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Recommendations to Help Prevent Mental Disorders and Limit Stigma

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ABSTRACT

Barriers to better mental health outcomes are stigma and sub-optimal mental health literacy. Mental health outcomes are being suppressed by both the residual eugenics beliefs and Nazi propaganda, that continue to dominate global public perceptions. As health professionals we need to move past these Nazi atrocities to modernize the delivery of mental health services. Mental health literacy is an invaluable resource. Many mental disorders are known to arise from the brain adapting to a low energy environment. An integrative literary review of brain energy requirements was conducted, to extrapolate recommendations that can inform clinical practice and policy. The quality of life aspect of mental health should emphasize cognitive well-being. Stress depletes our brain's iron and magnesium reserves. Smoking tobacco, heavy alcohol or marijuana use only worsens the damage that is occurring in the brain; from depleting levels of these important nutrients. Critically, do not fast for extended periods then eat ferociously or consistently skip meals. A variety of foods contain the recommended nutrients. Moreover, fish, seaweed or proteins possess higher levels of the nutrients required for thyroid hormone production to control energy expenditure. Eat iron, zinc and iodine rich foodstuffs and smaller portions of selenium, copper, magnesium and manganese to support your brain; especially if you have ever had a mild to severe hypoxic brain injury. Micronutrient preparations comprising of these nutrients may help to prevent mental disorders. See your doctor to have any gastro-intestinal or thyroid problems treated.

Keywords: eugenics, health, psychosis, mood, anxiety, post-traumatic stress disorder

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INTRODUCTION

Sub-optimal mental health literacy and stigma surrounding mental disorders remain a barrier for people seeking better mental health outcomes. This problem is multifactorial. Resistance to change within mental health services and ingrained public perceptions are clear concerns. Because of the Nuremberg trial for doctors that followed World War II, ethical codes for researchers were established in 1947 to address medical experimentation (Shuster, 1997). Unfortunately, people with mental disorders were largely forgotten and continued to be starved to death in mental institutions until 1949, as beds needed to be freed up for wounded soldiers (Seeman, 2006). Mental health services were only legislated to control detention and prevent professional negligence (Zielase & Gaebel, 2015). A Munich psychiatrist eloquently described how people with mental disorders continued to be subjected to ad hoc treatments, suffer in a culture that produced powerlessness, lack of hope and stifled innovative research (Hafner, 2013).

Amongst the public, residual Nazi propaganda that is derived from the eugenics movement is enabling stigma to persist. Eugenic beliefs infiltrated multiple areas of society in many countries following a lecture that Sir Francis Galton delivered, giving rise to the idea of selective human breeding (Galton, 1904). Winston Churchill was one of those in the audience and he promptly introduced the concept to Britain, where it spread to the USA (Barnett, 2004). Across the world, the eugenics movement took many forms against the weakest of society. Mixed marriages, unmarried mothers, crippled and intellectually disabled and mentally ill peoples were initially targeted (Galton, 1909). There were laws to force sterilisations, adopt out children, and segregate couples.

The German manifestation began with Professor Ernst Rudin who had moved on from being Dr Emil Kraepelin's laboratory assistant to become the Director of Wilhelm Keizer Institute in Munich. Professor Rudin and his brother-in-law, Alfred Poletz formed a political pressure group that aimed to implement their ideology using psychiatric genetics as pseudoscientific rationale (Eckart, 2006). Alfred Poletz coined the term racial hygiene. The starvation of people with mental disorders began in 1914, continuing with falsified records (Benedict & Sheilds, 2014; Foth, 2011). When Adolf Hitler rose to power it was a small step to promote these ideas to the Nazi Party.

There was a rise in the population of people with mental disorders, housed in German mental institutions following World War l (Torrey & Yolken, 2009). Germany had been forced to pay compensation following World War I in an agreement outlined by the Treaty of Versailles. So, Germany was left with insufficient funds to care for the rising numbers of mentally ill people (Torrey & Yolken, 2009). To solve the problem, Professor Ernst Rudin

suggested that people with mental disorders could be incinerated. The implementation of Aktion T-4 was accompanied by a propaganda campaign. The patients saw their treating physician who deemed them 'life unworthy of living'. Nurses carried out physician orders to alleviate suffering (Benedict & Shields, 2014). Most were given morphine injections, some were gassed with carbon monoxide and others starved, then their bodies were thrown into furnaces adjoining the mental institution (Torrey & Yolken, 2009). The holocaust commenced with children with deformities (Benedict & Shields, 2014), then those with schizophrenia, bipolar disorder, depression, alcoholism, were epileptic, homosexual, had Huntington's chorea, blindness, deafness, had physical disabilities or deformities, were gypsies, had mixed marriages, were conscientious objectors or Jewish (Torrey & Yolken, 2009). The killing of patients was trivialised and normalised by state sponsored systemic propaganda that was permissive (Foth, 2011) and therefore viewed as a legitimate part of caring for the state (Bendict & Shields, 2014). Nurses were provided with financial incentives and nursing notes were controlled by the psychiatrist so that nurses were active participants in the starvation programs (Foth, 2011).

Yet the eugenics movement should not have found a footing in the first place. The term eugenics was coined by Sir Francis Galton (Galton, 2016). Sir Francis was a half cousin of Charles Darwin; sharing the same grandfather. He was a respected anthropologist and statistician, who suffered from an anxiety disorder (Brave, 2014). He devoted a large portion of his life to studying family pedigrees and the human interpretation of Darwin's concept of natural selection (Galton, 2016). Sir Galton conducted a blood transfusion study to disprove Darwin's hypothesis that pangenesis was the mechanism for the observed gene expression (Darwin, 1871). Charles Darwin refuted Sir Francis' claim as a misinterpretation, stating that he had been talking about "protozoa that has no blood" (Darwin, 1871). Unfortunately, these protests fell on deaf ears. Sir Francis conducted further pea and Bassett hound studies before proposing his law of ancestral heredity in 1896 (Swinburne, 1965). This law was discounted by the scientific community of the time as it fails to explain individual variation (Bulmer, 1998). The heredity hypothesis had not been tested in humans. Dr Emil Kraepelin was about to change that.

Dr Emil Kraepelin is probably best remembered for his work classifying mental illnesses (Noll, 2007). There has been limited acknowledgement of his attempts to treat dementia praecox (now renamed schizophrenia). Dr Kraepelin tested the oral administration of desiccated thyroid gland (Noll, 2007), which is the frontrunner to thyroid hormones. When this approach to elevating thyroid hormone levels had limited success, Dr Kraepelin considered the influence of hereditary genetics (Noll, 2007). In 1916 Dr Kraepelin and his

laboratory assistant, Dr Ernst Rudin published findings indicating that there was no recessive gene in schizophrenia (Rudin, 1916).

Instead, the epigenetic changes to RNA processes are found to be diet driven. Over successive generations poor eating patterns have been consistently reported amongst people with mental disorders. Dr Emil Kraepelin documented skipping meals and ferocious eating (Strassnig et al, 2005). Poor appetite, skipping meals and a desire for sweet foods are still prevalent today (Rao et al, 2008). It is now a century since Dr Emil Kraepelin realised that schizophrenia was a metabolic disorder (Noll, 2007). In the interim dementia praecox was relabelled schizophrenia, manic-depression became bipolar disorder and the international diagnostic criteria that defines mental disorders has undergone generations of reworking.

There is an overarching mechanism that is common to psychosis, mood disturbances and to a lesser extent anxiety disorders. Inadequate nutrients disrupt the production of thyroid hormones that control energy expenditure to alter energy and lipid metabolism. Following a mild hypoxic event, this disruption occurs at the signalling level for the metabolic enzymes and protein transporters. Because of these disruptions, transcriptional factors and RNA processes are altered (Laupu, 2017). These epigenetic changes are nutrient or oxygen driven alterations that silence the expression of some of the gene sequence. This silencing changes the way the gene sequence is read. Without adequate energy for the brain tissues to function, cognitive functioning declines. Imagining studies clearly demonstrate the progressive nature of damage to the brain organelle. There are varying structural changes that occur as the brain cells become unable to reprogram and begin to die (Laupu, 2017). The numbers of ribosomal proteins decrease, dendritic sprouting and density are reduced as neurons shrink, affecting the brain's volume in the thalamus, grey and white matter (Laupu, 2017). Mitochondrial findings on post mortem examination of people who had a diagnosis of schizophrenia indicate the brain’s energy requirements remained unmet throughout their life span (Kung & Roberts, 1999).

Our collective understanding of neuropathology leading to the development of mental disorders can inform our understanding of ‘at risk’ behaviours and identify vulnerable populations. Knowledge of the brain’s energy requirements for these ‘at risk' populations would provide recommendations for practice and inform policy makers. With this assumption in mind, an integrated review of the available literature was conducted.

MATERIALS AND METHOD

Large arrays of people are potentially susceptible to insufficient energy for their brain tissues to perform cognitive functions, adequately. Obvious populations are likely to have nutrient deficiencies, have sustained a past hypoxic brain injury, or have an underlying

gastrointestinal disorder that promotes malabsorption. Those with a nutrient dietary deficiency are likely to be people who have experienced famine, natural disaster, an eating disorder, or have poor eating patterns (restrictive diets, executives and busy professionals who skip meals, those who participate in heavy alcohol, heavy tobacco, marijuana use or other illicit substances). People with ongoing gastrointestinal disorders are known to be prone to the effects of post-traumatic stress disorder (Wang et al, 2015). Those who have experienced a traumatic head injury or hypoxic episode, in the past are susceptible to developing a mood disorder (Zhao et al, 2017). Examples of this group are people who have had a perinatal hypoxic injury, sporting concussions or road trauma.

Moreover, there is a subtle population of people who have an imbalance between the supply and demand of metabolic processes for brain energy. Fever associated with infection is known to increase metabolic demand (Hercus et al, **1**925). Schizophrenia and bipolar disorder have been linked to raised inflammatory markers (Tanaka et al, 2017), without the benefit of genome wide signals (Avramopoulos et al, 2015). Endurance athletes are at risk if their training methods cause nutrient or hypoxic imbalance comparative to brain energy demands. However, a cross-sectional survey of (N=224) elite athletes suggests that International training methods are adequate (Gulliver et al, 2015). The incidence of mental disorders amongst athletes are like the wider community; that is 27% depression, 23% eating disorders, 15% social anxiety, 7% with general anxiety disorder and 4% with a panic disorder (Gulliver et al, 2015). There are differential labels placed on people who experience alterations to sensory perception. For example, alterations to the sense of smell is known as multiple chemical sensitivity (English et al, 2015).

This integrative review made use of Google scholar and PubMed to search for papers alluding to requirements for healthy brain tissues in the identifiable vulnerable populations. Following a search of the literature, the articles were grouped under emergent themes. Each article was critiqued for validity and obvious methodological errors. The main finding of papers that were deemed to have methodological errors was withheld. Rather than dismissing these papers outright, a critique was incorporated into the results dialogue to provide the reader with some context of trends in nutritional psychiatry. There are so many papers on mental disorders; an overview critiquing approaches tested and their methodological flaws may provide the reader with a degree of clarity. The main findings for remaining papers were synthesised and recommendations for practice were discussed.

RESULTS AND DISCUSSION

It has been well documented that dietary intake amongst people with diagnosed depression, bipolar disorder, schizophrenia and the anxiety disorder, obsessive compulsive disorder lack

nutrients, essential vitamins and omega-3 fatty acids (Rao et al, 2008). A re-analysis of historical goitre studies examined environmental data, public health and the dietary habits of New Zealanders in 1925. This is an era where produce was grown and sold locally, in chemical free, local soils. There were pockets of New Zealand where local diets were deficient in seafood, protein and devoid of seaweed; comparative to the local mental hospitals where healthier diets and exercise were treatments (Laupu, 2016). Encouraging more traditional and healthier food choices have been suggested to prevent depression (Opie et al, 2017). However, if we examine government records across the breath of diagnoses and over a ten-year period, there was a documented annual discharge rate at the Christchurch mental hospital of 8.8% at best (McKillop in Young, 1925-1936). Caution should be applied. Studies reporting the influence of specific diets for mental disorders such as, the SMILES trial may be confounded by the improved dietary pattern inherent in the study design (Jacka et al, 2017).

The use of vitamins and minerals to treat mental disorders was proposed as individualised care by the orthomolecular psychiatry movement. There has been limited robust evidence to support this approach. While there is evidence that micronutrients such as, iron and zinc improve symptoms of low mood and attention-deficit hyperactivity disorder (Akhondzadeh et al, 2013) this role is minimal. At a cellular level, the biochemical activity of micronutrients is dependent on nutrients working together in a cofactor arrangement to account for the minimal effectiveness of single nutrient supplements. In addition, methodological flaws make study interpretation difficult. For instance, the magnitude of change within the group was not quantified which diminishes claims of reduced symptoms (Parletta et al, 2017).

Various combinations of micronutrients that are required for brain health have shown positive signals (N=1713) across multiple studies using sub-clinical anxiety, stress and low mood indicators (Table 1). Meta-analysis of 20 placebo controlled trials show reduced stress and sub-clinical low mood (Rucklidge et al, 2013). There is a modest effect on mood with branded multivitamin supplementation (Harris et al, 2011). The preventative benefit of combination micronutrients is well supported; to reduce sub-clinical symptoms amongst vulnerable groups with poor nutrient intake or gastrointestinal disorders. In one study, selenium supplementation was administered as a prophylaxis prior to defence force personal being sent into active service. Post engagement, parameters for oxidative stress were 6% lower and the prevalence of post-traumatic stress symptoms was reduced by 46% comparative to previously (Voitsekhovskis et al, 2014). Meta-analysis of 25 studies show magnesium supplementation may prevent depression (Derom et al, 2013). An inverse association between magnesium intake and depression has been found in a large Norwegian

study (N=5708) (Jacka et al, 2009). However, magnesium and fish oil treatment was inconclusive (Derom et al, 2013; Deacon et al, 2017). Magnesium deficiency has been linked to chronic consumption of alcohol (Oberleas et al, 1972), impaired intestinal absorption (Ananth & Yassu, 1979) and to chronic stress (N=30) (Takase et al, 2004). Case studies point to the improvement of recovery times for people experiencing depression when taking high doses of magnesium (Eby & Eby, 2006).

Table 1: Participants given brain nutrients vs sub-clinical indicators

Study	Anxiety	Low mood	Stress
Harris et al, 2011	N=50	N=50	N=50
Jacka et al, 2009	N=874	N=517	N=50
Kaplan et al, 2015	N=18	N=18	N=18
Rucklidge et al, 2011		N=16	
Rucklidge et al, 2012	N=61		N=61
Rucklidge et al, 2014	N=64		N=64
Voitsekhovskis et al, 2015	N=63		N=63
Totals (1713)	N=1130	N=601	N=306

These findings are of significance in the context of hypoxic injury as magnesium, copper and manganese are essential components of antioxidant enzyme and protein transporters for metabolic signalling pathways (Laupu, 2017; Vural et al, 2010; Tapiero & Tew, 2003). Hypoxic episodes are known to reduce the wet weight of the brain, heart and liver in chicken embryos and reduce membrane zinc and iron levels (Richards et al, 1991). Whereas, in severe obstructive sleep apnoea studies where hypoxic is a chronic issue, (N=97) adaptation to micronutrient fluctuations appears to occur (Asker et al, 2015). Magnesium supplementation has also been assessed in cardiac hypoxia due to a cardio-protective benefit and is germane to the neuropathology which is present in mental disorders. This activity involves energy conservation, the prevention of intracellular calcium influx and the opening of mitochondrial potassium ATP channels (Watanabe et al, 2004).

Work testing the use of micronutrients around natural disasters, has included New Zealand earthquakes and Canadian floods. Micronutrients having been identified as deficient in the neuropathology of mental disorders, were present in the preparations being tested (Rucklidge et al, 2012). That is, iron, zinc, selenium iodine (Triggiani et al, 2009) and copper, manganese and magnesium (Bourre, 2006). These micronutrients reduced stress and anxiety (large effect size of 0.69) amongst a cohort with attention-deficit hyperactivity disorder (Rucklidge et al, 2011). By three months post-earthquake, people self-administrating supplements were experiencing better outcomes than controls with regards to stress and anxiety (Rucklidge et al, 2012). A one year follow up study in the same cohort (N=64) revealed improvement in disaster survivors, in keeping with previous work. However large effect sizes (up to 1.31)

supported claims of significant improvement in the people who continued to take micronutrients (Rucklidge et al, 2014). Interestingly 10% of people demonstrated symptoms of post-traumatic stress disorder (Rucklidge et al, 2014). Following severe flooding, a study was conducted in (N=56) adults, who self-reported low mood, stress and anxiety symptoms. There were large effect sizes between a vitamin B complex group of participants (with regards to reduced stress and anxiety) and those taking a broad-spectrum mineral and vitamin formulation (Kaplan et al, 2015). A cohort (N=23) with no vitamin B complex deficiencies were supplemented and showed 3.4% improvement in mental tests over the placebo (Simonson et al, 1942). The vitamin B complex improved working capacity during central nervous system fatigue (Simonson et al, 1942); unrelated to depression scores (HAM-D) (Bell et al, 1991). The prevailing scientific thinking in disaster medicine is that over time new presentations of mental disorders drop off as adaptation to the new environment occurs. An interesting observation can be extrapolated from the post-earthquake comparison for rates of mental illness, between Christchurch and Kaikoura on New Zealand's east coast of the South Island. Christchurch had a 37% increase in mental disorder presentations by five years with a corresponding 60% rise in suicide attempts (CDHB, 2016), whereas Kaikoura had a rise in presentation near 5-10% (Gluckman, 2016). These figures remind me of the Ngai Tuhoe in the 1920s, which Sir Charles Hercus encountered while working as a public health physician. History records the tribe was divided by a loss of assess to the sea, leaving coastal and inland Maori to live separate lives. Iodine deficient goitres were absent in the seafood loving coastal peoples (Laupu, 2016). Similarly, Kaikoura is known for its seafood and Christchurch for its 64% incidence of iodine deficient goitre in the 1920s (Hercus et al, 1931).

Table 2: Studies reporting effect of brain nutrients for mental health

Study	Design	Participants	Outcome measures	Main finding
Rucklidge et al, 2013	Meta-analysis	N=27 studies		Nutrients not able to treat mental disorders
Derom, 2013	Meta-analysis	N=25 studies		High dose magnesium useful for depression
Harris et al, 2011	RCT/placebo	N=50 over 8 weeks	DASS, GHQ, PSS, POMS, VOMS	Effect 0.06-0.31 in favour of nutrients; mood, anxiety & stress improved
Jacka et al, 2009	RCT/placebo	N=5708	Hospital anxiety & depression scale	Relationship between low magnesium & depression
Kaplan et al, 2013	RCT/placebo	N=56	DASS, CGI-I, IES-R	Effect 0.76-1.08 in favour of nutrients; stress & anxiety improved
Rucklidge et al, 2011	RCT/placebo	N=33 over 2-3 months	MADRS, CAARS, GAF, CGI-S, DASS	Effect 0.73-1.01 in favour of nutrients; MADRS effect 1.96
Rucklidge et al, 2012	RCT/placebo	N=91		Improved mood, anxiety & energy
Rucklidge et al,	RCT/placebo	N=85		Effect 0.69-1.31 in favour of

2014 Voitsekhovskis et al, 2015	RCT/placebo	N=143 over 6 months	Oxidative stress parameters, PCL-M	nutrients; 10% developed PTSD 100 u/g selenium reduce PSTD markers (5.85%) & limit prevalence of PTSD by 46.03%. 8-9% developed PTSD

Poor eating patterns are also associated with habitual lifestyle factors in mental disorders. Rodent models demonstrating marijuana use, have poor food and water intake (Drewnowski et al, 1978). The heavy use of alcohol, tobacco products, marijuana or illicit substances is widely acknowledged amongst populations utilising mental health services, where these lifestyle factors are thought to exacerbate symptoms. Moreover, alcohol use (Wu & Cederbaum, 2003), marijuana use (Sarafian et al, 1999) or a meta-analysis of 36 studies using tobacco; demonstrate significantly increased oxidative stress that damages DNA (Ellegaard et al, 2016).

Changing poor dietary patterns is not easy. Neurobiological evidence supports promoting healthier brain tissues for cognitive well-being. Micronutrient treatments are found to have a limited role in established mental disorders.

The supplementation of combination micronutrients may prevent the development of mental disorders under sub-optimal conditions. There have been multiple studies to date, examining various combinations of multivitamins to provide robust grounds for recommendations. The findings show positive signals across all three dimensions of sub-clinical indicators (anxiety, low mood and stress). Micronutrient combinations with positive signals, have included nutrients required for thyroid hormone production and to maintain enzymatic and transporter signalling. Thyroid hormone production requires selenium, iron, zinc and iodine (Triggiani et al, 2009); whereas, metabolic enzymes and transporters need magnesium, copper and manganese (Bourre, 2006; Laupu, 2017). Copper, zinc and manganese also maintain oxidant/ antioxidant defences so a deficiency in any one, affects the others (Evans & Halliwell, 2001). Various foodstuffs contain these micronutrients. However, seafood, seaweed or protein has the highest nutrient levels for thyroid hormone production (Laupu, 2016). Magnesium supplementation may protect brain tissues following mild hypoxic injury and have clinical benefit for low mood. Lifestyle factors that have a negative impact on the severity of mental disorders are heavy use of alcohol, tobacco, marijuana and/or other illicit substances as they increase oxidative stress to damage DNA.

At present, early interventions aimed at secondary preventative measures are in clinical use across several countries. The early detection of symptoms and intensive follow-up for up to 5 years, aims to prevent relapse and influence the chronicity of mental disorders. Intervention usually consists of low dose pharmaceuticals and psycho-education. However, the field of

nutritional psychiatry is shedding new light on this metabolic disorder. The present work recommends a role for combination micronutrients to reverse sub-clinical symptoms and prevent mental illness. In the context of early intervention this work is ground breaking as it targets primary prevention, verifying at risk behaviours and providing a platform for mental literacy to inform vulnerable populations. Much of this landmark work has been undertaken by Julia Rucklidge and her group at Canterbury University.

Mental health viewed from a quality of life perspective, seeks to empower individuals to become active participants in their cognitive well-being. To be told you are irreversibly mentally retarded or that your symptoms are genetic must be sole destroying. High rates of suicide amongst this cohort are well documented. These people deserve to be given hope. Eugenics must be demystified and degraded to provide the hope that we all deserve as humans. As hope becomes reality we should all be asking ourselves a simple question. Is your brain healthy?

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