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Counselors' Neuroscience Conceptualizations of Depression

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The authors conducted the first-ever study into counselor conceptualization of client problems using neuroscience theories. The authors selected an embedded mixed-methods design. Participants (N = 334) provided quantitative demographic information and responded to an open-ended qualitative question regarding a hypothetical situation of a client asking the counselor to explain depression from a neuroscience perspective. The authors coded, tallied, and transformed qualitative responses to quantitative data via frequency counts. Kappa coefficients for the coding team exceeded the threshold for acceptable reliability. Approximately half of the counselors applied neuroscience theories to explain client experiences of depression (57.7%, n = 194), and some counselors integrated multiple neuroscience theories in their response (23.2%, n = 45). The monoamine and neuroplasticity theories were the two most common neuroscience theories for depression. Implications for research and training are discussed.

Clinical mental health counselors have the ethical responsibility to keep up-to-date with newly emerging scientific findings and apply such information in clinical practice with clients (American Counseling Association [ACA], 2014; American Mental Health Counselors Association [AMHCA], 2015). During the past 5 years, the counseling field has increasingly sought to integrate neuroscience into clinical practice (Beeson & Field, 2017). Reflective of this trend, counselors are beginning to use forms of neuroeducation (Miller, 2016) to help clients better understand clinical problems from a neuroscience perspective. As counselors strive to incorporate neuroeducation in their prac-

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tice, studies are needed to explore how counselors conceptualize and then respond to client requests for an explanation of their symptoms. No studies currently exist into counselor conceptualizations of client problems from a neuroscience perspective, nor into the use of neuroeducation among practicing counselors. Furthermore, the authors could find no existing studies at the time of writing that examined other allied mental health professionals' neuroscience conceptualizations of client problems (e.g., psychologists, social workers).

NEUROSCIENCE CONCEPTUALIZATIONS OF DEPRESSION

Major depressive disorder (MDD) is one of the most common diagnoses treated by U.S. mental health professionals, with 10.6 million U.S. adults seeking treatment of the 16.2 million diagnosed (Substance Use and Mental Health Services Administration, 2017). MDD is difficult to diagnose, as no objective biomarker has yet been detected (Jentsch et al., 2015). In this vacuum, five neuroscience theories have emerged for conceptualizing MDD etiology.

The Monoamine Theory

Early understandings about the neurological underpinnings of mental disorders, including depression, were largely derived from drug studies (Lacasse & Leo, 2005; Sanacora, Treccani, & Popoli, 2012). The monoamine theory of depression emerged from drug research during the 1950s. Imipramine was known to increase serotonin neurotransmission via reuptake inhibition, and subjects taking the drug reported reduced depression symptoms (Schildkraut, 1965). This led to the hypothesis that reduced transmission of serotonin was a possible mechanism underlying the neurobiology of depression and could be treated through agents that enhanced serotonin transmission (Mulinari, 2012). During the ensuing 50 years, the hypothesis was revised to include other monoamine deficits as the cause of MDD, such as dopamine and norepinephrine (Liu, Liu, Wang, Zhang, & Li, 2017). Subsequent MDD medications that upregulated dopamine, norepinephrine, serotonin, or a combination of these were introduced to the market (Sanacora et al., 2012). Monoamines have multiple roles and functions. For example, serotonin is associated with sleep, appetite, and temperature modulation, in addition to mood regulation (Sanacora et al., 2012). The monoamine theory for depression became widely accepted and until recently was largely uncontested (Mulinari, 2012).

The Neuroplasticity Theory

During the past decade, critics have claimed that the monoamine hypothesis is insufficient to explain the complexity of depression, citing that healthy controls do not seem to develop depressive symptoms when serotonin is lowered experimentally, serotonergic medications do not help all clients with depression, and medications unrelated to serotonin transmission appear to have antidepressant effects (Cowen & Browning, 2015; Lacasse & Leo, 2005; Liu et al., 2017; Racagni & Popoli, 2008). During the same time period, a new hypothesis was proposed to explain the neural basis of depression. The theory, called the neuroplasticity hypothesis, emerged from epigenetic research into the role of environmental stress and transcription of genetic material in the development of depression (Pittenger & Duman, 2008; Racagni & Popoli, 2008; Sanacora, Zarate, Krystal, & Manji, 2008). Authors have used other titles for this theory, such as the diathesis–stress model of depression (e.g., Colodro-Conde et al., 2017) and the neurodevelopmental theory of depression (e.g., Gałecki & Talarowska, 2018). In brief, epigenetics postulates that environmental events alter which parts of a person's DNA are expressed and not expressed through complex processes known as methylation and transcription (Pittenger & Duman, 2008).

Chronic stress states have been a particular focus of epigenetic research, as persisting stress has been associated with neural death (necrosis) and reductions in birth of new neurons (neurogenesis) via down-regulation of brain-derived neurotropic factor (BDNF; Moser et al., 2015). Chronic stress in early childhood has been associated with impaired functioning of the hypothalamic–pituitary–adrenal (HPA) axis (Romens, McDonald, Svaren, & Pollack, 2015). A well-known study by Meaney and Szyf (2005) found that rat pups born to attentive mothers but raised by inattentive mothers developed symptoms of depression and anxiety, whereas rat pups born to inattentive mothers but raised by attentive mothers did not demonstrate symptoms of depression and anxiety. Rats raised by inattentive mothers had greater methylation of the NR3C1 gene. This gene codes for the glucocorticoid receptor, associated with the modulation of the HPA axis. The HPA axis is responsible for the release of hormones such as cortisol via the sympathetic branch of the autonomic nervous system. Such findings have been replicated in children exposed to early life trauma (Romens et al., 2015).

Dysregulation of the HPA axis has been associated with depressive disorders, as well as reductions of hippocampal volume (Anacker et al., 2011; Palma-Gudiel, Cordova-Palomera, Leza, & Fañanás, 2015; Romens et al., 2015). The dysregulation of the stress response system (i.e., HPA axis) through epigenetic modifications described above is theorized to cause chronic inflam-

mation responses within the enteric nervous system, such as the gastrointestinal tract (Hall, Jones, Tyson, Woods, & Keltz, 2016; Raison, Capuron, & Miller, 2006). Selective serotonin reuptake inhibitor (SSRI) medications have been found to combat reductions in hippocampal volume through enhanced production of BDNF (which stimulates neurogenesis) and also appear to reduce inflammation associated with depression (Raison et al., 2006; Stockmeier et al., 2004). SSRIs thus have a role in both monoamine and neuroplasticity theories of depression.

The Glutamate Theory

After the monoamine theory was developed, two amino acids were identified as the major excitatory and inhibitory neurotransmitters in the nervous system (Shabel, Proulx, Piriz, & Malinow, 2014). These two amino acids, gamma-aminobutyric acid (GABA) and glutamate, have different effects on neurotransmission. GABA mediates inhibitory transmission, whereas glutamate mediates excitatory transmission (Shabel et al., 2014). Excitatory neurotransmitters increase the likelihood that the postsynaptic neuron will itself experience an action potential, and inhibitory neurotransmitters reduce the probability of the postsynaptic neuron experiencing an action potential. Whereas monoamines regulate neurotransmission, excitatory neurotransmitters such as glutamate mediate the *frequency* of neurotransmission (Sanacora et al., 2012). Glutamate is involved in one neuron's signaling to another neuron, which results in the opening of ion channels leading to the release of neurotransmitters into the synaptic cleft (Shabel et al., 2014). Glutamate is therefore very important to neurotransmission, and increasing glutamate can result in synaptogenesis (the creation of new synaptic connections) and long-term potentiation (Holtmaat & Svoboda, 2009).

It has been long known that inescapable stress is associated with inhibition of the glutamate receptor, especially its NMDA subtypes (e.g., Trullas & Skolnick, 1990). Inescapable stress is defined as a stressful situation that a person cannot escape or avoid. Martin Seligman's (1972) research into the learned helplessness of shocked dogs is a well-known example of inescapable stress. The inhibition of glutamate during inescapable stress has implications for a person's functioning. A decrease in glutamate can lead to reduced receptor signaling and fewer synaptic connections. This downregulation may contribute to long-term depression (LTD) over time (Liu et al., 2017). Some studies have found associated volumetric reductions in glial cells in the same brain regions, suggesting that glutamate and glial cells may both be implicated in depression (Bernard et al., 2011; Sequeira et al., 2009). Because glutamate influences LTD, this theory has been referred to as the *metaplasticity hypothe-*

sis, as changes in glutamate and NMDA can shape future synaptic change and plasticity (Hulme, Jones, & Abraham, 2013).

Research using multiple assessment methods (plasma samples, magnetic resonance imaging, postmortem studies) has found that MDD is associated with the dysregulation of glutamatergic receptor signaling, particularly in the frontal cortex, cingulate, and limbic regions (and especially the hippocampus; Koolschijn et al., 2009; Lorenzetti, Allen, Fornito, & Yücel, 2009). Glutamate dysregulation varies by mood disorder type, age, and brain region. For example, higher levels of glutamate in the frontal cortex were found in persons with late-life depression, post-stroke depressive disorders, and bipolar disorders (Binesh, Kumar, Hwang, Mintz, & Thomas, 2004; Port, Unal, Mrazek, & Marcus, 2008; Wang et al., 2012).

The emergence of the glutamate hypothesis for depression has received substantial attention within the past decade because of ketamine trials for MDD (e.g., Li et al., 2011; Zarate et al., 2006). In 2019, the U.S. Food and Drug Administration approved a variant of ketamine called esketamine for the treatment of MDD. Glutamate works through several receptors, including N-methyl-D-aspartate (NMDA-R), α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA-R), and kainite, alongside the metabotropic receptors. Ketamine is an NMDA-R antagonist and increases the neurotransmission of glutamate. It is believed that NMDA-R antagonists enhance glutamate because they increase AMPA-R, which in turn increases production of BDNF (Autry et al., 2011).

Structural and Functional Abnormalities

MDD has been associated with abnormalities in the structure and function of various cortical and subcortical structures such as the amygdala, anterior cingulate, hippocampus, and prefrontal cortex (e.g., orbitofrontal cortex; Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Rolls, 2016), alongside networks such as the default mode network (Hamilton, Farmer, Fogelman, & Gotlib, 2015). Structural and functional damage to the brain caused by head injury and stroke has also been linked to the development of depressive symptoms (Cnossen et al., 2017). Hudak et al. (2011) found head trauma to cause atrophy in the same brain regions as "spontaneous depression" episodes (p. 160), referring to MDD episodes not precipitated by head trauma. Fortunately, these volumetric changes are often corrected through neurogenesis of neurons and glial cells (Medaglia, 2017). These abnormalities do not appear to have a direct connection to epigenetic changes caused by an overactive stress-response system, and their hypothesized association with depressive symptoms means

that structural and functional abnormalities must be considered a separate neuroscience theory for depression.

Medical Conditions

Hormonal alterations and vitamin deficiencies have been associated with MDD, such as hypothyroidism (Dayan & Panicker, 2013) and vitamin D deficiency (Parker, Brotchie, & Graham, 2017). The downregulation of thyroid-stimulating hormone (TSH) has been associated with enhanced release of cortisol via the HPA axis (Fugger et al., 2018). As described earlier, chronically high levels of cortisol are associated with LTD and reduced gray matter, particularly in the hippocampus, which has been associated with MDD. Hypothesized linkages to depressive symptoms, such as the TSH-cortisol-LTD pathway, make medical conditions a legitimate fifth neuroscience-informed theory for depression.

PURPOSE OF THE STUDY

Emerging neuroscience theories for depression have increased the complexity of how depressive disorders are conceptualized. A need exists for authors to explore how counselors are using these theories to conceptualize and treat depressive disorders. The extent to which mental health counselors are applying these theories in their work with clients remains unknown. No current studies into counselor utilization of neuroscience conceptualizations for client problems exist, and none exist specifically for the utilization of these conceptualizations for treatment of depression. The purpose of the study therefore was to explore how counselors are conceptualizing client depression from a neuroscience perspective and to gain insight into how counselors are communicating this information to clients. The research question guiding this study was: How are counselors currently conceptualizing client depression from a neuroscience perspective?

METHODS

To address the research question, the authors selected an embedded mixed-methods design (Creswell & Plano-Clark, 2017). The authors chose a mixed methodology because of the need to categorize, tally, and transform qualitative data into quantitative data. The authors were guided by post-positivist philosophy (Creswell & Creswell, 2017) and sought to recruit a large sample, compute interrater reliability for coding, and understand the relative frequency of response themes through statistical analysis (Creswell & Creswell,

2017). In regard to timing and mixing (Creswell & Plano-Clark, 2017), the authors used an online survey tool to collect both quantitative demographic information data and qualitative data through an open-ended question. They sought to categorize, tally, and transform the qualitative data into quantitative data via frequency counts. The data were thus merged for quantitative analysis (Creswell & Plano-Clark, 2017).

Greater weight was attributed to the quantitative data in the study. While qualitative data were collected, the authors did not approach the data from a constructivist position (i.e., that qualitative responses should be given equal weighting regardless of frequency because all reality is co-constructed; Lincoln & Guba, 2013).

Participants

For inclusion in the study, the authors required participants to possess an identity as a counselor, evaluated through either (1) current licensure as a counselor, (2) board certification through the National Board of Certified Counselors, (3) current membership in a counseling organization, or (4) current status as a faculty member or student in a counselor education program. All respondents met at least one of these criteria. Because the integration of neuroscience into counseling practice affects counselors at different developmental stages, the authors included counselors-in-training within the sample. The authors recruited participants in the study through several channels: (1) invitations on listservs and discussion boards of the neuroscience interest networks of the ACA, AMHCA, and Association for Counselor Education and Supervision (ACES); (2) invitation e-mails on counseling listservs, such as CESNET-L; (3) e-mails to program directors of counselor education programs, with requests for the program directors to share the recruitment e-mail with their faculty and students. Because of the convenience and snowball sampling recruitment approaches, the authors were unable to estimate the response rate for the survey.

All participants electronically consented to participate after reading an informed consent statement that was approved by the first author's university institutional review board (IRB). Participants could skip questions and could decline participation at any time. Participants who completed the survey had the option of being entered into a drawing for two signed textbooks on neuroscience in counseling. The final page of the survey contained a link for participants to enter their e-mail address in a separate online collector to ensure participant anonymity.

The overall sample size was $N = 336$. All but nine participants (97.3%, $n = 327$) reported their self-identified gender and ethnicity. Of those who

self-reported, the majority of respondents identified as female (75.6%, n = 254), followed by male (19.9%, n = 67) and nonbinary (0.6%, n = 2). In regard to race/ethnicity, respondents primarily identified as White (77.7%, n = 261), followed by Asian American (6.3%, n = 21), multiracial (6.3%, n = 21), African American/Black (3.6%, n = 12), Hispanic or Latinx (2.7%, n = 9), Arab American or Middle Eastern (0.6%, n = 2), and American Indian or Alaska Native (0.3%, n = 1). The average age of respondents was 42.8 years (SD = 13.6).

Of those participants who reported education level (92.3%, n = 310), 25.9% (n = 87) were current master's-level students, 34.2% (n = 115) possessed a master's degree, 11.6% (n = 39) identified as doctoral students, and 20.5% (n = 69) possessed a doctoral degree. The majority of respondents had attended a Council for Accreditation of Counseling and Related Educational Programs (CACREP)-accredited counseling program for their master's and/or doctoral degrees (72.9%, n = 245). A few participants (3.1%, n = 10) possessed a doctoral degree from another discipline (e.g., psychology) and had not attended a CACREP-accredited program at the master's or doctoral level. In regard to neuroscience preparation, a large number of respondents reported having neuroscience coursework in their graduate-level counseling degree program (39.3%, n = 132). Fewer participants had completed advanced training, with only seven participants (2.1%) possessing previous experience in a neuroscience research lab and only seven participants (2.1%) currently Board Certified in Neurofeedback.

The sample had a diverse amount of experience, ranging from 0 to 40 years with an average of 9.3 years (SD = 10.6). Half of respondents were independently licensed as mental health professionals (53.0%, n = 178). The majority of respondents were members of state or national counseling organizations (81.0%, n = 272). Fewer participants were members of the ACA, ACES, or AMHCA Neuroscience Interest Networks (24.4%, n = 82).

Procedure

The authors used an online survey platform to collect quantitative and qualitative data through a series of demographic questions and a single open-ended question about neuroscience conceptualizations of depression. Informed by Patton's (2014) guidelines for developing open-ended qualitative questions, the authors created and tested a question that would directly ask about how the participant would hypothetically conceptualize a client's depression from a neuroscience perspective. The authors chose an open-ended question to prevent leading participants to possible conceptualizations and to elicit more accurate data regarding participants' understanding of neurosci-

entific conceptual models for depression. The question read thus: "The next question pertains to your understanding of depression from a brain and body perspective. Imagine that a client asks you: 'What is going on in my brain, that I feel so depressed?' What answer might you provide them in response?" The authors included the first sentence to inform participants that a different type of question was about to be asked, following the demographic section. The authors piloted the survey questions via a test administration with faculty, graduate students, and practitioners. The survey was sent to the CESNET-L listserv and received 77 responses. The authors modified several demographic questions for clarity and deleted demographic questions that were extraneous to the research question. No modifications were made to the open-ended survey question about neuroscience conceptualizations of depression.

The authors used Survey Monkey (2019) to collect online responses. The authors selected this collector because Survey Monkey automatically provides secure encryption for each participant survey response. The authors noted in the informed consent document that response data would be stored on an encrypted online collector to protect participant privacy.

Following IRB approval, the authors sent recruitment invitations to the three channels listed above. Recruitment invitations included a link to the online survey. The authors sent two additional reminders before data collection concluded. The online survey portal was open for 6 weeks. After the portal closed, the first author randomly selected two participants for the book drawing who had submitted their e-mail address in the separate online collector.

After data collection ended, the first author downloaded the raw data in Excel for data coding and analysis using SPSS 24.0. A coding team made up of the five authors met regularly for several months to code the qualitative responses to the open-ended question regarding neuroscience conceptualizations of depression. Each of the five authors had completed doctoral-level training in qualitative coding and analysis. To address the first research question regarding how counselors currently conceptualize depression from a neuroscience perspective, the coding team developed a codebook that contained both a priori and emergent codes (Merriam & Tisdell, 2017). Codes were categorized using the constant comparative method from grounded theory qualitative research (Corbin & Strauss, 2014), as is typical for mixed-methods studies (Creswell & Plano-Clark, 2017). The a priori codes consisted of the five neuroscience conceptualizations for depression that were described earlier in this article.

During coding, the research team also identified emergent codes. Three of these were categorized as neuroscience conceptualizations for depression: polyvagal theory, brainwave dysregulation, and interpersonal neurobiology.

While both polyvagal and brainwave dysregulation theories have less empirical support, both were also fairly well-established neuroscience theories for depression (e.g., Fernandez et al., 2017; Hampson, 2017), and thus the research team decided to include them in the neuroscience-conceptualizations category. For example, studies have found that nonverbal behaviors have been associated with increased vagal activity in MDD, consistent with polyvagal theory (Fernandez et al., 2017). Neurofeedback has developed and empirically tested protocols for the treatment of depression and is in the process of investigating potential mechanisms of action for treatment efficacy (Hampson, 2017). Interpersonal neurobiology, while less directly related to depression, has a clear neurobiological basis for the theory (Miller & Barrio-Minton, 2016) and was therefore included. Other emergent codes were identified that did not have a clear neuroscience basis, such as cognitive-behavioral conceptualizations for depression. These emergent codes were not categorized as neuroscience conceptualizations. Once the research team had coded data and tallied the frequency of codes, descriptive statistics were performed to determine the frequency of participants who provided a response that fell within the category of neuroscience conceptualizations.

Validity

The authors sought to follow several recommendations by Merriam and Tisdell (2017) to enhance credibility, trustworthiness, and transferability when coding qualitative data. First, the authors ensured that coding team members possessed both etic and emic perspectives. The first, second, and third authors were very familiar with neuroscience theories of depression (emic positioning), whereas the fourth and fifth authors had little exposure prior to the research project (etic positioning). All authors were current faculty members in counselor education programs from different universities and different regions of the United States. Authors represented different gender and racial/ethnic backgrounds and were also at different stages of expertise regarding neuroscience as a whole. This diversity among the coding team helped reduce the potential for bias in the coding of responses. None of the authors had direct contact with participants, whose identity was anonymous. During coding, the team was blinded from participant demographic information.

Second, the coding team created a common codebook to ensure consistency in coding. The authors conducted two rounds of coding for the entire data set to improve consistency. After the first round of coding, the team met to discuss divergent codes and refine the coding protocol to enhance consistency. The coding team then coded the data set for the second time and computed interrater reliability for second-round coding. Any remaining discrepancies in

coding were resolved via consensus. Following the second round of coding, the first author inserted codes into SPSS to reduce errors in manual tallying when seeking to understand the frequency of coded responses.

Third, the authors recorded memos during team meetings when coding and reviewing data, and the team kept an audit trail of the process (Merriam & Tisdell, 2017). The authors were unable to conduct member checks because participant data were collected anonymously.

Inter-Coder Consistency

The authors evaluated inter-coder consistency by computing kappa correlation coefficients for multiple raters (McHugh, 2012) using the arithmetic mean of kappa coefficients to compute the corrected coefficient (Light, 1971). Prior to conducting the kappa coefficient, the authors ensured that all prerequisites were met, which included pairing coders with the same observation, and ensuring each team member coded data independent of other members. The authors followed Hallgren's (2012) guidelines of 67% agreement and Cohen's kappa of .67 as the cutoff for acceptable consistency.

RESULTS

The coding team's kappa coefficient for coded responses to the open-ended question was very high for second-round coding ($\kappa = .97$). More than half of the sample (57.7%, $n = 194$) provided a neuroscience conceptualization of depression, such as the monoamine or neuroplasticity hypothesis. The remainder of participants either reported that the reasons for depression were too individualized to comment (12.2%, $n = 41$), felt unqualified to answer the question and needed to research neuroscience conceptualizations of depression (6.5%, $n = 22$), or provided alternative conceptualizations that were not clearly connected to a neuroscience hypothesis for depression. Some of the non-neuroscience conceptualizations were fairly common, such as the cognitive-behavioral theory of depression (8.9%, $n = 30$).

Of participants who provided a neuroscience conceptualization ($n = 194$), the monoamine (65.5%, $n = 127$) theory was the most common. Of participants who provided a neuroscience conceptualization for depression ($n = 194$), a minority mentioned medication referral in their response (12.8%, $n = 43$), and few responses proposed only medication referral without consideration for counseling (2.3%, $n = 8$). Importantly, the monoamine hypothesis was cited in conjunction with medication referral in two-thirds of medication responses (67.4%, $n = 29$). In contrast, only 22.8% of monoamine responses included a medication referral suggestion. This suggests that while participants do not

necessarily associate the monoamine hypothesis with medication referral, those who did make medication referrals did so in part because of the monoamine hypothesis. This was apparent in the following sample response: "I might talk about how neurotransmitters and uptake work. We might also have a discussion about sleep habits. If they were already on antidepressants, then the conversation would relate directly to how that particular prescription functions and whether this is working or not for that client."

The neuroplasticity hypothesis (43.8%, $n = 85$) was the second most common. Responses in this category tended to be more complex and often included considerations for different factors that may be causing depression. The following response was representative in this regard: "The brain is an incredibly complex organ. It is shaped by all kinds of experiences as well as our individual predispositions we inherit from our parents. So it's hard to say right now exactly what all has contributed to your depression. It could be traumas, environmental factors, and biology."

The other neuroscience theories for depression were far less common and included polyvagal theory (9.8%, $n = 19$), brainwave dysregulation (9.8%, $n = 19$), structural and functional abnormalities (6.7%, $n = 13$), medical conditions (5.7%, $n = 11$), and interpersonal neurobiology (0.1%, $n = 2$). No participants cited the glutamate theory. In contrast to the other categories, the polyvagal and interpersonal neurobiology responses often sought to describe impacts of depression rather than causes of depression (e.g., "I would use the hand model to explain the various areas that are affected by depression") and thus appeared to be among the weaker explanations. The total number of individual neuroscience theories exceeded the total number of responses with neuroscience conceptualizations ($n = 194$) when combined, as respondents sometimes gave more than one neuroscience theory in their response.

Less than one quarter of participants who provided a neuroscience conceptualization of depression ($n = 194$) gave more than one conceptual model in their response (23.2%, $n = 45$). The complexity of participant neuroscience conceptualizations of depression was therefore low.

DISCUSSION

The purpose of the study was to understand how counselors currently conceptualize depression from a neuroscience perspective. The research team's inter-coder consistency exceeded the criteria for adequate reliability for the second-round coding of both participant responses and for the complexity of responses, lending some degree of confidence in the reliability of the coded data. Only half of participants provided a neuroscience conceptualization for

depression, and it was unclear why this occurred, considering the question prompt. Even when accounting for the subset of participants who refused to provide any conceptualization because of beliefs that each client presents with different issues and needs individualized care, more than one third of participants did not provide a neuroscience conceptualization in response to the prompt. In addition, the complexity of participant responses was fairly low. Of participants who provided a neuroscience conceptualization for depression, only 23.2% identified more than one neuroscience theory. This is of some concern, as mental health professionals are increasingly encouraged to assess and conceptualize client problems from multiple perspectives as evidenced by research domain criteria (Insel et al., 2010) of the National Institute of Mental Health, which encourages a multidimensional approach to understanding client symptomology.

Common Neuroscience Conceptualizations for Depression

The monoamine and neuroplasticity theories were the most common neuroscience conceptualizations used by participants. The research team was not surprised by monoamine's popularity, as it is a fairly well-established theory. The research team was encouraged to see the frequent application of the neuroplasticity theory during case conceptualization, as it represents newer thought regarding the neuroscience of depression. However, the newest theory for depression within the neuroscience field, the glutamate theory, was not cited by any participants, likely suggesting a lack of familiarity. Furthermore, relatively few participants who provided a neuroscience conceptualization of depression cited structural and functional abnormalities or medical conditions (6.7% and 5.7%, respectively). The authors noted that responses across categories contained inaccurate information, suggesting that further training is needed.

Model Response to Client Inquiries About the Neuroscience of Depression

Following the coding and analysis process, the research team developed an exemplary response to the question posed to participants. The research team believed that the counselor's best response would first validate the client's question and demonstrate active listening to understand their experience before providing education about the brain. The client might feel discouraged or demoralized, based on the question prompt (i.e., "What is going on in my brain?"), and this should be addressed by the counselor. In short, counselors should still use humanistic principles and attend to the therapeutic relationship when providing neuroeducation. Counselors should explore the client's

lived experience with their symptoms rather than explain them prematurely. After validating the client's question, the counselor should seek to clarify the question being asked and seek to better understand the problem. For example, the type of question being asked and/or the type of depression the client is experiencing might guide the counselor to provide different neuroeducation based on the question and problem. The client's degree of familiarity with neuroscience may also inform how neuroeducation is provided.

When providing neuroeducation, the counselor should directly respond to the question asked by the client and ground neuroeducation in the symptoms that most affect daily experience, as assessed earlier. The counselor must therefore be collaborative and flexible when providing information to the client. Ideally, neuroeducation should attempt to integrate the different models of depression that exist (e.g., monoamine, neuroplastic, glutamine). Neuroeducation should also be based on empirical findings rather than conceptualizations that lack supporting data (e.g., polyvagal theory). Counselors should therefore attempt to stay current with emerging research that is evolving in regard to the neuroscience of mental disorders.

While treatment options are important to eventually discuss with the client, the counselor should postpone presenting options until they have received more information from the client first. In short, a client's question about why they feel depressed should not elicit the counselor's immediate description of treatment options, as this feels like "fix-it" problem-solving, which can be invaliding. Last, counselors should avoid justifying medication referrals through the misuse of the monoamine hypothesis.

Limitations

The authors identified several study imitations. The question prompt was of a hypothetical client scenario, and participant responses may have differed from how they would respond to the same question by an actual client in a counseling session. The written nature of the survey may also have biased responses, as counselors may have responded differently to verbal rather than written communication. Furthermore, the phrasing of the prompt may have biased counselors. For example, the prompt did not ask for multiple neuroscience conceptualizations, which may have biased the responses. If more direction were provided, more counselors might have provided multiple neuroscience conceptualizations. Because of convenience sampling, it was impossible to know the response rate for the survey and also whether responses reflected self-selectivity bias (e.g., whether the survey was completed by counselors who were already familiar and comfortable with neuroscience). The

sample was overrepresented by White female respondents, and responses may not be representative of all counselors.

Implications for Practice and Further Research

The results of the first-ever study into the frequency of counselors' neuroscience conceptualizations for client problems indicate that many counselors are able to apply neuroscience theories to help explain client experiences. Regarding depression specifically, it appears that the neuroplasticity hypothesis is gaining popularity among counselors, though the glutamate hypothesis remains largely unknown. Trainings are needed to help counselors develop familiarity with neuroscience models of depression, as a sizable number of participants felt unqualified or unprepared to respond, responses across categories included inaccurate information, and a minority of counselors provided more than one conceptualization in their response. Counselors should develop a nuanced understanding of how these theories might be applicable in some clinical presentations than others, in order to individualize their approach.

While insufficient evidence exists for the impact of counseling on epigenetic modifications such as upregulation of BDNF, substantial evidence has linked counseling to the reduction of cortisol (Fischer & Cleare, 2017). Counseling may therefore impact the stress-response system. In addition to counseling, the counselor should consider the treatment options available, such as lifestyle changes (exercise, sleep) alongside medication evaluation referral. In addition to antidepressant medications (Musazzi et al., 2010), regular physical exercise has been found to stimulate BDNF and glutamate production, resulting in increased gray matter volume in the frontal lobe and hippocampus (Erickson et al., 2011) and reduced gray matter damage (Chaddock-Heyman et al., 2014). Sleep has also been found to have a role in hippocampal volume (Novati, Hulshof, Koolhaas, Lucassen, & Meerlo, 2011). Physical exercise and adequate sleep are known to upregulate serotonin (Young, 2007). Assessing a client's sleep quality and physical activity level and assisting them to make lifestyle changes that enhance aerobic physical activity and enhance sleep quality appear to be an empirically supported alternative to medication (Al-Qahtani, Shaikh, & Shaikh, 2018; Young, 2007).

Future research could evaluate the accuracy of the five neuroscience conceptual models for depression, as no published studies have compared the accuracy of these theories. Future research could also explore underlying factors that influenced response themes. For example, it is unknown why counselors chose certain conceptual models for depression over others. It is also possible that participants were aware of more than one neuroscience theory for depression, but other factors influenced their response, such as not aligning

with those theories, not investing enough time in their response to permit more complex written conceptualizations, feeling unable to articulate different neuroscience conceptualizations, or not remembering those theories when taking the survey. The "think-aloud" cognitive interviewing method (Willis, 2005) might be helpful to understand underlying intentions of participants when conceptualizing depression. Future research could also explore responses to a real-life client rather than a fictional case.

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