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Project for a Scientific Psychiatry: A Neurobiologically Informed, Phasic, Brain-Based ..Model of Integrated Psychotherapy

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Inthis paper we present a neurobiologically informed model ofpsychological "stability and growth"' to address a range of psychopathology. We provide a neuroscientific rationale for a proposed hierarchy of treatment that elucidates the "when, why, and what" of psychotherapeutic interventions based on the modulation, integration, and homeostatic rebalancing of affected subcortical and neural networks. We assert this hierarchical model of care has significant implications for understanding the pathogenesis of mental disorders. Furthermore, we suggest the model may inform prevention efforts that could potentially reduce the prevalence or burden of mental illness and has substantial implications for both psychiatric education and research.

Keywords: integrated psychotherapy, hierarchical model, neurobiology

There exist a range of effective and evidence-based treatment modalities for mental health disorders. Although outcome research suggests roughly equivalent effectiveness (Leichsenring & Leibing, 2003; Luborsky et al., 2002; Wampold et al., 1997), researchers often fail to examine specific components/techniques (e.g., proposed mechanisms of change) to determine which component best accounts for specific symptom improvement. Identifying these crucial relationships requires conceptualizing a model of psychopathology and therapeutic change thqt addresses both common and disorder speciffc features and change mechanisms.

Identifying shared (and unique) dysfunctional neurocircuitry may help elucidate these mechanisms. Since many psychiatric disorders are characterized by problems with emotion generation and/or regulation,. neural networks involved in these processes are likely a common link (Gross & Jazaieri, 2014). Dysfunctional prefrontal cortical and frontolimbic circuitry (decreased amygdala-prefrontal connectivity) predict developmental trajectories of emotional and behavioral dysregulation across disorders in youth (Bertocci et al., 2014). Functional imaging studies indicate that abnormal limbic and ventral-lateral prefrontal cortex activation combined with dysfunctional frontolimbic circuitry underlie the deficient emotion generation and regulation capacities in individuals with borderline personality disorder, depression, anxiety, schizophrenia, and posttraumatic stress disorder (Etkin & Wager, 2007; Hamilton et al., 2012; Holzel et al., 2013; Krause-Utz, Winter, Niedtfeld, & Schmahl, 2014; Rabinak et al., 2014; Radaelli et al., 2015; Townsend & Altshuler, 2012; Williams et al., 2007). Early life stress and attachment trauma negatively influence neural circuitry involved in emotional processing and regulation, further increasing the risk of developing psychiatric disorders (Puetz et al., 2014).

Despite common or overlapping neuronal features and symptoms, patterns of emotion generation, reactivity, and modulation also vary across disorders and likely involve differing neurocircuitry (Etkin & Wager, 2007; Krause et al., 2014; Oathes, Patenaude, Schatzberg, & Etkin, 2015).

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Hyperactivity of the amygdala and insula in response to emotionally arousing and/or neutral stimuli (i.e., an exaggerated fear or arousal response) may represent shared neurobiology, while level of activation of, and/or connectivity with, prefrontal areas involved in salience processing, emotion modulation, and response inhibition vary across disorders (Anticevic, Repovs & Barch, 2012; Etkin & Wager, 2007; Hamilton et al., 2012; Krause et al., 2014; Townsend & Altshuler, 2012).

A number of existing therapies target maladaptive emotion generation and regulation processes including cognitive-behavioral therapy, dialectical-behavioral therapy (DBT), mindfulness-based approaches, emotion-focused therapies, and psychodynamic interventions. Findings from neuroimaging studies indicate that mindfulness training and DBT produce a decrease in amygdala hyper-responsivity to negative emotional stimuli, while cognitive-behavioral and psychodynamic treatments modify neural circuits involved in the regulation of negative emotions and fear extinction (Beutel, Stark, Pan, Sibersweig, & Dietrich, 2010; Desbordes et al., 2014; Goldin, McRae, Ram.el, & Gross, 2008; Goodman et al., 2014; Nechvatal & Lyons, 2013; Porto et al., 2009; Reinecke, Thilo, Filippini, Croft, & Harmer, 2014; Yang, Kircher, & Straube, 2014). Thus, there may be a general effect of psychotherapy in normalizing the dysfunctional limbic activation often observed in mental illness (Etkin & Wager, 2007; Fonzo et al., 2014; Hamilton et al., 2012; Holzel et al., 2013; Krause-Utz et al., 2014; Townsend & Altshuler, 2012), while some approaches may also target cortical-limbic disintegration. This normalization and integration may represent important mechanisms of change that may enhance treatment outcomes if more explicitly and systematically targeted in psychotherapy.

Inthis paper we present a preliminary brain-based model of change in psychotherapy based on systematically normalizing dysfunctional cortical-limbic activation and dysintegration. Our theoretical model rests on the following assumptions: (a) dysfunctional neuronal networks involved in the generation and regulation of emotions represent a general risk factor for the development of psychopathology, (b) there exist common or overlapping neural network dysfunctions across mental health disorders involved in the generation and regulation of emotions (Etkin & Wager, 2007; Hamilton, Chen, Waugh, Joorman, & Gotlib, 2015; Hamilton et al., 2012; Oathes et al., 2015), and (c) these dysfunctions can be modified by psychotherapy. Furthermore we assert neuroscience findings provide tentative support for a phase-based approach to psychotherapy, elucidating the "when, why, and what" of psychotherapeutic interventions based on their potential role in the regulation, integration, and homeostatic rebalancing of subcortical and neural networks involved in emotion generation and regulation (Cozolino, 2010). At each phase of our model we discuss the following: (a) what clinical concerns are prominent, (b) why these concerns are present (present a neuroscientific rationale), (c) the underlying biological imperative that inform interventions at each stage, and (d) what needs to be accomplished prior to moving forward in the treatment hierarchy.

Hierarchical Phase Model of Integrated Psychotherapy

Phase One: Ensuring Safety of the Patient and Others

What clinical concerns are prominent? A patient in this phase of illness will display unstable and disorganized behavior, and cognitive and behavioral dysregulation (e.g., quasi-psychotic symptoms, extreme emotional distress/dysregulation, self-harm or aggressive behaviors). When pressured to look more closely at emotions and associated bodily experiences, thoughts, and behavioral responses, the patient will become easily overwhelmed and display psychological fragility through projection, acting out, regressive or dissociative responses (i.e., freeze or shut-down). These patients may report psychotic like episodes and intense/persistent suicidal/homicidal ideation and fantasies.

Why are these concerns present (a neuroscientific rationale)? At this phase, emotional experiences are perceived as threatening, uncontrollable and overwhelming. Triggered changes in the hypothalamus and brainstem associated with excessive, amygdala activation result in increased automomic arousal and the release of stress hormones (cortisol, adrenaline, and noradrenaline) in the brain and body. The

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What is the underlying biological imperative that informs interventions at this phase? As the patient at this phase of illness will display unstable/disorganized cognition and behavior, the immediate overarching goal is to provide supports that ensure safety and stabilize functioning. These supports include expressing empathy for the patient's distress, enhancing the social engagement system, providing cognitive containment (assisting with reality testing, helping the patient accurately differentiate and evaluate self-generated internal and external sensory information) and deterring potentially destructive behaviors (suicidal/parasuicidal and aggressive/destructive acts). These interventions temporally dampen intense arousal (decrease amygdala activation) by reducing negative salience (activating the ventral-lateral prefrontal cortex (VLPFC; Kohn et al., 2014). This may enhance integration of limbic structures (insula, amygdala) with prefrontal regulatory areas (dorsomedial prefrontal cortex), improving emotion processing and self-awareness.

What needs to be accomplished prior to moving forward in the treatment hierarchy? At this point, the patient's negative emotional responses are modulated primarily by external or synthetic influences (i.e., the individual is highly dependent on external/environmental or biological substances to regulate emotions). Safety of self and/or others is largely achieved through efforts designed to calm the nervous system, including attuned/calming therapeutic interactions (modulating negative affect arousal), cognitive containment, enhancing external structure (e.g., inpatient care or mobilization of family and support systems), and artificially enhancing internal structure with medication. These interventions temporarily reduce unstable and disorganized behavior, and result in improved cognitive and behavioral regulation (e.g., psychotic symptoms will decrease as will self-harm or aggressive behaviors).

Phase Two: Containment of Anxiety, Negative Emotion, and Self Sabotaging Behavior

What clinical concerns are prominent? Patients at this phase of illness often report confusion and lack of clarity regarding emotional experience, avoidant/self-harrnful/destructive behaviors, and/or somatization. Behaviors continue to appear contradictory and irrational as deficits exist in the awareness of, and/or conscious/integrated processing of, somatosensory/interoceptive responses. Thus the individual experiences difficulties linking implicit emotional responses to situational appraisals and action tendencies (i.e., lacks both cognitive and emotional insight). Cognitive representation/categorization of emotional experience may be undifferentiated and lack meaning ("I feel overwhelmed"). The cognitive schemas used to interpret neurophysiological arousal are poorly differentiated, and there is a "blurring" or overlap of emotions (emotions are similarly

valenced) due to poor differentiation along the arousal axis (Posner, Russell, & Peterson, 2005). The patient will report moderate to high anxiety (e.g., smooth and striated muscle tension), low tolerance for emotional/physiological distress, and maladaptive methods to regulate emotional experience (i.e., experiential avoidance, substance use, nonsuicidal self-injury). When pressured to experience and differentiate core affect the patient will feel challenged and may display regressive, defensive responding such as turning against themselves (e.g., present as passive/helpless/weepy) or turning against the therapist (e.g., present as dismissive/hostile/sarcastic/suspicious).

Why are these concerns present (a neuroscientific rationale)? At this phase, the ventral vagus nerve "vagal calming system" is retracted, and the sympathetic nervous system overactivated, supporting fight or flight behaviors (defense through increases in mobilization) (Porges, 2009) Self-harm may occur during dysregulated states, triggering a release of endorphins calming the amygdala and placing these individuals at risk for further episodes of self-injury (Weinberg & Klonsky, 2012). Substance use, dysfunctional relationships, and other harmful behaviors (e.g., disordered eating) can also function to dampen distressing amygdala activation, contributing further to seemingly irrational and maladaptive behaviors. Deficits exist in the conscious awareness of, and/or integration of, somatosensory/interoceptive experience. For some individuals, the subcortical structures are activated and they may be aware of this limbic activation, but this sensory information is not sufficiently integrated with the cortical circuits to make sense of and modify these visceral and behavioral reactions (a_ctivation and conscious awareness but limited integration = dysregulation). Others lack conscious awareness of this activation and these autonomic/interoceptive responses (decreased activation of anterior insula, dorsal anterior cingulate and medial prefrontal cortex = alexithymia) (Kano & Fukuda, 2013). For the latter group, emotions are experienced in a very rudimental form (i.e., chronic pain/somatization), as awareness of emotion requires active/conscious interpretations of changes in physiology (Seth, Suzuki, & Critchley, 2011). Both conditions result in decreased insight/awareness which contributes to a lack of perceived agency, with patients reporting feeling "out of control" and "unable" to change.

What is the underlying biological imperative that informs interventions at this phase? During the second phase of the "hierarchy of intervention," the goal is to help the patient develop awareness of (by increased attunement to) the neurophysiological aspect of emotional experience, decrease fear/avoidance of distressing interoceptive experiences, dampen intense arousal (activate the parasympathetic nervous system), and restore behavioral control. Areas of the prefrontal cortex involved in situational threat appraisal are activated (left VLPFC) to reduce intense physiological arousal and deter dissociative and impulsive tendencies which occur when implicit processes take over, and explicit mechanisms or cortical circuits are unbalanced (i.e., decreased negative coupling between the anterior cingulate cortex and amygdala, and enhanced connectivity between areas of the prefrontal cortex involved in treat detection, "the dorsal inferior frontal gyrus and fear networks") (Posner et al., 2009; Yang et al., 2014). Both limbic activation and integration of limbic responses with higher level cortical processes is required to make meaning of lower level sensory information (Gu, Hof, Friston, & Fan, 2013).

The patient in Phase Two is taught awareness of (to differentiate) the physiological markers of anxiety, and relaxation strategies to reduce the physiological intensity of this experience. Graduated interoceptive exposure exercises can be implemented to reduce fear of, and the intensity of distressing bodily states (i.e., panic symptoms). Although activation of limbic responses is required to integrate and regulate this lower level sensory information with cortical circuitry, a graded approach is needed because this activation must not be excessive or it will interfere with treatment (Bryant et al., 2008; Gu et al., 2013). The individual is encouraged to monitor/differentiate fluctuations in the physiological component of emotions. This mapping of internal states forms the basis for the creation and conscious recognition of specific emotions (making sense of and modulating arousal) (Gu et al., 2013). The self-monitoring encouraged during the application of relaxation techniques, and new learning around the meaning of physiological responses helps change the individual's relationship with distressing bodily states.

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Paying attention to these physiological cues shifts the response from avoidance to curiosity about triggers and approach/acceptance rather than experimental avoidance (Hayes-Skelton, Roemer, Orsillo, & Borkovec, 2013). Additionally, affect labeling activates areas in the prefrontal cortex associated with regulatory processes (i.e., dorsolateral and dorsomedial prefrontal cortex), diminishing limbic responses (Burklund, Creswell, Irwin, & Lieberman, 2014; Kashdan, Barrett, & McKnight, 2015). Mindfulness methods can be employed to create the opportunity to monitor and attend to emotions (rather than avoid emotional experience) and decenter from unhelpful, distressing thoughts. This allows the individual to appreci- · ate both the adaptive and fluctuating aspects of these responses, reducing negative salience and amygdala activation (Desbordes et al., 2012; Holzel et al., 2013).

What needs to be accomplished prior to moving forward in the treatment hierarchy? Interventions are focused on reducing distressing arousal and noting/altering patterns of impulsive/harmful or avoidant behaviors in response to intense emotional distress. Applied guided relaxation techniques, and the mindfulness components of DBT may be particularly effective interventions at this phase of treatment (Hayes-Skelton et al., 2013; Linehan, 1993). Cognitive-behavioral interventions that help restructure catastrophic interpretations of sympathetic responses and facilitate interoceptive desensitization may also be helpful. Generic therapeutic interventions including expressing empathy for distress, instilling beliefs about the capacity for change, encouraging agency, promoting adaptive help seeking, and building therapeutic rapport are also essential at this phase to increase motivation. Reflective awareness of emotion (i.e., conceptual representation of emotional experience) is not a primary focus until regulatory skills to manage intense arousal are mastered.

Phase Three: Internal Regulation and Curious, Active Engagement With Emotion to Guide Intention and Behavior

What clinical concerns are prominent? Patients presenting in this phase of illness experience difficulties linking or differentiating the different components of their emotional responses (bodily sensations, thoughts, and behavioral responses). The individual can tolerate and dampen intense emotional arousal without engaging in self-destructive behaviors when pressured to experience core affect (hurt, rage), and as a result they report/display less intense/better modulated anxiety (i.e., striated and occasional smooth muscle response). Although they will likely report emotional experience as distressing and may describe avoidance, they typically do not respond in harmful or destructive ways to negative emotional experience and display "higher" level defensive responses, including repressive (minimization, intellectualization, rationalization) and tactical defenses (e.g., vagueness, diversification) when pressed to experience negative emotions.

Why are these concerns present (a neuroscientific rationale)? Top-down circuitry shapes our experience of emotions (e.g., anxiety is associated with reduced top-down modulation of threat cues, while depression is associated with excessive attention to negative aspects of the environment or "biased activation of right prefrontal regions" (Cozolino, 2010, p. 255; Ritchey, Dolcos, Eddington, Strauman, & Cabeza, 2011). Conscious processing/categorization of emotion and conscious attemp"ts at emotion modification through reappraisal have a "top-down" modulating effect on the subcortical experience of emotion (Lee, Heller, Reekum, Nelson, & Davidson, 2012; Ritchey et al., 2011). Evidence suggests a reciprocal relationship between prefrontal and amygdala functional connectivity, with amygdala activation declining with increased contextualization and · processing of valence (Freed, Yanagihara, Hirsch, & Mann, 2009; Posner et al., 2009; Sotres-Bayon, Cain, & LeDoux, 2006). This reciprocal relationship may serve to regulate, modulate, or dampen affective responses (Freed et al., 2009; Ochsner et al., 2002). In order for this modulation to occur, however, the underly- ing implicit/physiological emotional experience must first be accessed, linked to appraisal (va- lence) dimensions, and assigned to an emotion category. This clear emotional differentiation enables the *highfidelity* experience of core emo- tion. Heightened experiencing allows enhanced access to, and clarity of, emotional information, resulting in greater coherence.

Conscious processing allows the individual to better influence/regulate and respond to emo-

tional states (becomes the precondition for the most functional act that can be taken) (Kashdan et al., 2015). Effective problem solving/ adaptive functioning requires both emotional regulation and cognitive flexibility. Negative emotions that are repeatedly activated (prolonged, undifferentiated physiological activation), but not consciously processed (linked with an emotional state) and modulated, may be experienced as distressing somatic symptoms (Deary, Chalder, & Sharpe, 2007; Stonnington, Locke, Hsu, Ritenbaugh, & Lane, 2013) and contribute to physical health concerns, learning dysfunctions, and memory concerns (Cozolino, 2010, pp. 249-253). Linking embodied emotional information with conceptual knowledge binds or *grounds* the physical state to an event so that is becomes more than just a physical sensation (Barrett, 2012).

What is the underlying biological imperative that informs interventions at this phase? During this phase of treatment, the focus is on activating the implicit (subcortical) circuitry while explicitly engaging the conscious, reflective, experience of emotion (i.e., cognitive representations of emotions or emotional valence) to further (a) dampen the implicit/physiological arousal associated with core emotional experience; (b) alter beliefs regarding the role of emotions (view emotions as helpful allies that provide important formation and are temporal) to facilitate acceptance of emotional experience; (c) to increase the capacity to tolerate distressing emotions without defensive responding or the use of maladaptive emotion regulation strategies; and (d) decrease behavioral avoidance and reduce symptoms. The primary goal is to assist the individual in utilizing the conscious linguistic processes of the prefrontal cortex to modify implicit emotional experience through more balanced emotional appraisals (Cozolino, 2010, pp. 254-256; Ritchey et al., 2011). "Bottom-up" engagement of the insula region is required so emotional experience can be processed and transformed (Reinecke et al., 2014). Core affect must be experienced at moderate levels (high levels trigger overwhelming anxiety and block cognition) to integrate the cognitive and physiological aspects of emotion (Bryant et al., 2008). In a sense, the therapist must "strike while the iron is cold" (i.e., arnygdala activation is modulated). Active attempts at regulation through balanced activation of the prefrontal cortex (decreasing right-biased activation) can then occur (Abler et al., 2010). This top-down integration creates new neural associations that keep the arnygdala from triggering a sympathetic response.

What needs to be accomplished prior to moving forward in the treatment hierarchy? Phase Three of treatment involves deepening the link between the physiological component of emotional experience and the conscious or explicit component (i.e., cognitive· representations of emotions), thus, both "top down" and "bottom-up" techniques are required. The focus is on assisting the patient in noticing/understanding/altering the associations between situational appraisals, emotional reactions, and associated behaviors. Specifically the patient is taught to (a) mobilize and track the changing physiological aspects of emotional experience (since implicit affect needs to be accessed to make it available for explicit processing); (b) develop adaptive emotion regulation skills, especially those related to tolerance and modification/reappraisal (Berking et al., 2008); and (c) identify valued goals (to enhance motivation, facilitate emotion tolerance, encourage effective problem solving and increase "approach behaviors"). Components of cognitive-behavioral, acceptance-commitment and emotionfocused approaches can effectively address these skill deficits (Greenberg, 2008; Hofmann, Asmundson & Beck, 2013; Paivio, 2013; Twohig, 2012).

Phase Four: Remembering and Working Through-Exploration and Change of Maladaptive Self and Interpersonal Schema

What clinical concerns are prominent? Patients at this phase of illness have learned skills to monitor and alter the physiological, cognitive, and behavioral aspects of emotion. Despite this, they continue to have issues with interpersonal sensitivity, difficulties tolerating interpersonal vulnerability, and hyperreactivity. They often defend against intense core emotional experiences related to past attachment traumas using less adaptive, more immature, image distorting defenses (e.g., regression, projection) (Bond, 2004). These patients may also struggle with mentalization deficits and pervasive and enduring maladaptive schema or ways of perceiving and relating to self and others neurotic, dysfunctional and result in adaptive f diagnosed with persom ley & Jang, 2005).

Why are these conct scientific rationale)? pacts cortical networks ir tional perception, attr intentions to others, self- self-awareness (Teicher, Polcari, 2014). Accessin ries often involves transf tions associated with atta, experience of grief. Al memories triggers intenst allows full conscious m Prelimiimy research indi acti'(ates an area of the b dial prefrontal cortex) tha inhibitory role on arousal sympathetic nervous syst Weiss, 2013). Thus deac1 increase in arousal. The l apy is to access this core shift the emotional respo pain" to "grief or loss." core .affect associated wi suits in a shift from i1 sadness and maladaptive sponses, to a conscio1 attachment cannot be rest gagement, acceptance) a tive salience (Freed & 1' ence of grief does nc approach/withdraw respc renouncement of an thought rather than acti drop in arousal, allowing tion of this implicit, sut prefrontal circuitry (i.e., , prefrontal areas) for co processing, and reapprais Kohn et al., 2014). This t1 correlates with neurologi, increased psychological

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ing to self and others that drive repetitive, neurotic, dysfunctional interpersonal patterns and result in adaptive failures. They are often diagnosed with personality pathology (Livesley & Jang, 2005).

Why are these concerns present (a neuroscientific rationale)? Trauma negatively impacts cortical networks involved in internal emotional perception, attributing thoughts and intentions to others, self-referential thinking, and self-awareness (Teicher, Anderson, Ohashi, & Polcari, 2014). Accessing trauma-related memories often involves transforming distressing emotions associated with attachment traumas with the experience of grief. Accessing trauma-related memories triggers intense psychological pain and allows full conscious awareness of core affect. Preliminary research indicates that such pain deactivates an area of the brain (the right ventromedial prefrontal cortex) that is considered to have an inhibitory role on arousal (by activating the parasympathetic nervous system) (Meerwijk:, Ford, & Weiss, 2013). Thus deactivation contributes to an increase in arousal. The goal at this point in therapy is to access this core implicit affect and then shift the emotional response from "psychological pain" to "grief or loss." Increased attunement to core affect associated with attachment trauma results in a shift from intense protest/yearning/ sadness and maladaptive secondary emotional responses, to a conscious awareness that the attachment cannot be restored (detachment, disengagement, acceptance) and a reduction in incentive salience (Freed & Mann, 2007). The experience of grief does not elicit flight/fight or approach/withdraw responses, rather a conscious renouncement of an attachment (motivates thought rather than action), facilitating a rapid drop in arousal, allowing more extensive integration of this implicit, subcortical experience with prefrontal circuitry (i.e., dorso- and ventral-lateral prefrontal areas) for contextualization, salience processing, and reappraisal (Freed & Mann, 2007; Kohn et al., 2014). This transformation of emotion correlates with neurological changes that facilitate increased psychological strength and resilience.

What is the underlying biological imperative that informs interventions at this phase? The goal of this phase of treatment is to access, understand the origins of, and alter internal working models/maladaptive self/other schemas (e.g., polarizing and self-invalidating patterns of thinking and behaving) and repetitive dysfunctional interpersonal patterns. Treatment at this phase also addresses deficits in mentalization resulting from disrupted early attachments and encourages behaviors that disconfirm maladaptive schema. At this stage, patients are required to *work through* core emotional conflicts, grieve attachment ruptures, and create more adaptive self/other schemas. As in Phase Three, the underlying physiological emotional experience must be activated; however, the accessed affect is,often implicit (unconscious) and specifically associated with painful attachment disruptions or trauma. This requires bringing psychological defense to conscious awareness (blocking defense) so that subcortical networks associated with attachment traumas are activated and made accessible for integration with cortical circuits (Schore, 2009b). This process of accessing and transforming painful emotions f!Sociated with early attachment trauma requires reactivation of limbic areas to access implicit memories. As psychological fragility is resolved (i.e., the capacity to tolerate painful core affect increased), individuals can access implicit neural networks associated with early attachment trauma and integrate this arousal with cortical structures to create meaning and alter dysfunctional psychoneurotic structures without disrupting psychological homeostatic balance.

What needs to be accomplished prior to moving forward in the treatment hierarchy? Although cognitive interventions continue to be helpful at this stage, other therapeutic methods drawn from psychodynamic and emotion-focused modalities are required to work through core emotional conflicts, grieve attachment ruptures, and create more adaptive self/other schemas. Addi-· tionally, mentalization approaches can be employed to develop awareness and understanding of the emotional drivers and mental states/intentions of self and others (i.e., address reflective functioning deficits that are often at the core of personality pathology; Bateman & Fonagy, 2010; Stonnington et al., 2013), paving the way for compassionate understanding and forgiveness.

Phase Five: Integration and Synthesis of a More Adaptive Self-Structure and Life Script

What clinical concerns are prominent? Patients at this phase of illness typically recognize maladaptive patterns of thinking and are able to engage in the process of establishing

more adaptive schema and associated behaviors; however, challenging circumstances may overwhelm their adaptive capacity. They are generally able to recognize and understand the mental/emotional states and associated intentions of self and others. Clinical concerns, including fluctuations in mood and low level anxiety are situationally triggered and temporary.

Why are these concerns present (a neuroscientific rationale)? At this phase, subcortical responses are contained (i.e., diminished sympathetic arousal) and can be managed through explicit modulation utilizing reappraisal strategies. Modulation of emotion requires ongoing attunement to the physiological signals associated with emotional arousal (i.e., implicit/interoceptive component of emotional experience-insular activation) and integration with higher cognitive networks (Ochsner & Gross, 2005). The ongoing tracking of the different components of emotional experience (physiology, thoughts, and behaviors), allows individuals to develop awareness of conflictual psychological needs and establish reciprocal, empathetic, respectful communication and mutuality in relationships.

What is the underlying biological imperative that informs interventions at this phase? During this phase, individuals are encouraged to routinely attend to the implicit/interoceptive component of emotional experience (insular activation) and associated orbital and medial prefrontal cortex (OMPFC) and dorsolateral prefrontal cortex linkages to better ascertain, understand and respond to intra and interpersonal psychological needs. Emotional experience and higher cognition networks are continually integrated/balanced, so effective problem solving and adaptive responses can occur. This involves encouraging ongoing attunement to the implicit/interoceptive component of emotional experience (insular activation) and prefrontal circuitry. Interventions focus primarily on tracking the components of emotional experience, identifying/removing obstacles to consolidating more adaptive self and other narratives, encouraging more productive functioning (e.g., enhancing agency, effective communication and problem solving skills) and facilitating corrective, "often relational" experiences. Other therapy tasks include discouraging maladaptive emotions (e.g., shame, guilt) from thwarting intimacy with others.

Embracing adaptive self and interpersonal schema leads to more positive and attuned social in,teractions. We suggest this new learning essentially reorganizes connections between the amygdala and the OMPFC to reconceptualize what is safe and dangerous (Cozolino, 2010, p. 213). The OMPFC interprets positive social interactions as rewarding, and over time reorganizes attachment schema in the direction of social approach/engagement (Cozolino, 2010, p. 229). Resulting changes in interpersonal functioning highlight the role of healthy social connection for well-being, and creates the expectation of positive outcomes in relationships.

What needs to be accomplished prior to moving forward in the treatment hierarchy? Individuals need to maintain/consolidate a more adaptive and integrated representation of self and others, and develop a greater capacity for self-directedness. Effective therapeutic approaches include cognitive- behavioral and interpersonal modalities that increase agency and awareness through conscious reflection and ongoing self-correction, as well as emotion-focused approaches that encourage ongoing attunement to emotional experience to guide adaptive functioning. More traditional psychodynarnic approaches relying heavily on interpretation that enhance mentalization, increase empathy, and promote reflection are also effective in this phase. The overarching goal is to encourage new, adaptive patterns of thinking and behavior that promote ongoing compassion for self and others, resulting in personal well-being and interpersonal effectiveness.

Summary and Implications for Psychiatric Education, Practice, Research, and Service Delivery

With advances in neuroimaging research on mental illness and psychotherapy, we can now make tentative hypotheses regarding both neural correlates of symptom presentation, treatment outcomes, and possible mechanisms of change. We have used these preliminary research findings to develop our phasic, transdiagnostic, brain-based model of stability and growth. We propose that this model has the capacity to provide a vital link between psychological and neurological phenomena: the elusive and essential mind-body connection.

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The model supports a phasic approach to care highlighting awareness, agency, and ultimately, self-regulation. Initial targets in this. stepped approach include the physiology of hyperarousal, and inhibition or mobilization of extremes of sympathetic activation (to facilitate later integrated processing of these autonomic responses). Once stabilized, core emotional pathology is targeted and, when necessary, attachment ruptures grieved, resulting in the reintegration of dysregulated circuitry, enhanced reflective capacity, and the establishment and consolidation of adaptive regulatory mechanisms.

This model of psychotherapy has significant implications for service delivery, suggesting a defined and sequential course of specific therapeutic interventions with the primary goals/interventions at each phase of illness informed by the neuronal dysfunctions that are prominent. Time spent at each phase of the hierarchy would vary depending on psychological profile. Some individuals/populations may start treatment at a later phase, or alternatively require substantial or ongoing care at an earlier phase of treatment (display slower than typical transition through the hierarchy of care) depending on the level of impairment (dysintegration) of associated neural networks. Early relational trauma, in particular, may disrupt the development of neuronal circuitry required to tolerate and self-regulate/modulate the intensity and duration of negative emotional states (Schore, 2009a, 2009b).

The implications of this model also relate to the pathogenesis of mental disorder, suggesting the design of prevention and early intervention efforts focused on modulation of subcortical (autonomic) responses, and encouragement of balanced cortical integration to enhance cognitive flexibility/psychological resilience. Additionally, the model supports a neuroscience training curriculum for psychiatry residents that is antireductionistic, and integrates principles of neurobiology and psychology (Lacy & Hughes, 2006). This curriculum should include therapeutic approaches that address both maladaptive defensive functioning/responding, and emotion generation/regulation deficits, while also building a foundational understanding of the neurological processes that may underlie psychiatric symptoms and mechanisms of change in psychotherapy.

Although there is tentative support for phasic, brain-based model of integrated psychotherapy, further research is required to substantiate the common neurological underpinnings of mental illness proposed in this theory (i.e., dysfunctions in emotion generation, regulation, and integration with cortical circuits), the suggested neurobiological mechanisms of change (i.e., modulation, integration, and homeostatic rebalancing of affected subcortical, and neural networks), and the recommended phase-based approach to intervention (i.e., external safety/containment, internal modulation of arousal, integration/regulation, access/integration of implicit/unconscious emotional experience, and consolidation of balanced integration). Longitudinal studies, in particular, may clarify commonalities and differences in neurodevelopment trajectories across disorders and symptom c;lusters. This research may enhance our understanding of the development of healthy emotion regulation and reflective functioning capacities, potentially informing the design and delivery of effective preventive and early intervention efforts (Bertocci et al., 2014; Gee et al., 2014; Puetz et al., 2014). Ultimately, interventions based on this model and related research may help modulate the genetic diathesis and comorbidities of mental illness, increase psychological resilience, and reduce the prevalence or burden of mental health disorders.

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