

## White Paper: Brain Scan Research

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Recently, there have been intense debates and controversies within the mental health field regarding the validity of the current diagnostic system, how to best work with individuals in distress, and what constitutes "mental illness." The British Psychological Society (British Psychological Society, 2013) and the International Society for Ethical Psychology & Psychiatry (ISEPP, May, 2013) both have issued statements asserting that "mental illnesses" are best conceptualized as problems in living that result from traumatic and societal ills which overwhelm one's capacity to cope, and that "treatment" should be based in a psychosocial framework that honors individuality. On the other hand, the mainstream mental health field, and biological psychiatry in particular, in conjunction with various political and corporate powers, is pushing further in a direction of conceiving emotional distress as a brain disease; a direction often referred to as "biological reductionism".

The most prominent movement within biological psychiatry is calling for more brain science and a shift from a system of many discrete mental disorders based upon somewhat arbitrary lists of symptoms to a system that retains the framework of discrete mental disorders but shifts the diagnostic criteria to differentiations within brain activity and/or brain structure. Conversely, others have come to believe that applying further funding and resources towards strengthening a disease-based diagnostic system of "mental illnesses," rather than psychosocial interventions and prevention methods, is profoundly misguided and potentially detrimental to those vulnerable individuals mental health professionals are purportedly trying to help.

Much of this controversy began with the newest edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association, 2013), the DSM-5. This greatly expanded version of the DSM is the latest series of ongoing revisions to a diagnostic system that has emerged from a tremendous effort spanning over 150 years to classify and categorize subjective human distress. Controversially, at the time of DSM-5's release in April 2013, Thomas Insel, director of the National Institute of Mental Health (NIMH) released a statement saying that the DSM's "weakness is its lack of validity" and that "patients with mental disorders deserve better." This was part of an announcement proclaiming a shift in funding research supported by the NIMH to be centered on the Research Domain Criteria (RDoC). This is proposed to "transform diagnosis by incorporating genetics, imaging, cognitive science, and other levels of information to lay the foundation for a new classification system . . . not constrained by the current DSM categories" (Insel, 2013). The Chairman of the DSM-5 responded: "We've been telling patients for several decades that we are waiting for biomarkers. We're still waiting" (Kupfer, 2013). This statement by Dr. Kupfer strongly questions the continued narrow focus on thus-far non-existent biomarkers, particularly when it

is done so at the expense of other frameworks even if those approaches (i.e., the DSM) are flawed.

Also in April 2013, United States President Barack Obama announced the launch of the BRAIN initiative. This federally funded program, which has over \$100 million committed to it, mostly by pharmaceutical companies and companies that design brain scanning technologies, was established in order to support "the development and application of innovative technologies that can create a dynamic understanding of brain function" (see [www.whitehouse.gov/brain](http://www.whitehouse.gov/brain)). Similar initiatives are being developed in other countries throughout the world (see [www.humanbrainproject.eu](http://www.humanbrainproject.eu)).

These events show a powerful movement towards a new system of mental disorder diagnoses based on biomarkers and brain scans that follows in the footsteps of traditional medical diagnoses. This line of research has been promoted as an exciting step towards a more scientifically advanced understanding of human suffering and behavior.

The motivation driving this movement is understandable. It is clear that many people experience severe forms of psychological distress, many of whom experience significant torment and disability as a result. It is also clear that the current system is doing a poor job of adequately addressing this problem. The hope for many is that a brain-based approach will provide a more evidence-based understanding of these disorders. Further, a brain-based approach to sometimes difficult-to-understand behaviors and experiences of those in extreme distress may be seen as a desperate effort to explain such phenomena without blaming anybody or insisting that someone "just get over it." By attributing "mental illness" to biology, it may be thought that stigma surrounding mental health difficulties may be reduced. Additionally, brain-based accounts of mental illness could allow for the creation of more effective psychopharmacological treatments and to identify those who have the most need for our limited resources. Lastly, when professionals charge insurance companies and federal agencies for reimbursement for services rendered, people want to know that the money is being spent effectively.

The problem with this approach, however, is that the brain-based initiatives for clinical research rely on a disease model that is based on erroneous logic, a faulty reductionistic view of human nature, and a contradiction of the most robust research findings within the mental health field. The brain research conducted thus far actually appears to indicate that most of the conditions referred to as "mental illnesses" are likely otherwise healthy adaptive processes in response to extreme environmental experiences. So while it appears that such adaptive processes often do correlate with changes within the brain, and that they may lead to certain long-term problems for the individual, these changes do not necessarily signify biological disease. Furthermore, brain research has ironically reinforced the benefits of certain psychosocial interventions, such as yoga, meditation, and psychotherapy, thereby negating the assumption that the resolution of such distressing conditions requires psychopharmacological or other related biological interventions.

Yet in spite of the robust evidence pointing to the importance of psychosocial factors in both the onset and the resolution of distressing psychological conditions, we find the mainstream mental health field pursuing biologically-based research ever more doggedly while increasingly neglecting psychosocial research and support. While it may at first appear counterintuitive that the field continues down a path that contradicts much of the recovery research, the reasoning for it becomes clearer if we keep in mind that this path is strongly encouraged by powerful vested interests (Whitaker & Cosgrove, 2015) and that people are suffering and we want answers that are unambiguous and easily implemented. Regardless of the reasoning, the fact remains that in spite of the great technological advances within the emerging brain-based initiatives, because they retain the same flawed core assumptions that gave rise to the DSM system—particularly conflating psychological distress and/or brain anomalies with biological disease—it is likely that they will leave us with little more than the further entrenchment of these flawed assumptions to the detriment of many. So while the ongoing research of neurological and genetic correlates of psychological distress may be valuable as an academic exercise, we fear the consequences that are clearly implicated in this pursuit.

### Current Findings and Flawed Assumptions

The last several decades have seen hundreds of studies reporting discoveries correlating brain activity with human emotion, behavior, and experience. Yet, the general consensus is that there has yet to be any replicable findings that show distinct differences or abnormalities associated with any DSM category, as evidenced by this statement by Thomas Insel: "We cannot design a system based on biomarkers or cognitive performance because we lack the data... This is what we have been doing for decades when we reject a biomarker because it does not detect a DSM category" (Insel, 2013). Although reports continue to be published suggesting associations between particular brain activity and specific disorders, these fail to hold significance when looked at in the aggregate across diagnostic categories. Further, such findings fail to delineate between those individuals who have experienced trauma and chronic stress, particularly childhood trauma, but who do not have clinically significant "symptoms," and those who are significantly disabled by such distressing conditions.

For instance, it is often said that "schizophrenia" is known to be a debilitating, life-long, brain-based disease. Studies show significantly reduced brain volume in particular areas of the brain in individuals diagnosed with schizophrenia, (e.g., Douaud, et al., 2007). However, these same abnormalities are also shown more generally to be directly associated with severe stress in early childhood (Howell, et al., 2013), severe emotional deprivation in early childhood (Eluvathingal, et al., 2006), and/or chronic social stress and discrimination (Akdeniz, Tost, Streit, & Haddad, 2014). Additionally, it has been reluctantly revealed that most studies indicating a reduced brain volume show that this is directly due to the effects of antipsychotic drugs (Andreasen, Liu, Ziebell, Vora, & Ho, 2013; Szeszko, et al., 2014). Undermining the brain-disease assumption even further are studies in which brain scans of children diagnosed with schizophrenia show similar abnormalities as their healthy siblings; as adults, those diagnosed with schizophrenia still have similar brain abnormalities while their non-diagnosed siblings went on to have "normal" brain scans as adults (e.g., Gogtay, 2008; Gogtay &

Rapoport, 2008). This shows not only that the structure of the brain can change for various reasons over time, but also that abnormalities in the brain do not necessarily equate with abnormal behavior. Rather, there is likely some other shared variable involved with particular types of psychological distress, such as the home in which these siblings were raised.

A similar pattern of brain anomalies and changes associated with environmental and psychotropic influences can be found for other diagnostic categories thought to be brain-based diseases, such as attention-deficit disorder (ADHD) and depression. The reason for this pattern of findings is that brain activity appears to be most closely associated with certain subjective phenomena, for instance hearing voices, seeing visions, impulsivity, fatigue, etc., that do not uniquely map onto DSM-defined categories and are associated with a variety of environmental influences.

Many studies have suggested a link between inflammation and serious emotional distress (e.g., Khandaker et al., 2015; Papakostas et al., 2013; Raison & Miller, 2011), yet inflammation is known to be a direct result of chronic stress and/or poor diet. Furthermore, chronic stress and trauma have been found to not only change the structure and activity of the brain (Read, Fosse, Moskowitz, & Perry, 2014; Read, Perry, Moskowitz, & Connolly, 2001; van der Kolk, 2003), but that of gene expression as well (Epel et al., 2004; Labonte et al., 2012). In fact, the most consistent finding across all diagnostic categories is abnormalities in the hypothalamic-pituitary-adrenal (HPA) axis, which is an area of the brain that is specifically associated with trauma and stress (e.g., van der Kolk, 2003). Although many tend to take for granted that posttraumatic stress disorder (PTSD) is the only syndrome directly caused by traumatic events, we cannot say that trauma has any more direct causal link with PTSD than it does with other phenomena such as hyperactivity, psychosis, or depression (Brown, et al., 2014; Dorrington, et al., 2014; Janssen et al., 2004). To conflate distressing experiences and the body's natural reaction to such experiences with biological disease is not only a giant leap in logic, but could also result in minimization and denial of the traumatic and stressful experiences that initially led to the emotional distress.

In the same way that the environment may negatively affect the structure of the brain, so too may there be positive influences. Training that involves the active participation of parents has been shown to change the brains of children who are diagnosed with ADHD (Neville, et al., 2013), while psychotherapy, in general, has been shown to be associated with changes in the brain consistent with changes in behavior (Abbass, Nowoweiski, Bernier, Tarzwell, & Beutel, 2014; Roffman, Gerber, & Click, 2012). In addition to more formal psychotherapeutic intervention, other healthy practices have also been shown to have a direct effect on the brain. For instance, meditation has been shown to increase brain volume in as little as eight weeks (Hölzel, et al., 2010), aerobic exercise can increase the volume in an area of the brain directly associated with the effects of trauma (ten Brinke, et al., 2014), and eating healthily may reverse brain abnormalities associated with addiction to food (Deckersbach, et al., 2014).

But, is it really necessary to have "evidence" from brain scans to know that meditation, exercise, and eating healthily have beneficial effects on one's wellbeing? The only thing this research really seems to show is how much the brain is constantly adapting to its environment. In fact, one could even interpret the findings of many of the brain differences in traumatized and distressed individuals as signs of adaptive functioning— the complete opposite of disease!

In sum, the only consistent and replicable findings in brain-based mental health research have been those showing the relationship of early childhood stress, neglect, trauma, poor diet, and/or lack of exercise on the structure and neural connectivity within the brain. Similar brain abnormalities can be identified in siblings where one sibling has been diagnosed with a psychological disorder and the other has never shown any sign of psychological dysfunction. This alone should make people question the utility of assuming biological disease based simply on brain differences. Additionally, healthy behaviors and psychotherapeutic interventions have significant and measurable effects on the brain, further underscoring the importance of understanding environmental effects on developing children and of intervening at familial and systemic levels. At this time, there is no evidence of any disease process in any of the so-called mental disorders, except that which is associated with the long-term use of medications (e.g., Deacon, 2013; Whitaker, 2010).

#### Consequences of the Continued Search for "Disease" in the Brain

Three prominent negative consequences of focusing on biological, brain-based etiologies of "mental illness" are that it results in skewed research funding, biased treatment preferences, and clinically harmful impacts. First, it problematically skews research funding. Although brain-based research has received a far disproportionate share of mental health funding, there have been little-to-no advancements in symptom or functional outcomes for individuals diagnosed with serious mental illness within the biological/disease model. This leads us to question the logic of continuing to fund this line of research particularly when it is at the expense of alternative theories. Frances (2014) described this funding/research situation as follows:

NIMH was at the center of the neuroscience enthusiasm, dubbing the 1990s the "decade of the brain" and betting the house on a narrow biological agenda to replace what previously had been a more balanced portfolio of research into not only the basic sciences, but also into treatments and health services. In effect, NIMH turned itself into a "brain institute" rather than an "institute of mental health." Its efforts have succeeded in producing wonderful science, but have failed in helping patients. (p, 47)

This problem will likely become more prevalent as the RDOC gains momentum. Frances (2014) worried that the RDoC "will almost certainly deliver nothing of practical import within this decade. My guess is that it will consist of a slow, steady slog of tiny steps, more characterized by frustrating blind alleys than by any great leaps forward" (p. 48). This was echoed by Phillips (2014) who argued, "The RDoC initiative, though intellectually appealing (to neuroscientists), is tone deaf to the current global trajectory of mental health. . . . This high-

profile focus of NIMH funding on the very long-term goal of establishing biologically-based diagnostic categories...may ultimately prove impossible for a large proportion of the persons we currently treat" (p. 40).

Additionally, emphasizing biological, reductionistic etiologies of mental illness prejudices clinicians and patients to believe medication is the most effective form of treatment (Lebowitz & Ahn, 2014). While medication may sometimes be useful, it also has a variety of negative side effects, some of which can be very serious. For example, some of the side-effects of antidepressants include: increased risk of relapse after discontinuation, negative effects on attention, a variety of gastrointestinal problems, disruption of the blood clotting process and abnormal bleeding, impaired sexual functioning, and negative effects on embryos and fetuses in pregnant women consuming antidepressants (Andrews et al., 2012). Some of the negative side-effects of antipsychotics include: irreversible alterations in brain size, structure, and function, tardive dyskinesia, obesity and associated conditions such as diabetes, heart disease, and stroke, and other cardiovascular problems (Moncrief, 2013). In other words, many of the biological anomalies that one finds with chronic sufferers of "mental illness" are directly caused by the very biological interventions thought necessary to decrease distress.

There is also increasing research demonstrating the severe and prolonged symptoms of withdrawal from these drugs (Fava et al., 2015; Nielsen, Hansen, & Gozsche, 2012). Often, when people choose to discontinue the use of these addictive substances, the changes in their brains can lead to a "super-sensitivity" to particular experiences (Hyman, 1996). So, for instance, when one desires to stop taking an antipsychotic that has been prescribed long-term, and the person does not taper slowly, the person is very likely to experience a kind of rebound psychosis. This withdrawal effect of brain-altering drugs is then taken as "proof" that the drugs were necessary and that the person does, in fact, have a biological disease (Whitaker, 2010). This circular reasoning, which is not based in the actual scientific long-term research, can trap a person in a life-long cycle of extreme distress and disability.

Critically, many studies have found treatment alternatives to medication are effective. For example, evidence suggests that psychotherapy is as effective as antidepressant medication as well as psychotherapy plus antidepressant medication for the treatment of depression (Khan, Faucett, Lichtenberg, Kirsch, & Brown, 2012). And recent longitudinal research has shown that those who do not take antipsychotic medication long-term have better outcomes than those who remain on these drugs continuously (Harrow, Jobe, & Faull, 2012; Wunderink et al., 2013). In short, while many of these drugs may provide temporary relief, the underlying problems in living must be acknowledged and addressed to gain increased quality of life and functional recovery.

The final prominent consequence of the emphasis on brain-based etiologies of emotional distress is that this framework can have significant negative clinical impacts. In the past five years, research has consistently suggested that conceptualizing mental illness as exclusively or primarily biologically caused can lead to increased prognostic pessimism (Lebowitz, 2014). Since clients' expectancies to improve in treatment have a significant impact on how much they actually improve, conceptualizations that increase prognostic pessimism can negatively

impact clinical outcomes (Constantino, 2012; Tambling, 2012). Furthermore, multiple studies evaluating the effects of a disease model on stigma have found that biological conceptualizations of emotional distress are associated with increased stigma and negative opinions versus conceptualizations based on the psychosocial circumstances that led to the distress (Pescosolido, et al., 2010; Schomerus, Matschinger, & Angermeyer, 2014). So not only are brain-based etiologies of psychological distress unsupported by the evidence and related to the excessive use of dangerous medications, they also have powerful psychological impacts that can adversely influence treatment.

### Conclusion

Having reviewed the relevant research, and having considered the pros and cons of the general trend of devoting increasing resources towards seeking the etiologies and remedies for psychological distress within the brain, we have come to the conclusion that this trend is misguided and seriously problematic. Attempts to arrive at a biologically-based diagnostic system for those conditions of human distress not clearly associated with a genuine medical condition (so called “mental illnesses”) have so far has been costly and unsuccessful. Biological interventions for such conditions have consistently been associated with worse long-term outcomes and increased chronicity of distress.

Poverty, trauma, child abuse and neglect, discrimination, loneliness, bullying, drug use, and inequality are directly associated with such conditions, and are also correlated with certain kinds of observable changes within our brains. Psychosocial support that directly addresses these issues has been consistently associated with long-term benefits superior to those of biological interventions. Finally, we need to acknowledge that the resources available for mental health research and care are limited, and that every dollar and person-hour spent pursuing brain-based solutions to psychological distress comes at a direct cost to those resources available for psychosocial research and support.

It seems that reducing rates of "mental illness" and disability would require that we start at the source. We need to identify persons in need of food, housing, and employment and help them to support themselves instead of judging and condemning them. We should identify mothers who are distressed, particularly when pregnant, and help them to learn how to bond with their child and form healthy attachments. We should put resources towards decreasing poverty and racism, while increasing social inclusion and empowerment. But, even if we cannot change the world, we can certainly put our efforts and resources towards individual interventions that have been shown to work based off the extensive support showing that people breakdown and despair for a reason.

So what exactly would such a shift in emphasis within the mental health care field look like? When we consider the vast disparity between the predominant research and interventions within the mental health field on one hand, and the actual needs of distressed human beings on the other hand, we recognize that our mental health field is in dire need of a radical paradigm shift—from trying to make sense of psychological distress from a biologically reductionistic framework to one that is more humanistic and needs-based. This essentially

involves shifting the general stance within the mental health field from “diagnosis and treatment” to one of “assessing needs and offering support.” This would mean focusing our resources on providing psychosocial support for individuals, families, and communities and working towards a social system in which meaningful and rewarding activity, education, and work is accessible to everyone.

Even in those cases in which the specific needs or other causal factors are unable to be identified, the evidence suggests that when a person’s basic needs are addressed, such conditions of psychological distress tend to naturally recede over time. And in those rare cases where such factors are unable to be identified and addressed, and in which the condition does not naturally recede over time, some psychoactive drug support may be beneficial, as long as it is used in minimal dosage for minimal duration and only with the individual’s fully informed consent; nutritional modifications may be an apt intervention as well.

One final important component of such a system that needs to be mentioned is that of prevention—identifying and eliminating the seeds of potential psychological distress. Considering that factors such as poverty, inequality, family disharmony, and various forms of social discrimination lie right at the root of so much of those distressing conditions that are called “mental illness,” this forces us to acknowledge that if we really want to address “mental illness,” then we really need to work together as a society and explore how we can address these broader social problems. This is no easy task, for sure, but if we honestly want to ask the question, “What causes ‘mental illness’ and how do we best ‘treat’ it,” then we need to be willing to look honestly at the research and accept the answers that emerge.

## References

- Abbass, A. A., Nowoweiski, S. J., Bernier, D., Tarzwell, & Beutel, M. E. (2014). Review of psychodynamic psychotherapy neuroimaging studies. *Psychotherapy and Psychosomatics*, 83, 142-147.
- Akdeniz, C. Tost, H., Streit, F., & Haddad, L. (2014). Neuroimaging evidence for a role of neural social stress processing in ethnic minority-associated environmental risk. *JAMA Psychiatry*, Online April 16, 2014. Doi:10.1001/jamapsychiatry.2014.35
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. Washington, DC: Author.
- Andreasen, N. C., Liu, D., Ziebell, S., Vora, A., & Ho, B. C. (2013). Relapse duration, treatment intensity, and brain tissue loss in schizophrenia: A prospective longitudinal MRI study. *American Journal of Psychiatry*, 170, 609-615.
- Andrews, P. W., Thomson Jr, J. A., Amstadter, A., & Neale, M. C. (2012). Primum non nocere: an evolutionary analysis of whether antidepressants do more harm than good. *Frontiers in psychology*, 3.
- British Psychological Society. (2013). *Division of Clinical Psychology position statement on the classification of behaviour and experience in relation to functional psychiatric diagnoses: Time for a paradigm shift*. Leicester, United Kingdom: British Psychological Society. Retrieved from <http://dxrevisionwatch.files.wordpress.com/2013/05/position-statement-on-diagnosis-master-doc.pdf>.
- Brown, R. C., Berenz, E. C., Aggen, S. H., Gardner, C. O., Knudsen, G. P., Reichborn-Kjennerud, T., Kendler, K. S., & Amstadter, A. B. (2014). Trauma exposure and Axis I psychopathology: A cotwin control analysis in Norwegian young adults. *Psychological Trauma: Theory, Research, Practice, and Policy*, 6(6), 652-660.
- Constantino, M. J. (2012). Believing is seeing: an evolving research program on patients'psychotherapy expectations. *Psychotherapy Research*, 22(2), 127-138.
- Deacon, B. J. (2013). The biomedical model of mental disorder: A critical analysis of its validity, utility, and effects on psychotherapy research. *Clinical Psychology Review*, 33(7), 846-861.
- Deckersbach, T., Das, S. K., Urban, L. E., Salinardi, T., Batra, P., Rodman, A. M., Arulpragasam, A. R., Dougherty, D. D., & Roberts, S. B. (2014). Pilot randomized trial demonstrating reversal of obesity-related abnormalities in reward system responsivity to food cues with a behavioral intervention. *Nutrition & Diabetes*, 4, e129; doi:10.1038/nutd.2014.26

Dorrington, S., Zavos, H., Ball, H., McGuffin, P., Rijdsdijk, F., Siribaddana, S., Sumathipala, A., Hotopf. (2014). Trauma, post-traumatic stress disorder and psychiatric disorders in a middle-income setting: prevalence and comorbidity. *The British Journal of Psychiatry*, 205, 383-389.

Douaud., G., Smith, S., Jenkinson, M., Behrens, T., Johansen-Berg, H., Vickers, J., James, S., Voets, N., Watkins, K., Matthews, P. M., & James, A. (2007). Anatomically related grey and white matter abnormalities in adolescent-onset schizophrenia. *Brain*, 130, 2375-2386.

Eluvathingal, T. J., Chugani, H. T., Behen, M. E., Juhász, C., Muzik, O., Maqbool, M., Chugani, D. C., & Makki, M. (2006). Abnormal brain connectivity in children after early severe socioemotional deprivation: A diffusion tensor imaging study. *Pediatrics*, 117(6), 2093-2100.

Epel, E. S., Blackburn, E. H., Lin, J., Dhabhar, F. S., Adler, N. E., Morrow, J. D., & Cawthon, R. M. (2004). Accelerated telomere shortening in response to life stress. *Proceedings of the National Academy of Sciences of the United States of America*, 101(49), 17312-17315.

Fava, G. A., Gatti, A., Belaise, C., Guidi, J., & Offidani, E. (2015). Withdrawal symptoms after selective serotonin reuptake inhibitor discontinuation: A systematic review. *Psychotherapy and Psychosomatics*, 84(2), 72-81.

Frances, A. (2014). RDoC is necessary, but very oversold. *World Psychiatry*, 13(1), 47-49.

Gogtay, N. (2008). Cortical brain development in schizophrenia: Insights from neuroimaging studies in childhood-onset schizophrenia. *Schizophrenia Bulletin*, 34(1), 30-36.

Gogtay, N., & Rapoport, J. L. (2008). Childhood-onset schizophrenia: Insights from neuroimaging studies. *Journal of the American Academy of Child and Adolescent Psychiatry*, 47(10), 1120-1124.

Harrow, M., Jobe, T. H., & Faull, R. N. (2012). Do all schizophrenia patients need antipsychotic treatment continuously throughout their lifetime? A 20-year longitudinal study. *Psychological Medicine*, 42(10), 2145-2155.

Hölzel, B. K., Carmody, J., Vangel, M., Congleton, C., Yerramsetti, S. M., Gard, T., & Lazar, S. W. (2010). Mindfulness practice leads to increases in regional brain gray matter density. *Psychiatry Research: Neuroimaging*, 191, 36-43.

Houenou, J., Duhamel, A., Amad, A., De Weijer, A. D., Ćurčić-Blake, B., Linden, D. E., Thomas, P., & Jardri, R. (2014). The arcuate fasciculus in auditory-verbal hallucinations: A meta-analysis of diffusion-tensor imaging studies. *Schizophrenia Research*, 159(1), 234-237.

Howell, B. R., McCormack, K. M., Grand, A. P., Sawyer, N. T., Zhang, X., Maestriperi, D., Hu, X., & Sanchez, M. M. (2013). Brain white matter microstructure alterations in adolescent rhesus monkeys exposed to early life stress: Associations with high cortisol during infancy. *Biology of Mood & Anxiety Disorders*, 3; doi:10.1186/2045-5380-3-21.

Hyman, S. (1996). Initiation and adaptation: A paradigm for understanding psychotropic drug action. *American Journal of Psychiatry*, 153, 151-161.

Insel, T. (2013, April). *Director's blog: Transforming diagnosis*. National Institute of Mental Health. Retrieved from <http://www.nimh.nih.gov/about/director/2013/transforming-diagnosis.shtml>

International Society for Ethical Psychology & Psychiatry. (May, 2013). Statement supporting the British Psychological Society's Statement on Functional Psychiatric Diagnoses [Public Statement]. Retrieved from: <http://psychintegrity.org/wp-content/uploads/2015/08/Supporting-the-British-Psychological-Society%E2%80%99s-Statement-on-Functional-Psychiatric-Diagnoses-May-16-2013.pdf>

Janssen, I., Krabbendam, L., Bak, M., Hanssen, M., Vollebergh, W., De Graaf, R., & van Os, J. (2004). Childhood abuse as a risk factor for psychotic experiences. *Acta Psychiatrica Scandinavica*, 109(1), 38-45.

Khan, A., Faucett, J., Lichtenberg, P., Kirsch, I., & Brown, W. A. (2012). A systematic review of comparative efficacy of treatments and controls for depression. *PLoS One*, 7(7), e41778.

Khandaker, G. M., Cousins, L., Deakin, J., Lennox, B. R., Yolken, R., & Jones, P. B. (2015). Inflammation and immunity in schizophrenia: Implications for pathophysiology and treatment. *The Lancet*, 2(3), 258-270.

Kupfer, D. (2013). *News release: Chair of DSM-5 task force discusses future of mental health research*. American Psychiatric Association. Retrieved from <http://www.psychiatry.org>.

Labonte, B., Suderman, M., Maussion, G., Navaro, L., Yerko, V., Mahar, I., ... Turecki, G. (2012). Genome-wide epigenetic regulation by early-life trauma. *Archives of General Psychiatry*, 69, 722-731.

Lebowitz, M. S. (2014). Biological conceptualizations of mental disorders among affected individuals: A review of correlates and consequences. *Clinical Psychology: Science and Practice*, 21(1), 67-83.

Lebowitz, M. S., & Ahn, W. K. (2014). Effects of biological explanations for mental disorders on clinicians' empathy. *Proceedings of the National Academy of Sciences*, 111(50), 17786-17790.

Moncrieff, J. (2013). *The bitterest pills: The troubling story of antipsychotic drugs*. New York, NY: Palgrave Macmillan.

Neville, H. J., Stevens, C., Pakulak, E., Bell, T. A., Fanning, J., Klein, S., & Isbell, E (2013). Family-based training program improves brain function, cognition, and behavior in lower

socioeconomic status preschoolers. *Proceedings of the National Academy of Sciences of the United States of America*, 110(29), 12138-12143.

Nielsen, M., Hansen, E., & Gozsche, P. C. (2012). What is the difference between dependence and withdrawal reactions? A comparison of benzodiazepines and selective serotonin reuptake inhibitors. *Addiction*, 107, 900-908.

Papakostas, G. I., Shelton, R. C., Kinrys, G., Henry, M. E., Bakow, B. R., Lipkin, S. H., Pi, B., Thurmond, L., & Billello, J. A. (2013). Assessment of a multi-assay, serum-based biological diagnostic test for major depressive disorder: A pilot and replication study. *Molecular Psychiatry*, 18, 332-339.

Pescosolido, B. A., Martin, J. K., Long, J. S., Medina, T. R., Phelan, J. C., & Link, B. G. (2010). "A disease like any other"? A decade of change in public reactions to schizophrenia, depression, and alcohol dependence. *American Journal of Psychiatry*, 167, 1321-1330.

Phillips, M. R. (2014). Will RDoC hasten the decline of America's global leadership role in mental health? *World Psychiatry*, 13(1): 40–41.

Raison, C. L., & Miller, A. H. (2011). Is depression an inflammatory disorder? *Current Psychiatry Reports*, 13(6), 467-475.

Read, J., Fosse, R., Moskowitz, A., & Perry, B. (2014). The traumagenic neurodevelopmental model of psychosis revisited. *Neuropsychiatry*, 4(1), 65-79.

Read, J., Perry, B. D., Moskowitz, A., & Connolly, J. (2001). The contribution of early traumatic events to schizophrenia in some patients: A traumagenic neurodevelopmental model. *Psychiatry*, 64, 319-345.

Roffman, J. L., Gerber, A. J., & Click, D. M. (2012). Neural models of psychodynamic concepts and treatments: Implications for psychodynamic psychotherapy. In R. A. Levy, J. S. Ablon, H. Kächele (Eds.), *Psychodynamic psychotherapy research: Evidence-based practice and practice-based evidence* (pp. 193-218). Totowa, NJ, US: Humana Press.  
doi:10.1007/978-1-60761-792-1\_11.

Schomerus, G., Matschinger, H., & Angermeyer, M. C. (2014). Causal beliefs of the public and social acceptance of persons with mental illness: a comparative analysis of schizophrenia, depression and alcohol dependence. *Psychological Medicine*, 44(2), 303-314.

Szeszko, P. R., Robinson, D. G., Ikuta, T., Peters, B. D., Gallego, J. A., Kane, J., & Malhotra, A. K. (2014). White matter changes associated with antipsychotic treatment in first-episode psychosis. *Neuropsychopharmacology*, 39, 1324-1331.

Tambling, R. B. (2012). A literature review of therapeutic expectancy effects. *Contemporary Family Therapy*, 34(3), 402-415.

Ten Brinke, L. F., Bolandzadeh, N., Nagamatsu, L. S., Hsu, C. L., Davis, J. C., Miran-Khan, K., & Liu-Ambrose, T. (2014). Aerobic exercise increases hippocampal volume in older women with probable mild cognitive impairment: A 6-month randomised controlled trial. *British Journal of Sports Medicine*, doi:10.1136/bjsports-2013-093184

Van der Kolk, B. A. (2003). The neurobiology of childhood trauma and abuse. *Child and Adolescent Psychiatric Clinics of North America*, 12, 293-317.

Whitaker, R. (2010). *Anatomy of an epidemic: Magic bullets, psychiatric drugs, and the astonishing rise of mental illness in America*. New York, NY: Crown Publishers.

Whitaker, R., & Cosgrove, L. (2015). *Psychiatry under the influence: Institutional corruption, social injury, and prescriptions for reform*. New York, NY: Palgrave Macmillan.

Wunderink, L., Nieboer, R. M., Wiersma, D., Sytema, S., & Nienhuis, F. J. (2013). Recovery in remitted first-episode psychosis at 7 years of follow-up of an early dose reduction/discontinuation or maintenance treatment strategy: Long-term follow-up of a 2-year randomized clinical trial. *JAMA Psychiatry*, 70(9), 913-920.

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