IMPORTANCE  Major depressive disorder (MDD) has been linked to imbalanced communication among large-scale brain networks, as reflected by abnormal resting-state functional connectivity (rsFC). However, given variable methods and results across studies, identifying consistent patterns of network dysfunction in MDD has been elusive.

OBJECTIVE  To investigate network dysfunction in MDD through a meta-analysis of rsFC studies.

DATA SOURCES  Seed-based voxelwise rsFC studies comparing individuals with MDD with healthy controls (published before June 30, 2014) were retrieved from electronic databases (PubMed, Web of Science, and EMBASE) and authors contacted for additional data.

STUDY SELECTION  Twenty-seven seed-based voxel-wise rsFC data sets from 25 publications (556 individuals with MDD and 518 healthy controls) were included in the meta-analysis.

DATA EXTRACTION AND SYNTHESIS  Coordinates of seed regions of interest and between-group effects were extracted. Seeds were categorized into seed-networks by their location within a priori functional networks. Multilevel kernel density analysis of between-group effects identified brain systems in which MDD was associated with hyperconnectivity (increased positive or reduced negative connectivity) or hypoconnectivity (increased negative or reduced positive connectivity) with each seed-network.

RESULTS  Major depressive disorder was characterized by hypoconnectivity within the frontoparietal network, a set of regions involved in cognitive control of attention and emotion regulation, and hypoconnectivity between frontoparietal systems and parietal regions of the dorsal attention network involved in attending to the external environment. Major depressive disorder was also associated with hyperconnectivity within the default network, a network believed to support internally oriented and self-referential thought, and hyperconnectivity between frontoparietal control systems and regions of the default network. Finally, the MDD groups exhibited hypoconnectivity between neural systems involved in processing emotion or salience and midline cortical regions that may mediate top-down regulation of such functions.

CONCLUSIONS AND RELEVANCE  Reduced connectivity within frontoparietal control systems and imbalanced connectivity between control systems and networks involved in internal or external attention may reflect depressive biases toward internal thoughts at the cost of engaging with the external world. Meanwhile, altered connectivity between neural systems involved in cognitive control and those that support salience or emotion processing may relate to deficits regulating mood. These findings provide an empirical foundation for a neurocognitive model in which network dysfunction underlies core cognitive and affective abnormalities in depression.
Major depressive disorder (MDD) is a psychiatric illness with devastating social, personal, and medical consequences. Moreover, MDD is ubiquitous, affecting more than 16 million people in the United States and 350 million people worldwide each year. Although significant progress has been made in understanding MDD and developing treatments, much is unknown about the pathophysiology of the disease, and rates of recurrence remain high. Exploring the neurobiological signature of MDD from new perspectives has the potential to transform current conceptualizations of the disease and sharpen the search for treatment targets.

Researchers have become increasingly interested in the role of abnormal communication among large-scale functional brain networks in the pathophysiology of MDD. Functional networks can be defined as distributed sets of brain regions that exhibit correlated activity at rest, that is, resting-state functional connectivity (rsFC), or during task performance. The recruitment of a highly synchronized network, in response to task demands or at rest, is believed to reflect distinct cognitive or emotional processes or mental states (eg, mind-wandering), although these relationships are complex and remain a rapidly evolving field of study. Of particular relevance are networks putatively related to processes affected in depression, such as the frontoparietal network (FN), involved in top-down regulation of attention and emotion; the default network (DN) and the dorsal attention network (DAN), involved in internally or externally oriented attention, respectively; and the affective network (AN) and the ventral attention network (VAN) (sometimes together called the salience network) involved in processing emotion or monitoring for salient events.

Abnormal communication within the FN may underlie deficits in cognitive control, which are commonly observed in depression and may contribute to symptoms such as difficulty concentrating or regulating emotions. Likewise, aberrant communication between the FN and DN may reflect ongoing rumination or an underlying bias for control systems to allocate resources toward internal thoughts at the cost of engaging with the external world. Hence, specific patterns of network dysfunction may contribute to core deficits in cognitive and affective functioning that are believed to underlie clinical symptoms.

Investigation of functional networks has surged in recent years, in particular in the domain of rsFC. Initial findings support the view that MDD is characterized by abnormal rsFC, but inconsistency in the location and nature of effects makes it difficult to unify this research. Variability across studies may emerge for several reasons, including small sample sizes or differences in the networks selected for study. For example, prior research using seed-based rsFC, the most common analytic strategy, varies considerably in the location of seed regions of interest (ROIs). Although a spatially extensive set of seed ROIs provides a comprehensive view of rsFC across the brain, organizing results into a coherent model of network functioning is challenging. A theoretically informed strategy for categorizing seed ROIs and related findings (eg, by the location of seed ROIs within functional networks) would help organize the diverse set of findings and allow for a direct test of replication across studies. Meta-analysis is arguably the most powerful tool for synthesizing this research because it is capable of evaluating whether effects are robust across differences in methodologic details and disentangling consistent effects from false-positive results. However, although rsFC abnormalities related to MDD have been reviewed, meta-analysis of this burgeoning literature has, to our knowledge, never been performed.

The present study aimed to fill this important gap by conducting a meta-analysis of seed-based rsFC studies and unifying findings in a neurocognitive model of depression. Primary analyses tested for consistency in the location of brain systems exhibiting depression-related hyperconnectivity or hypococonnectivity with seed ROIs, which in turn were categorized within a priori networks. On the basis of evidence for broad deficits in cognitive control in MDD, it was predicted that seed ROIs located within the FN would exhibit reduced connectivity with other areas of the FN. In addition, on the basis of the central role of ruminative, self-referential thinking in cognitive models of depression, it was predicted that seed ROIs located within the DN would exhibit increased connectivity with other DN regions and increased connectivity with prefrontal regions of the FN involved in directing attention. Secondary analyses tested whether rsFC abnormalities were moderated by seed anatomy or by demographic or clinical factors.

**Methods**

**Literature Search**

A comprehensive literature search was conducted in Web of Science, PubMed, and EMBASE for articles as of June 30, 2014, using the keywords rest(-ing), connect(-ivity), and depress(-ion, -ive). Manual searches were conducted within the reference sections of empirical and review articles and for publications that cited those articles. Original functional magnetic resonance imaging studies using whole-brain seed-based rsFC to compare individuals with MDD with a healthy control (HC) group were eligible for inclusion (other rsFC methods, such as independent components analysis, adopt a distinct statistical approach that cannot be aggregated with seed-based data). If a published study did not report whole-brain effects or did not provide seed ROI or peak effect coordinates, authors were contacted for this information. Exclusion criteria were as follows: (1) no HC group or no current MDD group; (2) non–seed-based method; (3) whole-brain results could not be retrieved or did not survive correction (meta-analyses of functional magnetic resonance imaging data test for consistency in the spatial location of significant effects across studies; thus, only studies that reported group differences in rsFC were eligible for inclusion); (4) entirely overlapping sample and seed ROIs reported in another publication; or (5) seed ROI or peak effect coordinates could not be retrieved (eFigure in the Supplement). Publications reporting on the same sample but using different seed ROIs were coded as a single study; publications in which distinct MDD groups were each compared with a single HC group were coded as distinct studies, and supplementary analyses were conducted to address the issue of partial nonindependence. These searches and inclusion criteria yielded a sample of 27 studies from 25 publications that reported on 556 individuals with MDD and 518 healthy controls in the HC group (eTable 1 and eTable 2 in the Supplement).
Data Extraction and Coding

The present meta-analysis was coordinate based, with coordinates reflecting the locations of significant group differences in functional connectivity at the time series level. Data extraction and coding included the following. First, coordinates for the center of mass of each seed ROI (91 seeds) and the peak of each significant between-group effect (346 effects) were extracted for each study and converted to Montreal Neurological Institute space as needed. If the seed ROI was an anatomical region from a mask or standard brain atlas, the center of mass was calculated to obtain a representative coordinate. Second, each seed ROI was categorized into a seed–network based on the location of its center of mass within a priori rsFC networks defined by a previous whole-brain network parcellation in 1000 participants (eTable 3 in the Supplement). This network parcellation was selected given its full coverage of cortex, cerebellum, and striatum; its definition in a large sample; its replication across an independent sample; and its close correspondence with networks derived from alternative rsFC analytic strategies and task-based patterns of coactivation.

Effects were also categorized based on the direction of effect (ie, hyperconnectivity or hypoconnectivity in MDD groups). In previous work, hyperconnectivity has been defined as larger positive or reduced negative rsFC in individuals with MDD compared with healthy controls; hypoconnectivity has been defined as larger negative or reduced positive rsFC in individuals with MDD compared with healthy controls. Because the distinction between enhanced and weakened connectivity was inconsistently reported in the studies reviewed, it was not possible to test these forms of rsFC abnormality separately. However, when reported in the original publication, patterns of abnormal rsFC related to stronger or weaker connectivity in individuals with MDD are noted in the Results.

Multilevel Kernel Density Analysis

Meta-analysis was performed using the multilevel kernel density analysis toolbox (http://wagerlab.colorado.edu), a Matlab (MathWorks) toolbox that incorporates tools from Statistical Parametric Mapping (http://www.fil.ion.ucl.ac.uk/spm/). Coordinates for peak effects from each study and seed-network comparison were convolved with a spherical kernel (r = 15 mm) and thresholded at a maximum value of 1, yielding an indicator map in which a value of 1 indicated a significant effect in the neighborhood and a value of 0 indicated no significant effect. Next, the density of effects across studies was computed by averaging the indicator maps, weighted by study sample size. The resulting density maps showed the proportion of studies in which hyperconnectivity or hypoconnectivity with each seed–network was observed in MDD within 15 mm of each voxel. Differences between density maps were calculated to test for directional effects (eg, either consistent hyperconnectivity or hypoconnectivity in MDD; unless otherwise noted, all effects were specific to one direction).

A Monte Carlo simulation was performed to establish the familywise error rate threshold used to correct for multiple comparisons. In this simulation, the locations of significant effects from indicator maps were randomized within a gray-matter mask in 15 000 iterations, yielding an estimate of the maximum density of effects predicted to occur by chance. A familywise error rate threshold of P < .05 was met when the density statistic exceeded the maximum null in 95% of the Monte Carlo maps. Density maps can be thresholded based on height (density at that voxel exceeds the maximum expected over the entire brain by chance) or extent (density at multiple contiguous voxels exceeds the maximum expected in a cluster of that size by chance). Because these thresholds provide complementary information, both are reported. Findings are discussed in terms of within-network abnormalities (effects fall within the same functional network as seed ROIs) or between-network abnormalities (effects fall outside the functional network in which seed ROIs are located).

Post Hoc Analyses

Three categories of post hoc tests were conducted. First, jackknife analyses were conducted to assess whether the inclusion of any partially nonindependent study disproportionately affected the results. To accomplish this, the density statistic for each significant cluster was iteratively recalculated leaving out each partially nonindependent study, and a χ² test or Fisher exact test was performed between the original density statistic and the leave-one-out density statistic. Because these analyses failed to reveal disproportionate effects of any individual study, results reported here include all studies. Second, Fisher exact tests were conducted to investigate whether a specific anatomical region contributed more strongly to a significant effect than other regions of the same network. Although the primary analytic approach of grouping regions into functional networks made meta-analysis possible by boosting power across studies, this network-level approach made the assumption that distinct regions within each functional network show similar abnormalities in MDD. Therefore, post hoc region-level analyses were conducted by calculating the likelihood of a particular effect for seeds in distinct anatomical regions of a functional network and testing the difference in effect likelihood among regions. Third, analyses were performed to investigate moderation of effects by clinical and demographic factors (eTable 1 in the Supplement), including severity of depression (mild, moderate, or severe), medication status (medication use or no use of medication in the MDD group), or age (teen, adult, or elder). For these analyses, the proportion of studies within each clinical or demographic group reporting the effect was calculated, and differences in proportions were tested between groups.

Results

Within- Network Abnormalities

Hypoconnectivity Within the FN

Major depressive disorder was associated with hypoconnectivity between the FN seeds and bilateral posterior parietal cortex (PPC), regions involved in attending to goal-relevant stimuli or features of an internal representation (Figure 1A and Table). Examining the original empirical studies revealed that, when reported, hypoconnectivity was related to weaker positive connectivity between the FN seeds and the PPC. Specifically, the FN seeds in the dorsolateral prefrontal cortex (DLPFC) or cerebel-
lum exhibited hypoconnectivity with the PPC, and post hoc testing indicated that seeds in the DLPFC were more likely than cerebellar seeds to exhibit hypoconnectivity with right PPC (likelihood ratio, 5.29; \( P = .04 \)), although no differences were detected for the left PPC (\( P = .53 \)). Hypoconnectivity within the FN was not moderated by age, depression severity, or medication status (\( P > .05 \) for all; eTable 4 and eTable 5 in Supplement).

### Hyperconnectivity Within the DN

Major depressive disorder was characterized by hyperconnectivity between the DN seeds and regions of the hippocampus extending to the middle temporal gyrus and prefrontal areas of the medial prefrontal cortex (MPFC) (Figure 1B). These areas are believed to support internal mentation (eg, self-referential thinking and affective decision making).61 When reported in the original studies, within-DN hyperconnectivity was related to enhanced positive connectivity in MDD (\( P > .05 \) for all; eTable 4 and eTable 5 in Supplement). Neither age nor depression severity predicted DN hyperconnectivity (\( P > .05 \); eTable 4 and eTable 5 in Supplement), although trends emerged for greater likelihood of hyperconnectivity in unmedicated MDD between the DN seeds and the hippocampus (likelihood ratio, 6.01; \( P = .09 \)) or the MPFC (likelihood ratio, 3.18; \( P = .12 \)).

### Between-Network Abnormalities

#### Altered Connectivity Between the FN and Regions of the DAN or DN Involved in Externally or Internally Oriented Attention

As reported above, MDD was associated with weaker rsFC between the FN seeds and regions of bilateral parietal cortex; these clusters extended to regions of the superior parietal lobule involved in attending to perceptual cues in the environment60 that fall within the DAN (Figure 1A). In addition, MDD was associated with hyperconnectivity between the DN seeds and a region of left DLPFC believed to be critical for goal-directed regulation of attention and emotion60,62-64 (Figure 1B). When reported, DN hyperconnectivity with lateral prefrontal regions was predominantly related to enhanced positive32,34,45 but also weaker negative31 connectivity in MDD. No differences were detected among anatomical regions of the DN in the likelihood of hyperconnectivity with DLPFC (\( P > .05 \); eTable 4 and eTable 5 in Supplement), and effects were not moderated by clinical or demographic variables (\( P > .05 \); eTable 4 and eTable 5 in Supplement).

#### Altered Connectivity Between the AN and Regions of the DN Involved in Mediating Emotion Regulation

Hypoconnectivity was observed between the AN seeds and regions of the MPFC involved in mediating emotion regulation65 (Figure 1C). When reported, hypoconnectivity was related to both weaker positive (between the nucleus accumbens and the

---

### Table. Results of the Meta-analysis of Resting-State Functional Connectivity in Major Depressive Disorder

<table>
<thead>
<tr>
<th>Seed-Network (and Thresholding)</th>
<th>Seed Anatomy</th>
<th>Effect Network</th>
<th>Effect Anatomy</th>
<th>x</th>
<th>y</th>
<th>z</th>
<th>Voxels</th>
<th>Maximum P</th>
</tr>
</thead>
<tbody>
<tr>
<td>FN</td>
<td>Caudate, cerebellum, DLPFC</td>
<td>FN</td>
<td>Right PPC</td>
<td>44</td>
<td>50</td>
<td>162</td>
<td>.54</td>
<td></td>
</tr>
<tr>
<td>MDD &gt; HC (hb)</td>
<td>ACC, caudate, cerebellum, hippocampus, IPL, MPFC, MTG, PCC</td>
<td>DN</td>
<td>Right hippocampus extending to MTG</td>
<td>38</td>
<td>30</td>
<td>6</td>
<td>148</td>
<td>.30</td>
</tr>
<tr>
<td>MDD &gt; HC (eb)</td>
<td>DN</td>
<td>MPFC</td>
<td>-2</td>
<td>38</td>
<td>12</td>
<td>7456</td>
<td>.20</td>
<td></td>
</tr>
<tr>
<td>MDD &gt; HC (hb)</td>
<td>FN</td>
<td>Left DLPFC</td>
<td>-38</td>
<td>34</td>
<td>36</td>
<td>1125</td>
<td>.18</td>
<td></td>
</tr>
<tr>
<td>MDD &gt; HC (eb)</td>
<td>FN</td>
<td>Left DLPFC</td>
<td>-34</td>
<td>34</td>
<td>36</td>
<td>1125</td>
<td>.18</td>
<td></td>
</tr>
<tr>
<td>MDD &lt; HC (eb)</td>
<td>VAN</td>
<td>Midcingulate extending to thalamus and putamen</td>
<td>-2</td>
<td>4</td>
<td>22</td>
<td>2476</td>
<td>.26</td>
<td></td>
</tr>
<tr>
<td>AN</td>
<td>ACC, amygdala, NACC</td>
<td>DN</td>
<td>MPFC</td>
<td>-2</td>
<td>46</td>
<td>16</td>
<td>3118</td>
<td>.37</td>
</tr>
<tr>
<td>MDD &lt; HC (eb)</td>
<td>ACC, cerebellum, insula, putamen</td>
<td>FN, DN, visual network</td>
<td>Precuneus extending to PCC and occipital cortex</td>
<td>18</td>
<td>-66</td>
<td>34</td>
<td>6194</td>
<td>.32</td>
</tr>
</tbody>
</table>

Abbreviations: ACC, anterior cingulate cortex; AN, affective network; DAN, dorsal attention network; DLPFC, dorsolateral prefrontal cortex; DN, default network; eb, extent based; FN, frontoparietal network; hb, height based; HC, healthy control; MDD, major depressive disorder; MPFC, medial prefrontal cortex; MTG, middle temporal gyrus; NACC, nucleus accumbens; VAN, ventral attention network. *Coordinates are Montreal Neurological Institute standard stereotaxic spaces. Voxels indicate the number of 1×1×1 mm3 voxels. Maximum P is the maximum proportion of studies exhibiting the effect at the peak density weighted by sample size.*
Figure 1. Meta-analysis of Abnormal Resting-State Function Connectivity (rsFC) in Major Depressive Disorder (MDD)

Shown are seed regions of interest categorized by a priori functional network and brain regions in which abnormal rsFC was observed in MDD compared with healthy control (HC) individuals. A, Individuals with MDD exhibited hypoconnectivity within the frontoparietal network (FN) between the FN seeds and the posterior parietal cortex (PPC) and hypoconnectivity between the FN seeds and a region of superior parietal lobule (SPL) within the dorsal attention network (DAN). B, MDD was associated with hyperconnectivity within the default network (DN) between the DN seeds and the medial prefrontal cortex (MPFC) and hippocampus and hyperconnectivity between the DN seeds and the dorsolateral prefrontal cortex (DLPFC), a key hub of the FN. C, MDD was linked to hypoconnectivity between seeds in the affective network (AN) and regions of the MPFC. D, MDD was related to hypoconnectivity between the ventral attention network (VAN) seeds and the precuneus extending to the occipital and posterior cingulate cortex (PCC), although post hoc analyses also indicated hyperconnectivity between the VAN and posterior regions. Shown here are results of both height-based (hb) thresholding (proportion of studies reporting an effect at that voxel exceeds chance) and extent-based (eb) thresholding (proportion of studies reporting an effect at contiguous voxels exceeds chance). All results are significant at $P < .05$, corrected for familywise error rate.
and enhanced negative (between the amygdala and the MPFC) connectivity. The likelihood of MPFC hypoconnectivity did not differ among anatomical regions of the AN ($P > .05$; eTable 4 in Supplement) and was not moderated by clinical or demographic variables ($P > .05$; eTable 5 in Supplement).

Altered Connectivity Between the VAN and Regions of the FN or DN
Major depressive disorder was linked to hypoconnectivity between the VAN seeds and regions of the precuneus extending to the occipital and posterior cingulate cortex (Figure 1D), a functionally diverse set of regions involved in visual attention and internal thought.60,61 There was no difference in likelihood of hyperconnectivity vs hypoconnectivity, suggesting generally abnormal connectivity between the VAN and posterior systems. Hypoconnectivity was also observed between the DN seeds and a region of the midcingulate extending to the thalamus and putamen (Figure 1B), areas involved in relaying information about salience and somatosensation.13,36 When reported, such hypoconnectivity was related to weaker positive connectivity in MDD.26,34 Post hoc analyses failed to reveal differences among anatomical seeds in the likelihood of abnormal rsFC ($P > .05$; eTable 4 in Supplement) or moderation by clinical or demographic variables ($P > .05$; eTable 5 in Supplement).

Discussion
The present study provides the first meta-analytic evidence, to our knowledge, that individuals with MDD exhibit abnormal connectivity within and between brain networks involved in internally (DN) or externally (DAN) oriented attention, processing of emotion (AN) or salience (VAN), and goal-directed regulation of these functions (FN) (Figure 2). These findings motivate a neurocognitive model in which network dysfunction is tightly linked to deficits regulating attention and mood.6,7,23 In this model, reduced coordination among brain systems critical for cognitive control and altered communication between such control systems and other networks engaged for internal thought or emotional regulation may underlie the biased cognitive style and persistent negative mood that characterize MDD.

Reduced connectivity was observed in individuals with MDD among frontoparietal systems involved in cognitive control, and imbalanced connectivity was observed between control systems and regions engaged for externally directed attention or internal mentation. These findings converge with theoretical models in which depression is defined by the tendency to become mired in negative rumination,24 which in turn stems from abnor-
Two general patterns emerged in this meta-analysis. First, the sources of abnormal connectivity within seed-networks tended to be spatially distributed, highlighting the importance of considering anatomical regions within functional networks. However, given the low frequency of any single seed ROI implemented across studies, the absence of anatomical specificity should be interpreted with caution. Second, network abnormalities were similar across demographic and clinical groups. However, these analyses could only compare differences in the likelihood (but not magnitude) of network abnormalities between clinical or demographic groups and only for groups that were consistently identified across the original studies. Future studies investigating additional clinical constructs will provide a more nuanced view of rsFC in depression.

Several limitations warrant attention and suggest directions for future research. First, the present meta-analysis was necessarily limited to seed-based rsFC studies and seed ROIs selected by those studies (eTable 3 in the Supplement). Hence, particular networks and anatomical regions were better represented than others. In addition, it was not possible to include findings from studies that adopted alternative analytic methods (eg, independent components analysis). Because relatively few prior studies have implemented these methods with MDD samples (eTable 2 in the Supplement), separate meta-analyses for each analytic approach could not be conducted. However, as this literature increases, an important next step will be to test the replicability of rsFC abnormalities across other analytic methods and network parcellations.

Second, because rsFC is a rapidly evolving field, standards for data acquisition and processing varied considerably among the studies reviewed here (eTable 6 in the Supplement). Differences in motion correction or instructions to rest with eyes open vs closed may substantially affect results. Unfortunately, it was not possible to test the moderating effects of such variables because of the low frequency of studies within methodologic categories, but these effects merit future investigation.

Third, an important question unanswered by the present meta-analysis is the extent to which aberrant functional connectivity could be related to structural abnormalities. For example, decreased cortical thickness has been associated with altered functional connectivity in depressed adults. Future studies that integrate structural and functional perspectives may provide a more comprehensive view of neurobiological abnormalities in mood disorders.

Fourth, it is unclear to what extent depression-related abnormalities in rsFC would persist during performance of other tasks. Resting-state functional connectivity appears to reflect both static (eg, related to anatomical connections) and dynamic (eg, related to changing goals or states of arousal) components, but the precise contribution of these components to rsFC is unknown. Abnormal rsFC in MDD may be a transient consequence of internally biased attention, related to ruminating while resting in the scanner, rather than a persistent cause for biased or poorly controlled attention when engaged in other tasks. To disentangle these non-mutually exclusive possibilities, studies will be required that compare network functioning at rest and during tasks that challenge attention and mood regulation.
Conclusions

To our knowledge, this study provides the first meta-analytic evidence of large-scale network dysfunction in MDD, including imbalanced connectivity among networks involved in regulating attention to the internal or external world and decreased connectivity among networks involved in regulating or responding to emotion or salience. These findings are consistent with a neurocognitive model of MDD in which abnormal communication among functional networks may mediate the core cognitive and affective biases that characterize this serious disorder.

ARTICLE INFORMATION

Submitted for Publication: January 3, 2015; final revision received January 3, 2015; accepted January 20, 2015.

Published Online: March 18, 2015. doi:10.1001/jamapsychiatry.2015.0071

Author Contributions: Drs Kaiser and Pizzagalli had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

Study concept and design: Kaiser, Wagner, Pizzagalli.

Acquisition, analysis, or interpretation of data: All authors.

Drafting of the manuscript: Kaiser, Andrews-Hanna.

Critical revision of the manuscript: All authors.


Obtained funding: Kaiser, Pizzagalli.

Administrative, technical, or material support: Andrews-Hanna, Pizzagalli.

Study supervision: Wagner, Pizzagalli.

Conflict of Interest Disclosures: Dr Pizzagalli has reported receiving honoraria and consulting fees from Advanced Neuro Technology North America, AstraZeneca, Otsuka Pharmaceutical, Pfizer, and Servier for activities unrelated to this project. No other disclosures were reported.

Funding/Support: This project was partially supported by grants R01 MH068376 and R01 MH101521 (Dr Kaiser), R01 MH101521 (Drs Wagner and Andrews-Hanna), and R01 MH076136 (Drs Wagner and Andrews-Hanna) from the National Institute of Mental Health and by The Phyllis and David S. Kaiser Mental Health Research, McLean Hospital, Belmont, Massachusetts, provided interrater checks on the database. No compensation was provided. We thank the authors of the included studies, with special thanks to authors who generously shared unpublished data from whole-brain analyses and seed ROI and peak coordinates for inclusion in this meta-analysis.

Additional Contributions: Franziska Goer, BA, Center for Depression, Anxiety, and Stress Research, McLean Hospital, Belmont, Massachusetts, provided interrater checks on the database. No compensation was provided. We thank the authors of the included studies, with special thanks to authors who generously shared unpublished data from whole-brain analyses and seed ROI and peak coordinates for inclusion in this meta-analysis.

REFERENCES


3. Substance Abuse and Mental Health Services Administration. Results From the 2012 National Survey on Drug Use and Health: Mental Health Findings. Rockville, MD: Substance Abuse and Mental Health Services Administration; 2013.


Meta-analysis of Network Dysfunction in Depression

Original Investigation Research


