Neurological and Psychiatric Diseases and Their Unique Cognitive Profiles: Implications for Nursing Practice and Research

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ABSTRACT

To successfully negotiate and interact with one's environment, optimal cognitive functioning is needed. Unfortunately, many neurological and psychiatric diseases impede certain cognitive abilities such as executive functioning or speed of processing; this can produce a poor fit between the patient and the cognitive demands of his or her environment. Such nondementia diseases include bipolar disorder, schizophrenia, post-traumatic stress syndrome, depression, and anxiety disorders, just to name a few. Each of these diseases negatively affects particular areas of the brain, resulting in distinct cognitive profiles (e.g., deficits in executive functioning but normal speed of processing as seen in schizophrenia). In fact, it is from these cognitive deficits in which such behavioral and emotional symptoms may manifest (e.g., delusions, paranoia). This article highlights the distinct cognitive profiles of such common neurological and psychiatric diseases. An understanding of such disease-specific cognitive profiles can assist nurses in providing care to patients by knowing what cognitive deficits are associated with each disease and how these cognitive deficits impact everyday functioning and social interactions. Implications for nursing practice and research are posited within the framework of cognitive reserve and neuroplasticity.

Keywords: cognition, cognitive remediation, cognitive reserve, comorbidity, executive functioning, memory, speed of processing

Cognition emerges from the complex interaction of neurons, astrocytes (by far the most numerous glial cells), and other glial cells in the brain and is shaped by the person's interaction with his or her environment (Fields, 2009). But cognition is also composed of unique interactions of different cognitive abilities such as executive functioning (i.e., reasoning, planning, judgment) and attention, memory, speed of processing, and psychomotor ability. But with some neurological and psychiatric diseases, mild to moderate declines in particular cognitive abilities are observed. Studies show that even small nonpathological deficits in certain cognitive domains can negatively impact how people perform tasks for everyday functioning (McGuire, Ford, & Arjani, 2006). For example, visual speed of processing is an essential cognitive ability needed for safe driving (Ball, Edwards, & Ross, 2007).

The purpose of this article is to provide a general overview of some of those unique cognitive deficits found in some of the common, nondementia neurological and psychiatric diseases. Thus, a basic review of each disease, along with their unique cognitive profile, is provided. A cognitive profile is simply a listing of what cognitive abilities are generally compromised for each disease. It is important for nurses and nurse practitioners as caregivers and health educators to understand the cognitive profiles of their patients so that they possess a better awareness of why patients behave and emote as they do. As a guide to the reader, Figure 1 captures the primary points for each of the neurological and psychiatric diseases reviewed. Implications for nursing practice and areas of potential research are provided within the framework of cognitive reserve and neuroplasticity (Figure. 2).

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Common Neurological and Psychiatric Diseases and Cognitive Profiles

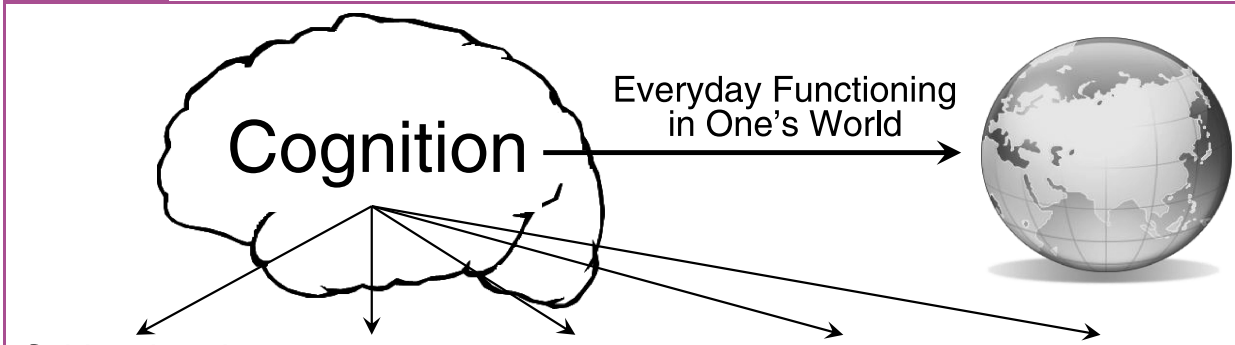
Cognition is composed of several separate mental abilities or domains that interact to form global cognition.

These mental abilities are generally categorized into executive functioning, memory/attention, speed of processing, language, and psychomotor functioning. Even within each of these larger abilities, subsets of these mental abilities are observed. For example, memory can be categorized into declarative memory, episodic memory, semantic memory, procedural memory, visual memory, verbal memory, and prospective memory; furthermore, within each of these memory types, further distinctions can be made (Vance, Graham, Fazeli, Heaton, & Moneyham, 2012). Different brain regions and structures are responsible for each of these particular mental abilities. Thus, if these brain regions and structures are compromised by disease-related insults, this will produce cognitive deficits in certain mental abilities.

A number of diseases such as hypertension, heart disease, and diabetes are known to negatively impact neurological functioning, which in turn reduces cognitive efficiency and creates specific cognitive deficits (Vance, Larsen, Eagerton, & Wright, 2011). The patterns of cognitive deficits can be referred to as cognitive profiles. For example, in hypertension, white matter lesions are common in the frontal lobes of the brain, the region largely responsible for executive functioning and reasoning ability. Thus, those with uncontrolled hypertension may be more likely to experience a cognitive profile reflective of poorer executive function (Hajjar et al., 2009). Thus, executive function and reasoning deficits can result in poorer impulse control, inhibition, and frustration in figuring out complex problems or situations, which can lead to poor emotional homeostasis (McLaughlin & Nolen-Hoeksema, 2011). Similarly, neurological and psychiatric diseases produce

Cognitive efficiency and deficits may emerge in individuals with a number of diseases such as hypertension, heart disease, and diabetes as a result of the negative impact of these disorders on neurological function.

FIGURE 1 The Cognitive Profiles of Select Neurological and Psychiatric Diseases



Schizophrenia
- Executive function deficits are considered a phenotypic hallmark.
- Global deficits in attention, memory, and speed of processing are also observed.
- Disorganized language (e.g., word salad) is a characteristic cognitive deficit during psychotic episodes.
- Cognitive improvement are generally not observed with medication.

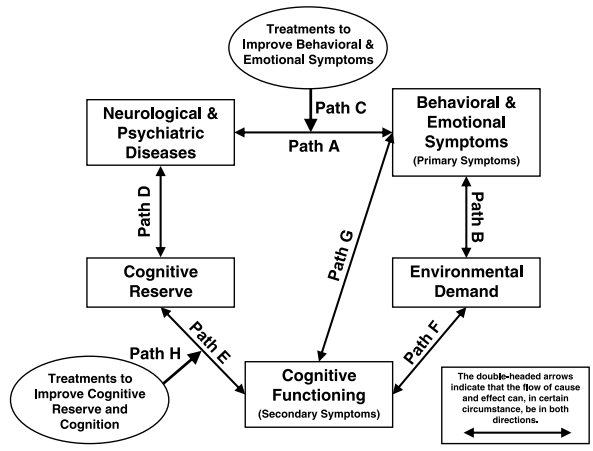
Bipolar Disorder
- Executive function deficits may be the most prominent cognitive deficit; but, it is not as severe as seen in schizophrenia.
- Global deficits in attention, memory, and speed of processing are also observed.
- Disorganized language (e.g., rapid or rambling speech or poverty of speech) is a characteristic cognitive deficit during psychotic episodes.
- In recovery phase, global cognitive deficits are still observed.

PTSD
- Global deficits in attention and memory are observed.
- No executive function or language deficits are normally observed.
- Medication therapies have proven effective in mitigating such attention and memory deficits.

Depression
- Global deficits in attention, memory, executive function, and speed of processing are observed.
- No language deficits are normally observed.
- Cognitive deficits may range from mild to severe.
- It is not certain whether disease-modifying medications mitigate such cognitive deficits.

Anxiety Disorder
- Global deficits in attention, memory, executive function, and speed of processing are observed.
- No language deficits are normally observed.
- Cognitive deficits may range from mild to severe.
- Attention processes are captured by threatening or negative emotions, thereby slowing down or impairing overall cognitive performance.
- It is not certain whether disease-modifying medications mitigate such cognitive deficits.

FIGURE 2 The Dynamic Interactions Between Neurological and Psychiatric Diseases With Behavioral and Emotional Symptoms and Cognitive Functioning



different cognitive profiles. As investigated in a previous article on cognitive profiles in common medical comorbidities (Vance et al., 2011), this article provides a follow-up with a focus on prevalent neurological and psychiatric diseases known to exert alterations and deficits in cognitive functioning. Thus, this article focuses specifically on schizophrenia, bipolar disorder, post-traumatic stress disorder, depression, and anxiety.

Schizophrenia and Cognition

Schizophrenia is a progressive and degenerative disease affecting approximately 1% of the adult population. Particularly devastating, the onset typically occurs in late adolescence or early adulthood, a period normally associated with accelerated social and intellectual maturation (Keltner, 2011a). Traditionally, the descriptive profile of schizophrenia has been divided between positive and negative symptomatology. Positive symptoms are those associated with exaggerated perception, or heightened responses to normal sensory stimuli (e.g., delusions of grandeur, hostility, or auditory hallucinations). Negative symptoms include behavior that may be viewed as ''lacking'' or ''diminished'' from what one might expect to be ''normal'' (e.g., flat affect, poverty of speech, or catatonia; Keltner, 2011a). The negative symptoms are largely understood to manifest from cognitive deficits resulting from the dysfunction of frontal and temporal lobes of the brain (Lesh, Niendam, Minzenberg, & Carter, 2011), areas typically associated with executive functioning and language. Patients present with varying degrees of both positive and negative symptoms and require an individualized approach to treatment.

On the basis of an analysis of pen-and-paper tests commonly used to assess cognition of those with schizophrenia, the Measurement and Treatment Research to Improve Cognition in Schizophrenia has been used to identify seven specific cognitive domains that are affected in schizophrenia: attention, speed of processing, verbal memory, visual memory, reasoning, problem-solving, and social cognition (Marder & Fenton, 2004). This represents widespread cognitive deficits, which may explain some of the symptomatology associated with this disease; however, executive function deficits appear to be particularly salient with schizophrenia.

Executive function is a multifaceted, higher cognitive domain that is partially dependent on intact speed of processing and attention for optimal function (Vance et al., 2012). Executive function deficits are generally considered the cognitive hallmark of schizophrenia and are even viewed by some as the ''phenotypic marker'' of the disorder (Thurston-Snoha & Lewine, 2006). These patients typically score low on tests of logic and reasoning and present with symptoms of disorganization as well as poor response inhibition and mental flexibility (Tan, 2009). These deficits are thought to stem from miscommunication between the temporal lobe and prefrontal cortex. In general, the prefrontal cortex acts to organize conflicting behavioral responses to new stimuli by directing the brain away from socially unacceptable behavior and toward behavior considered appropriate for the given situation (Lesh et al., 2011).

In addition to executive function deficits and other cognitive domains, social cognition deficits are also common in persons with schizophrenia. Impairments in social cognition affect a person's ability to accurately perceive the motives, intentions, and emotions of others. Patients are often unable to interpret facial expressions and gestures accurately or may misinterpret them altogether. Those having difficulties identifying social cues typically have poor social outcomes. These poor interactions in one's social environment typically interfere with the affected person's ability to maintain close relationships or employment, which can

further compromise emotional homeostasis (Harvey & Penn, 2010).

Fortunately, the introduction of atypical antipsychotics offers the promise of improved compliance to medical regimens, decreased adverse affective profiles, and improved treatment of negative symptoms compared with traditional medications. However, in one of the largest independent double-blind comparisons to date on the effectiveness of atypical versus typical antipsychotic medications, the National Institute of Mental Health released the Clinical Antipsychotic Trials of Intervention Effectiveness Study and was among the first to challenge the claims of antipsychotic manufacturers. The study compared the efficacy of multiple atypical medications (i.e., olanzapine, risperidone, quetiapine, and ziprasidone) against the older typical antipsychotic perphenazine in 1,493 adults with schizophrenia. From this study, it was found that only olanzapine outperformed perphenazine in terms of adherence but actually carried a worst side effect profile including weight gain, increased cholesterol, and other metabolic disturbances. Unfortunately, cognitive improvements with the atypicals were only marginal at best and may only have been secondary to improvements in positive symptoms (Lieberman et al., 2005).

Current treatment options offer only limited improvement in cognitive function (Lesh et al., 2011). Because of these modest improvements in cognitive and the related negative symptoms, a new glutamate model of schizophrenia is being extensively studied. Although a discussion of this model lies beyond the scope of this article, its insertion reflects the thrust of this paradigm (i.e., the amelioration of cognitive/negative symptoms; Citrome, 2011). With the help of initiatives (i.e., Cognitive Neuroscience Treatment Research to Improve Cognition in Schizophrenia), the Federal Drug Administration agreed to include improvements in cognitive dysfunction as a discrete criterion for future drug approval (Buchanan et al., 2005). These initiatives are essential in changing how the scientific community thinks about the treatment of schizophrenia.

Bipolar Disorder and Cognition

Following a wave of interest in the neurocognitive profile of schizophrenia, similar interests have emerged in the study of bipolar disorder (Latalova, Prasko, Diveky, & Velartova, 2011). Bipolar disorder is estimated to affect about 2.6% of the adult population and affects men and women equally (Keltner, 2011b; Kessler & Wang, 2008). Like schizophrenia, the onset is typically in the early 20s and is likely to begin with a major depressive episode (American Psychiatric Association, 2002; Keltner, 2011b). Bipolar disorder is characterized by mood swings of variable severity between manic or hypomanic episodes and major depression. During a manic episode, patients may complain of racing thoughts or may be observed speaking or behaving outlandishly. A grandiose sense of self is common. Auditory hallucinations may be present. This episode may be followed by a period of severe depression where the patient may withdraw from his or her environment. In fact, assessments for suicidal ideation are necessarily early in these periods. These episodes may swing from one extreme to the other but are often separated by a refractory period (Keltner, 2011b).

Studies of cognition and bipolar disorder have focused overwhelmingly on identifying deficits in patients diagnosed with bipolar disorder I (i.e., manic episodes) and bipolar disorder II (i.e., no manic episodes but multiple major depression episodes), despite there being two other major variations (i.e., cyclothymic and mixed; Latalova et al., 2011). Current data suggest that, even during euthymic periods (i.e., without current mania or depressive episode), cognitive deficits are present in both bipolar disorders I and II compared with healthy controls. For example, researchers from the University of Barcelona examined functional outcomes on 172 euthymic bipolar patients (bipolar disorder I, $n = 106$ and bipolar disorder II, $n = 66$) and 61 healthy controls using the Hamilton Depression Rating Scale, the Young Mania Rating Scale, and the Functioning Assessment Short Test. Cognitive deficits were observed in both bipolar disorders I and II patients compared with healthy controls. A post hoc analysis identified global cognitive deficits in both types of bipolar disorder, which is consistent with previous data (Rosa et al., 2010; Tabares-Seisdedos et al., 2008).

The types of cognitive deficits in patients with bipolar disorder are similar to those found in schizophrenic patients, although they are typically less severe. It has proven difficult to identify specific cognitive deficits that are inherent to the disorder and not because of confounding factors such as medication, manic episodes, or residual depression. Therefore, cognitive presentation may be dependent on mood state and can even be affected by seasonal changes (Beyer, Kuchibhatla, & Payne, 2004). In a meta-analysis of 185 studies of cognition and bipolar disorder, Bora, Yucel, and Pantelis (2009) identified verbal memory, executive function, and sustained attention as being the most frequently reported deficits in euthymic bipolar disorder patients; however, response inhibition, a component of executive function, may be the most prominent cognitive impairment.

Similar to schizophrenia, ventricular, temporal, and dorsolateral prefrontal cortex abnormalities have been observed in bipolar disorder patients using magnetic resonance imaging (MRI) scans. Ventricular enlargement

is suggestive of tissue degeneration, and these abnormalities are consistent with findings of verbal memory and executive function impairments (Bruno, Barker, Cercignani, Symms, & Ron, 2004). The previously mentioned studies suggest that bipolar disorder patients in the ''recovery'' phase of the disorder still exhibit cognitive deficits. Finally, the severity of such cognitive deficits may also be dependent when the onset of the disease occurs. Late onset bipolar disorder (60+ years) is associated with greater cognitive impairments compared with patients who develop the disorder before the age of 40 (Schouws et al., 2009).

Post-Traumatic Stress Disorder and Cognition

Post-traumatic stress disorder (PTSD) is a common clinical disorder that occurs in response to being exposed to a severely traumatic stressor. According to the Diagnostic Statistical Manual (American Psychological Association, 2000), a PTSD diagnosis includes reexperiencing the stressor, avoiding situations that remind one of the stressor, and hyperarousal. Other symptoms include feeling numb, flat affect, and a feeling of detachment. In addition, patients with PTSD often report cognitive disruptions (i.e., deficits in concentration, attention, and memory).

Researchers have observed that PTSD patients exhibit poorer cognitive functioning. Vasterling and colleagues (2002) investigated the association between PTSD and cognitive performance within a group of 47 Vietnam veterans. They observed that PTSD severity was negatively associated with performance on tasks of sustained attention, working memory, initial learning, and estimated premorbid intelligence. Jelinek and colleagues (2006) examined memory functioning in a group of 80 individuals with and without PTSD. These researchers observed deficits in both verbal and nonverbal memory in PTSD participants compared with those without PTSD.

Over the past two decades, a growing interest in the neuroanatomy and neurochemistry of PTSD has developed. Advances in neuroimaging techniques have made it possible to study the primary brain structures believed to be affected by PTSD. Many researchers have reported decreases in hippocampal volume (Gilbertson et al., 2002; Villarreal et al., 2002) as well as reduced concentrations of the neuronal marker *N*-acetyl aspartate (Schuff et al., 2001) in PTSD patients. *N*-acetyl aspartate occurs in high concentrations in neurons but is virtually undetectable in other tissues (Birken & Oldendorf, 1989). It is accepted that *N*-acetyl aspartate levels reflect neuronal metabolism and that decreased hippocampal *N*-acetyl aspartate might be an indication of hippocampal damage in PTSD patients (Neylan et al., 2004; Schuff et al., 2001). In addition, researchers have observed that comorbid alcohol and substance abuse are associated with decreased hippocampal volume (Laakso et al., 2000) and lower performance on cognitive testing (Goldman, Brown, Christiansen, & Smith, 1991) and may, therefore, confound the results of studies of functional and structural domains in PTSD. Consequently, it is critical that researchers investigating associations between *N*-acetyl aspartate, hippocampal volume, and cognition carefully control for such comorbid conditions that may confound study results (Neylan et al., 2004).

Even so, researchers focusing on the treatment of PTSD have observed some positive results because of therapies employing the drugs paroxetine and phenytoin. Such therapies have resulted in increases in hippocampal volume and memory function (Bremner, 2006). Paroxetine is classified as a selective serotonin reuptake inhibitor, and evidence shows that it helps to promote the growth of neurons in the hippocampus as well as improve verbal and declarative memory in PTSD patients (Vermetten, Vythilingam, Southwick, Charney, & Bremner, 2003). Phenytoin is a commonly used anticonvulsant and is generally prescribed to control abnormal electrical brain activity. However, researchers have observed improvements in PTSD symptoms through the use of phenytoin and believe that these improvements are because of the observed increases in right hippocampal and right cerebral volume that accompany the use of phenytoin (Bremner et al., 2005).

Depression and Cognition

Depression is a prevalent medical condition, with nearly 20% of primary care patients presenting symptoms (Patten et al., 2005). There is also a growing awareness in the medical community that a close association exists with depression and a pattern of cognitive impairment. Marazziti, Consoli, Picchetti, Carlini, and Faravelli (2010) suggest that patients experiencing major depression present a variety of cognitive deficits such as deficits in attention, working memory, and executive function. The specific executive function difficulties include cognitive inhibition and problem and task planning. Baune and colleagues (2010) found impairments in all tested cognitive domains: attention, immediate and delayed memory, language, and visuospatial construction. Similarly, Andersson, Lovdahl, and Malt (2010) found that patients with recurrent brief depression were significantly impaired across all domains of cognition except for verbal learning and nonsemantic verbal fluency. Cognitive deficits such as these can contribute to lower social and occupational functioning and a diminished quality of life.

There is currently significant interest in creating a neurological cognitive profile of the anatomy and physiology of brain function in regards to depression. These

studies suggest that the limbic system, including the amygdale, hippocampus, and some of the anterior cingulated cortex may be involved with both emotional states and depression (Gotlieb & Hamilton, 2008; Ochsner & Gross, 2008). Eugene, Joormann, Cooney, Atlas, and Gotlib (2010) highlight the importance of assessing activation in the rostral anterior cingulated cortex, whereas Johnstone, van Reekum, Urry, Kalin, and Davidson (2007) found that depressed individuals were characterized by increased dorsolateral prefrontal cortex activation (cognition and negative symptoms) and decreased amygdale activation that appeared to be mediated by the ventromedial prefrontal cortex (affect and negative symptoms). Eventually, it is anticipated that a functional neuropathology of depressive disorders will be identified, with cognitive deficits shown to be an intrinsic expression of the brain changes that occur with depression.

A number of studies have addressed whether treatment may improve cognitive functioning. In fact, the possibility that cognitive deficits may linger after the depressive episode has been treated is an important medical consideration. A study by Nebes and colleagues (2003) administered patients and controls measures of working memory, speed of processing, episodic memory, and attention five times over the course of 12 weeks. Results revealed that cognitive dysfunction persisted in older depressed patients even after the administration of antidepressants improved their moods. In a sample of 19 adults with major depression disorder, Hammar and colleagues (2010) showed that patients with major depression disorder were significantly impaired when performing cognitive demanding tasks and showed no improvement on the tasks even after significant improvement in their depressive symptoms. Baune and colleagues (2010) also showed that impairments in cognitive processes such as executive control, attention, and memory persist after a depressed person has recovered. However, a review by Castaneda, Tuulio-Henriksson, Marttunen, Suvisaari, and Lönngvist (2008) concluded that, although deficits in certain aspects of executive control and attentional deficits were characteristic of depressed individuals, the evidence for learning and memory deficits is mixed. They suggest this might be because of the significant variability of the subtypes of depression (with deficits being most prominent in psychotic depression) and to the age of the participants (with older depressed adults displaying the most deficits). As patients with depression typically experience a mixture of psychological, behavioral, physical, and cognitive symptoms, management of symptoms in regards to cognition is difficult and complicated. More research is needed in this area to better treat and manage cognitive dysfunction in patients with depressive disorders.

Anxiety Disorders and Cognition

Epidemiological and clinical research clearly indicates that patients with anxiety disorders experience a significant reduction in their quality of life and psychosocial functioning (Bystritsky, 2006; Mendlowicz & Stein, 2000). Anxiety disorders are also characterized by cognitive deficits, with different types of anxiety disorders (e.g., phobias) associated with particular cognitive profiles (Engels et al., 2007; Nitschke & Heller, 2002). Anxiety disorders encompass a heterogeneous group, with each variant differing in etiology, outcome, and physiological characteristics. They are typically divided into panic disorder, phobias, generalized anxiety disorder, and obsessive compulsive disorders (Kessler, Chiu, Demler, Merikangas, & Walters, 2005; Nitschke, Heller, & Miller, 2000).

Ferreri, Lapp, and Peretti (2011) classify cognitive deficits within anxiety disorders into four different domains. The first domain consists of the executive functions, with attentional processes being the most severely affected. The second domain concerns memory, which includes autobiographical memory and deficits in working and episodic memory. The third domain concerns maladaptive cognitions, thoughts, and beliefs as is typically seen with phobias (e.g., an irrational and uncontrollable fear of dogs). Finally, a growing area of research in obsessive–compulsive disorder concerns thoughts and beliefs about one's own thoughts and beliefs or metacognitions; for example, hoarding behavior is often associated with an unrealistic belief that every hoarded item is useful, meaningful, and worthy of saving. All of these cognitive deficits may help maintain or aggravate anxiety disorders. Joorman and Gotlib (2008) found a correlation between rumination (i.e., stuck in a thought pattern) and the ability to control irrelevant negative material from working memory. These studies give empirical support for the hypothesis that rumination is related to the executive control component of working memory and that deficits in executive control and inhibition are related to and delay recovery from negative effect. In fact, McLaughlin and Nolen-Hoeksems (2011) propose that recent evidence actually implicates rumination in the development of such anxiety disorders.

In general, there are some wide-ranging cognitive profiles that are associated with all anxiety disorders, specifically cognitive biases toward threat and attentional biases. However, Nitschke and colleagues (2000) suggest that a more useful way to distinguish between types of anxiety is to limit the cognitive profiles to two—anxious apprehension and anxious arousal. Anxious apprehension is characterized primarily by worry and reliance on the left hemisphere processes. Anxious arousal, on the other hand, is characterized by immediate fear and panic symptoms and closely

aligned with the emotional surveillance system of the right hemisphere. This hypothesis was supported by Engels and colleagues (2007), who found that the two different types of cognitive profiles suggested by Nitschke and colleagues was supported by fMRI mapping of the specific brain areas. Specifically, the anxious apprehension group showed increased activity in the region of the left inferior frontal lobe associated with speech production, and the anxious arousal group showed increased activity in a region of the right hemisphere inferior temporal lobe that is associated with tracking and responding to information signaling danger.

Possible treatments for cognitive deficits in anxiety include drugs, cognitive therapy, meditation, mindfulness, electric shock therapy, and neurosurgery. Specific treatment targeting cognitive disorders in anxiety include the adaptation of an emotional regulation clinical perspective (Mennin, Heimberg, Turk, & Fresco, 2002) and the combination of selective serotonin reuptake inhibitors with cognitive behavioral treatment (Stein, 2002), with both of these approaches finding cognitive improvement. Unfortunately, one of three patients will be treatment resistance, rendering this a somewhat intractable psychiatric condition (Bystritsky, 2006). This treatment resistance may be because of multiple factors such as genetics, social factors, and comorbidities such as substance abuse (Bystritsky, 2006). Another important consideration is that a complex patient may require a multifactorial approach rather than a brief behavioral strategy, which is not always addressed during patient assessment. As anxiety disorders are the most prevalent psychiatric condition in the United States, understanding these cognitive profiles is important for improving current strategies and treatment modalities.

Implications for Nursing Practice and Research

As nurses and nurse scientists provide care to patients with neurological and psychiatric diseases, it is important for them to remain cognizant of how the primary symptoms of behavioral and emotional dysregulation can affect the secondary symptoms of cognitive functioning and vice versa. Likewise, it is important to consider how these symptoms can exert on their patients' everyday functioning such as remembering clinic appointments, adhering to medication schedules, and engaging in social and occupational environments. All of this can be interpreted within the context of cognitive reserve and neuroplasticity.

Cognitive Reserve and Neuroplasticity

Cognitive reserve is a well-supported concept that refers to the brain's ability to maintain a certain level of cognitive functioning in lieu of insults from physiological sequlae whether it be inflammation, excitotoxicity, or age-related insults (Vance & Wright, 2009). In general, the degree of cognitive reserve rests on (a) the viability of neurons; (b) the quantity, sophistication, and strength of connections between these neurons; and (c) the ability to repair and rewire neural connections so neural communication can continue unimpeded. The underlying principle of cognitive reserve is that (a) cognition emerges from such neural communication and (b) as long as neural communication flows efficiently, cognition will be maintained.

Cognitive reserve is either up-regulated or down-regulated through two related processes, respectively—positive neuroplasticity and negative neuroplasticity (Vance & Wright, 2009). Positive neuroplasticity refers to the strengthening of neural connections by adapting to novel and challenging stimulation (e.g., learning a foreign language); in so doing, this builds cognitive reserve. Likewise, negative neuroplasticity refers to the weakening of neural connections by lacking novel and challenging stimulation (e.g., not practicing the foreign language one has previously learned); in so doing, neural connections atrophy, reducing cognitive reserve. The interaction of cognitive reserve with positive and negative neuroplasticity has been documented in animal and human studies.

This interaction is clearly showed in the classic enriched environmental paradigm (Diamond, 1993). In the enriched environmental paradigm, rats from the same colony are randomly assigned to live in one of three environments—enriched, standard, and impoverished. In the enriched environment, rats are placed in a large cage with other rats and toys and objects to explore. In addition, these toys and objects are replaced periodically so the rats always are exposed to novel stimuli. In the standard environment, rats are placed three to a small cage but with no toys or objects to explore. In the impoverished environment, rats are placed in isolation, with no other rats with which to interact and no toys or objects to explore. After exposure to these environments for a period of time, rats in the enriched environment are observed to develop bigger brains, have more dendritic connections between neurons, and exhibit better maze test performance than rats in the other environments; likewise, rats in the standard environment exhibit similar outcomes compared with the rats in the impoverished environment. The enriched environment exemplifies positive neuroplasticity, whereas the impoverished environment exemplifies negative neuroplasticity.

Positive and negative neuroplasticity is also observed in humans. In the seminal study by Boyke, Drimeyer, Gaser, Büchel, and May (2008), 69 older adults (M_{age} = 60 years) were given instructions on how to learn to juggle using a standard three-ball

cascade. MRI scans were conducted at baseline (before they learned to juggle), approximately 3 months later (after they learned to juggle), and approximately 3 months later (after they ceased juggling practice). Of the 69 participants, only 25 achieved juggling competency as assessed by maintaining a three-ball cascade for at least 1 minute. From the MRIs of these 25 participants, from baseline to 3 months of juggling, there was a significant volume increase in the nucleus accumbens and hippocampus (the brain structures needed for memory); this signifies positive neuroplasticity. However, from this 3-month period when participants could juggle to 3 months later when participants no longer practiced juggling and lost this ability, there was a significant volume decrease in these same brain structures; this signifies negative neuroplasticity. In essence, this study highlights that cognitive reserve, as exhibited by the volume of brain structures, can be either increased or decreased by exposing people to either novel and challenging stimuli/activities or less novel and challenging stimuli/activities, respectively.

Given that such neurological and psychiatric diseases result in such distinct cognitive deficits, there is concern that these diseases may reduce cognitive reserve. As seen in Figure 2, how this plays out is proposed in the following dynamic model. Typically, the primary symptoms of neurological and psychiatric diseases is often conceptualized as being behavioral and emotional in nature (path A) and that such behavioral and emotional symptoms result in poor adaptation to environmental demands (path B). For example, if one experiences anxiety in social situations, it will be difficult to excel in the work environment where interacting with customers or engaging in meetings is required. Thus, treatments (path C) to improve the behavioral and emotional symptoms (i.e., counseling, antidepressants, and so forth) are prescribed to mitigate these primary symptoms so one can successfully negotiate his or her environment. Albeit, as reviewed, neurological and psychiatric diseases can result in secondary symptoms, specifically poor cognitive reserve (path D) and poor cognitive functioning (path E) that results in poor adaptation to environmental demands (path F). For example, if one's executive functioning and attention is compromised by his or her disease, this could impair one's ability to stay focused on conversation in a social environment. As such, poor fit with the environment may exacerbate behavioral and emotional symptoms and lead to isolation from the environment; just as in the enriched environmental paradigm, such social isolation can result in negative neuroplasticity. More importantly, cognition (path G) may be considered as either a way of controlling behavioral and emotional symptoms (that is when cognition is functioning well) or as a cause of such behavioral and emotional symptoms (that is when there are cognitive deficits and dysfunctions). For example, if executive deficits are presented, one may not be able to organize his or her thoughts well and may be more prone to perseveration and rumination; such cognitive deficits can result in negative mood and the formation of delusions. As such, treatments (path H) to bolster cognitive reserve and cognition may serve as a novel and innovative means to abate or mitigate such behavioral and emotional symptoms.

Cognitive remediation therapy may be one of many ways to improve cognitive reserve and cognition in adults with such neurological and psychiatric diseases and, in turn, may prove helpful in reducing behavioral and emotional symptoms. Cognitive remediation therapy is a series of specific exercises, administered via pencil or paper techniques, computer software, or videotapes, designed to improve either global cognition or particular domains such as memory, executive functioning, or speed of processing (Vance & Wright, 2009). In a subsample (n = 1,606) of the largest clinical trial (N = 2,802) of cognitive remediation therapy called ACTIVE (Advanced Cognitive Training for Independent and Vital Elderly), Wolinsky and colleagues (2009) found that normal, community-dwelling older adults administered 10 hours of visual speed of processing training over a 6-week period were 38% less likely to show signs of clinical depression 1 year later compared with those who did not receive this training. Likewise, in a pilot study (n = 32), Hudak and colleagues (2011) found that adults with Parkinson's disease who were administered 20 hours of visual speed of processing training over a 12-week period had significantly fewer depressive symptoms compared with those who did not receive this training. Such studies suggest that improving cognition, at least in visual speed of processing, may result in improved behavioral and emotional regulation.

As additional therapies are developed to address the behavioral and emotional symptoms of such neurological and psychiatric diseases, nurse scientists may also consider ways of improving cognition in their patients to investigate whether this may also mitigate the manifestations of behavioral and emotional symptoms. By bolstering cognitive abilities, this may help patients to better negotiate their environment, thereby reducing the stress created by a poor fit between themselves and their environmental demands. Again, this is reflected in the model by the double-headed arrows exemplifying that the environment also affects behavioral and emotional symptoms as well as behavioral and emotional symptoms impact how well one negotiates his or her environment.

Some common lifestyle strategies to bolster cognitive function (and promote better mood) that may also be considered are intellectual exercise, physical exercise,

proper nutrition, good sleep hygiene, and avoidance of substances such as drugs and alcohol. The avoidance of substances is particularly important given that self-medication with alcohol and drugs is used by many to cope with their behavioral and emotional symptoms; unfortunately, excessive alcohol and drug use is associated with poorer cognitive functioning (Vance, Roberson, McGuiness, & Fazeli, 2010), which may compromise one's ability to cope with his or her environment (path F) and behavioral and emotional symptoms (path G). Generally, it is considered that what is good for the body is likewise good for the brain (Colcombe & Kramer, 2003; Férat et al., 2009). Furthermore, Vance and colleagues (2010) developed an individualized cognitive prescription that focuses on behavioral goals of exercise, nutrition, sleep, intellectual stimulation, and mood support designed to promote successful cognitive health in older adults; such a tool could be safely used in patients with neurological and psychiatric diseases to improve their cognitive health as well. On the basis of the model in Figure 2, by incorporating activities that support optimal cognitive functioning, this may in turn increase cognitive reserve and cognitive functioning, reducing cognitive deficits, thus allowing one to better control behavioral and emotional symptoms to negotiate one's environment.

Conclusion

Clearly, cognitive functioning is necessary to negotiate social and environmental demands to function in life. For someone with either a neurological or psychiatric disease, such cognitive resources may be compromised, thus compromising their ability to fully engage in life. Fortunately, medications to treat some of these comorbidities can result in emotional equilibrium and stabilization or primary symptom mitigation; likewise, the secondary symptoms of cognitive deficits may also be abated. Unfortunately, this is not always the case, and despite control of the primary symptoms, such secondary symptoms of cognitive deficits may persist as seen with executive functioning in schizophrenia. For those with such persistent cognitive problems, there remain concerns that as one ages with their particular disease, age-related neurological and psychiatric insults may exacerbate such cognitive deficits, which may make primary symptom management more difficult. But with a host of cognitive strategies to improve cognition (i.e., cognitive remediation therapy, exercise) and mitigate cognitive losses (i.e., spaced-retrieval method; method of loci, external mnemonics) found in the gerontological literature (Vance et al., 2008), such strategies may be effective in improving primary and secondary symptoms in these different clinical populations; however, further evidence-based research is needed to establish whether such strategies are clinically feasible and effective.

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