Neurofeedback for Psychosis

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Abstract:

Psychosis causes major disease burden, individual, family, social and economic pain. Medications are of limited effectiveness and cause many unpleasant side effects. Technology is now allowing us to examine the organ being treated, to better personalize therapies, including neurofeedback operant conditioning. The epigenetic effects of developmental trauma in the causation of psychosis are highly significant and responsive to quantitative electroencephalography (qEEG) guided neurofeedback, with very promising results. While there are many questions to be answered, it is time to implement this therapy more widely.

Keywords: psychosis, schizophrenia, epigenetics, qEEG, neurofeedback, developmental trauma, burden of disease, recovery

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1. Introduction*:*

There is considerable evidence that childhood and adolescent trauma has major epigenetic effects in causing psychosis, including schizophrenia (review by Popovic et al 2019). In schizophrenia, about 8,300 single nucleotide polymorphisms have been estimated to contribute to a common genetic risk of only 32% (Ripke et al 2013), suggesting that in addition to genetic background, environmental factors may be the basis of pathophysiological processes (Manolio et al 2009). The review of neuroimaging by Teicher and Samson (2016) shows the plastic brain changes in response to childhood traumas (e.g. neglect, poverty, poor attachment, emotional abuse, and physical and sexual abuse) which are evolutionary protective mechanisms to enable survival in a toxic environment until puberty, to produce the next generation. The brain then switches from growth to pruning for efficiency, and behavioral programming to competing with peers for the best mate and resources to support children. The previous protective changes cease to be helpful and may be harmful. Further traumas at sensitive stages of development, such as bullying at the ages of 13-14, when competing with peers is most important, can also cause brain dysregulation. Psychopathology may emerge, due to the mismatch between the world the brain was modified to survive in, and the world it finds itself in, during subsequent developmental stages. These changes may remain throughout life unless actively modified, and medication does not do it.

Psychiatry has had to rely on symptom clusters for diagnosis, as direct examination of the organ being treated was not possible. The profound effects of developmental trauma have not been recognized or controlled for in research, and this has led to highly confounded results, and unfortunately this often continues. Now technology is enabling better diagnosis, particularly through functional magnetic resonance imaging (fMRI) (Xiaoyan & Rongjun 2015) and quantitative electroencephalography (qEEG) (Kropotov 2016). While academics have access to very expensive fMRI machines, qEEG is cheap and quite effective.

The prospect of personalized treatment is now being realized for medication (Gunkelman 2014) and brain operant conditioning through neurofeedback (Sitaram et al 2017). While neurofeedback has long been used by psychologists for performance enhancement and ADHD treatment, it is now effectively used for the treatment of trauma in refugees (Askovic et al 2017 & in press), chronic Post Traumatic Stress Disorder (PTSD) (Van Der Kolk et al 2016) and schizophrenia.

Brand et al (2017) did a systemic review of trauma focused cognitive behavior therapy (CBT) interventions for people with psychosis, who also had a diagnosis of PTSD, and across 25 studies found low effect sizes, poorly sustained when treatment ceased. This should not be surprising, as PTSD that responds to exposure treatment usually follows discrete highly threatening events, rather than the more insidious emotional abuse, physical neglect and attachment failures in the developmental period, which many in the trials would also have experienced. Raio et al (2013) found that stress markedly impairs cognitive regulation of emotion and highlights critical limitations of this technique to control affective responses under stress. Re-regulation of the brain to reduce the effects of stress is needed at the start of treatment to enable more effective psychotherapy outcomes.

Developmental trauma has an effect on the severity of mental health disorders (Bailie et al 2018). A systematic review of the association with the severity of hallucinations and delusions in psychotic disorders showed significant dose related correlation with the severity of hallucinations and delusions, but not correlated with the severity of negative symptoms. Severity of childhood neglect was correlated with negative symptoms, which is logical, as there are very different protective brain changes, and failures of brain development, due to the lack of appropriate attachment and stimulation. There is evidence that the emotional, physical and sexual abuse types of trauma generally lead to high anxiety and abnormalities in the left hemisphere on the qEEG. Children who experience extreme levels of social neglect early in life, show diminished electrical activity in the brain as measured by EEG (National Scientific Council on the Developing Child 2012, Vanderwert et al 2010) However some episodes of trauma can lead to dissociative shut-down (Schalinski & Teicher 2015). Thus neurofeedback protocols need to vary with guidance from the qEEG.

Gruzelier (2000) showed that people with schizophrenia can learn to self-regulate their brain activity with operant conditioning, such as neurofeedback. Treatment of schizophrenia using qEEG or rtfMRI guided neurofeedback, designed before the understanding of the evolutionary protective brain changes, showed significant benefits (Surmeli et al 2012, Bolea 2010, Schummer & von Stietz 2013, Orlov et al 2018, Nan et al 2017). The qEEG differences between those with developmental trauma and the normative database would have led to protocols directed at those differences, as well as the psychosis.

Surmeli (2012) treated 51 people with chronic schizophrenia with neurofeedback. They had Positive and Negative Syndrome Scale (PANSS) scores within the range 76-156, mean 110.24 (SD 21.62). 47 out of 48 final participants showed clinical improvement, as the mean PANSS score decreased to 19.56 (SD 26.78) which was statistically significant ($P< 0.01$), along with significant improvements in Minnesota Multiphasic Personality Inventory (MMPI) ($P< 0.01$). There were also significant improvements in the Test of Variables of Attention (TOVA) measurements for the 33 whose initial symptom levels allowed for pre testing ($P<0.01$). They were followed for around 2 years with the mean reduction in PANSS scores of 82%. Above 20% is considered good for antipsychotic medications (aripiprazole 28.6%, placebo 21.2% in adolescents – US FDA approval data). 19 ceased to meet criteria for schizophrenia, 27 did not need medication and the remaining 24 required about half their previous dosage and were more functional. The post qEEG changes were consistent with these results. In the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) study, the efficacy of the pharmacological treatment on the primary measure, staying in the study until completion, was only 26% (Kane, Janicak 2006). In this study, not only did all but 3 people adhere to the neurofeedback regimen (94%), but of those that needed medication, 68% of them adhered to their medication treatment when followed up to 2 years.

Bolea (2010) reported on his neurofeedback treatment of 70 people with severe chronic hospitalized schizophrenia, resistant to all other treatments, who responded very well and most were discharged to the community, with reduced medication. One recent trial of intensive neurofeedback over 4 days, for a person with chronic schizophrenia, with geographic difficulty to complete long term training, produced an excellent result (Nan et al 2017). The follow up assessments have shown that the effects are sustained, unlike medication effects that cease with the medication.

2. Discussion:

It could be argued that the efficacy of the neurofeedback was due to reversing the effects of developmental trauma, and those with a stronger genetic risk remaining on medication, while functionally much improved. These results point to a significant reduction in the burden of disease and the cost of effective treatment, despite the intervention being initially intensive.

The headspace Youth Early Psychosis Program in Western Sydney is caring for young people 12-25 with either first episode psychosis or who are at ultra-high risk of developing psychosis. Conus et al (2010) found that 83% of the young people with first episode psychosis, admitted to the Early Psychosis Prevention and Intervention Centre (EPPIC) in Melbourne, had been exposed to at least one stressful event and 34% to sexual or physical abuse. Our experience is that when we look for developmental trauma in the first episode group, two thirds appear to have had significant trauma and almost all of the ultra-high risk group. Retrospective reports by people will often fail to retrieve memories of neglect and emotional abuse, which often occurred before the development of language, while parents are preoccupied with sedation from drugs and alcohol, their own symptoms of PTSD, depression, psychosis, physical illness, depravation, developmental disability and domestic discord (Murphy et al 2018). Thus the rates of developmental trauma are likely to be higher than reported, another reason to look at the qEEG.

3. Conclusions:

As we see the limitations of medications with our young people and the apparent benefits of actually treating developmental trauma, the time has come to start using qEEG and neurofeedback in our services, to enable full uptake of the recovery approach to care. This would be consistent with the interventionist-causal paradigm method proposed by Brand et al (2017) to untangle the relationship between developmental trauma and psychosis. It is postulated that this will have a major beneficial effect across the ultra-high risk and first episode psychosis spectrums, considering the dose related correlation of symptoms and trauma (Bailey et al 2018).

As 68% of causation of psychosis is epigenetic, and developmental trauma plastic changes affect physical health functions as much as mental functioning (Lanius et al 2010), I hypothesize that applying neurofeedback, based on qEEG signs of developmental trauma, will show significant benefits for mental and physical health, reducing personal, family, social and economic pain. It should be immensely cost effective by reducing the burden of disease and enabling greater functional recovery and social inclusion.

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