al., 2001). A more recent PET report using the dopamine precursor fluorodihydroxyphenyl-L-alanine (^{18}F-DOPA) as radioligand to compare presynaptic striatal dopaminergic function in bipolar disorder and schizophrenia found no between-group differences in dopamine synthesis capacity in the striatum (Jauhar et al., 2017). Higher white matter glucose metabolic rates have been reported in patients with bipolar disorder than schizophrenia (Altamura et al., 2013; Shinto et al., 2015).

A detailed review of the more numerous fMRI studies (limited to reports published before 2011) concluded that overactivation of the medial temporal and limbic structures during tasks of emotional and memory processing in patients with bipolar disorder may differentiate them from patients with schizophrenia and define differences in neurobiological substrates of the two disorders (Whalley et al., 2012). Indeed, studies using emotional, reward or memory tasks showed greater activation in patients with bipolar disorder than schizophrenia in the hippocampus (Whalley et al., 2009; Hall et al., 2010), right parahippocampal and left medial frontal gyri (Mitchell et al., 2004), were less conclusive for the anterior cingulate (Mitchell et al., 2004; Whalley et al., 2009) and tended to show greater activation in schizophrenia patients in the prefrontal regions (Abler et al., 2008; Hall et al., 2010). Executive function and language tasks have been more commonly used and showed lower activation in patients with schizophrenia than bipolar disorder in the more posterior cortical regions (Curtis et al., 2001; McIntosh et al., 2008b) and greater activation in patients with schizophrenia in the prefrontal cortex, including Brodmann areas 32, 44, 45, 6 (Milanovic et al., 2011; Costafreda et al., 2009, 2011; Hamilton et al., 2009), with less conclusive results in area 9 (McIntosh et al., 2008b; Costafreda et al., 2011) and no significant differences in dorsolateral prefrontal and superior parietal regions (continuous performance task, Smucny et al., 2018). No differences between schizophrenia, bipolar and healthy subjects were found in the lateral occipital activation during neural tuning for object features of visually presented stimuli (Reavis et al., 2017a).

The fMRI comparisons using resting-state activity and default mode functioning paradigms tended to show greater resting activity in patients with bipolar disorder than schizophrenia in the temporal (Calhoun et al., 2008; Öngür et al., 2010), parietal and occipital regions (Öngür et al., 2010), but greater activity in patients with schizophrenia in the frontal cortex (Öngür et al., 2010; Chai et al., 2011). Resting-state functional connectivity was decreased in both patient groups, including the overlapping frontoparietal networks (Baker et al., 2014, but see opposite results in Skåtun et al., 2016), with possibly greater dysconnectivity in schizophrenia than bipolar disorder in the paracingulate gyrus and right thalamus (Argyelan et al., 2014), amygdala and dorsolateral prefrontal cortex (Liu et al., 2014), and cingulo-cerebellar network (Mamah et al., 2013), and with lower connectivity in patients with bipolar disorder than schizophrenia between the anterior insula and left middle frontal gyrus (Li et al., 2017a). No connectivity differences between two patient groups were revealed during explicit self or close other reflection (both groups showed reduced connectivity in several regions, Zhang et al., 2016). Aberrant thalamocortical connectivity patterns were found in both patients with schizophrenia (decreased intrathalamic and thalamo-frontoparietal) and bipolar disorder (increased thalamo-somatomotor, Skåtun et al., 2018).

3.5. Diagnostic classification

First attempt at using neuroimaging data for diagnostic classification between schizophrenia and affective disorders was by Al-Mousawi et al (1996), who stratified patients based on ^{18}F-fluorodeoxyglucose PET and network abnormalities into schizophrenia (with most abnormal networks), depression (with least abnormalities) and mania (in the intermediate position). Further attempts have steadily grown in sophistication and accuracy of classification, eventually adopting various computational and machine learning approaches.

In a small study, Pardo et al. (2006) reported 96% accuracy of classification by using linear discriminant analysis to ascribe adolescent subjects into three diagnostic groups (schizophrenia, bipolar, healthy) with a combination of structural MRI and neuropsychological data. Another study attempted to discriminate patients with schizophrenia and bipolar disorder with a combined fMRI (contrast maps) and diffusion tensor imaging data; the authors found that two diagnostic groups were separated by functional differences in the medial frontal and visual cortex and integrity of the fronto-occipital tracts (Sui et al., 2011).

Schnack et al. (2014) used voxel-based analysis of structural MRI data and support vector machine learning approach for diagnostic classification; the authors achieved high average accuracy of separation (88%) and determined that their discrimination was driven by differences in the superior frontal and parietal gray matter, and was more accurate with 3T as compared to 1.5T MRI scanner. In a large, data-driven fusion sample of 233 schizophrenia, 190 bipolar disorder, and 284 healthy subjects, Doan et al. (2017) reached 58% accuracy of classification using machine learning to identify combinations of morphological patterns of gray matter density and cortical thickness for diagnostic group discrimination. Palaniyappan et al. (2018) used a multimodal machine learning approach with a combination of effective connectivity (fMRI), voxel-based morphometry and clinical symptom scores in a small sample of patients with schizophrenia and bipolar disorder, and reported a 100% accuracy of discrimination.

4. Schizophrenia and major depressive disorder

Clinical overlap between negative symptoms in schizophrenia and core depressive symptomatology is sometimes cited as rationale for their comparison (Wei et al., 2017). In the case of major depressive disorder with psychotic features, the dimensional alternative to Kraepelinian dichotomy or interest in shared neural correlates of psychoses irrespective of their etiology underlie the comparative approach.

4.1. Structural neuroimaging

As early as in 1989, a computerized tomography study found that patients with schizophrenia had a greater ventricle-to-brain ratio than healthy subjects and that patients with major depressive disorder occupied an intermediate position, with no significant differences between the two patient groups (Rossi et al., 1989). In the first direct MRI

comparison of patients with first-episode schizophrenia and major depressive disorder (with and without psychotic features) as well as healthy participants, Salokangas et al. (2002) found smaller frontal lobe gray matter volumes and overall white matter volumes in patients with schizophrenia compared to all other diagnostic groups. Comparative white matter volume reductions in schizophrenia were also reported by Maller et al. (2012). A small longitudinal study of patients with schizophrenia and major depressive disorder with psychotic features, concentrating on subgenual prefrontal cortex, found smaller volumes in patients with major depressive disorder in the left posterior portion of the subgenual cortex both at baseline and at 2-8-year follow-up (Coryell et al., 2005). Smaller volumes in patients with schizophrenia than MDD were also reported in the pineal gland (Findikli et al., 2015) and callosal subregions (splenium, midportion as well as total mid-sagittal area), with no differences in total volume of the corpus callosum (Sun et al., 2009).

Other reports found that in comparison to patients with MDD, those with schizophrenia had reduced volumes of the hippocampus in both hemispheres (Meisenzahl et al., 2009) and specifically in its dentate gyrus (Ota et al., 2017), but contradictory results were obtained by Maller et al. (2012), who found lower-than-normal hippocampal volumes in patients with both schizophrenia and treatment-resistant MDD, yet they were significantly smaller in MDD than schizophrenia patients both in absolute measurements and relative to the total brain volume.

Chuang et al. (2014) evaluated correlations between negative symptoms and morphometric data in both patient groups: negative symptoms inversely correlated with cerebellar gray matter volumes in patients with MDD and with white matter volumes in the anterior limb of the internal capsule and anterior thalamic radiation in patients with schizophrenia. The authors concluded that negative symptoms in these two disorders are pathophysiologically distinct.

4.2. Functional neuroimaging

During the working memory tasks, schizophrenia participants failed to activate right dorsolateral prefrontal cortex whereas depressed patients activated both left and right dorsolateral prefrontal, inferior and superior frontal cortex (Barch et al., 2003), and right cerebellum (Walter et al., 2007); greater activation in patients with schizophrenia was observed in the temporal lobes, including the superior temporal gyrus (Walter et al., 2007). Patients with schizophrenia displayed lower activation in the right middle frontal gyrus (Brodmann area 9) than patients with MDD during a context processing task, which is associated with both working memory and attention (Holmes et al., 2005). Schizophrenia patients also recruited more areas than patients with MDD in the precentral and postcentral cortical region during the overnight motor memory consolidation following a finger-tapping task but displayed less pronounced overnight decreases in activation in the basal ganglia (Genzel et al., 2015).

During an unexpected reward receipt via a simulated slot-machine game, both patient groups showed lower-than-normal activation in the orbitofrontal, inferior temporal and occipital cortex as well as the ventral striatum, but schizophrenia patients showed significantly lower activation than patients with MDD in the medial frontal cortex (Segarra et al., 2016). Higher activations in patients with schizophrenia than MDD were recorded during mental arithmetic and vigilance tasks in the superior frontal and posterior cingulate cortex (Hugdahl et al., 2004).

Several studies assessed resting-state functional connectivity and found lower-than-normal but similar connectivity in both patient groups between amygdala and dorsolateral prefrontal cortex (first-episode patients, Wei et al., 2017), between the cuneus and bilateral superior parietal lobe (Schilbach et al., 2015), in salience and default mode networks (Chen et al., 2017), positive connectivity within the fronto-parietal networks (left and right dorsolateral prefrontal, superior parietal lobule, middle temporal gyrus, inferior parietal lobe) and negative connectivity between the left dorsolateral prefrontal, inferior parietal, middle temporal cortex and the visual network (cuneus, posterior precuneus, lingual and fusiform gyrus, Wu et al., 2017). Reduced functional connectivity in schizophrenia compared to MDD was reported between the precuneus/posterior cingulate and parietal operculum (Schilbach et al., 2015), between the medial prefrontal cortex and the basal ganglia/thalamocortical regions (before correction for multiple comparisons, Penner et al., 2016), interpreted by the latter authors as anomalous directed effort in patients with schizophrenia. Aberrant connectivity in opposite directions between patients with schizophrenia and MDD in relation to healthy subjects was reported between default mode and central executive networks as well as salience and central executive networks (enhanced in schizophrenia, reduced in MDD, Chen et al., 2017). Jiang et al. (2017) also found enhanced functional connectivity in patients with schizophrenia between default mode and central executive networks, but (in contrast to Chen et al., 2017)—enhanced connectivity in patients with MDD between salience and central executive networks.

7T MR spectroscopy was recently applied in direct comparison of patients with schizophrenia and major depressive disorder and found lower glycine concentrations in schizophrenia than MDD and lower myo-inositol concentrations in MDD than schizophrenia in the anterior cingulate cortex and thalamus (Taylor et al., 2017), whereas no intergroup differences were found in the anterior cingulate in a novel application of functional MR spectroscopy for dynamic monitoring of glutamate during performance of the Stroop task (Taylor et al., 2015).

4.3. Diagnostic classification

Ota et al. (2013) utilizing the stepwise discrimination analysis of volumetric and diffusion tensor imaging data in female patients with schizophrenia and MDD achieved 80% classification accuracy for schizophrenia and 76% accuracy for MDD based on the combination of lateral ventricular volumes, gray matter volumes (insula, thalamus, anterior cingulate cortex) and white matter integrity indices in the corpus callosum. Another structural MRI analysis of the multivariate pattern classification in 158 first-episode and recurrent schizophrenia and 104 major depressive disorder patients achieved correct diagnosis in 72% of patients with schizophrenia and 80% of patients with MDD, with 76% overall separability of these diagnostic groups based on the prefronto-temporo-limbic reductions and premotor-somatosensory and subcortical increases in schizophrenia compared to MDD (Koutsouleris et al., 2015). Recurrently ill patients had much lower rate of correct classification than those in the first outbreak, possibly due to the differential aging effects.

In the multiclass classification of fMRI resting-state connectivity patterns using intrinsic discriminative analysis Yu et al. (2013) reported diagnostic accuracy of 80.9%, based on the patient similarities in the medial prefrontal cortex, anterior cingulate, thalamus, hippocampus, and cerebellum, and on differences in the prefrontal cortex, amygdala, and temporal poles. Sasaki et al. (2010) attempted to measure neuromelanin content in dopaminergic and noradrenergic neurons using the fast spin-echo T_1 weighted 3T MRI but despite achieving good specificity showed relatively low sensitivity and were unable to differentiate participants with major depressive disorder from those with schizophrenia.

5. Schizophrenia and autism spectrum disorder

Nosological separation of autism spectrum disorder from the more inclusive concept of schizophrenia had been a gradual process (Sukhareva, 1925; Ssucharewa, 1926; Kanner, 1943; Asperger, 1944) until infantile autism was finally included in DSM-III in 1980. Since then it has been increasingly recognized that there are overlapping deficits in social functioning in these two disorders, ascribed to their

shared pathophysiological involvement of the so-called social brain (Craddock and Owens, 2010). Indeed, Stefanik et al. (2017), comparing endophenotypes based on diffusion tensor, morphometric and cognitive/demographic data in patients with schizophrenia, autism spectrum and bipolar disorders, found that ASD and schizophrenia patients tended to cluster in one group. A competing evolutionary hypothesis was advanced by Crespi and Badcock (2008), describing psychotic and autistic disorders as diametrically opposite extremes of the same spectrum. Paradoxical though it may sound, the two views are not mutually exclusive since distal evolutionary trade-offs (expressed in diametrical structural features of the brain) may proximally converge on the shared functional mechanisms (Crespi and Go, 2015; Mitelman et al., 2017). Both structural and functional neuroimaging, therefore, naturally lends itself to testing of these theoretical models.

5.1. Structural imaging

Despite the arguably solid rationale, studies with direct comparisons of patients with psychotic disorders and ASD have only begun to appear in the past several years. In a voxel-based morphometry study, reduced gray matter density in subjects with ASD in comparison to subjects with schizophrenia was detected in the left insula but surprisingly no differences were found between subjects with schizophrenia and healthy participants (Radeloff et al., 2014). In contrast (but in line with accumulated morphometric data for each of the disorders), Katz et al. (2016) reported greater gray matter volumes in subjects with high-functioning autism than in schizophrenia, with the opposite changes in the two patient groups in relation to healthy controls: increased prefrontal and anterior cingulate volumes in autism and decreased prefrontal and left temporal volumes in schizophrenia. Differences between healthy subjects and patients with schizophrenia in this study were more widespread than between healthy subjects and patients with ASD. Cortical thickening was also recently reported in patients with ASD as opposed to cortical thinning in schizophrenia (Park et al., 2018). In comparison of children with autism spectrum and bipolar disorders, volumes of the left anterior cingulate cortex were found to be smaller in patients with bipolar disorder than ASD (Chiu et al., 2008).

In a study that explicitly set out to evaluate the Crespi and Badcock hypothesis of diametrical relationship between autistic and psychotic disorders, Mitelman et al. (2017) observed that in patients with schizophrenia and ASD volumetric changes in both gray and white matter clustered in nearly identical regions but were in the opposite directions in relation to healthy comparison subjects: greater-than-normal in ASD and smaller-than-normal in schizophrenia in the gray matter, with reverse pattern in the white matter. In the gray matter, these patterns were seen in the total gray matter volume and in each cerebral lobe as well as the cingulate gyrus, with post-hoc localization to the polar, rostromedial and posterior frontal, postcentral, dorsal anterior cingulate, middle and superior temporal, fusiform, and primary visual occipital cortex. In the white matter, diametrical patterns were seen in the total white matter volume as well as the temporal, frontal and parietal lobes (in the latter two lobes primarily localizable to the precentral/postcentral region). As in the study by Katz et al. (2016), in relation to healthy subjects volumetric changes in this report were more pronounced in schizophrenia than ASD. Given the nearly identical topography and opposite direction of changes in these two disorders, the authors concluded that these findings were consistent with the Crespi and Badcock's proposal that from the evolutionary perspective schizophrenia and autism may represent the opposite extremes of the same phenotypic continuum, rather than a mere overlap in their impairment of the social brain.

5.2. Functional imaging

Several fMRI studies compared activation patterns in patients with ASD and schizophrenia, with specific interest in mechanisms that may underlie overlapping impairments in social functioning documented in both disorders. During visual perspective-taking task (evaluating social cognitive processing) greater reductions in activation were observed in patients with ASD than schizophrenia in the medial prefrontal cortex and at the temporo-parietal junction (Eack et al., 2017). Functional connectivity in this study was higher in patients with ASD than schizophrenia in the orbitofrontal cortex, despite similar impairments in task performance, suggesting that different mechanisms may underlie similar deficits in the two disorders. Similar conclusion was reached by Stanfield et al. (2017), who directly compared patients with ASD and schizotypal personality disorder during Ekman 60 faces task and found that schizotypal patients showed greater activation during social compared to gender judgments in the amygdala, posterior cerebellum, temporal and fusiform gyri. In a combined structural and fMRI comparison of cortical thickness and surface area and their alignment with functional resting-state connectivity in patients with ASD, ADHD and schizophrenia, Park et al. (2018) found that regional neuroanatomical deficits were enriched within the functional networks (frontoparietal and limbic for ASD and schizophrenia), rather than simply spatially overlapping with the functional deficits. Consequently, common deficits in these disorders may also arise from similar functional network involvement, rather than regional overlaps.

The only study that used ^{18}F-fluorodeoxyglucose positron emission tomography to compare patients with schizophrenia and ASD found opposite metabolic changes in each disorder in relation to healthy subjects in the primary motor and somatosensory cortex, anterior cingulate, and hypothalamus (Mitelman et al., 2018a). Similar metabolic changes in both patient groups were observed in the prefrontal and occipital cortex, inferior parietal lobule, hippocampus, amygdala, and basal ganglia. The authors viewed these overlaps as indicating that similar physiological mechanisms may be responsible for some of the shared social deficits in both patient groups. The same research group found increased white matter metabolism in both patients with schizophrenia and ASD in comparison to healthy subjects in all regions of interest included in the study (internal capsule, corpus callosum, and frontotemporal white matter), with the highest increases in the anterior limb of the internal capsule and prefrontal white matter (Mitelman et al., 2018b). In contrast to almost invariably more pronounced decreases in gray matter glucose metabolism in patients with schizophrenia than ASD, increases in white matter metabolic rates were more pronounced and more widespread in patients with ASD than schizophrenia. Given that increased white matter metabolic rates have been reported in schizophrenia (Buchsbaum et al., 2007), ASD (Mitelman et al., 2018b), Alzheimer's disease (Jeong et al., 2017) as well as bipolar disorder in comparison to schizophrenia (Altamura et al., 2013; Shinto et al., 2015), the authors proposed that this may be a common feature of all developmental and neurodegenerative disorders, reflecting disrupted connectivity and inefficient metabolic expenditures in these disorders.

In a resting-state fMRI study of adolescents with first-episode schizophrenia and ASD, Chen et al. (2017) using multivariate pattern analysis achieved 93% classification accuracy for schizophrenia and 80% for ASD, based on shared atypical connections in the default mode and salience networks.

In studies comparing children with high-functioning autism and DSM-IV Asperger's disorder, patients with high-functioning autism displayed smaller volumes in subcortical (globus pallidus, putamen, thalamus), precuneus and posterior cingulate gray matter (McAlonan et al., 2008) and greater white matter volumes around the basal ganglia, with both groups having greater volumes than healthy subjects (McAlonan et al., 2009). In a similar comparison of adults with high-functioning autism and Asperger's disorder, no significant intergroup differences were found in either gray or white matter volumes, with a trend towards greater white matter volumes in a region underlying auditory cortex in patients with Asperger's disorder (Mitelman et al.,

2017).

6. Schizophrenia and obsessive-compulsive disorder

The rationale for comparing these two disorders is the increased incidence of obsessive-compulsive symptoms in schizophrenia and existence of an obsessive-compulsive disorder with poor insight, which led to the description of a "schizo-obsessive disorder" (Poyurovsky et al., 2012, 2013; its imaging findings reviewed in Attademo et al., 2016) and more recently "schizo-obsessive spectrum disorders" (reviewed in Scotti-Muzzi and Saide, 2017), implying a dimensional overlap (Adler and Strakowski, 2003). The morphometric studies that compared patients with schizophrenia and obsessive-compulsive disorder reported similar reductions in hippocampal volumes in both groups (Kwon et al., 2003), no differences in total volumes of the thalamus (Kwon et al., 2003) but differential thalamic shape deformities—posteromedial in schizophrenia and anterior in OCD (Kang et al., 2008). Left amygdalar volume was increased in patients with obsessive-compulsive disorder but not in schizophrenia (Kwon et al., 2003). Using fractal dimension of the cortical surface as the measure of shape complexity in the whole brain and individual hemispheres, Ha et al. (2005) found the lowest fractal dimension in patients with schizophrenia, intermediate in patients with obsessive-compulsive disorder, and the highest in healthy subjects.

Using an fMRI during a task of auditory verb generation, Bleich-Cohen et al. (2014c) compared regional activation patterns during language processing in four diagnostic groups: patients with schizophrenia, obsessive-compulsive disorder, combined symptoms of OCD and schizophrenia ("schizo-obsessive"), and healthy subjects. They found language processing in patients with OCD to be similar to that in healthy subjects, whereas two schizophrenia groups displayed similar reductions in lateralization in the inferior frontal gyrus and diminished interhemispheric functional connectivity. The authors concluded that language processing in patients with combined symptoms of OCD and schizophrenia was similar to schizophrenia rather than OCD. The same authors described similarly disrupted activation patterns in patients with schizophrenia and schizo-obsessive disorder based on a working memory task (Bleich-Cohen et al., 2014a). Applying machine learning approach to diagnostic classification based on multivoxel analysis of fMRI under a working memory task, the authors were nonetheless able to separate two putative schizophrenia subgroups for correct classification (Bleich-Cohen et al., 2014b).

7. Autism spectrum and obsessive-compulsive disorder

Obsessive preoccupations, stereotyped and repetitive behaviors are core features of autism, yet neuroimaging comparisons with obsessive-compulsive disorder have been scant. In a series of fMRI comparisons of adolescent boys with obsessive-compulsive and autism spectrum disorders, Carlisi and coworkers set out to elucidate similarities and differences in neural mechanisms underlying stereotyped and compulsive behaviors intrinsic to both disorders. In one such experiment, regional activation patterns were compared during a gambling task (Carlisi et al., 2017a). Patients with ASD and OCD showed similar task performance (with the exception of perseverance deficits that were specific to ASD) and shared underactivation in the lateral inferior and orbitofrontal cortex and ventral striatum during decision-making. During reward-receipt part of the task, patients with ASD showed higher activation in the inferior striatum and insular regions in comparison to both patients with OCD and healthy subjects. During temporal discounting task, which also involves reward-based decision-making, shared reductions in activations in both patient groups were observed in the right ventromedial and lateral orbitofrontal cortex, posterior cingulate, precuneus, and cerebellum in delayed-immediate choices part of the task (Carlisi et al., 2017c). During immediate-delayed part, patients with ASD showed a trend towards lower activation than patients with OCD in the anterior cingulate, ventromedial prefrontal cortex, and left caudate (as well as greater choice-impulsivity), and both groups had lower activation than healthy participants.

During psychomotor vigilance task, involving sustained attention, patients with OCD exhibited decreased activation in the left insula and inferior frontal gyrus in comparison to both patients with ASD and healthy subjects, with no between-group differences in actual task performance (Carlisi et al., 2017b). Patients with OCD also showed an exaggerated normal response (more pronounced decreases in activation) with increasing attention load in inferior parietal lobe, superior and middle temporal gyri, and progressively increased activation in the anterior cingulate and medial prefrontal cortex, as compared to other diagnostic groups. The authors interpreted this latter finding as compensatory recruitment to attain comparable task performance.

In a multigroup diffusion tensor imaging study, children with autism spectrum disorder showed lowed fractional anisotropy compared to children with OCD in widespread cortico-cortical, interhemispheric and corticostriatal fibers (Ameis et al., 2016).

8. Attention-deficit/hyperactivity disorder comparisons

Attention-deficit/hyperactivity disorder is a frequent comorbidity in autism spectrum and conduct disorders, so that direct comparisons of pure, non-comorbid conditions may help to disentangle unique neural correlates of each of them. Relationship between ADHD and obsessive-compulsive disorder is less well understood and in children may be confounded by other comorbidities, in particular by tic disorders (Abramovitch et al., 2015).

8.1. Attention-deficit/hyperactivity and obsessive-compulsive disorders

Several fMRI studies from the same research group compared activation patterns during performance of various neuropsychological tasks in children with ADHD and OCD. During a tracking Stroop task, which measures inhibition and stopping failure, both patient groups shared dysfunction in the right orbitofrontal and left dorsolateral prefrontal cortices (Rubia et al., 2010a). During the inference inhibition condition of the Simon task, adolescent boys with ADHD and OCD shared underactivation in the anterior cingulate and superior parietal cortex (Rubia et al., 2011). During temporal discounting task (a reward decision-making assessment), shared deficits in activation were found in the fronto-striato-insular and cerebellar regions (Norman et al., 2017a). During decision-making under ambiguity, both groups displayed underactivation in the ventral striatum, while adolescents with OCD showed diagnosis-specific underactivation in the ventromedial orbitofrontal cortex during advantageous choices part of the task (Norman et al., 2018). During outcome evaluation part of this task, both groups shared underactivation to losses in the medial prefrontal cortex and to wins in the left caudate and putamen, while disorder-specific excessive activation to losses in boys with ADHD was seen in the right caudate and putamen, where healthy participants showed more activation to wins (Norman et al., 2018). Activation patterns during sustained attention showed shared deficits in adolescents with both ADHD and OCD—underactivation in the left insula and ventral inferior frontal gyrus, overactivation in the posterior default mode network (Norman et al., 2017b).

In a diffusion tensor imaging study, Ameis et al. (2016) reported lower fractional anisotropy in children with ADHD than OCD in the anterior thalamic radiation, genu of the corpus callosum, cortico-spinal tract, acruate and inferior fronto-occipital fasciculi.

8.2. Attention-deficit/hyperactivity and conduct disorders

Differences in fMRI activation patterns between boys with non-comorbid ADHD and conduct disorder have been investigated during several tasks of selective attention and inhibitory control. As compared

to patient with conduct disorder and healthy children, boys with ADHD exhibited underactivation in the inferior prefrontal cortex during a visual-spatial Switch task (cognitive flexibility/inhibitory control, Rubia et al., 2010b) and during the oddball condition in the Simon task (Rubia et al., 2009a), underactivation in the posterior cingulate during the reward condition of a rewarded continuous performance task, underactivation in the vetrolateral cortex and overactivation in the cerebellum during the sustained attention condition (Rubia et al., 2009b). Boys with noncomorbid conduct disorder in comparison to the other groups displayed underactivation at the temporoparietal junction during a tracking stop task (Rubia et al., 2008), in the orbitofrontal cortex during the reward condition of a rewarded CPT and in the insula, hippocampus, anterior cingulate, and cerebellum during the sustained attention condition (Rubia et al., 2009b).

The only morphometric study directly comparing these groups found widespread gray matter density reductions throughout the cortex and subcortical structures in children with conduct disorder (post-hoc estimated at 13% of the gray matter) and no differences between patients with ADHD and healthy subjects (Stevens and Haney-Caron, 2012).

8.3. Attention-deficit/hyperactivity and autism spectrum disorders

Lim et al. (2015) in a voxel-based morphometry study, reported lower gray matter density in the right posterior cerebellum in patients with ADHD than ASD. Two studies used diffusion tensor imaging to compare patients with ADHD and autism spectrum disorder. Chiang et al. (2017) reported lower fractional anisotropy in the latter group in the right arcuate fasciculus, right cingulum, anterior commissure and corpus callosum; patients with ADHD did not differ from healthy control subjects (Chiang et al., 2017). Ameis et al. (2016) found no differences in fractional anisotropy between the two patient groups.

Christakou et al. (2013) using fMRI observed underactivation in the left dorsolateral prefrontal cortex during a sustained attention task in both patients with ADHD and ASD, significantly more pronounced in the former group; increased cerebellar activity, as compared to both healthy and ADHD participants, was peculiar to patients with ASD. Lower activation in patients with ASD than ADHD during a temporal discounting task was reported in the left lingual and right inferior frontal gyri, whereas lower activation in patients with ADHD than ASD was recorded in the left inferior parietal, pre- and postcentral cortex (Chantiluke et al., 2014).

9. Bipolar and unipolar depression

Considerable interest in both structural and functional comparisons of bipolar and major depressive disorders stems primarily from difficulties in their clinical differentiation, particularly at first depressive episode in adolescents and young adults (Cardoso de Almeida and Phillips, 2013). Practical implications of reliable classification by means of neuroimaging are therefore hard to overestimate.

9.1. Structural and diffusion imaging

Since the first study directly comparing patients with bipolar and unipolar depression reported no differences in volumes of the thalamus (Caetano et al., 2001), several other comparisons found no differences in the volumes of the pituitary glands (both patient groups had larger-than-normal volumes, MacMaster et al., 2008), habenula (Savitz et al., 2011), amygdala (Sublette et al., 2016), left dorsolateral prefronal cortex and right supplementary motor area (both medication-naïve patient groups showed decreased volumes, Chang et al., 2018). With the inclusion of more regions of interest or using voxel-based morphometry, other researchers reported smaller volumes of the hippocampus and amygdala (Redlich et al., 2014) and lower gray matter density in the right inferior frontal gyrus (Cai et al., 2015) in bipolar than unipolar depression. Some reports highlighted the cingulate gyrus for differential changes in unipolar and bipolar depression (Matsubara et al., 2016), describing smaller midcingulate volumes in patients with bipolar depression (Cai et al., 2015) or, in contrast, smaller anterior cingulate gray matter volumes (Redlich et al., 2014) and anterior cingulate white matter volumes (MacMaster et al., 2014) in patients with unipolar depression.

Lower cortical thickness in patients with bipolar disorder than major depressive disorder has been reported in the left and right middle frontal gyrus (Lan et al., 2014; Niu et al., 2017), right lateral orbitofrontal region, pars triangularis, and lateral occipital cortex (Niu et al., 2017), precuneus and left inferior parietal lobule (Lan et al., 2014). Greater cortical surface area in patients with bipolar disorder was also reported in some of these and other regions—precuneus and inferior parietal lobule as well as precentral, superior parietal, and middle temporal cortex (Fung et al., 2015).

White matter evaluations using diffusion tensor imaging found lower fractional anisotropy in patients with bipolar than unipolar depression in the left (Versace et al., 2010) and right (Repple et al., 2017) superior longitudinal fasciculi and cingulum (Repple et al., 2017), corpus callosum (Repple et al., 2017; anterior only in Matsuoka et al., 2017), although Yamada et al. (2015) reported similar anisotropy reductions in the anterior corpus callosum in both patient groups. In a three-dimensional arterial spin labeling imaging study, Zhao et al. (2016) found lower diffusion kurtosis and perfusion values in the right superior cerebellar peduncle in unipolar than bipolar depression.

9.2. Functional imaging

The majority of the fMRI studies comparing patients with bipolar disorder and major depressive disorder concentrated on group differences in the processing of emotions (as reviewed in Han et al., 2018). In the first such study, that involved viewing mild and intense emotional faces with expressions of fear, happiness, sadness, and neutral, patients with bipolar disorder, as compared to both depressed and healthy participants, showed increased activation in the left amygdala in response to mild and neutral facial expressions (Almeida et al., 2010). Since then, several other authors have focused on the amygdala for differentiation between the affective disorders. Greater activation in patients with MDD than bipolar disorder was observed in the left amygdala as main region of interest in response to anger condition during implicit emotion-faces task (angry, fearful, sad, and happy), while the whole-brain analyses in the same study registered greater activation to sad faces in bipolar patients in the middle and superior temporal gyri, insula and parietal cortex, and in patients with MDD to angry, fearful, and happy faces in overlapping regions (Fournier et al., 2013). The authors interpreted these findings as mood-congruent increases in activation in patients with bipolar disorder and mood-incongruent overactivation in patients with MDD. When these subjects were evaluated for within-session adaptation to repeated performance of the same task, linear decreases in BOLD signal were observed in the left amygdala, right temporoparietal and fronto-insular regions in all diagnostic groups, but bipolar patients displayed greater linear decreases in posterior insula as compared to patients with MDD despite their comparable task performance (Fournier et al., 2016). Another study, specifically investigating amygdalar excitability during processing of the sad, happy and neutral faces found that amygdalar response to sad > neutral faces was increased in patients with MDD as compared to bipolar participants, and reverse was true for happy > neutral faces, with greater response in patients with bipolar disorder (notably, most activations in both diagnostic groups were abnormal, Grotegerd et al., 2014).

Bertocci et al. (2012) investigated the role of the anterior cingulate in the processing of emotional faces with distracters under different memory loads and reported that high memory loads with neutral face

distracters elicited greater activation in the dorsal anterior midcingulate in patients with MDD and greater putaminal activation in those with bipolar disorder. This was interpreted as suggestive of abnormal recruitment of attentional cingulate circuitry in patients with MDD. In another experiment, patients with bipolar disorder exhibited decreased activation to emotional faces in comparison to patients with MDD and healthy subjects in the middle occipital, lingual and middle temporal gyri (Cerullo et al., 2014). Adolescent patients with bipolar disorder displayed similarly lower activation in response to both intense happy (in the insula and temporal cortex) and intense fearful faces (precentral cortex) in comparison to adolescents with MDD (Diler et al., 2013); no differences between these patients were found during a response inhibition Go/NoGo task (Diler et al., 2014). In a comparison of patients in different phases of their illness, heightened activation to both happy and sad faces was seen in remitted patients with bipolar disorder (as compared to remitted patients with MDD and healthy subjects); in actively depressed patients differences in activation were confined to the posterior cingulate (Rive et al., 2015).

Activation studies aimed at investigation of reward processing circuitry have been less numerous and found no significant between-group differences in activation to social reward (Sharma et al., 2016), or reduced activation during reward expectancy in the anterior cingulate in both patients with bipolar disorder and MDD, whereas anticipation-related activation was heightened in the left ventrolateral prefrontal cortex in patients with bipolar disorder as compared to MDD and healthy participants (Chase et al., 2013). Lower activation in bipolar than unipolar patients during reward processing was described in the prefrontal cortex, insula, thalamus, caudate and putamen (Redlich et al., 2015). Manelis et al. (2016) in their study of anticipation-related brain connectivity during reward task found that participants with MDD and bipolar disorder differed in the density of connections, connectivity path length and direction as a function of win or loss in reward anticipation. Resting-state functional connectivity in reward networks was found to be higher in patients with bipolar than unipolar depression, and it was diminished in proportion to depression severity in both patient groups (i.e. possibly being a common feature of the depressed state, Satterthwaite et al., 2015).

In a motor activation task, patients with bipolar II disorder showed greater activation in the right posterior cingulate (used as the seed region) and right parietal/insular regions as compared to patients with MDD (Marchand et al., 2013). During a visuospatial planning (Tower of London) task increased activity was recorded in the frontostriatal circuits in depressed patients with bipolar disorder as compared to patients with MDD; visuospatial load in this task was associated with increased activity in the parietal cortex in depressed patients with bipolar disorder, followed by actively depressed patients with MDD, with the lowest activity in remitted unipolar depression (Rive et al., 2016a). During a working memory task, both unipolar and bipolar participants failed to deactivate the medial frontal cortex (part of the default mode network), and this failure was significantly more pronounced in patients with bipolar than unipolar depression (Rodríguez-Cano et al., 2017).

In the resting-state investigations, abnormal baseline brain activity was documented in both the unipolar and bipolar depression. Both groups showed reduced corticolimbic resting-state connectivity (Anand et al., 2009), with no intergroup differences in interhemispheric connectivity (Wang et al., 2015). Lower resting-state connectivity was reported in patients with bipolar disorder than MDD in the left insula and right frontopolar cortex, whereas lower connectivity in patients with MDD than in those with bipolar disorder and healthy subjects was found in the right amygdala and left anterior hippocampus (Ambrosi et al., 2017). Stronger resting-state connectivity in patients with bipolar disorder than MDD was also reported in the default mode network (in the medial prefrontal, posterior cingulate and right inferior parietal cortex, left hippocampus and right insula, Liu et al., 2015). Resting-state amplitude of low-frequency fluctuations (ALFF) was found to be lower in patients with bipolar than unipolar depression in the left superior parietal lobule and posterior insula (Liu et al., 2012) and higher in patients with bipolar disorder in the right dorsal anterior insula (Liu et al., 2012), left precuneus, middle temporal and lingual gyri (Qiu et al., 2018). Regional homogeneity differences in resting-state activity between the two diagnostic groups were documented in widespread cerebral regions, including the right ventrolateral middle frontal and parahippocampal gyri, posterior cingulate, dorsal and ventral anterior insula, cerebellum (Liu et al., 2013) as well as left insula and superior temporal gyrus (Yao et al., 2018). Wang et al. (2017a) used the graph theory approach to evaluate strength of the functional connectivity and reported lower connectivity in patients with bipolar than unipolar depression in bilateral precuneus and stronger connectivity in bipolar depression in the right middle temporal gyrus (long-range connectivity) and bilateral thalamus (short-range connectivity). Both groups showed reduced global efficiency, disrupted connectivity in default mode network and limbic system (Wang et al., 2017b). The main differences between the bipolar and unipolar participants with this novel approach were seen in the precuneus and the temporal pole (Wang et al., 2017b).

9.3. Diagnostic classification

The attempts to differentiate between bipolar and unipolar depression by means of neuroimaging have begun to appear only recently and have been predominantly based on functional MRI or combination data. Grotegerd et al. (2013) utilized regional activation in response to facial expressions of opposite valence and achieved 90% accuracy of diagnostic classification, with weighted contribution of the medial prefrontal and orbitofrontal regions in unipolar depression and the dorsolateral prefrontal cortex in bipolar depression. In addition, the authors found that in the amygdala heightened responses to negative faces were sensitive to unipolar depression and heightened responses to positive faces were sensitive to bipolar depression. Bürger et al. (2017) employed a multivariate classification scheme based on activation of the amygdala and anterior cingulate cortex in response to presentation of happy or fearful faces in conjunction with whole-brain analysis, attaining 72% classification accuracy weighted by the anterior cingulate response to fearful faces. Increased activation in response to fearful faces in the right frontal and parietal cortex was sensitive in bipolar depression, whereas reduced activation to both happy and fearful faces in the anterior cingulate was sensitive in unipolar depression.

Goya-Maldonado et al. (2016) also attained a strong diagnostic differentiation by using large-scale network analyses based on increased resting-state connectivity in the fronto-parietal networks in bipolar disorder and in the default mode network (specifically in the cingulo-opercular region) in unipolar depression. 86% overall differentiation accuracy was achieved by Li et al. (2017b) based on decreased connectivity in patients with bipolar disorder in the insula and increased connectivity in bilateral precuneus. He et al. (2016) employed a combination of the resting-state fusion network analysis of functional connectivity and voxel-based gray matter density to achieve a very high accuracy of diagnostic discrimination (99.5%). Another combined analysis of resting-state functional connectivity and structural MRI in actively depressed and remitted bipolar and unipolar patients achieved a moderately accurate discrimination (69.1%) in actively depressed patients only, concluding that current symptomatic status may have a strong impact on classification efforts (Rive et al., 2016b).

Only four published reports based their classification efforts on the structural data alone. Discriminant analysis of six regions of interest achieved 81% classification in one study (MacMaster et al., 2014) and a voxel-based morphometry pattern classification reported a 69% accuracy of classification when the discriminating algorithm was machine trained at one research site and tested at another (Redlich et al., 2014). Based on the cortical surface area and using support vector machine algorithm, Fung et al. (2015) reported 74.3% overall classification

accuracy. Wise et al. (2018) also employed voxel-based morphometry and machine learning to predict depressive and hypomanic symptoms based on the gray matter density patterns in a mix of patients with major depressive disorder and bipolar disorder with depressive and self-reported hypomanic episodes. Severity of both unipolar and bipolar depressive, but no hypomanic symptoms was indeed predicted by gray matter density in the anteroventral insula, and the authors concluded that the insula may thus be associated with depressive symptoms in both major depressive and bipolar disorders.

9.4. Other comparisons of major depressive disorder

In the single ^{18}F-fluorodeoxyglucose PET study with direct comparison of female patients with bulimia nervosa and major depressive episode during a continuous performance task, Hagman et al. (1990) found that bulimic patients displayed a less asymmetrical temporal lobe activity than patients with MDD (especially in the middle and inferior temporal gyri), whereas patients with MDD showed lower activity in the basal ganglia and thalamus (and especially in the left hemisphere). Both patient groups showed decreased metabolism in the medial frontal cortex, more pronounced in the right hemisphere, and increased lateralization in the occipital lobes, with no differences in the overall cortical or lobar metabolism.

Another study analyzed cortical thickness and gray matter density in patients with major depressive disorder and social anxiety (Zhao et al., 2017). The authors reported no differences in gray matter density or cortical thickness between groups, but both patient groups had overlapping reductions in density in the orbitofrontal cortex, putamen and thalamus in comparison to healthy participants. In addition, patients with major depressive disorder displayed greater-than-normal thickness in the superior frontal cortex and thinness in the left post-central cortex.

10. Eating disorders

With a single exception, both structural and functional comparisons of patients with eating disorders focused on anorexia and bulimia nervosa. In a direct voxel-based morphometry comparison, patients with bulimia nervosa had lower gray matter density than patients with anorexia in the cerebellum, fusiform gyrus, supplementary motor region, and occipital cortex (Amianto et al., 2013). Bulimic patients had greater density than both anorectic and healthy participants in the caudate, and both patient groups had greater-than-normal density in bilateral somatosensory cortex (Amianto et al., 2013). In another voxel-based morphometry study without direct comparison of two patient groups, patients with anorexia nervosa showed decreased gray matter density in the anterior cingulate, frontal operculum, precuneus and left temporo-parietal junction, whereas bulimic patients did not differ from healthy participants (Joos et al., 2010). An earlier study found no differences in gray or white matter volumes between any of the patient groups and healthy subjects (Wagner et al., 2006).

In a voxel-based morphometry comparison of patients with binge-eating disorder and bulimia nervosa of purging type, Schäfer et al. (2010) found that both patient groups had greater-than-normal volumes in the medial orbitofrontal cortex, but bulimic patients also displayed increased volumes in the ventral striatum.

A combined diffusion tensor imaging and fMRI study of patients with anorexia and bulimia nervosa reported that both patient groups showed increased DTI connectivity in the tracts connecting the insula, orbitofrontal region and ventral striatum, and decreased connectivity in the tracts subserving the orbitofrontal region, amygdala and hypothalamus (Frank et al., 2016). Functional (effective) connectivity in both patient groups went from the anterior cingulate to hypothalamus via ventral striatum, whereas reverse direction was observed in healthy subjects—from the hypothalamus to ventral striatum (Frank et al., 2016). Another combined diffusion-tensor and fMRI study, focusing on interhemispheric resting-state connectivity and the corpus callosum, found reduced voxel-mirrored homotopic connectivity in patients with anorexia nervosa in the cerebellum, insula, and precentral cortex, and in bulimia nervosa—in the dorsolateral prefrontal and orbitofrontal cortex (Canna et al., 2017). No intergroup differences in diffusion tensor imaging were seen in the corpus callosum. Lee et al. (2014) evaluated resting-state synchrony in patients with anorexia and bulimia nervosa centered on the anterior cingulate. The authors found stronger-than-normal synchronous activity in patients with anorexia nervosa between the dorsal anterior cingulate and retrosplenial cortex and in patients with bulimia nervosa—between the dorsal anterior cingulate and medial orbital cortex; both groups shared increased synchronization between the dorsal anterior cingulate and precuneus, which the authors viewed as reflective of eating preoccupations in both patient groups.

In an fMRI study of responses to pleasant and aversive taste stimuli in patients with anorexia and bulimia nervosa, Monteleone et al. (2017) found that, opposite to the patterns seen in healthy subjects, sucrose solution (sweet taste) induced higher activation in patients with anorexia than quinine solution (bitter taste), in particular in the left orbitofrontal cortex and caudate and right dorsolateral prefrontal cortex, anterior insula, thalamus and putamen. Patients with bulimia nervosa similarly displayed a stronger response to sweet than bitter stimuli, in contrast to responses observed in healthy subjects, in particular in the left dorsolateral prefrontal cortex, bilateral brainstem, and right post-central gyrus. Diminished response to bitter stimuli in patients with anorexia was observed in the right amygdala and left anterior cingulate, and in patients with bulimia—in the right amygdala and left insula.

11. Conclusions

The oft-stated transdiagnostic mantra notwithstanding, as seen in this review different diagnostic comparisons in neuroimaging appear to have been pursued with heterogeneous goals, supported by eclectic theoretical models and seldom explicitly focused on dimensionality. Some showed primary interest in similarities between disorders (overlapping social brain involvement in autism spectrum disorder and schizophrenia, common basis for psychosis across diagnostic entities), others—in their differences (bipolar and unipolar depression, diagnostic classification studies in general). Among the supporting theoretical models are such distinct hypotheses as the historical Kraepelinian dichotomy and evolutionary conceptualization of autism and psychotic spectra as opposite extremes of the same distribution of psychological traits in a population (trade-off diseases) by Crespi and Badcock (2008). Whether this miscellany of rationales and assumptions eventually fulfills the promise of transcending the limitations of current psychiatric nosologies remains to be seen, but it is inarguable that transdiagnostic comparisons greatly advance our understanding of physiological and neuroanatomical bases of the presently recognized psychiatric disorders.

Conflict of interests

The author has no conflicts of interest to declare.

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Supplementary materials

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