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Network Analysis-Based Psychoeducation for GAD

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NETWORK ANALYSIS BASED PSYCHOEDUCATION FOR GAD 1

Running head: NETWORK ANALYSIS BASED PSYCHOEDUCATION FOR GAD: A FIRST STEP

Network Analysis Based Psychoeducation for GAD

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Abstract

Psychoeducation, providing essential information for a client about psychological and physiological processes, is typically the first step in cognitive-behavioral therapy (CBT) but its content and implementation is currently underdeveloped. Generalized anxiety disorder (GAD) is a prevalent mental disorder in the United States. Often, persons with GAD and their primary care providers and family do not recognize or understand the meaning of their symptoms because of the diffuse and imprecise nature of the diagnosis. This thesis aims to investigate a systems network analysis of generalized anxiety disorder to explore suggestions for enhancing psychoeducation intervention as an initial intervention for GAD clients. A network analysis based psychoeducation intervention could also work as a first-step intervention for GAD clients on a wait-lists for CBT. The thesis concludes with a proposal for a pilot study testing the effectiveness of a more developed psychoeducation intervention.

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Introduction

This thesis is a literature-based investigation into a network analysis understanding and potential of psychoeducation for clients diagnosed with generalized anxiety disorder (GAD) who are entering cognitive-behavioral therapy (CBT). People can be directly and/or indirectly affected by anxiety disorder, specifically GAD. Individuals raised in a family dynamic where one or more members have been clinically diagnosed with an anxiety disorder, but him- or herself not having been so diagnosed, may still be exposed to a network of anxiety symptoms. Such individuals may be affected indirectly by the modeling of emotional and intellectual regulations, coping mechanisms and, use of language.

Many adults have a basic understanding of the phenomena of anxiety due to personal experience, dialogue with others, and media portrayals. However, adults often don't have an understanding of the meaning of anxiety disorder from a network systems perspective-- a complex, interactive system of interdependent symptoms. Persons with anxiety disorder may

behave in ways that are deviant from the norm of the social bell curve. Oftentimes behavior that is not considered normal is stigmatized. An example of a general stigmatization of anxiety disorder expression would be assuming someone has a weak character because they exhibit signs of tension and nervousness in larger social groups. Individuals who exhibit a network of GAD symptoms may be uncontrollably focused on perceived and objective negative aspects of their life and consequentially may talk excessively about negatively perceived experiences excessively. This is an example of a behavior that may not fit the social norm and that may cause distress in relationships. Often times big life changes or stressful events may trigger an onset of developing a network of anxious symptoms. People, who do not have an informed knowledge of anxiety disorder from a networks perspective, may have a hard time processing their chronically stressful and self-reinforcing experience.

Pathological anxiety (and other mental disorders) from a network analysis perspective, is a webbed interaction among causes and symptoms. From this complex network approach we are better able to do justice to the understanding of pathological anxiety as a unified, dynamic phenomena. This means that teaching about network analysis of pathological anxiety to GAD clients could be a key component in the development of psychoeducation in CBT. In contast, the simple symptom list approach is to say to a client, "Okay, you have generalized anxiety disorder because you have been irritable, fatigued, and have had problems sleeping and concentrating for six months now". A network analysis approach would say, "It sounds like you have some symptoms that are interacting with each other in a way that has been causing you distress for a while now. Some symptoms that I'm hearing in your dialogue are problems with sleep, which may be effecting your ability to concentrate, which might be influencing you to feel more irritable and fatigued. Also, your experience of chronic irritability might be making it more

difficult for you to concentrate and sleep. All of these symptoms are interacting with and liking amplifying each other. Let's talk about why you might be experiencing these symptoms". These examples illuminate how a deeper level of processing one's experience of anxiety as the expression of a network of interactive causes instead of a passive expression of a set of symptoms that someone "has", as if they're stuck with them until they're "cured".

The theme of this thesis is that a network analysis approach to psychoeducation-- being more comprehensive and dependent on understanding cause-and-effect of symptoms-- could lay the essential ground work for optimal CBT. This would provide the information, resources, and dialog necessary for clients' initial understanding and tolerance of their symptom origins and expression. If GAD clients are able to identify and accept, their unique symptoms, without negative self-judgment, and understand the complexity of anxiety experiences then clients may be better able to benefit from the ensuing cognitive behavioral therapy. Psychoeducation could be developed into a more in-depth and interactive first step that primes clients for long-term commitment of multidimensional understanding of symptoms, as well as a commitment to developing healthy coping strategies.

Since a system network analysis is the guiding framework for this model it should be well-defined for the client. Network analysis rejects the illness ideology or disease model that the DSM 5 and all previous Diagnostic and Statistical Manual (DSM) editions have driven in research, health care, health insurance, laws, culture, and language. Currently people are diagnosed with disorders from the DSM 5, such as Major Depression Disorder, which enumerates that the symptoms persons experience (i.e. fatigue, impaired concentration, weight loss or gain etc.) are caused by the illness of major depression. Network analysis rejects mental disorders being illnesses the way cancer is an illness that can be measured and treated. Instead,

networks, comprised of complex interactions between symptoms. "Most scholars agree that this currently dominant paradigm [disease model] is problematic." (Borsboom & Cramer, 2013).

In order to move forward with a network analysis systems approach to GAD we must accept two concepts. First, we cannot conclude that individual symptoms of a labeled disorder are caused by the same psychological or biological basis. Second, symptoms of disorder-causing distress, dysfunction, deviance, and danger-causally interact with and influence each other (Borsboom & Cramer, 2013).

The model this thesis proposes aims to move the American psychology paradigm towards a network analysis systems understanding and treatment of disorder through psychoeducation intervention. The concept of psychoeducation for this investigation is a composite of multiple therapeutic elements designed to teach clients with mental disorder, and their families, about the fundamental understanding and acceptance of personally unique networks of interacting symptoms for the purpose of committing clients to long-term involvement with the coping processes. The model provides a foundation for a deeper appreciation of symptoms and their causes rather than merely a passive acceptance of symptoms as products of an illness.

While psychoeducation is not yet a prevalent antecedent of therapy, there is some support for its current structure and use. A meta-analysis of psychoeducation for depression and psychological distress was published in 2009 using Cochrane, PsycInfo and PubMed databases for randomized controlled trials testing the effects of psychoeducation; four articles were found out of 9,010 abstracts identified. Brief passive psychoeducational interventions alone are shown to reduce symptoms of distress with small but significant effect (Donker, Griffiths, Cuijpers, Christensen, 2009). Psychoeducation is currently an inexpensive and easily accessible first-step

intervention in CBT for understanding and coping. The accessibility of psychoeducation begs the question of how it can be developed deeper, yielding more powerful effects in research investigations.

Currently, cognitive behavioral therapy is the most empirically supported psychotherapy for depression and anxiety disorders (Forman, Shaw, Goetter, Herbert, Park, 2012). An example of empirical support is the following, in a randomized controlled clinical trial with the treatment group receiving 16-weekly sessions and the control group being on the wait list for treatment. 77% of the treatment group did not meet GAD diagnostic criteria at the 12-month follow up session (Ladouceur et al., 2000). CBT requires the client to bring the insider information, the thoughts, feelings and problem behaviors and the therapist to bring the evidence-based techniques, the clinical experience and a more objective perspective. Tying together these two contributions to the therapeutic process in antecedent psychoeducation could be invaluable. Typically CBT sessions last from 8-16 weeks. CBT usually begins with psychoeducation, followed by cognitive and then behavioral interventions. Environmental factors that the CBT structure takes into consideration in order to tailor therapy to individuals are family history, health, financial, marital and housing status, and level of education (Sheldon, 2011, p. 47). CBT is a primed pair for a network analysis based psychoeducation because it is already designed to factor in the complex environmental network factors that contribute to the four hallmark criteria of psychological disorder: distress, danger, deviance, and dysfunction.

Overview of Literature

Anxiety and Anxiety Disorder as a Concepts

Anxiety is a multi-dimensional phenomena that is collectively thought to be an evolutionary process advantageous for protecting the self from challenge, harm or threat by activating the sympathetic nervous system as well as other physiology. An example of another main physiological player in the system of response to stress is the HPA axis (hypothalamic-pituitary-adrenal axis). The HPA axis is a major component of the neuroendocrine system that is involved in controlling reactions to stress. As this implies, anxiety is a stress-response. Anxiety enables animals to either avoid a potentially dangerous situation or to approach it with heightened awareness of the environment (Corr, 2011). For humans, anxiety is an emotion, a subjective conscious experience leading to physiological responses.

A question encountered in the discussion of anxiety is whether or not it is a subset of fear. It is important to define anxiety in relation to fear for the purpose of research and intervention development. One way to investigate the possible distinctions between fear and anxiety is with anxiolytics. Researchers have tested anxiolytics on rat and mice behaviors in what is thought to be an anxiety producing situation and another situation which is thought to be fear inducing. If the rats and mice respond the same way, while on the drug, then it can be concluded that the behaviors (fear and anxiety) are not actually different (McNaughton, 2010). Neural receptors have evolved across species so we can conclude that the way anxiolytics affects rats and mice will be a similar effect on humans (McNaughton, 2010). This study found that mice respond differently to fear and anxiety-provoking situations when on anxiolytics and panicolytics (anti-

panic medication). This suggests that fear and anxiety are actually discrete phenomena. Often in literature anxiety is described as an element of fear. The contradiction of this study with the description of anxiety as fear in common literature also sheds light on the limitations of our linguistically symbolized understanding of our experiences.

Anxiety is first an automatic, reflexive process before it enters consciousness, which is a controlled process. Corr proposes that anxiety, as a whole process, should be understood as an interaction between the automatic and conscious processes. When stimulus error reaches consciousness, controlled cognitive processes are able to analyze and resolve the stimulus error situation so that future interactions may be acted upon differently to avoid error. Stimulus error is the consciousness recognizing an environmental stimulus as error-triggering (challenging, threatening or harmful). When aversive stimuli reach consciousness, and controlled processes take over, imagination comes into play. The conscious either stops the personal behavior that is contributing to the environmental-error or it produces a detailed and controlled analysis of the perceived stimulus error. This controlled analysis involves imagining why the detected error is an error. Imagination can create aversive scenarios that are detached from the external world; an example is secondary appraisal. Secondary appraisal is the conscious assessing how well the self is able to cope with the detected error. An example of this is if a person registers stimulus A as an error and then imagines that stimulus A is an error because of X, Y, and Z. Then the person proceeds to imagine that they will not be able to adequately respond or handle stimulus A because of L, N, and M.

The conscious process of imagination is evolutionarily advantageous for social interactions; it enables individuals to work out "the best" decision for action based on the imaginable probable outcomes. For anxious-prone persons the process of secondary appraisal

with the use of imagination can be the source of self-generated error-signals. If an individual imagines his or her self to be incapable of being able to adequately respond to an external stimuli he or she may experience excessive worry, difficulty controlling worry and difficulty concentrating.

Anxiety disorder is often an unintentional, unconscious emotional experience that is often difficult for clients that have never been psychoeducated on the phenomenon, to explain what they feel and why and when they began feeling different (Sheldon, pg. 137). GAD is also substantially under-diagnosed and mistreated in the primary care setting, which calls for more research into the causes of GAD, the criteria for identifying GAD and effective treatments for the symptoms. The herein proposed pilot study will incorporate a network analysis understanding of the role of conscious, unconscious, physical, mental, and emotional experience of anxiety disorder.

Excessive Attention and Appraisal to Potentially Threatening Stimuli

Empirical research supports the hypothesis that GAD clients have an attentional bias for negative or threatening stimuli. In a study, in which GAD and control participants were asked to click a button to indicate the location of a word on a screen, GAD participants had a longer response time than control participants when the words were negative or threatening (Mathews et al., 1989). These findings imply that GAD clients are prone to being distracted by their appraisal of non-threatening stimuli as threatening or negative which means that they have a more difficult time with communication, relationships and occupational roles.

If an individual expresses GAD they are more likely to perceive neutral stimuli as threatening. An example of negative primary and secondary appraisal is if person A asks person B, "How are you", and person B has GAD, they may interpret persons A's objectively neutral

tone as negative and threatening (primary appraisal). Person B's reaction to person A may be to imagine that they cannot adequately respond to the negative stimuli (secondary appraisal) and therefore act by avoiding the perceived threat by avoiding eye contact and walking away quickly after saying, "Fine". This interaction could be perceived as rude by person B if they do not know that person A is excessively anxious, thus increasing tension in their relationship.

In a study of GAD participants with a comparison control group, both tasked with selecting a word that is pronounced the same but with different meaning (i.e. flu and flew) showed that GAD participants were more likely to choose the threatening words (Mathews et al., 1989). The findings presented above show GAD as cyclic disorder. Primary appraisal of an external stimuli as negative followed by secondary appraisal of the self being incapable of adequately responding to the stimuli will increase the likelihood of avoidance, and the more the external stimuli is avoided the more likely a person will be to have negative secondary appraisal, which increases the likelihood of future negative primary appraisal. The information in this section works to address the conscious and unconscious cognitive patterns of anxiety disorder for the use of network analysis based psychoeducation.

Impact and Prevalence of Anxiety Disorder

Establishing epidemiological significance of pathological anxiety in general and GAD specifically is instrumental in establishing the relevance of the topic at hand in the context of United States prevalence. Out of all the physically disabled Americans, there are an estimated 3 million (one-third of disabled persons) with a reported mental disorder contributing to their disability. Anxiety disorders are the most prevalent manifestations of mental disorder in the

United States (Druss et al., 2000), meaning that the probability of physically disabled Americans experiencing pathological anxiety in concurrence with their physical disabilities is high.

Generalized anxiety disorder is very prevalent, with 1.2% - 6.4% annually affected in the U.S. (Hoge, Oppenheimer, Naomi, 2004). The National Comorbidity Survey used DSM-III criteria to determine that the average lifetime rate of GAD is 5.1% (Wittchen, Zhao, Kessler, Eaton, 1994).

GAD is typically more common among women than men; in community samples female lifetime prevalence of GAD was 7% and male lifetime prevalence was 4% (Wittchen et al., 1994).

The World Health Organization (WHO) conducted a 14-country study on the prevalence of GAD, the mean results were a lifelong prevalence of 9.2% among women and a 5.7% among men however there was clearly a cultural and/or genetic component with huge variability between countries. In Brazil GAD prevalence was 26% for women and 14% for men and in China prevalence for women was 2.1% and men was 1.7% (Kessler & Wittchen, 2002). One hypothesis is that GAD typically occurs later in the lifespan development because it results from chronic stressors, which usually increase with age, however there are reports of anxiety disorders being common among children. Some issues with socially defining anxiety presented above are: defining the average age of onset, relation to sex, gender and culture, whether GAD is a discrete disorder or whether it is bridged with other disorder symptoms, and it's relation to personality. This evolving social construction of mental disorder creates an inconsistency in research that should be kept in mind when contextualizing the statistical data on GAD and other enumerations of mental health disorder.

GAD can result in significant occupational and social impairment, with individuals diagnosed with GAD missing or shortening an average of 6.3 days of work a month (Hoge et al., 2004). The WHO Collaborative Study on Psychological Problems in General Health Care pooled

from 14 countries to survey disability associated with GAD. The results showed that 38% of GAD participants have moderate to severe occupational disability. Anxiety disorders are correlated with personal suffering, high economic costs, and reduced quality of life (Donker et al., 2009).

In the DSM-IV and DSM-5, criteria for a GAD diagnosis are uncontrollable worry or anxiety for more days than not over the course of 6 months. The experienced anxiety has to be associated with 3 or more of the following symptoms: feeling chronically tense or keyed up, easily fatigued, irritable, difficulty concentrating or the mind randomly going blank, muscle tension and sleep disturbance. GAD can be difficult to clinically treat because of its diffuse nature and because of its high rate of comorbidity with major depressive disorder clinically and in the community (MDD) (Hoge et al., 2004). Taking a network analysis approach we can understand comorbid symptoms as those having effects on other symptoms, or for a symptom to create a *bridge* (rather than comorbidity) to another symptom. An example would be excessive worry (anxiety) increasing issues with concentrating which increases stress level which bridges to insomnia, which is often a symptom thought to be found "in" depression. Also, as mentioned previously, it is common for persons with a physical disability to experience anxiety disorder. This too is an example of a difficult life circumstance bridging with anxiety disorder.

The Role of Diagnostic and Statistical Manual GAD Criteria

How society defines GAD largely determines who will be considered to be experiencing anxiety disorder, how research into anxiety disorder is performed, how clinicians work with clients, insurance coverage, how anxiety is conveyed in media, cultural stigmatization, etc.

Generalized anxiety disorder (GAD) was first introduced in DSM-III in 1987, defined as a diffuse and uncontrolled chronic worry for 1 month or longer, with psychophysiological symptoms such as muscle tension, being easily fatigued, hot and cold spells, difficulty concentrating, irritability and clammy hands. Other DSM-III criteria for the diagnosis of GAD included the provision that these symptoms cannot exist in conjunction with other psychotic disorders or be due with medications. DSM-IV changed the GAD diagnostic requirement to a duration requirement of 6-months because research was finding that GAD was highly comorbid with depression and physical disabilities. There is still debate on whether GAD duration requirements should be 1-month or 6-month, or something else entirely. Some researchers argue that there is empirical evidence for persons having anxious temperaments early in life which could be an underlying factor for GAD. Someone with an anxious temperament could begin with having acute episodes that increase in duration over time (Kessler & Wittchen, 2002).

Since the 1987 introduction of GAD as a diagnosis there has been debate on whether GAD is a precursor, a bridge (comorbid), or residual of another primary diagnosis, such as major depressive disorder (MDD). Research has found that GAD can exist as a precursor, a bridge, a residual and a separate diagnosable disorder. GAD has been shown to be highly bridged (approximately 95%) with MDD in the National Comorbidity Survey. When GAD is bridged with another disorder symptoms are more severe, activities of daily life are impaired to a greater degree, and treatment results are poorer. "Pure" GAD (persons with no other diagnosis) are found in 10% - 18% of GAD clients (Kessler & Wittchen, 2002).

Anxiety is something that everyone experiences. It is not necessarily a problem, as previously stated; it is an evolutionary adaptation. Nevertheless, we know that anxiety can be a problem to varying degrees. While we know that anxiety is a real part of our experience and is a

potential problem, we also must recognize that the language with which we choose to discuss anxiety as a problem is socially constructed and the language that we use to define anxiety as a problem influences how we think and talk about working through problem anxiety. A concrete example of this is how the DSM-III differs from IV and 5, which are the same, with requirements for symptom duration for GAD diagnosis.

Statistical data, shaped by how the DSM enumerates disorders, is currently the closest we can get to empirical support for our social constructions of mental disorder. We should use statistical measures with precise calculation, respectful caution and all the while keeping it in context of its limited correlational contribution. DSM criteria for GAD is useful in its standardization of clinical diagnosis and research but it is not a perfect system.

Genetics & Neurotransmitters

A network analysis based psychoeducation for GAD should incorporate what is known about the physical manifestation of anxiety within the brain as well as the genetic components that may be influencing the brain's disposition to experience disorder. The body is a complex, dynamic system, it cannot be said that *a gene* causes *a symptom*. At the same time it should be recognized that neuroscience is beginning to find more correlations between genes, neurotransmitters and hormones in their effects in regulating our physiological and psychological functions. Epigenetics, heritable changes to gene activity that are not due to change in DNA sequencing, is another emerging field of study important to this complex dynamical system that should be considered but that will not be covered in this article.

Psychological disorder cannot be clearly defined by the physical elements seen within the brain. In part, science cannot provide such clear distinctions because technology and research has not yet evolved that far, but also, it is questionable as to whether neuroscience will ever be able to evolve so far as the brain is plastic, constantly changing with experience, and experience is vastly complex. However genetics and neurotransmitters can show physical patterns to disorder that are consistently demonstrated in replicated research.

Family studies on GAD patients and their first-degree relatives (parents, siblings, children) show that if a person has a first-degree relative with an anxiety disorder that they are six times more likely to have an anxiety disorder during their lifespan compared to someone that does not have a first-degree relative with an anxiety disorder, suggesting that there is a strong genetic or exposure component to GAD. GAD is also highly bridged (comorbid) with depressive symptom networks, which indicates that there may be a shared genetic and electrochemical component, which will be addressed further. Clients simply should not be urged to "will away" excessive anxious dispositions but rather should be enlightened on the complexity of our behavior. The following information on neurological components of anxiety should be incorporated in psychoeducation for GAD clients to gain a basic understanding of the genetic and electro-chemical processes involved in anxiety.

In the research literature the GABA receptor system has been a main focus for anxiety and depression disorder. GABAergic neurons are the highest population of inhibitory neurons in the brain, "GABA is the primary mediator of inhibitory transmission in the mammalian central nervous system" (Kalueff & Nutt, 2007). At a basic level this means that GABA prevents over excitation. Anxiolytics work as GABA agonists, helping individuals to receive more GABA and therefore inhibit or relax more. Incorporating poignant education of neural networks and action

potentials in relation to feeling, thinking, and acting in general and then in relation to anxiety would provide clients with a way to physically understand their experience of anxiety as well as its correlation to depression.

Some genes, neurotransmitters, and hormones have been identified as having some influence on the experience of anxiety. Looking at these findings side-by-side for the purpose of a network based psychoeducation is to elucidate the complexity of emotion, cognition and behavior. In one study genes coding both α 3 and α 6 (nicotinic acetylcholine receptors) are found to be correlated with anxiety and depressive behaviors in animals (Kalueff & Nutt, 2007). A human study showed a correlation between a mutation in the β1 subunit of GABA-A receptor and treatment-resistant anxiety and depression (Kosel et al., 2004). In a third study, both depression and anxiety are correlated with abnormalities in secretions of adrenocorticotropic hormone, cortisol/corticosterone, corticotropin-releasing hormone, adrenal catecholamines, oxytocin, prolactin, and rennin (Carrasco & Van de Kar, 2003). In a fourth study (rodent models) 5-HT₂A (serotonin receptors) regulates anxiety behaviors. Genetic deletion of this receptor in mice has shown an anxiolytic effect (Heisler et. al., 2007; Weisstaub et al., 2006). Three neurotransmitter systems and seven hormones have been mentioned so far in relation to anxiety and depression, which elucidates the complexity of looking at behavior as an electro-chemical process. The concert of anxiety is not a solo act from one genetic component or one neurotransmitters distribution but, rather is a symphony of physical interactions at play and each individual's symphony will sound different because its members are unique.

For the study of genetic and neurotransmitter activity with anxiety researchers often use mice and/or rats as subjects to alter because of the ethical issues of working with humans and because we largely have similar brain structure and neurotransmitter systems. Rat and mice

research can contribute to a bootstrapping approach of understanding the difference between anxiety and fear in humans (McNaughton, 2010). We cannot equate human anxiety to rat and mice anxiety but we can measure the physiological patterns of rats and mice internal responses to harm, threat or challenge.

In one study a group of mice was genetically manipulated to have partial impairment of GABA A-receptor function. When the manipulation group was compared to a control group of wild-mice with the task of exploring a new compartment, the genetically manipulated mice were less likely to explore new areas of the compartment. The genetically manipulated mice also showed a freeze response when presented ambiguous stimuli (Crestani, Lorez, Baer, Essrich, Benke, Laurent et al., 1999). The results of this study was the finding that behavioral inhibition to naturally aversive stimuli is attributed to the hippocampus and cerebral cortex in both animals and humans, meaning they may be involved in the process of anxiety as anxiety is often inhibition of behavior in some regard (Crestani et al., 1999).

Other factors are likely to contribute to the phenomena of GAD as well. Neuroimaging offers some vague insight into the electro-chemical activity of anxiety disorders. A PET study that measured the cerebral glucose use in 18 GAD participants during a passive viewing task found increased metabolism in the occipital lobe (vision), temporal lobe (language and auditory function), frontal lobe (decision making), basal ganglia (voluntary movement, habits, procedural learning and emotion) and right parietal lobe (sensory information and spatial awareness) compared to control participants (Wu et al., 1991). The participants of this study were also prompted with an active vigilance task which showed a correlation between anxiety diagnoses and intensity of basal ganglia activation (Wu et al., 1991). This study illustrates the complex activity of anxiety in the brain.

An introduction to the complex coordination of genetics, epigenetics, and neurotransmitters activity as mentioned above, for GAD clients within a psychoeducation intervention has the potential to provide a foundation of physiological understanding of their experience of disorder. A base foundation is necessary for incorporating biofeedback technology into the psychoeducation intervention because clients would need to understand what it is that they're seeing. The biofeedback technology to be discussed later would also work to reinforce clients understanding of anxiety as a physiological and psychological experience.

Anxiolytics in relation to anxiety disorder relief

Benzodiazepines are currently the most common form of pharmaceutical agents for treating anxiety and they work on GABA receptors as agonists (Hoge et al., 2004). Anxiolytics are targeted and fast-acting. Alcohol also works as an agonist for GABA receptors. Clients of the age 21+ could be informed of this within a psychoeducation intervention to gain a better understanding of how and why anxiolytics work the way they do. Antidepressants, while less targeted for specific receptors, are also shown to increase GABAergic functions in both humans and animals (Kalueff & Nutt, 2007). Just like alcohol, anxiolytic drugs have side effects too, which include drowsiness, dizziness, lowered blood pressure, slowed breathing, and problems with memory. The longer anxiolytics are used, the worse the side effects are. Withdrawal from anxiolytics typically produces: anxiety, insomnia, confusion, shaking and sweating. The reason why withdrawal is experienced when chronic use of anxiolytics is stopped is because the anxiolytics change the genetic expression of GABAergic neurons to have fewer receptors. When there are fewer GABA receptors there is a decreased ability to inhibit action potential.

A psychoeducation intervention with a network analysis approach will work to bring awareness to the variety of factors that may be influencing the experience of anxiety disorder as well as how possible actions will influence how said factors interact with each other. An example of how a network analysis approach could influence an individual to make well thought through decisions on how to nurture their mental and physical health would be to explain how anxiolytics work similar to alcohol in that they can increase inhibition, promoting more relaxing states but that there is a real risk for abuse which could have long-term consequences. With this understanding an individual may decide to use anxiolytics selectively, as many do with alcohol, during the appropriate time and place and to an appropriate degree.

Psychoeducation

Since the mid-1980s researchers in Germany have implemented a psychoeducation-based intervention in the framework of CBT for the empowerment of directly and indirectly affected individuals (Bauml, Frobose, Kraemer, Rentrop, Pitschel-Walz, 2006). CBT is effective at addressing GAD clients' negative bias in part because it facilitates clients and therapists working together to identify the dysfunctional beliefs and automatic thoughts that create negative interpretations and then working towards finding rational alternatives for the beliefs and thoughts. Current research on psychoeducation interventions define the concept as having three main components which are the following; 1. Therapeutic interaction 2. Clarification 3. Enhancement, of coping competence (Bauml et al., 2006).

Therapeutic interaction is the quality of relationship between therapist and client that is of the utmost importance. Therapists should communicate appreciation, respect, a sense of

calculability and unfailing optimism. Therapeutic interaction can also involve group therapy so that clients may process a sense of unity in their common struggles and goals. *Clarification* is the need to convey the fundamental information for understanding of the disorder as well as the impact of the disorder on the clients' behavior. For clarification to be optimal the fundamental information for disorders should be consistent across therapies, this consistency may require a kind of psychotherapy alliance in the field. Clarification should address topics with positive overtones as well as topics with negative overtones as well as normalization of relapse and destigmatization of the disorder at hand. *Enhancement of coping competence* involves providing information on treatment options, which is a form of tangible assistance.

There is mounting evidence and support for psychoeducational interventions for a variety of categorized psychological disorders in countries around the world, but not in the United States. A randomized study in Munich, Germany, showed that a two-year psychoeducation based program for schizophrenic clients resulted in significant reduction of re-hospitalization, from 58% to 41% and a shortening of hospital days, from 78 to 39 days (Bauml et al., 2006). The guidelines of the American Psychiatric Association (APA) and the German Society for Psychiatry, Psychotherapy, and Neurology (DGPPN) label psychoeducation as a standard aspect of therapy programs for acute and post-acute schizophrenic clients. In a meta-analysis of psychoeducation for schizophrenia and other mental disorder, the combined results of 44 articles with a total of 5,142 participants, showed that psychoeducation facilitates relapse prevention, reduction in re-admission to hospitals, encourages compliance to medication and reduces the length of hospital stays (Xia, Merinder, Belgamwar, 2011). A quasi-experimental design conducted with 117 patients (suffering from various psychiatric disorders) at a psychiatry clinic in Uganda showed that compared to patients that received the usual clinical care, patients that

received formalized psychoeducation sessions at each visit were more likely to adhere to medication and had significantly higher understanding and knowledge of their disorder (Prost, Musisi, Okello, Hopman, 2013). In an open-trial in the Netherlands 21 self-referred participants, that met DSM criteria for hypochondriasis completed six 2-hour small group psychoeducation sessions aimed at gaining a greater understanding of hypochondriasis. Pre- and post-test measures found that participants had improved their hypochondriasis and depression at 4 weeks and 6 months follow-up (Bouman, 2002).

Psychoeducation serves as a basic orientation, facilitating interpretation to help clients and their families to become experts of their mental disorder symptoms. Psychoeducation provides optimal facilitation of the combination of self-help and professional help, ideally for first-time clients. Clients of GAD should be provided support through psychoeducation for optimal understanding of the meaning and processing of GAD as well as the psychosocial, pharmacotherapeutic, and psychotherapeutic process of treatment. If clients are not first offered this supportive education they may be at a suboptimal level throughout their treatment processes.

Most people have natural psychological coping mechanisms for depressive and anxious moods. However when people first experience excessive and prolonged symptoms of uncontrollable worry with physiological symptoms (GAD symptoms) individuals may not be aware of or understand what they are suffering through and why. Professionally provided psychoeducation offers an enabling of understanding and accepting GAD symptoms through informational brochures, books, and videos, (such as, the "Treating Anxiety Disorders: Educational Videos" offered on adaa.org), and most importantly dialogical support and supervision. The dialogical support should not be supplemented with media materials because

GAD is a diffuse condition and highly specific to individuals. Dialog is necessary for personal customization, adaptation and facilitating questions and answers.

In addition psychoeducation also works to de-stigmatize GAD for clients by explaining the conceptual and physiological processes that produce the complex network of symptoms. This internal de-stigmatization is important because often public perception stigmatizes mental disorders, including GAD. When public perception stigmatizes a psychological condition, it often makes it more difficult for individuals diagnosed with said condition to be positively affected by treatment because of external and internal shame (Fung, Tsang, Cheung, 2011). CBT has been shown to decrease and in some cases eliminate internal shame in social anxiety disorder (SAD) patients (Hedman, Strom, Stunkel, Mortberg, 2013), which has implications for CBT effectiveness on decreasing stigmatization of generalized anxiety disorder.

Psychoeducation of Physiology with Biofeedback

Biofeedback is typically a three-step process that includes, becoming aware of physiological arousal, learning to control the arousal response, and transferring the practice of controlling the physiological response to everyday life situations. Biofeedback has been shown to be an effective tool for clients with GAD. In a controlled randomized clinical trial, GAD participants that received biofeedback in addition to counseling showed statistically significantly greater reductions in Beck Anxiety Inventory scores compared to control group participants that only received counseling (Ratanasiripong, Sverduk, Prince, Hayashino, 2012). Biofeedback devices are found to be very easy to use and noninvasive to daily life by researchers and clinical participants. Biofeedback training is also easy to facilitate, it does not need to be led by a licensed practitioner. The cost of biofeedback devices are also reducing as technology advances.

Two common forms of biofeedback that are statistically and clinically significantly supported in the literature are based on EMG (electromyograph) and EEG (electroencephalograph) monitoring. EEG is a form of neurofeedback. Electrodes are placed on a person's scalp and they are instructed to monitor a screen which indicates brain waves through visual and/or sound display. EMG is a form of muscle contraction feedback. Electrodes are placed over target skeletal muscles and muscle action potentials are monitored. The feedback can detect irregular muscular and neural activity. Anxiety clients may monitor their feedback and be told to control or maintain amplified activity without being told how. Surprisingly, more often than not, clients are able to do this. In a controlled randomized study of GAD participants, significant improvement in control of physiological symptoms was found with both the EMG and the EEG technologies (Agnihotri, Paul, Sandhu, 2008).

Incorporating EEG and EMG biofeedback training in psychoeducation sessions could be very instrumental in training GAD clients to pay attention to their sympathetic nervous system responses in order to gain greater control over their arousal. GAD clients could potentially be provided with portable biofeedback devices to use outside of the clinic while participating in network analysis based psychoeducation interventions as a learning component for the physiological aspect of anxiety symptoms.

Pilot Study Proposal

The objective of the foregoing literature review is to gain an understanding for the meaning and potential of psychoeducation in cognitive behavioral therapy for generalized anxiety disorder. The study proposal is a suggested method for future development of a

psychoeducation intervention based on the components covered in the literature review, for first-time diagnosed GAD clients.

This suggested pilot study is quantitative, comparing control and experimental groups.

Participants could be recruited over the course of a year from primary care referrals in the

Portland metro area, from doctors who suspect clients have GAD. The experiment would require recruitment of 100 GAD participants. All procedures would be approved by the Institutional Review Board of Portland State University.

Participants

Each participant must meet the DSM-5 GAD criteria and be of mild to moderate degree of GAD diagnosis, determined by the Generalized Anxiety Disorder Severity Scale (GADSS). Participant recruitment would include a diverse range of age, gender, social economic status and level of education in order to support generalizability. A diversity demographic quota would be established prior to start of recruitment to set requirements. All participants would have to fill out a consent form before step-one, being assessed for GAD with DSM-5 criteria. Potential participants would be assessed for GAD by clinician, psychologist or licensed clinical social worker that is not involved in the experiment. Participants would not be excluded if they met other DSM-5 diagnoses in conjunction with GAD because oftentimes anxiety is highly bridged with other disabilities and disorders.

Methods

Once 100 participants have signed consent forms and have met mild-moderate DSM-5 GAD diagnosis, participants will be randomly assigned to their group using a computer-generated randomized list. Participants would not know of the different treatment groups and

would not know which one they would be in. Other measurement scales would need to be researched and used at pre-, post- and follow-up sessions. Other measurements might include quality of life, life stressors, and social support self-report measures.

This pilot study would have four comparative groups. Group 1 would participate in 8 weekly 1.5 hour sessions of CBT, without any pre-therapy psychoeducation involved. Group 2 would participate in 8 weekly 1.5 hour sessions of CBT with only one passive pre-therapy psychoeducation session, which would include an informational brochure, a video and a book on generalized anxiety disorder. Group 3 would participate in 8 weekly 1.5 hour sessions of CBT with one pre-therapy moderate psycho-education session, receiving the informational media provided to group 2 and having psychoeducational dialog. Group 3's one session of psychoeducation dialog would include education of the following: the concept of anxiety, the concept of generalized anxiety disorder, excessive attention and appraisal to potentially threatening stimuli, and the role of the DSM defined GAD criteria. Group 4 would participate in 8 weekly 1.5 hour sessions with in-depth psychoeducation, involving informational media and 2 pre-therapy sessions based on psychoeducational dialog. The two sessions of psychoeducational dialog in group 4 would include education on the following: the concept of anxiety, the concept of psychological disorder, the concept of generalized anxiety disorder, excessive attention and appraisal to potentially threatening stimuli, the role of the DSM defined GAD criteria, the role of genetics and neurotransmitters, the drug actions and effects of anxiolytics, biofeedback using EEG and EMG, and the role of psychoeducation with a network analysis approach.

All participants would be tested for DSM-5 criteria for GAD after the last CBT session and at a 12-month follow up to test for long-term efficacy of each treatment group. In order for participants results to be included they must have attended all 8 classes and have completed all

pre-, post- and follow up measures. Follow up measures might include a survey to test for how useful the participants perceived their designated training to be. The persons administering the CBT sessions would have to be certified CBT psychotherapists, social workers and nurses with prior experience working with GAD clients.

After the completion of the 12-month follow-up session participants would be debriefed on the design and purpose of the study. After debriefing participants would be asked to attend a group discussion with their designated experimental group (1,2,3 or 4). The discussion group would facilitate feedback for the researchers and group support for the participants. For the hypothesis to be supported the results would need to show clinical and statistical significance between group 1 and 4.

Hypothesis

This pilot study aims to examine whether psychoeducation can be constructed with a network analysis approach so as optimize teaching clients about the complex dimensionality of anxiety, with the client's and practitioner's goal of facilitating increased awareness and consequently increased control over their network of symptoms. If anxiety is an emotional, cognitive and physical phenomenon, then clients experiencing anxiety disorder can be psychoeducated on the symbolic understanding of anxiety (labels and constructs of symptoms), the limitations of labels and the importance of holding labels in the context of social construction, the potential predispositions to emotional reactivity (electro-chemical movement and epigenetics in personal history), and physical manifestations, with clinical and statistical significance (Borsboom & Cramer, 2013).

Conclusion

While psychoeducation is successfully implemented in Germany and the Netherlands as an important stand-alone mediation, it is highly under developed in the United States within CBT and as a stand-alone intervention. Anxiety over past, present and future interactions is a part of the human experience. Anxiety in moderation can help motivate people to approach or to avoid potentially challenging interactions. However, the rate of anxiety disorder in the United States is high. This thesis aims to continue a contemporary conversation on the potential of psychoeducation, with a network analysis approach, for psychological disorder, by focusing specifically on the potential impact of psychoeducational mediation for generalized anxiety disorder.

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