An Affective Neuroscience Model of Boosting Resilience in Adults

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Abstract

Although research has identified dozens of behavioral and psychosocial strategies for boosting resilience in adults, little is known about the common underlying pathways. A comprehensive review of these strategies using an affective neuroscience approach indicates three distinct general routes to resilience (Fig. 1A): 1) down-regulating the negative (e.g., exposure, cognitive reappraisal) by reducing distress-related responses of the amygdala, hypothalamic-pituitary-adrenal axis, and autonomic nervous system; 2) up-regulating the positive (e.g., optimism, social connectedness) by activating mesostriatal reward pathways, which in turn can buffer the effects of stress; and 3) transcending the self (e.g., mindfulness, religious engagement) by reducing activation in the default mode network, a network associated with self-reflection, mind-wandering, and rumination. Some strategies (e.g., social support) can boost resilience via more than one pathway. Under- or over-stimulation of a pathway can result in vulnerability, such as over-stimulation of the reward pathway through substance abuse. This tripartite model of resilience-building is testable, accounts for a large body of data on adult resilience, and makes new predictions with implications for practice.

Keywords: resilience; well-being; emotion regulation; affective neuroscience; amygdala; stress; reward; default mode

1. Introduction

Whether surviving a natural disaster, loss of a loved one, or violence, most people experience a traumatic event at some point in their lives. In addition to these types of acute stressors, many people also often experience some form of chronic stressor such as marital problems, a debilitating disease, low socioeconomic status, or work-related stress. While some individuals recover from or continue to thrive in the face of such stressors, others fall into the grips of depression, anxiety, or other chronic disease, and many report poor psychological and physical well-being. Decades of research have identified dozens of behavioral and psychosocial strategies for buffering stress and boosting resilience. However, to date little is known about the common pathways underlying the various strategies. This integrative review is an attempt at identifying the few general pathways that are shared among these numerous strategies. Taking an affective neuroscience approach, here I propose a tripartite model of resilience building, identifying three distinct major pathways to building resilience in adulthood.

Resilience has been defined in the literature in many different ways (Dunkel Schetter & Dolbier, 2011; Kalisch et al., 2017). In this paper, resilience is defined broadly as adapting well in the face of chronic or acute adversity. Consistent with prior reviews (Southwick, Vythilingam, & Charney, 2005; Tabibnia & Radecki, 2018), in the review that follows, I include the following outcome measures as indices of resilience: mental health and related indices, including self-reported affect and quality of life; physical health and related indices, including immune assays and

pain management; and longevity and mortality, particularly following trauma.

I begin with a brief overview of the brain networks that are most relevant to resilience. Then I present the tripartite model of resilience-building that is based on this current knowledge of functional neuroanatomy. Next, I present a selective review of evidence in support of the model. Specifically, I review affective neuroscience studies of over two-dozen resilience-building strategies and demonstrate how each strategy A) boosts resilience and B) recruits at least one of the 3 major pathways in the tripartite model. I focus on behavioral and psychosocial, rather than pharmacological or genetic or environmental, factors that affect resilience, with an emphasis on studies published within the last 5-10 years. Finally, I discuss practical implications and suggestions for future research.

2. Neurocircuitry of resilience

It has generally been postulated that the neural correlates of resilience overlap with the brain circuitry involved in fear and stress (henceforth, distress) and their regulation (Charney, 2004; Russo, Murrough, Han, Charney, & Nestler, 2012; van der Werff, van den Berg, Pannekoek, Elzinga, & van der Wee, 2013). The mesolimbic reward circuit has also gained attention as an important network in resilience (Charney, 2004; Dutcher & Creswell, 2018; Krishnan et al., 2007; Russo et al., 2012). More recently, a pattern is emerging in the literature relating an additional neural circuit, namely the default mode network, to resilience (Whitfield-

Gabrieli & Ford, 2012). In this section, I review the roles of the following networks in resilience: 1) distress (fear and stress) networks, 3) reward networks, and 3) default mode network. I also discuss the involvement of the prefrontal cortex (PFC), particularly lateral prefrontal cortex (LPFC), medial prefrontal cortex (MPFC), and anterior cingulate cortex (ACC), in these 3 categories of networks. While other brain regions, such as anterior insula (Waugh, Wager, Fredrickson, Noll, & Taylor, 2008) and hippocampus (A. P. King, 2018), also play important roles in resilience, here I demonstrate that a simple model limited to these 3 categories of brain networks can account for a vast array of behavioral and psychosocial strategies of resilience-building in adult humans.

2.1. Amygdala fear network

The central role of the amygdala in emotion, particularly fear learning, has been well-established and reviewed elsewhere (Adolphs, 2013; Davis, 1992; LeDoux, 2000). Briefly, because of its afferent connections from sensory cortices and efferent connections to brainstem and hypothalamic nuclei that can mobilize autonomic (e.g., fight-or-flight) and endocrine (e.g., stress) responses, the amygdala is uniquely positioned to learn about and respond to salient stimuli. These complex circuits, along with microcircuits within the amygdala and extended amygdala, help orchestrate physiological and behavioral responses to stimuli that are pertinent to self-preservation, including stimuli that predict potential threat, thus mediating states of fear and anxiety (Janak & Tye, 2015; Tovote, Fadok, & Luthi, 2015), but also

rewarding stimuli such as food and social partners (Tovote, Fadok, & Luthi, 2015). Based on animal models and parallel findings in human neuroimaging studies, hyper-responsiveness of the amygdala, along with dysfunction in MPFC regions that can help inhibit it, is posited to underlie many anxiety disorders, including specific phobias, generalized anxiety disorder, and PTSD (Andrewes & Jenkins, 2019; Rauch, Shin, & Wright, 2003; Shin & Liberzon, 2010). Amygdala abnormalities are central to other psychopathologies, including mood disorders, as well (Drevets, 2001; Nestler et al., 2002). In particular, hyperactivity of the amygdala to threatening stimuli (Etkin & Wager, 2007; Rauch et al., 2003; Shin & Liberzon, 2010; Siegle, Thompson, Carter, Steinhauer, & Thase, 2007) and at rest (Leaver et al., 2018; Shin & Liberzon, 2010), as well as anatomical abnormalities in the amygdala (Bora, Fornito, Pantelis, & Yucel, 2012; Hamilton, Siemer, & Gotlib, 2008; Hilbert, Lueken, & Beesdo-Baum, 2014), have been reported in most anxiety disorders and depression. There are also multiple reports of amygdala hyperactivation during explicit emotion regulation in major depressive disorder (Rive et al., 2013; Zilverstand, Parvaz, & Goldstein, 2017). Conversely, medications that reduce amygdala activity help alleviate psychiatric symptoms (Arnone et al., 2012; Labuschagne et al., 2010; Sheline et al., 2001). Given the comorbidity of mood and anxiety disorders with cardiovascular disease, it is perhaps not surprising that amygdala hyperactivity has also been implicated in cardiovascular disease (Fiechter et al., 2019; Kraynak, Marsland, & Gianaros, 2018). Taken together, these findings suggest that interventions that can reduce amygdala hyperactivity can help boost psychiatric and physical health resilience.

2.2. Autonomic and neuroendocrine stress networks

Stressful experience has long been implicated in the etiology of many chronic physical and mental health conditions (S. Cohen, Janicki-Deverts, & Miller, 2007). In the current paper, stress is defined broadly as an actual or anticipated disruption of homeostasis or well-being (Ulrich-Lai & Herman, 2009). Depending on the type of stressor, the brain can initiate a fast autonomic nervous system (ANS) response, involving the sympathetic and parasympathetic systems, and/or a slow endocrine response, involving the hypothalamic pituitary adrenal (HPA) axis, resulting in elevation of circulating glucocorticoids like cortisol (Russell & Lightman, 2019; Ulrich-Lai & Herman, 2009). The fight-or-flight autonomic response and the HPA endocrine response are adaptive in the short term, but their prolonged activation leads to endocrine, immune, and cardiovascular dysregulation, which in turn can contribute not only to infectious (e.g., viral) and inflammatory (e.g., atherosclerosis) disease, but also to mental illness like depression (Dantzer, Cohen, Russo, & Dinan, 2018; Hunter, Gray, & McEwen, 2018; Russell & Lightman, 2019). Prolonged elevation of glucocorticoids also impairs brain structure and function that can affect mental health. Specifically, chronic elevation of stress or stress hormones can cause abnormal cell growth in the amygdala, as well as neural damage in the hippocampus and prefrontal cortex, compromising cognitive and affective function, including emotion regulation, and increasing vulnerability to psychopathology, including substance abuse, depression, and anxiety (Arnsten, 2009; Cathomas, Murrough, Nestler, Han, & Russo, 2019; Lupien, McEwen, Gunnar, & Heim, 2009; Russell &

Lightman, 2019). Increased HPA activity, including elevated levels of cortisol, has long been linked with depression (Ali & Nemeroff, 2020; Pariante & Lightman, 2008; Russell & Lightman, 2019; Stetler & Miller, 2011), although more recent meta-analyses and models associate elevated HPA activity with only subgroups of patients (Gold, 2015; Lombardo et al., 2019; Menke, 2019), such as with melancholic but not atypical depression (Gold, 2015). Successful treatment of depression tends to normalize the HPA axis disruptions (reviewed in Ali & Nemeroff, 2020), and, importantly, reduction of stress or cortisol can reverse the neural and psychological impairments caused by elevated HPA activity (Lupien et al., 2009; McEwen & Gianaros, 2011). Thus, interventions that can reduce stress and excess sympathetic and HPA activity are considered particularly effective at boosting resilience.

2.3. Mesostriatal reward networks

The canonical reward network in the brain, known as the mesolimbic reward pathway, is that of the midbrain dopamine neurons in the ventral tegmental area (VTA) projecting onto the nucleus accumbens in the ventral striatum and other forebrain areas; another related pathway is the mesocortical pathway connecting the VTA dopamine neurons to ventromedial PFC and other MPFC regions (Bjorklund & Dunnett, 2007). A third and final dopaminergic pathway originating in the midbrain is the nigrostriatal pathway, which, although primarily associated with motor function, has also been implicated in reward processing (Schultz, Dayan, & Montague, 1997). I refer to the mesolimbic, mesocortical, and nigrostriatal reward

pathways collectively as the "mesostriatal" reward pathway. The midbrain dopamine neurons in the mesostriatal pathway respond to reward such as food and even to anticipation of reward, and they are inhibited by aversive stimuli and by omission of anticipated reward (Schultz, 2007). Although there is no single unidirectional association between reward and resilience across the board (Russo et al., 2012), a growing literature links activation of mesostriatal reward networks with better physical and mental health, implicating reward networks as a relatively novel target for boosting resilience (Dutcher & Creswell, 2018). Briefly, decreased activity in mesostriatal networks has been associated with anhedonia, major depression, and PTSD (Feder, Nestler, & Charney, 2009; Husain & Roiser, 2018). For example, reduced capacity to sustain positive emotion in major depression has been associated with diminished maintenance of mesostriatal activation in response to positive images (Heller et al., 2009). Conversely, in resilient individuals mesolimbic dopamine pathways might be more responsive to reward in the face of trauma (Charney, 2004). For example, special-forces soldiers show greater reactivity to positive events in reward-processing regions than healthy civilian controls (Vythilingam et al., 2009). Indeed, the mesostriatal reward network can directly buffer the effects of stress, as stimulation of the reward network can downregulate amygdala/HPA activity (reviewed in Dutcher & Creswell, 2018). Thus, reward-related striatal activity may buffer against the effect of stress on depressive or PTSD-related symptoms (Admon et al., 2013; Avinun et al., 2017; Corral-Frias et al., 2015; Nikolova, Bogdan, Brigidi, & Hariri, 2012; Tashjian & Galvan, 2018). Similarly, reward-related traits, such as sensitivity to reward, predict stress resilience, and the

relation between reward sensitivity and resilience is mediated by positive affect during or after stress (Corral-Frias, Nadel, Fellous, & Jacobs, 2016). Importantly, experimentally increasing mesolimbic dopamine neuron activity in vulnerable mice can boost immunity (Ben-Shaanan et al., 2018) and have an anti-depressant effect (Friedman et al., 2014). Similarly, deep brain stimulation of the ventral striatum in patients with treatment-resistant or refractory depression has an anti-depressant effect (Bewernick et al., 2010; Dougherty et al., 2015; Schlaepfer et al., 2008). Together these findings highlight the importance of positive affect and the reward pathway in stress resilience and suggest that one way to boost resilience is to employ strategies that boost positive affect and reward pathway activity.

2.4. Default mode network (DMN)

DMN dysfunction has emerged as a new area of interest in mental health research. The DMN is commonly described as a distributed network of regions, including MPFC and posterior cingulate cortex (PCC), that tend to be co-activated when a person is not engaged in tasks that demand external attention (Buckner & DiNicola, 2019; Raichle et al., 2001). These regions in the DMN also get activated during tasks of self-referential thinking, prospection, and social cognition, suggesting that when the mind is not otherwise engaged, its default is to wander, to think about one's past, plan for one's future, and think about others (Buckner & DiNicola, 2019; Christoff, Irving, Fox, Spreng, & Andrews-Hanna, 2016). The DMN may in fact be comprised of multiple different but interwoven networks, but they

share the general role of supporting internally-oriented (i.e., stimulus-independent) thought (Buckner & DiNicola, 2019). While mind-wandering and other self-focused thought is commonly associated with the DMN, this brain network may also play a more fundamental function, such as a commanding role in the large-scale functional organization of the brain's ongoing intrinsic activity (Raichle, 2015). According to one model, hyperactivity in a default network called DN_{CORE}, which includes MPFC and PCC, leads to increased automatic constraints in thought, particularly when functional connectivity between these networks is high, resulting in ruminative and obsessive thinking that characterize mood and anxiety disorders (Christoff et al., 2016). Indeed, hyperactivity in the DMN has been associated with psychiatric illness, including depression and schizophrenia (reviewed in Andrews-Hanna, Smallwood, & Spreng, 2014; Anticevic et al., 2012; Broyd et al., 2009; Whitfield-Gabrieli & Ford, 2012), as well as addiction (DeWitt, Ketcherside, McQueeny, Dunlop, & Filbey, 2015). For example, increased dominance of DMN over the executive network in major depressive disorder has been linked to maladaptive rumination (Hamilton et al., 2011). Furthermore, increased mind wandering (Killingsworth & Gilbert, 2010) and MPFC-related self-referential thinking (Brewer et al., 2011) are associated with unhappiness, while decreased activity in DMN is associated with happiness (Luo, Kong, Qi, You, & Huang, 2016). Although the exact nature of DMN dysfunction in psychiatric disease is still not clear and varies across diseases and even across studies of the same disease, meta-analyses of depression report increased connectivity within DMN (Kaiser, Andrews-Hanna, Wager, & Pizzagalli, 2015), and within MPFC specifically (Iwabuchi et al., 2015; Mulders, van

Eijndhoven, Schene, Beckmann, & Tendolkar, 2015), which in turn seems associated with rumination (Hamilton, Farmer, Fogelman, & Gotlib, 2015; Zhu et al., 2012). Importantly, transcranial stimulation (Liston et al., 2014; Philip et al., 2018) and pharmaceutical (Dutta, McKie, & Deakin, 2014; Posner et al., 2013) interventions that successfully treat depression and PTSD tend to normalize DMN functional pathology. Together these results support the emerging idea that experiences that can lower excessive DMN activity, and promote disengagement from the default self-focused mind-wandering, may help boost resilience (Brewer et al., 2011; Wu, Wang, He, Mao, & Zhang, 2010).

2.5. Role of the PFC in these networks

The PFC is a large and heterogeneous region intricately involved in all the networks discussed so far. Here I focus on MPFC and LPFC. MPFC in this literature typically refers to the ventromedial wall of the PFC (hence also called ventromedial PFC or VMPFC), including Brodmann Areas 25 and 12, medial 10 and 11, and ventral 24 and 32 (Delgado et al., 2016; Roy, Shohamy, & Wager, 2012). A partially overlapping region is the anterior cingulate cortex (ACC), including rostral anterior cingulate (rACC; ventral 24 and 32), associated with induced emotion and emotion regulation, and dorsal anterior cingulate (dACC; dorsal 24 and 32), involved in physical and social distress (Eisenberger, 2015; Etkin, Egner, & Kalisch, 2011). In LPFC, I mainly focus on the association of self-regulation with ventrolateral PFC (VLPFC), including BA 44, 45, 47, lateral 11 and ventrolateral 10. Less discussed is

dorsolateral prefrontal cortex (DLPFC), which is part of the executive network and is involved in such higher cognitive processes as working memory and attention (E. K. Miller & Cohen, 2001). MPFC and LPFC are reciprocally connected to and can exert top-down control over multiple downstream networks in the brain, thus helping to orchestrate and execute goal-directed action (E. K. Miller & Cohen, 2001), including many of the resilience-building strategies reviewed here. Hence, strategies that facilitate function in these prefrontal regions tend to help boost resilience.

2.5.1. Medial prefrontal cortex. (MPFC)

According to recent reviews, three functions most commonly associated with MPFC are 1) interpreting affective information and modulating emotional response (affect regulation), 2) encoding reward and subjective value (reward network), and 3) thinking about oneself and others (default mode network) (Acikalin, Gorgolewski, & Poldrack, 2017; Delgado et al., 2016). The MPFC is well suited to regulate negative and positive affect and support self-reflective thinking, as this region connects systems involved in processing affective sensory cues, self and social cognition, and episodic memory with centers that regulate autonomic and endocrine responses such as the amygdala and hypothalamus (Roy et al., 2012). In this section, I focus primarily on the role of MPFC in regulating distress. As the MPFC sends robust projections to amygdala neurons that can inhibit the fear response (Milad, Rauch, Pitman, & Quirk, 2006), this region is involved in, and in fact necessary for, fear extinction memory in rodents (Do-Monte, Manzano-Nieves, Quinones-Laracuente, Ramos-Medina, & Quirk, 2015) and seems to play a similar role in human fear

extinction (Gottfried & Dolan, 2004; Milad et al., 2005; Phelps, Delgado, Nearing, & LeDoux, 2004). The MPFC can also regulate the HPA axis and stress responses (Eisenberger & Cole, 2012) via direct projections to the hypothalamus (Diorio, Viau, & Meaney, 1993; Radley, Arias, & Sawchenko, 2006). MPFC has been implicated in both down- and up-regulation of affect (Pezawas et al., 2005), including up-regulation of positive affect (S. H. Kim & Hamann, 2007). In studies of stress-related psychopathology, trauma-exposed healthy participants (resilient group) compared to trauma-exposed patients with PTSD show greater activation in MPFC during voluntary regulation of negative affect (New et al., 2009) and increased effective connectivity from MPFC to amygdala (F. Chen et al., 2018). Conversely, hypo-connectivity between MPFC and amygdala is associated with depression (Kaiser et al., 2015) and anxiety (Xu et al., 2019). Similarly, integrity of white fiber tracks that connect MPFC and amygdala moderates an association between self-reported use of cognitive emotion regulation and the experience of anxiety and depressive symptoms (d'Arbeloff et al., 2018).

2.5.2. Lateral prefrontal cortex. (LPFC)

While MPFC is an evolutionarily conserved PFC region involved in emotion regulation, enabling passive regulation of distress such as extinction learning across species, humans also possess evolutionarily newer cortical regions, particularly LPFC, a key component of the central executive network, enabling higher-order cognitive abilities, including such self-regulation strategies as cognitive reappraisal and affect labeling (discussed in detail in Section 4 below). According to meta-

analyses, cognitive regulation such as reappraisal consistently activates LPFC (Buhle et al., 2014; Frank et al., 2014; Klumpp, Bhaumik, Kinney, & Fitzgerald, 2018; Morawetz, Bode, Derntl, & Heekeren, 2017), as it reduces activation in amygdala (Buhle et al., 2014; Frank et al., 2014; Klumpp et al., 2018), a pattern of neural activity associated with successful regulation (Klumpp et al., 2018). This relationship of increased LPFC activation with decreased amygdala activation is sometimes mediated by increased activation in MPFC (Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007; Lieberman et al., 2007), prompting theories that LPFC may enable emotion-regulation by recruiting MPFC, which can then directly regulate the amygdala (Diekhof, Geier, Falkai, & Gruber, 2011; Kohn et al., 2014; Schiller & Delgado, 2010). The LPFC, particularly VLPFC, has been implicated in self-regulation across multiple different domains (Aron, Robbins, & Poldrack, 2014; J. R. Cohen & Lieberman, 2009), including regulation of motor impulses, negative affect, and craving in an overlapping region (Tabibnia et al., 2014; Tabibnia et al., 2011). Given its role in cognitive control and self-regulation, VLPFC deficits in structural integrity or function during self-regulation have been associated with various pathologies of self-regulation, including mood and anxiety disorders (Pico-Perez, Radua, Steward, Menchon, & Soriano-Mas, 2017; Zilverstand et al., 2017), as well as addiction (Aron et al., 2014; Feil et al., 2010; R. Z. Goldstein & Volkow, 2011).

3. A tripartite model of building resilience

To date, no single model of resilience-building can account for the dozens of commonly known seemingly diverse behavioral and psychosocial strategies of resilience-building in adulthood. Historically, psychological models have focused on negative aspects of human experience and functioning, including negative affect, as the root of psychopathology (Vazquez, 2017), along with a focus on reducing the negative rather than promoting the positive (Carl, Soskin, Kerns, & Barlow, 2013; Seligman, 2019). Not until recently has the field made a strong push towards also investigating the role of positive experience and functioning in well-being and resilience (Carl et al., 2013; Diener, 2000; Fredrickson, 2004; Seligman, 2019; Vazquez, 2017), generally recognizing that positive and negative affect are at least partly orthogonal constructs (B. D. Dunn, 2017; Ryff et al., 2006). This push in psychology has paralleled the burgeoning of neural models of resilience, which typically include not only the amygdala and HPA distress networks but also the mesostriatal reward network as important components in the pathway to resilience (Dutcher & Creswell, 2018; Feder et al., 2009; Franklin, Saab, & Mansuy, 2012; Kalisch, Muller, & Tuscher, 2015; Russo et al., 2012; Southwick & Charney, 2012; van der Werff et al., 2013). For example, clinical depression is marked by an abundance of negative affect and associated abnormalities in the amygdala fear circuitry, as well as by a paucity of positive affect and associated deficits in the brain's dopaminergic reward circuitry (Charney, 2004). Thus, a comprehensive approach to building resilience should tackle both distress-reducing and positivity-boosting pathways. While the distress and reward networks both play critical roles in resilience, in light of recent findings reviewed above, I suggest that DMN also be

included as a key network in resilience-building. I demonstrate here that a single model including these three pathways (distress networks, reward networks, and default network) can not only account for the more than two-dozen psychosocial strategies commonly known to boost resilience, but it can also make predictions about potential novel strategies.

3.1. Description of the Tripartite Model

According to the tripartite model of resilience-building (Fig. 1A), the three distinct general routes to resilience are 1) down-regulating the negative, 2) up-regulating the positive, and 3) transcending the self. First are strategies that directly target and down-regulate negative affective states (e.g., exposure and cognitive reappraisal) by reducing distress-related responses of the amygdala and HPA axis. The second route to resilience encompasses strategies that up-regulate positive affective states (e.g., optimism and social connectedness) by activating mesostriatal reward networks, which in turn can buffer the effects of stress. The emerging third route to resilience encompasses strategies that promote an experience of self-transcendence (e.g., mindfulness, religious engagement) and reduce activation in the DMN.

Top-down voluntary or implicit control over each of these three pathways can be exerted via LPFC and MPFC regions that support attention, self-regulation, and other executive function (Fig. 1B). Thus, strategies that recruit or improve PFC executive function can also impact resilience. These strategies are outside the scope

of the current review, but an example is psychological flexibility (Kashdan & Rottenberg, 2010). Also known as coping flexibility (Cheng, Lau, & Chan, 2014) or regulatory flexibility (Koch, Mars, Toni, & Roelofs, 2018), and related to cognitive flexibility (Dajani & Uddin, 2015), psychological flexibility is the ability to recognize which strategy to use or not use and the flexibility to select and implement, and even switch to, a strategy that fits changing situational demands. Another example of top-down resilience building approach is "brain training" or cognitive training, programs designed to directly boost PFC function, reviewed elsewhere (e.g., Simons et al., 2016). Importantly, as can be seen in Fig. 1B, according to the tripartite model, even strategies that target PFC executive function are hypothesized to have their impact on resilience via one of the 3 proximal pathways to resilience.

3.2. Nuances of the Tripartite Model

Although the three pathways of the tripartite model are distinct, they are not mutually exclusive, they overlap, and they can influence one another. Some strategies can boost resilience via more than one pathway. For example, while social connectedness boosts positive affect and involves neural pathways associated with reward, it also reduces negative states such as loneliness and activation in distress networks. In fact, as I review in Section 5 below, positive affect bolsters resilience in part because it helps build resources for coping with future adversity (Fredrickson, 2013). The pathways overlap at the neural level as well. While the MPFC plays a role in all 3 network, the amygdala too is not only an important component of the

distress network, but it has also been implicated in the reward network (Baxter & Murray, 2002; Cardinal, Parkinson, Hall, & Everitt, 2002; Janak & Tye, 2015) and has been considered part of the DMN by some (Sheline et al., 2009). Importantly, each region within a network is anatomically connected to multiple other regions within and outside the network, allowing networks to influence one another. For example, activating the reward network can reduce activity in the distress networks (Dutcher & Creswell, 2018).

It is important to note that the tripartite model promotes practices that reduce *over*-activation in distress networks or DMN or increase *under*-activation in reward networks. The model does not propose that activation in distress networks or DMN be eliminated entirely, as these networks evolved to serve adaptive functions and in fact their activation may boost resilience in some cases. For example, activating the fear network during exposure has been associated with successful extinction (Barad, Gean, & Lutz, 2006; Foa & Kozak, 1986), and DMN-related self-reflection can lead to positive outcomes like enhanced meaning in life (Waytz, Hershfield, & Tamir, 2015). Nor does the model propose that the more activation in the reward network the better, as countless addiction studies have demonstrated the devastating consequences of intense stimulation of the dopamine reward pathway (Di Chiara & Bassareo, 2007; Volkow & Morales, 2015). More likely, optimal functioning is associated with proper balance within (e.g., Grant & Schwartz, 2011) and between (e.g., Raichle, 2015) systems, including timely activation and recovery, such as in other networks in the brain (e.g., Arnsten, 2009) and body more generally (i.e., homeostasis) (McEwen & Gianaros, 2011). As reviewed in Section 2

above, imbalances typically associated with psychopathology and poor resilience tend to go in the direction of hyperactivity in distress networks and DMN and hypoactivity activity in reward networks. Hence, the tripartite model is a theory of how to counteract these imbalances that are typically associated with low resilience.

To support the tripartite model, in the sections that follow, I systematically review over two-dozen behavioral and psychosocial strategies that boost resilience and activate at least one of the 3 pathways in the tripartite model. This review is summarized in Table 1 and depicted in Fig. 1A.

4. Down-Regulating the Negative

Several different strategies can be used to counteract or directly cope with a threat or stressor. Coping refers to the behavioral and cognitive responses made in order to manage perceived threat. In general, behavioral coping strategies, such as those that involve some form of physical engagement with the environment (e.g., fight or flight), tend to be adaptive when the stressor is controllable or changeable, while cognitive coping strategies, such as reappraisal, tend to be adaptive when the stressor is not controllable.

4.1. Behavioral coping and taking control

Behavioral coping is protective against distress and psychopathology

(Hobfoll et al., 2007; Moos & Schaefer, 1993). The benefits and limitations of behavioral coping have been extensively discussed under various headings, including "situation selection" and "situation modification" (Gross, 2015), "proactive coping" (Koolhaas et al., 1999), "problem-focused coping" (Lazarus & Folkman, 1984), and "primary control" (Heckhausen, Wrosch, & Schulz, 2010; Rothbaum, Weisz, & Snyder, 1982). Here I use the broad term of behavioral strategies to refer to exposure and reconsolidation, active coping strategies like active avoidance and controlling the stressor, and the proactive strategy of stress inoculation.

4.1.1. Exposure and reconsolidation.

To the extent that a fear is irrational, the classic method of overcoming the fear is through repeated exposure in a safe environment until the fear is extinguished, a technique that has been extensively researched in animals and humans for decades (Asnaani, McLean, & Foa, 2016). Exposure therapy is well-established, effective, and an important aspect of most interventions for anxiety disorders (reviewed in Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014; Foa & McLean, 2016). Animal models indicate that extinction training does not erase the original fear memory but rather leads to formation of a new amygdala-based memory, associating the formerly feared stimulus with safety (reviewed in Furini, Myskiw, & Izquierdo, 2014). This new memory is consolidated and stored in MPFC, and it can inhibit the expression of the original fear response (reviewed in Milad & Quirk, 2012; Sotres-Bayon, Cain, & LeDoux, 2006; also see Section 2.5.1. above). Human neuroimaging (reviewed in Fullana et al., 2018) and transcranial stimulation

studies (Herrmann et al., 2017; Raij et al., 2017) also implicate a critical role of MPFC, as well as LPFC, during extinction learning, along with reduction in amygdala fear-network reactivity following extinction (reviewed in Hartley & Phelps, 2010; Sehlmeyer et al., 2009) and exposure therapy (Goossens, Sunaert, Peeters, Griez, & Schruers, 2007; Hauner, Mineka, Voss, & Paller, 2012). However, because the original fear memory is still present, the fear can return after successful extinction (e.g., spontaneously or in contexts that differ from the extinction context).

An emerging alternative intervention that can potentially weaken the fear memory itself, and hence reduce fear recovery, is reconsolidation interference. When a memory is recalled, it is rendered labile and can therefore be altered, or reconsolidated. Thus, fear memories can be altered if extinction training occurs following the mere act of retrieving the memory, when the memory has been rendered labile. Multiple animal and human studies have shown lasting benefits of memory reactivation prior to extinction training (reviewed in Baldi & Bucherelli, 2015; Beckers & Kindt, 2017; Kredlow, Unger, & Otto, 2016), resulting in less fear recovery, lower amygdala activation, and weakened fear circuit connectivity in the amygdala (e.g., Agren et al., 2012; Bjorkstrand et al., 2017; Schiller, Kanen, LeDoux, Monfils, & Phelps, 2013), although these results have not been consistently replicated, possibly due to "boundary conditions" that remain to be examined (Treanor, Brown, Rissman, & Craske, 2017). For a full discussion of the strengths and limitations of reconsolidation interference, see Beckers and Kindt (2017).

4.1.2. Active coping.

While exposure is relatively passive, also effective are more active forms of coping – i.e., engaging in actions that reduce negative emotional outcome (or increase positive emotional outcome, discussed in Section 5 below). Active coping can buffer stress (Bowen et al., 2014; Y. Ono et al., 2012) and is associated with better psychological and physical health, including lower vulnerability to depression and anxiety, as well as better pain management (Emmert et al., 2017; Koolhaas et al., 1999; Russo et al., 2012). For example, adult rats allowed to actively cope with an immobilization stress by chewing on a stick show reduced HPA activation compared to immobilized rats without access to the stick (Y. Ono et al., 2012). Similarly, chronic-pain patients who use active coping strategies, such as doing household chores, report lower psychological distress (Snow-Turek, Norris, & Tan, 1996).

4.1.2.1. Active avoidance. One paradigm for studying the neural basis of active coping is active avoidance, physically moving away from a predictor of stress. Animal models indicate that active avoidance disrupts fear-learning circuitry by directly changing connections in the amygdala, rerouting the amygdala output away from brainstem targets that promote passive coping such as freezing and towards a cortico-striatal pathway that integrates motivation and action (reviewed in LeDoux & Gorman, 2001; LeDoux, Moscarello, Sears, & Campese, 2017), reducing passive fear responding not only in the short- but also in the long-term (Cain & LeDoux, 2007). Active avoidance also recruits MPFC to suppress amygdala-based passive fear responding both during learning and in subsequent tests, even in novel environments, suggesting that the learned active coping can generalize across different environments (Moscarello & LeDoux, 2013). A similar neural pathway

underlies this type of active coping in humans, including reduced amygdala and autonomic reactivity following avoidance learning (Delgado, Jou, LeDoux, & Phelps, 2009). Thus, active coping can strengthen the neural pathway for active coping and hence reduce future anxiety or negative affect.

4.1.2.2. Controlling the stressor. Another method of active coping is controlling the stressor itself, when possible. Laboratory rats that can terminate a shock by turning a wheel, compared to rats that experience identical ("yoked") shocks but without control, show plasticity in MPFC. Specifically, exercising control activates MPFC, and this activation of MPFC in the presence of shock seemingly strengthens connections between MPFC and distressing stimuli, such that future distressing stimuli, even if they are uncontrollable, come to activate the MPFC and its downstream inhibitory connections, leading to a reduction of amygdala and brainstem stress responses and a reduction of subsequent depression-like behavior (Maier, 2015). Given that the shock is identical and equally stress-inducing in both groups, it is the exercise of control, rather than differences in shock, that leads to neural changes and facilitated coping. A similar mechanism seems to underlie this controllability-induced resilience in humans. Specifically, Hartley et al. (2014) found that actively escaping uncomfortable shocks by performing a computer task facilitates fear extinction learning a week later and prevents spontaneous return of fear, as measured by autonomic arousal. Furthermore, they found a strong correlation between self-reported perception of control over the shocks and the reduction in later fear, consistent with the notion that experiencing control strengthens the pathway for regulating future distress. Similarly, individuals with a

phobia of snakes exhibit greater MPFC activity during anticipation of snake videos when they have control over whether the videos are presented compared to when they do not have control; and this MPFC activation is negatively correlated with amygdala activation (D. L. Kerr, McLaren, Mathy, & Nitschke, 2012), consistent with Maier's model (2015) of controllability-induced MPFC inhibition of limbic fear response. Indeed, this controllability-induced resilience generalizes across different stressors, suggesting a potential pathway to preventative intervention, such as stress inoculation.

4.1.3. Stress inoculation (proactive coping).

Although exposure to trauma can render a person vulnerable to psychopathology, experiencing moderate stress can buffer against future stress (R. T. Liu, 2015; Seery, 2011) (Shapero et al., 2015). For example, in a large longitudinal study, people with a history of *some* traumatic experience reported lower distress and greater life satisfaction compared to people with a history of *high* or *no* trauma (Seery, Holman, & Silver, 2010). Since Levine (1957) serendipitously observed that briefly separating rat pups from their mother improves the pups' capacity to handle stressors later in life, it has been suggested that experiencing moderate stress has an effect of "steeling" (Rutter, 1981) or "toughening" (Dienstbier, 1989) the individual against future stressors. Consistent with this notion, Stress Inoculation Training (SIT) (Meichenbaum, 1977) has been used to successfully treat and prevent relapse of depression and anxiety disorders (reviewed in Meichenbaum, 2017), and it can reduce state anxiety and enhance performance under stress (reviewed in Saunders,

Driskell, Johnston, & Salas, 1996). Stress inoculation produces psychological and neurobiological changes, including in MPFC and HPA pathways that facilitate better management of subsequent stress (reviewed in Dienstbier, 1989; Levine & Mody, 2003; Maier, 2015; Parker & Maestripieri, 2011; Russo et al., 2012). In rodents, exposure to mild stress reduces subsequent anxiety-like behavior and plasma glucocorticoid stress response (Brockhurst, Cheleuitte-Nieves, Buckmaster, Schatzberg, & Lyons, 2015); and a functioning MPFC at the time of the initial stress exposure is necessary for the subsequent "behavioral immunization" (Amat, Paul, Zarza, Watkins, & Maier, 2006). Similarly, in squirrel monkeys, early exposure to mild separation stress leads to fewer signs of anxiety, better prefrontal-related cognitive control, and diminished plasma cortisol and ACTH stress response (Parker, Buckmaster, Schatzberg, & Lyons, 2004) and increases myelination and volume in MPFC (Katz et al., 2009) compared to non-inoculated peers. Even adult monkeys exposed to manageable stress can build resilience, as indicated by prevention of stress-induced anhedonia, reduced HPA reactivity, and increased glucocorticoid receptor gene expression in MPFC (A. G. Lee, Buckmaster, Yi, Schatzberg, & Lyons, 2014). Similarly, human neuroimaging studies indicate that such mild and phasic parental separation stress buffers HPA activity and promotes connectivity between MPFC and amygdala, resulting in reduced amygdala reactivity later in life (reviewed in Tottenham, 2015). Further supporting a role of stress-induced MPFC plasticity in resilience in humans, fMRI study participants whose MPFC showed adaptation during sustained stress reported greater active coping and lower maladaptive lifestyle behaviors such as alcohol intake (Sinha, Lacadie,

Constable, & Seo, 2016).

4.2. Cognitive coping and emotion regulation

Bearing in mind that physically confronting or changing a stressor is not always possible or sufficient, a complementary coping strategy is to change or regulate the way one attends to, interprets, or emotionally responds to the stressor. These types of cognitive strategies are commonly referred to as emotion regulation (Gross, 2015). Highlighting its role in resilience, emotion dysregulation is a core underlying process common across different psychopathologies (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Berking & Wupperman, 2012). Consistent with this transdiagnostic conceptualization of emotion regulation, maladaptive emotion regulation strategy use and overall emotion dysregulation decrease following effective psychological treatment across different disorders, as do symptoms of anxiety, depression, substance use, and eating pathology (Sloan et al., 2017). In this section, after I review cognitive behavioral therapy (CBT), a common and effective therapy for many mental health disorders, I discuss specific strategies that can be implemented during psychological therapy or in daily life. First I discuss strategies that involve some form of explicit acknowledgment of or mental confrontation with the negative affect or experience, namely acceptance, labeling, and disclosure. Then I review strategies that involve further cognitive processing, namely cognitive reappraisal and self-efficacy.

4.2.1. Cognitive behavioral therapy (CBT)

CBT is a structured psychotherapy that utilizes cognitive and behavioral strategies to change maladaptive tendencies that maintain psychiatric disorder (Clark & Beck, 2010). CBT is an effective treatment for depression (Cuijpers et al., 2013) and anxiety (Hofmann & Smits, 2008), often considered first-line treatment (Craske et al., 2017; Tolin, 2010) (cf Baardseth et al., 2013). CBT may be as effective as antidepressant medication in treating depression and anxiety in the short term, and possibly more effective in the long term (Clark & Beck, 2010; DeRubeis, Siegle, & Hollon, 2008; cf Johnsen & Friborg, 2015). A review of 10 neuroimaging studies has shown that CBT for various anxiety disorders reduces abnormal limbic reactivity in treatment responders (Porto et al., 2009). For example, CBT for phobia reduces reactivity in the amygdala and other limbic regions, and the degree of amygdala-limbic attenuation predicts symptom reduction (Furmark et al., 2002; Lipka, Hoffmann, Miltner, & Straube, 2014). Similarly, cognitive therapy for depression and anxiety generally reduces amygdala activation and enhances prefrontal function (Clark & Beck, 2010; DeRubeis et al., 2008). CBT can also increase LPFC gray matter volume, which correlates with improved performance on executive control tasks (de Lange et al., 2008). Furthermore, CBT can increase amygdala connectivity with LPFC-control network in both depression and PTSD (Shou et al., 2017).

4.2.2. Confrontation (vs. avoidance) strategies.

Although individual difference and situational factors moderate the

effectiveness of a given emotion regulation strategy (Gross, 2015; Segerstrom & Smith, 2019), in general explicit identification and expression of a distressing experience tends to be more adaptive than denial or suppression of it (Gross, 2002; Mund & Mitte, 2012; Webb, Miles, & Sheeran, 2012). This is consistent with behavioral coping strategies for anxiety disorders, such as exposure therapy, that emphasize fully confronting the feared situation and fully "processing", or working through, the negative experience (Foa & Kozak, 1986; Rachman, 1980). By writing or talking about an emotional experience, one is effectively confronting it and being exposed to it cognitively (Niles, Byrne Haltom, Lieberman, Hur, & Stanton, 2016).

On the other hand, avoidant strategies, though sometimes adaptive (Tabibnia & Radecki, 2018; Wortman & Silver, 2001), can often have negative emotional, cognitive, and social consequences. For example, inhibiting the outward expression of one's emotions, or emotion suppression, not only fails to decrease self-reported emotional experience, but it impairs memory and increases physiological arousal in both suppressors and their social partners (Gross, 2002). Furthermore, people who engage in avoidant coping strategies, such as denial and disengagement, tend to have lower hardiness (Maddi & Hightower, 1999) and are at greater risk for cancer and cardiovascular disease (Mund & Mitte, 2012).

4.2.2.1. Affect labeling. The simplest way to explicitly acknowledge an emotion is to put it into words. Merely labeling an emotional experience with a single word, or affect labeling, can reduce self-reported, autonomic, and amygdalar emotional arousal (Constantinou, Van Den Houte, Bogaerts, Van Diest, & Van den Bergh, 2014; Hariri, Mattay, Tessitore, Fera, & Weinberger, 2003; Lieberman,

Inagaki, Tabibnia, & Crockett, 2011). Affect labeling can also have lasting benefits, such as reducing autonomic reactivity to provocative cues a week after labeling (Tabibnia, Lieberman, & Craske, 2008) and enhancing outcome of exposure therapy (Kircanski, Lieberman, & Craske, 2012) (Niles, Craske, Lieberman, & Hur, 2015). Using emotion words (such as "afraid") to process evocative cues (such as a photo of a fearful face) activates LPFC, which in turn reduces amygdala activation (Lieberman et al., 2007; Torrisi, Lieberman, Bookheimer, & Altshuler, 2013). Because affective labels recruit LPFC regions that can downregulate the amygdala, affect labeling can dampen emotions even when people do not believe that affective labeling can be an effective emotion regulation strategy (Lieberman et al., 2011). Supporting the notion that affective labels can dampen emotions even in the absence of an explicit intention to dampen them, a recent meta-analysis of 386 neuroimaging studies of emotions shows that when emotion words (e.g., "anger," "disgust") are present in the experimental task, there is less activation in bilateral amygdala than when emotion words are not present (Brooks et al., 2017).

4.2.2.2. Emotion disclosure. Another language-based coping strategy is emotion disclosure, sometimes referred to as expressive writing. Verbal disclosure of traumatic experience, such as simply writing about the experience for 20 minutes, can improve physical and psychological well-being in the long-term (reviewed in Frattaroli, 2006; Hemenover, 2003; Pennebaker, 1997), including psychological health benefits among individuals with major depression (Krpan et al., 2013) and among caregivers (reviewed in J. P. Riddle, Smith, & Jones, 2016). The mechanisms underlying the benefits of disclosure are complex and varied, but they include

promotion of self-efficacy (Hemenover, 2003) and self-distancing (Park, Ayduk, & Kross, 2016) (see Sections 4.2.3. and 4.2.4. below). Although neuroimaging studies of expressive writing are challenging and still limited, current work suggests a neural mechanism that resembles that of affect labeling and reconsolidation (see Sections 4.1.1 and 4.2.2.1 above). Verbally re-experiencing past trauma allows reprocessing of the traumatic memory, thus allowing the formerly implicit amygdala-based memory to be re-encoded in explicit neocortex-based declarative memory, where it can be intentionally accessed and hence more easily regulated in the future (Brewin, 2001; Careaga, Girardi, & Suchecki, 2016). Thus, as with affect labeling, the idea is that words can help transfer emotion processing out of the reactive limbic system, where the default outcome is activation of distress circuits, and into the neocortex where control can be exerted. Consistent with this framework, self-reported tendency to self-disclose is correlated with gray matter volume in LPFC (Wang et al., 2014) and activity in LPFC and amygdala during affect labeling predicts disclosure effects on physical symptoms, depression, anxiety, and life satisfaction (Memarian, Torre, Haltom, Stanton, & Lieberman, 2017). Further supporting the beneficial impact of verbal disclosure on distress networks, writing a narrative of their marital separation improved newly separated adults' ANS function several months later (Bourassa, Allen, Mehl, & Sbarra, 2017).

4.2.2.3. Emotion acceptance. The opposite of experiential avoidance, acceptance has been described as a form of cognitive "exposure" and refers to the willingness to experience all emotions, physical sensations, and thoughts, even if they are negative (Hayes, 2004). Because it acts in the early stages of emotion

regulation (Dan-Glauser & Gross, 2015), acceptance can reduce ongoing emotional reactivity and the need for further regulation (Lindsay & Creswell, 2017). Compared to avoidance, acceptance predicts better mental health outcomes when faced with adversity, such as breast cancer (Carver et al., 1993), one's child undergoing bone marrow transplant (Manne et al., 2003), and a terrorist attack (Silver, Holman, McIntosh, Poulin, & Gil-Rivas, 2002). In randomized trials, acceptance and commitment therapy (ACT) is found to be as effective as cognitive therapies for treating anxiety and depressive disorders, and self-reported acceptance underlies reductions in symptom severity (Arch et al., 2012; Forman, Herbert, Moitra, Yeomans, & Geller, 2007). A meta-analysis of 30 experimental comparisons between acceptance and other emotion regulation strategies (e.g. suppression, distraction, reappraisal) found that acceptance is superior in boosting pain tolerance and at least as effective in reducing subjective pain intensity and negative affect (Kohl, Rief, & Glombiewski, 2012). The neurobiological underpinnings of emotion acceptance are still unclear, but existing studies point to involvement of an LPFC-amygdala network. In samples of remitted major depressive disorder and healthy control participants, acceptance versus passive viewing of sad images reduced self-reported negative affect and activated LPFC (Smoski et al., 2015). In another study, individuals with generalized anxiety disorder showed greater LPFC-amygdala functional connectivity during acceptance of personally relevant worry-inducing statements compared to either suppression of or worry about those statements (Ellard, Barlow, Whitfield-Gabrieli, Gabrieli, & Deckersbach, 2017). Similarly, greater inverse connectivity between LPFC and amygdala during affect labeling, an

emotion regulation strategy akin to acceptance, both predicts (K. S. Young et al., 2019) and follows (K. S. Young et al., 2017) greater symptom reduction following psychological therapy, including ACT, for social anxiety.

4.2.3. Cognitive reappraisal (incl. distancing).

Cognitive reappraisal refers to the reframing of a situation in order to alter its emotional impact (Gross, 2002). Most typical reappraisal tactics are *reinterpretation* and *distancing*. Reinterpretation involves construing an alternative outcome or meaning for a situation (e.g., reinterpreting being stuck in traffic as an opportunity to listen to a podcast), while distancing, aka "perspective taking", involves construing a perspective that increases psychological distance (e.g., appraising the situation as if it had happened to someone else) (Powers & LaBar, 2019). Meta-analyses of the effectiveness of various emotion regulation strategies identify cognitive reappraisal, particularly distancing, as one of the most effective (Augustine & Hemenover, 2009; Webb et al., 2012). (In the review by Webb and colleagues (2012), emotion acceptance was construed as a form of reappraisal, which, along with distancing, was among the most effective strategies.) People who report frequently using reappraisal exhibit better psychological health, while laboratory participants instructed to use cognitive reappraisal during a negative experience report less negative emotion and show lower autonomic arousal (reviewed in John & Gross, 2004). Cognitive reappraisal is an important component of well-established and effective therapies for affective and anxiety disorders, and it is associated with greater well-being in the face of stress (reviewed in Southwick &

Charney, 2012; Troy & Mauss, 2011).

A review of 48 neuroimaging studies reports that cognitive reappraisal reduces activation in the amygdala and increases it in LPFC regions associated with cognitive control, among other regions (Buhle et al., 2014), a neural pattern that has been associated with successful reappraisal (Klumpp et al., 2018). Functional (Morawetz, Bode, Baudewig, & Heekeren, 2017) and anatomical (d'Arbeloff et al., 2018) connectivity between LPFC and amygdala also play important roles in reappraisal. Supporting a causal role of LPFC in reappraisal success, transcranial stimulation of LPFC during reappraisal of negative images alters autonomic response and reduces self-reported emotional reactivity (Feeser, Prehn, Kazzer, Mungee, & Bajbouj, 2014; Marques, Morello, & Boggio, 2018). Reappraisal may also have long-term effects; repeatedly reappraising an aversive image reduces amygdala response to that image a week later compared to repeated exposure without reappraisal (Denny, Inhoff, Zerubavel, Davachi, & Ochsner, 2015). Neuroimaging studies targeting distancing specifically also generally show decreased amygdala and increased LPFC activation (reviewed in Powers & LaBar, 2019), some also showing reduced activation in MPFC, potentially explained by this region's involvement in rumination (Kross, Davidson, Weber, & Ochsner, 2009; Leitner et al., 2017; Moser et al., 2017).

4.2.4. Self-efficacy and perception of control.

Not only do appraisals of events affect emotional response and ultimately resilience, so do self-appraisals or beliefs about the self. In particular, self-efficacy,

the belief in one's ability to master life's challenges, is protective against psychiatric symptoms, including depression and posttraumatic stress, and facilitates recovery from a wide range of traumas (Benight & Bandura, 2004; Blackburn & Owens, 2015; Schwarzer & Warner, 2013). Self-efficacy and perceived control can also facilitate smoking cessation (Schnoll et al., 2011; Schuck, Otten, Kleinjan, Bricker, & Engels, 2014), reduce cigarette craving (M. Ono et al., 2017), mediate the effect of therapy on social anxiety disorder (Goldin et al., 2012), and improve fear extinction (Zlomuzica, Preusser, Schneider, & Margraf, 2015) and emotion regulation (Morina et al., 2018). Importantly, perception of control even in the absence of true control can be beneficial, such as by reducing the perception of pain (Bowers, 1968; Mackie, Coda, & Hill, 1991). Uncontrollable pain that is *perceived* as "controllable" results in reduced activation in the pain network, including the ACC, insula, and secondary somatosensory cortex (Salomons, Johnstone, Backonja, & Davidson, 2004). And this controllability-induced reduction in pain is driven by increased activation in LPFC (Brascher, Becker, Hoeppli, & Schweinhardt, 2016; Wiech et al., 2006).

Neuroimaging studies of self-efficacy also implicate a role of PFC. Consistent with the critical role of MPFC in active coping (see Section 4.1.2 above), self-reported self-efficacy and a larger MPFC are correlated with one another and with lower levels of psychopathology in women (Holz et al., 2016). Additionally, boosting self-efficacy alters resting state connectivity in MPFC and LPFC (Titcombe-Parekh et al., 2018), and regulating craving with a self-efficacy strategy activates MPFC and alters MPFC connectivity (M. Ono et al., 2017). Furthermore, reappraising negative self-beliefs decreases amygdala reactivity and increases PFC-amygdala connectivity

(Goldin, Manber-Ball, Werner, Heimberg, & Gross, 2009; Goldin et al., 2013).

4.2.5. Cognitive bias modification (CBM).

Negativity bias, or the tendency to attend to negative cues and interpret ambiguity in negative ways, can be modified with training, in turn reducing depression and anxiety symptoms (Hertel & Mathews, 2011). This training, broadly referred to as cognitive bias modification (CBM), involves repeated trials on computerized tasks originally developed to measure cognitive bias, such as the dot probe task which can both measure and manipulate negativity bias in attention (Hallion & Ruscio, 2011). Despite some inconsistencies in the literature, recent reviews suggest that CBM for interpretation may reduce depression symptoms (Koster & Hoorelbeke, 2015), while attention bias modification (ABM) may reduce anxiety symptoms (reviewed in Jones & Sharpe, 2017; Mogg, Waters, & Bradley, 2017). Due to the smaller literature on CBM for interpretation relative to ABM, here I focus on ABM. Neuroimaging studies of ABM have shown that training with modified dot probe tasks increases LPFC activation (Browning, Holmes, Murphy, Goodwin, & Harmer, 2010; Taylor et al., 2014) and decreases amygdala activation to affective stimuli (Hiland et al., 2019; Taylor et al., 2014) (cf Britton et al., 2015). Supporting a causal role of LPFC in ABM, anxious (Clarke, Browning, Hammond, Notebaert, & MacLeod, 2014; Heeren et al., 2017) and healthy (Ironside, O'Shea, Cowen, & Harmer, 2016) participants receiving transcranial stimulation over LPFC before or during training subsequently show greater ABM than participants receiving sham stimulation. In fact, transcranial stimulation over LPFC, when

repeated over several sessions, has been touted as a potentially useful therapeutic tool for reducing psychiatric symptoms (Kekic, Boysen, Campbell, & Schmidt, 2016), and ABM may mediate the positive effect of transcranial stimulation on emotional resilience (N. T. M. Chen, Basanovic, Notebaert, MacLeod, & Clarke, 2017).

4.3. Summary of strategies that down-regulate the negative.

The strategies reviewed in this section all show evidence of recruiting the distress-reduction pathway. Specifically, each strategy reduces the distress response of amygdala, HPA, and/or ANS. Many of the strategies (e.g., exposure, active coping, stress inoculation, and CBT) can also lead to neuroplasticity along the PFC-amygdala emotion-regulation pathway (Table 1).

5. Up-Regulating the Positive

In this section, I review strategies that have been shown to increase positive affect and activate mesostriatal reward pathways. The benefits of positive affect, ranging from longevity to better physical and psychological well-being, have been known for some time (S. Cohen & Pressman, 2006; Lyubomirsky, King, & Diener, 2005; Scheier & Carver, 1993; Steptoe, 2019) and are the focus of a subfield of psychology known as positive psychology (Seligman, 2019). Conversely, paucity of positive affect is a risk factor for psychiatric disease (Keyes, Dhingra, & Simoes,

2010). Similarly, excessively denying oneself life's indulgences and pleasures, such as foregoing vacation or a night out in favor of working, can hinder one's emotional, physical, and even economical well-being (Kivetz, Meng, & He, 2017). Thus, psychological interventions that incorporate positive psychology (reviewed in Bolier et al., 2013; in Seligman, 2019; and in Sin & Lyubomirsky, 2009), such as "well-being therapy" (Fava, 1999; Ryff, 2014) and "positive psychotherapy" (Seligman, 2019), can be more effective at preventing mental and physical disease and their relapse compared to interventions without positivity training.

Positive emotions can not only provide a respite from ongoing stress but they can also facilitate adaptation to future stress. In her broaden-and-build model, Fredrickson has argued that positive emotions broaden the individual's attentional focus (see Isen, 2008) and behavioral repertoire, prompting the individual to explore and experiment with novel or creative coping strategies, thus building over time an arsenal of physical, psychological, and social resources that can later be drawn upon for successful coping with a stressor (Fredrickson, 2004; cf Gable & Harmon-Jones, 2008). Examples of resources that positive emotions can help build include self-efficacy beliefs (see Section 4.2.4 above and Section 5.1.4 below), social support (see Sections 5.1.1 and 5.3 below), and vagal tone (Fredrickson, 2013). Positive affect can also facilitate learning, including fear extinction learning (Meulders, Meulders, & Vlaeyen, 2014; Zbozinek & Craske, 2018). Thus, an upward spiral of positivity can occur whereby momentary positive affect leads to broadened attention which can facilitate coping with adversity and build resources that promote future well-being and resilience (Fredrickson & Joiner, 2002).

There are numerous ways to boost positive emotions and well-being. The Blue Zone investigations, studies of geographic regions with the greatest longevity, consistently identify several lifestyle factors associated with longevity, including physical exercise, engagement in social and family life, and moderate caloric intake, among other factors (Buettner, 2012). Psychological exercises, such as writing letters of gratitude or practicing optimism, can also boost positive affect and resilience (Layous, Chancellor, Lyubomirsky, Wang, & Doraiswamy, 2011; Lyubomirsky, Sheldon, & Schkade, 2005). Below I separately review a number of different psychological, physical, and social practices that can activate reward pathways and boost positive affect and resilience.

5.1. Psychological strategies (positivity)

Resilient individuals are characterized by high positive emotionality and dispositional optimism (Carver & Scheier, 2014; S. Cohen & Pressman, 2006) and demonstrate more adaptive responding in the mesolimbic reward pathway during stress (Krishnan et al., 2007). These positivity-related characteristics are not immutable and can be cultivated, as reviewed below.

5.1.1. Optimism

Across numerous studies, optimism, or the tendency to expect favorable outcomes, has been associated with better psychological well-being during times of stress (even after controlling for previous well-being), along with increased success

in one's educational, economic, and social endeavors (reviewed in Carver & Scheier, 2014), as well as greater longevity (L. O. Lee et al., 2019) and physical health (reviewed in Scheier & Carver, 2018). These effects are not merely correlational. Learning to have a more optimistic explanatory style—attributing negative life events to forces that are temporary and confined—promotes resilience (Seligman et al., 1988), and incorporating optimism training, such as in the Penn Resilience Program, has proven particularly effective in treating and preventing depression (Reivich, Gillham, Chaplin, & Seligman, 2013; Seligman, Steen, Park, & Peterson, 2005). Even engaging in a simple exercise of imagining one's best possible self can increase optimism and subjective well-being, at least temporarily (L. A. King, 2001; Meevissen, Peters, & Alberts, 2011), and subsequently decrease pain perception (Hanssen, Peters, Vlaeyen, Meevissen, & Vancleef, 2013) and reduce the number of illness-related health center visits 5 months later (L. A. King, 2001). However, optimism does not always enhance resilience (Segerstrom, 2005), and unmitigated unrealistic optimism can even be harmful, such as in gambling. Thus, an adaptive outlook may be "mostly optimistic, tempered with small doses of realistic pessimism when needed" (Forgeard & Seligman, 2012).

Our understanding of the neural basis of optimism comes from a few disparate lines of research. Extensive animal studies have demonstrated that expectation of a positive outcome (i.e., the definition of optimism), such as anticipation of food reward, activates dopamine neurons in the mesostriatal reward pathway (Schultz et al., 1997), a finding paralleled by human neuroimaging studies (e.g., Knutson, Adams, Fong, & Hommer, 2001). Further supporting a role of

dopamine in optimism, Sharot et al. (2012) have shown that administration of the dopamine precursor L-DOPA increases an optimism bias. Similarly, mesostriatal reward networks, including regions such as MPFC and ventral striatum, have been implicated in the generation of optimistically biased self-related belief changes (e.g., lowering one's estimate of getting cancer) (Kuzmanovic, Jefferson, & Vogeley, 2016), as well as in the tendency to maintain optimistic self-appraisals (Yamada et al., 2013), even in the face of social evaluative threat (Flagan & Beer, 2013). Furthermore, several studies have shown that imagining future personally rewarding events also activate reward-related regions such as MPFC and ventral striatum (Benoit, Szpunar, & Schacter, 2014; D'Argembeau, Xue, Lu, Van der Linden, & Bechara, 2008; Gerlach, Spreng, Madore, & Schacter, 2014). Finally, a recent structural MRI study found that the only brain region whose gray matter associated with dispositional optimism is the putamen, a reward-related region of the striatum (Lai, Wang, Zhao, Qiu, & Gong, 2019).

5.1.2. Smiling

Consistent with Darwin's claim that the strength of emotional experience can be modulated by intensifying or inhibiting the expression of the emotion (Darwin, Ekman, & Prodger, 1998), the "facial feedback hypothesis" posits that facial expressions can affect emotional experience, even when people are not aware of making the expression (Damasio, Everitt, & Bishop, 1996; Tourangeau & Ellsworth, 1979). Numerous studies have reported covariance between smiling and positive affective experience (reviewed in Fernández-Dols & Crivelli, 2013; Reisenzein,

Studtmann, & Horstmann, 2013). Similarly, intensity of smiling in photographs predict life satisfaction (Seder & Oishi, 2011) and longevity (Abel & Kruger, 2010). Importantly, a recent meta-analysis indicates a small but reliable effect of facial expression on self-reported happiness and positive affect (Coles, Larsen, & Lench, 2019), and preliminary results from a multi-lab replication project corroborate these findings (N. Coles et al., 2019). For example, encouraging participants to smile without realizing that they are smiling (e.g., by instructing them to hold chopsticks between their teeth) can counteract reductions in positive affect during a stressful experience and lower heart rate during stress recovery (Kraft & Pressman, 2012). Indeed, voluntary smiling can activate the same patterns of regional brain activity as spontaneous smiling (Ekman & Davidson, 1993). There is also evidence that the mere act of forming a smile, whether by following instructions to pull up lip corners and relax the face (Hennenlotter et al., 2005) or by imitating another person's smile (Hsu, Sims, & Chakrabarti, 2018; T. W. Lee, Josephs, Dolan, & Critchley, 2006), uniquely activates the striatum, MPFC, and amygdala. Consistent with a role of the mesial dopaminergic network in smiling and mood, patients with Parkinson's disease, involving degeneration of the mesostriatal dopamine neurons, have deficits in smiling (Marsili et al., 2014) and in the experience of positive affect (Cummings, 1992).

5.1.3. Humor

Humor is another venue for increasing positive affect and boosting resilience. Using and appreciating humor have been associated with resilience in multiple

vulnerable populations, including combat veterans, firefighters, patients with serious illness, and mothers of children with serious illness (e.g., Sliter, Kale, & Yuan, 2014) (reviewed in Kuiper, 2012; and in Southwick et al., 2005). A longitudinal study of over 50K participants in Norway found that self-reported ability to recognize humor is associated with lower mortality (Romundstad, Svebak, Holen, & Holmen, 2016). Genuine laughter and smiling when talking about a recent loss is associated with better coping over time (Bonanno, 2004). Experimental studies also indicate a causal effect of humor on resilience (Kuiper, 2012). For example, using humor reduces state anxiety in response to a stressful event (Ford, Lappi, O'Connor, & Banos, 2017) and increases subjective well-being (Maiolino & Kuiper, 2016). Observing humorous material such as funny cartoons activates brain regions in mesostriatal reward pathways, including VTA, nucleus accumbens, amygdala, and MPFC (Chan et al., 2018; Goel & Dolan, 2001; Mobbs, Greicius, Abdel-Azim, Menon, & Reiss, 2003; Shibata, Terasawa, & Umeda, 2014), and subjective funniness of the observed material correlates positively with activation in these regions (Bartolo, Benuzzi, Nocetti, Baraldi, & Nichelli, 2006; Jaaskelainen et al., 2016; Mobbs et al., 2003). Generating humorous captions for cartoons also activates these reward regions (Amir & Biederman, 2016). Interestingly, the trait emotional stability (i.e., the opposite of neuroticism) correlates with increased activation in this network during observation of humorous material (Mobbs, Hagan, Azim, Menon, & Reiss, 2005), suggesting a connection between emotional resilience and mesostriatal activation to humor.

5.1.4. Self-affirmation

At the root of self-affirmation theory is the idea that people are deeply motivated to maintain self-integrity, or a perception of self as "morally and adaptively adequate" (G. L. Cohen & Sherman, 2014). Self-affirmation in an act designed to affirm one's self-integrity, typically by reflecting on sources of self-worth such as important personal values, personal qualities, or social relationships (reviewed in G. L. Cohen & Sherman, 2014; Steele, 1988). For example, writing about an important personal value can reduce negative affect and boost positive affect following social rejection (Hales, Wesselmann, & Williams, 2016). Self-affirmation can also facilitate executive functions that are important for resilience, such as problem-solving under stress (Creswell, Dutcher, Klein, Harris, & Levine, 2013) and exerting self-control (Churchill, Jessop, Green, & Harris, 2018; P. S. Harris, Harris, & Miles, 2017; Schmeichel & Vohs, 2009), even 2+ years after the intervention (Logel, Kathmandu, & Cohen, 2018). Similarly, writing about personal core values, such as close relationships or religion, can buffer against being negatively stereotyped and improve academic performance, even years after intervention (e.g., Brady et al., 2016). Neurobiologically, self-affirmation can buffer stress and be rewarding. Writing about important personal values prior to a very stressful exam can prevent the increase in urinary epinephrine levels, a marker of ANS stress response, associated with the exam (Sherman, Bunyan, Creswell, & Jaremka, 2009). Furthermore, reflecting on one's top values increases positive affect (Hales et al., 2016; Nelson, Fuller, Choi, & Lyubomirsky, 2014) and activates brain regions associated with reward, including ventral striatum and MPFC (Cascio et al.,

2016; Dutcher et al., 2016).

5.1.5. Reactivating positive memories

Another strategy for increasing positive affect and boosting resilience is recalling positive memories (i.e., remembering the good times). Autobiographical affective recall, or imagining a past emotional experience, is thought to reactivate the emotions associated with the original experience, and it is a common technique for mood induction in laboratory studies (Westermann, Spies, Stahl, & Hesse, 1996). Recalling positive autobiographical memories has also been used as a successful clinical tool, for increasing positive affect among individuals with schizophrenia (Johnson, Gooding, Wood, Fair, & Tarrier, 2013), PTSD (Panagioti, Gooding, & Tarrier, 2012), and major depression (Arditte Hall, De Raedt, Timpano, & Joormann, 2018). Positive reminiscence can also reduce distress. In a groundbreaking study, artificially reactivating positive memories in mice, by inducing the firing of hippocampal neurons that had been active during a prior positive experience, alleviated depression-like behavior through a hippocampus–amygdala–accumbens pathway (Ramirez et al., 2015), implicating an important role of the reward pathway in the beneficial effect of positive reminiscence. In humans, reminiscing about a past positive experience activates the mesostriatal reward pathway, including nucleus accumbens and MPFC (Speer & Delgado, 2017; Suardi, Sotgiu, Costa, Cauda, & Rusconi, 2016), and it reduces cortisol rise and negative affect in response to a laboratory stressor (Speer & Delgado, 2017). Activation in these reward-related regions during positive reminiscence is correlated with self-

reported positive affect, and, notably, with a self-reported measure of resilience (Speer, Bhanji, & Delgado, 2014).

5.2. Physical health strategies

Given the reciprocal relationship between the brain and body, improving physical health can help boost physical AND psychological resilience (McEwen & Gianaros, 2011). Chief among health-improving factors are sleep, exercise, and diet. I will not cover nutrition, as it is beyond the scope of this paper and is reviewed elsewhere (e.g., Gomez-Pinilla, 2008; Goyal, Iannotti, & Raichle, 2018). Instead, I will discuss two topics related to food intake and the digestive system, namely food restriction and gut microbiota, both of which can affect brain function and mood.

5.2.1. Sleep

Getting sufficient sleep is important for health. Conversely, sleep deprivation is a stressor, with negative consequences for the brain and the rest of the body. Chronic sleep deprivation increases blood pressure, as well as levels of cortisol, insulin, and pro-inflammatory cytokines (McEwen, 2006). Lack of sleep is also associated with increased markers of systemic inflammation, which in turn are linked with subsequent physical and mental health problems (Irwin, Olmstead, & Carroll, 2016). Several different lines of work suggest that sleep affects psychiatric health and well-being (reviewed in Rumble, White, & Benca, 2015; Vandekerckhove & Cluydts, 2010). For example, patients with insomnia symptoms are more likely to

develop future depression than those without insomnia symptoms, and treating sleep disturbance can help improve mood (Rumble et al., 2015). Similarly, improving sleep duration and quality is associated with better subsequent physical and emotional well-being (N. K. Tang, Fiecas, Afolalu, & Wolke, 2017). Sleep may also help with recovery from stress and trauma (A. N. Goldstein & Walker, 2014). The neurobiological pathways from sleep to resilience include the fear/stress and the dopaminergic reward networks (Krause et al., 2017). In rodents and humans, sleep restriction can elevate the activity of sympathetic and HPA systems (reviewed in Meerlo, Sgoifo, & Suchecki, 2008) and potentiate HPA stress reactivity (reviewed in van Dalfsen & Markus, 2018). Sleep deprivation also impairs amygdala discrimination of threat cues (Goldstein-Piekarski, Greer, Saletin, & Walker, 2015) and heightens amygdala response to such cues, along with a reduction in amygdala-MPFC functional connectivity (Motomura et al., 2013; Yoo, Gujar, Hu, Jolesz, & Walker, 2007) that is directly correlated with self-reported anxiety (Motomura et al., 2013). The dopaminergic reward network is also affected by sleep and is in fact activated during sleep (Perogamvros & Schwartz, 2012). Sleep deprivation leads to selective disruptions in MPFC-to-accumbens signaling (Z. Liu et al., 2016), reduces gray matter volume in nucleus accumbens (Whitman et al., 2017), and downregulates dopamine receptors in ventral striatum (Volkow et al., 2012), ultimately resulting in inaccurate reward coding (reviewed in Krause et al., 2017).

5.2.2. Exercise

The positive consequences of physical exercise have been well studied and

include improved physical health, mental health, and cognitive function (reviewed in Ashdown-Franks et al., 2020; Kramer & Erickson, 2007; Penedo & Dahn, 2005; Warburton & Bredin, 2017) [also see (Diamond & Ling, 2019)]. The pathway from exercise to these benefits is complex, but the mesostriatal reward network plays an important role. Physical exercise, whether acute or chronic, is generally considered rewarding, as evidenced by rodents' behavior when given the opportunity to exercise such as on a running wheel (Basso & Morrell, 2015; Belke & Wagner, 2005; Greenwood et al., 2011), by increased striatal dopamine activity following exercise (Basso & Morrell, 2015; Greenwood et al., 2011; Meeusen & Fontenelle, 2012), and by human self-report (Boecker et al., 2008; Saanijoki et al., 2018). For example, wheel running in rats induces a preference for the environment associated with the running, along with plasticity in the VTA and ventral striatum (Greenwood et al., 2011; Herrera et al., 2016). These changes in the reward network in turn may underlie the positive effect of exercise on stress resilience (Marais, Stein, & Daniels, 2009; Mul et al., 2018), as disruption of this reward pathway during exercise eliminates its stress-buffering effect (Mul et al., 2018). In humans, exercise modifies an estimated 80% of brain gray matter, including ventral striatum and MPFC (Batouli & Saba, 2017) and has a neuro-protective effect on striatal dopamine receptors (Dang et al., 2017; Robertson et al., 2016). One potential mechanism underlying the positive effect of exercise on resilience may be an ultimate dopaminergic input from VTA to MPFC leading to enhanced active coping (C. Chen et al., 2017). Exercise may also boost prefrontal executive function such as inhibitory control, along with a corresponding increase in LPFC activation (Byun et al., 2014)

and in LPFC and MPFC volume (Batouli & Saba, 2017; Kramer & Erickson, 2007).

5.2.3. Food restriction

Another form of physical challenge that can boost resilience is food restriction, including caloric restriction (reducing caloric intake while maintaining meal frequency) and fasting (skipping meals). Food restriction with adequate nutrient intake can slow down aging and age-related diseases, and improve mood and cognition (Longo & Mattson, 2014). From an evolutionary perspective, the physical and cognitive benefits of food restriction make sense because individuals whose brains function well during hunger would have a survival advantage (i.e., a hungry animal that can successfully outrun or outsmart its predator is more likely to survive and reproduce). Caloric restriction, typically implemented in the range of 20-40% decrease in daily intake, increases healthy lifespan in a range of species (C. Lee & Longo, 2016). A number of studies demonstrate the benefits of caloric restriction and fasting. Notably, a multi-site randomized controlled trial found that two years of ~10-20% caloric restriction in non-obese adults can improve mood, sleep, and quality of life (Martin et al., 2016), as well as some predictors of health span and longevity (Ravussin et al., 2015). Interestingly, no negative effects of caloric restriction were found in this study. Fasting (e.g., for 12-24 hours 2-4 times/week) is emerging as an effective alternative to caloric restriction, with potentially better compliance (Horne, Muhlestein, & Anderson, 2015). Although well-designed randomized controlled studies of fasting are limited, existing studies show improvements in weight and other health outcomes (Horne et al., 2015). Food

restriction may also facilitate extinction of traumatic memories (Shi et al., 2018), likely through serotonin-dependent (M. C. Riddle et al., 2013) and amygdala (Verma et al., 2016) pathways. Food restriction also affects mesostriatal dopamine pathways (Maalouf, Rho, & Mattson, 2009). In rhesus monkeys, 6 months of 30% caloric restriction has neuroprotective effects on midbrain dopamine neurons, likely by increasing striatal neurotropic factors (Maswood et al., 2004). Similarly, fasting has neuroprotective effects on striatal neurons in rats following excitotoxic and metabolic insults (Bruce‐Keller, Umberger, McFall, & Mattson, 1999; Marie, Bralet, Gueldry, & Bralet, 1990). Food restriction may also activate the mesostriatal reward pathway via orexin, a neuropeptide implicated in the regulation of natural and drug rewards (G. C. Harris & Aston-Jones, 2006). Fasting activates orexin neurons (Lutter et al., 2008), which heavily innervate dopamine-rich VTA and nucleus accumbens structures (G. C. Harris & Aston-Jones, 2006), and orexin signaling has been proposed as a potential mechanism underlying the antidepressant effect of food restriction (Lutter et al., 2008; Manchishi, Cui, Zou, Cheng, & Li, 2018; Zhang et al., 2015).

5.2.4. Brain-gut microbiome

Bacteria in the gut are important for normal health and functioning, impacting lifespan and health span (Spielman, Gibson, & Klegeris, 2018). There is bidirectional signaling between the brain and gut microbiome, and the latter can play a role in such brain-related diseases as anxiety, depression, and chronic pain (reviewed in Long-Smith et al., 2020; Mayer, Knight, Mazmanian, Cryan, & Tillisch,

2014; Pereira et al., 2020). For example, an imbalance in the mouse gut microbiome can induce depressive behavior like social withdrawal (Buffington et al., 2016; Gacias et al., 2016), a deficit that can be passed on to other mice by fecal transplant (Gacias et al., 2016) or reversed by selectively adding *Lactobacillus* bacteria to their drinking water (Buffington et al., 2016). Similarly in humans, altered gut microbiota has been associated with depression and anxiety (Jiang et al., 2015; Luna & Foster, 2015; Sharon, Sampson, Geschwind, & Mazmanian, 2016) and with neurodegenerative and metabolic diseases (Patterson et al., 2014; Sharon et al., 2016; Spielman et al., 2018). Importantly, consumption of probiotics, beneficial bacteria, can have antidepressant and anxiolytic effects (Butler, Cryan, & Dinan, 2019; R. T. Liu, Walsh, & Sheehan, 2019; Sarkar et al., 2016) along with physical health benefits (Long-Smith et al., 2020; Patterson et al., 2014), as can consumption of prebiotics, non-digestible fibers that support probiotics (Foster, Rinaman, & Cryan, 2017; Sarkar et al., 2016). Nonetheless, not all clinical trials have demonstrated beneficial effects of probiotics (Long-Smith et al., 2020). Additional strain-selection and large-scale longitudinal studies are needed to determine the specific impact of any given psychobiotic (Pereira et al., 2020).

Microbiata can have multitude and broad effects on the brain, including the fear/stress and the dopaminergic reward networks (Butler et al., 2019; Luczynski, McVey Neufeld, et al., 2016; Sarkar et al., 2018; Sharon et al., 2016). Several studies have shown that depleting gut microbiota can increase HPA activation, while pre- and probiotics can reduce it (reviewed in de Weerth, 2017; Foster et al., 2017). Microbiome also regulate amygdala fear-related function (Hoban et al., 2018), as

well as amygdala morphology (Luczynski, Whelan, et al., 2016) and receptors (reviewed in Sarkar et al., 2018). Emerging evidence also implicates the dopaminergic reward system in the path from gut microbiota to resilience. In mice, depletion of gut microbiota via antibiotics leads to anhedonia-like deficits in reward responding, along with abnormal microglia growth in the VTA (K. Lee et al., 2018), while treatment with *Lactobacillus* bacteria reduces anxiety- and depression-like behavior as it increases prefrontal (Wei et al., 2019) and striatal (Huang et al., 2018) dopamine. In humans, gut microbiota imbalance has been associated with elevated levels of an enzyme directly involved in the production of the dopamine precursor phenylalanine, along with abnormal reward anticipation response in the ventral striatum (Aarts et al., 2017). Additionally, fecal microbiota-derived tryptophan metabolites positively correlate with functional and anatomical connectivity of the reward network, including amygdala and nucleus accumbens (Osadchiy et al., 2018). Together these findings link gut microbiota with mental health and changes in the distress and reward networks.

5.3. Social strategies (social connectedness)

5.3.1. Receiving social validation and support

Decades of research have demonstrated that social relationships can positively impact mental and physical health (House, Landis, & Umberson, 1988), and recent large-scale epidemiological findings corroborate this effect (Buettner, 2012). Compared to social isolation, social integration (having social connections)

and social support (feeling loved and cared for) have consistently been associated with lower mortality and morbidity, including lower risks of depression and PTSD (Holt-Lunstad, Smith, & Layton, 2010; Thoits, 2011; Uchino, 2006). Positive social connectedness can benefit health and resilience via multiple different routes, such as increasing feelings of belonging and self-efficacy (Southwick et al., 2005; Thoits, 2011) and promoting subjective well-being through social contagion (Fowler & Christakis, 2008). Indeed, some researchers have suggested that even having a happy spouse may enhance health beyond the contribution of one's own happiness to health (Chopik & O'Brien, 2017).

Social integration and support activate reward circuitry and can buffer stress. Social interaction in rodents activates VTA stimulation of NAS neurons (Gunaydin et al., 2014) and dampens HPA response (reviewed in DeVries, Craft, Glasper, Neigh, & Alexander, 2007; Kiyokawa & Hennessy, 2018). In humans, viewing pictures of a supportive partner during the experience of physical or social pain reduces self-reported pain and activation in the brain's pain network, as it increases activation in MPFC (reviewed in Eisenberger, 2013). Simply viewing pictures of a supportive partner activates reward-related regions like the ventral striatum (Singer, Kiebel, Winston, Dolan, & Frith, 2004), as does receiving social validation (reviewed in Tabibnia & Lieberman, 2007), such as feeling understood by peers (Morelli, Torre, & Eisenberger, 2014) and being treated with fairness (Tabibnia, Satpute, & Lieberman, 2008).

A pathway for the rewarding and stress-buffering effects of social connection

is through oxytocin, a neurohormone that promotes social attachment and prosocial behavior, stimulates the mesostriatal reward pathway, and attenuates stress-related neuroendocrine and rACC responses (reviewed in Meyer-Lindenberg, Domes, Kirsch, & Heinrichs, 2011; Shamay-Tsoory & Abu-Akel, 2016; Skuse & Gallagher, 2009). Oxytocin action in the nucleus accumbens and VTA of rodents is critical for pair bonding and positive social interaction (Dolen, Darvishzadeh, Huang, & Malenka, 2013; Hung et al., 2017; L. J. Young & Wang, 2004). Receiving a dose of oxytocin enhances the stress-buffering effect of social support on stress, such as decreasing blood pressure, pain sensitivity, and stress hormone levels (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). Oxytocin release can be stimulated by trust, as well as by physical touch (Morhenn, Beavin, & Zak, 2012), important components of social connectedness and support. Being physically touched can be sufficient to reduce threat-related activity in the pain network, including anterior insula and rACC (Coan, Schaefer, & Davidson, 2006). In short, physical or psychological social connection can stimulate oxytocin release, which in turn can stimulate the mesostriatal reward pathway and attenuate stress-related response in the brain.

5.3.2. Giving social validation and support

Not only does receiving social validation and social support promote resilience, so do giving social support and being prosocial (Eisenberger, 2013; Inagaki et al., 2016). Supporting others during times of stress, such as volunteering during war or caregiving after the loss of a spouse, are consistently associated with

adaptive outcomes, including greater longevity, psychological well-being, and physical health (S. L. Brown, Brown, House, & Smith, 2008; Curry et al., 2018; Post, 2005; Rachman, 1979; Southwick et al., 2005; cf Whillans et al., 2017). In mice, stimulation of oxytocin neurons or their terminals in the VTA increases prosocial behavior (Hung et al., 2017). In a human neuroimaging study of support giving, holding the arm of a partner who is experiencing physical pain activates the support giver's ventral striatum and septal area, a region implicated in fear attenuation, and those who show greater activity in the septal area show reduced bilateral amygdala activity during support giving (Inagaki & Eisenberger, 2012). Giving social support also reduces sympathetic response to stress (Inagaki & Eisenberger, 2016). Generosity, such as spending money on others, also enhances subjective well-being (E. W. Dunn, Aknin, & Norton, 2008) and increases activation in the brain's reward pathways, including MPFC and ventral striatum (reviewed in Cutler & Campbell-Meiklejohn, 2019; Hubbard, Harbaugh, Srivastava, Degras, & Mayr, 2016). Similar reward-related activation is observed when giving social validation, such as when treating others fairly (Zaki & Mitchell, 2011) or cooperatively (Decety, Jackson, Sommerville, Chaminade, & Meltzoff, 2004).

5.3.2.1. Compassion. A special type of social support is compassion. In contrast to empathy, which refers to the vicarious experience of another's suffering, compassion refers to the feeling of warmth and care for the other's suffering, a feeling that motivates a desire to help (Goetz, Keltner, & Simon-Thomas, 2010; Singer & Klimecki, 2014). Compassion training can help build resilience resources, including decreased illness symptoms and increased social support (Fredrickson,

Cohn, Coffey, Pek, & Finkel, 2008), and be a useful tool for targeting such mental health problems as depression and anxiety (Hofmann, Grossman, & Hinton, 2011). Trait compassion (Cosley, McCoy, Saslow, & Epel, 2010), as well as compassion training (Engert, Kok, Papassotiriou, Chrousos, & Singer, 2017; Hofmann et al., 2011), can have buffering effects on the neuroendocrine response to social stress. While reducing negative affect (Hofmann et al., 2011), compassion training can also boost positive affect (Fredrickson et al., 2008; Hofmann et al., 2011; Klimecki, Leiberg, Ricard, & Singer, 2014) and increase activation in reward-related regions, including ventral striatum and MPFC (Klimecki et al., 2014; Preckel, Kanske, & Singer, 2018; Weng et al., 2013). Similarly, responding to another's suffering with compassion can boost positive affect (Engen & Singer, 2015) and activate these reward-related regions (Engen & Singer, 2015; J. W. Kim et al., 2009; for gender differences, see Mercadillo, Diaz, Pasaye, & Barrios, 2011).

5.3.2.2. Gratitude. Expressing gratitude can boost psychological and physical well-being (Hill, Allemand, & Roberts, 2013; Seligman et al., 2005). Effective gratitude strategies include expressing gratitude to another person (e.g., by writing a gratitude letter) or privately to oneself (e.g., by journaling about one's fortunes in a diary) (Kaczmarek et al., 2015). For example, in a 2-week intervention study of adults waiting to receive psychological treatment, daily journaling of things they were grateful for reduced anxiety and increased optimism and life-satisfaction, as did daily journaling of the kind acts they had committed (S. L. Kerr, O'Donovan, & Pepping, 2015). Similarly, in a randomized controlled study, psychotherapy clients who wrote gratitude letters to others reported better mental health 4 and 12 weeks

after therapy than clients who did expressive writing or therapy-only clients (Wong et al., 2018). Multiple neuroimaging studies have associated gratitude with MPFC function, along with other regions in the mesostriatal reward pathway. A 5-minute gratitude exercise can decrease autonomic arousal, and this measure correlates with functional connectivity of reward regions, including MPFC, ventral striatum, and amygdala (Kyeong, Kim, Kim, Kim, & Kim, 2017). Self-reported gratitude correlates with MPFC activation during the experience of gratitude (Yu, Gao, Zhou, & Zhou, 2018), as well as with activation in ventral striatum and MPFC during charitable giving (Karns, Moore, & Mayr, 2017), and gratitude journaling increases this MPFC response (Karns et al., 2017). Gratitude exercise can also have lasting effects on MPFC. A one-hour gratitude letter writing exercise increased generosity and increased activity in MPFC during charitable giving 3 months later (Kini, Wong, McInnis, Gabana, & Brown, 2016). Consistent with the notion that gratitude is socially rewarding (G. R. Fox, Kaplan, Damasio, & Damasio, 2015), another fMRI study has shown that summoning feelings of gratitude activates the basal forebrain (Zahn et al., 2009), a region that is critical for maternal caregiving in rodents and includes important components of the mesostriatal pathway, such as the ventral striatum and VTA.

5.4. Summary of strategies that up-regulate the positive

All the strategies reviewed in this section show evidence of activating some aspect of the reward networks, typically including the ventral striatum and MPFC.

Most of these strategies also impact some aspect of the distress pathways, such as giving social support, which reduces autonomic arousal, and positive reminiscence, sleep, and receiving social support, which reduce HPA stress response (Table 1).

6. Transcending the Self

Recent findings suggest that another way to enhance resilience and well-being is by transcending the self, disengaging from the default self-focused mind wandering and instead engaging in the present moment, such as by practicing mindfulness or by losing oneself to activities or experiences that are personally meaningful (Yaden, Haidt, Hood, Vago, & Newberg, 2017). Empirical research has consistently shown that having a sense of meaning in life is bedrock of human well-being (Reker, Peacock, & Wong, 1987; Ryff & Singer, 1998; Zika & Chamberlain, 1992). Meaning can emerge in many different ways, including having a purpose, religious faith, or engagement with nature. While the neural pathway from these various strategies to resilience is diverse and complex, they share the common feature of interfering with the DMN and facilitating a feeling of transcending the self, losing oneself to something larger, or a sense of union with the universe or greater entity.

6.1. Mindfulness

A well-examined strategy that promotes self-transcendence and resilience is mindfulness (reviewed in Holzel et al., 2011; Lin, Callahan, & Moser, 2018; Vago & Silbersweig, 2012), a process of purposefully and nonjudgmentally paying attention to the present moment (Kabat-Zinn, 2003), resulting in reduction in the mostly maladaptive mind-wandering about self-focused matters of the past and future (Mrazek, Franklin, Phillips, Baird, & Schooler, 2013). Mindfulness practice, even brief daily practice (Basso, McHale, Ende, Oberlin, & Suzuki, 2019), can buffer stress and thus ameliorate stress-related conditions, including depression, anxiety, inflammation, and drug abuse (Creswell & Lindsay, 2014; Wielgosz, Goldberg, Kral, Dunne, & Davidson, 2019), and it can improve executive function, subjective well-being, and relationship quality (Creswell, 2017). Although mindfulness intervention is not always beneficial (Reynolds, Bissett, Porter, & Consedine, 2017; Van Dam et al., 2017; Wielgosz et al., 2019), it has demonstrated efficacy as a treatment tool for certain psychiatric diseases, including depression and addiction (Goldberg et al., 2018). Neural plasticity associated with mindfulness training includes structural and functional changes in PFC-amygdala emotion-regulation circuitry (Guendelman, Medeiros, & Rampes, 2017; Y. Y. Tang, Holzel, & Posner, 2015), such as stronger MPFC-amygdala coupling and reduced amygdala reactivity (Kral et al., 2018), structural and functional changes in LPFC and ACC associated with executive tasks (K. C. Fox et al., 2016; Holzel et al., 2011; Y. Y. Tang et al., 2015), and reduced activation of DMN (Brewer et al., 2011; K. C. Fox et al., 2016; Guendelman et al., 2017; Y. Y. Tang et al., 2015). Given that each of these mindfulness-related psychological, social, and neural changes alone can boost resilience, mindfulness

may have a unique potential to boost resilience through multiple routes.

6.2. Purpose in life, religion, and spirituality

Having a sense of meaning or purpose in life, such as striving to achieve valued goals, has traditionally been deemed an integral part of psychological well-being (Reker et al., 1987; Zika & Chamberlain, 1992), particularly when feeling competent about attaining the goals (Ryan & Deci, 2001). For many people, meaning in life can be obtained by affiliating with a larger framework of beliefs or practices, such as a religion or philosophy of life (Inzlicht, Tullett, & Good, 2011; Southwick et al., 2005). According to various polls, religiously committed or religiously active people tend to rate themselves as happier (Myers, 2000). Furthermore, having a sense of purpose, such as through spiritual faith or religious activities, may be protective against physical and psychological illness and aid coping with illness (Alim et al., 2008; Cheadle & Dunkel Schetter, 2017; Southwick et al., 2005). Demonstrating a causal connection, several studies have shown that inducing a sense of self-transcendence, spiritual orientation, and increased purpose in life, by administration of psychedelic drugs such as psilocybin (found in "magic mushrooms") and ayahuasca (used in religious ceremonies by some indigenous peoples in the Americas), can improve mental health (reviewed in Bouso, Dos Santos, Alcazar-Corcoles, & Hallak, 2018; Nunes et al., 2016). For example, clinical trials have shown that a single dose of psilocybin can produce large and sustained decreases in depression and anxiety symptoms, an effect mediated by psilocybin-

induced mystical experience (Griffiths et al., 2016; Ross et al., 2016).

Having purpose in life (Van Reekum et al., 2007) and prioritizing self-transcendent values (Kang et al., 2017) are associated with reduced distress response in brain regions such as the amygdala. Similarly, religious conviction is associated with reduced distress-related reactivity in dACC (Inzlicht et al., 2011). The brain's reward network may also play a role in the salutatory effects of having a sense of purpose. In an fMRI study, people who showed sustained striatal activity to positive events, had lower cortisol output, and reported having a greater sense of purpose, meaning, and engagement with life (Heller et al., 2013). In addition to the distress and reward networks, the DMN also plays a role in religiosity and having purpose in life. When the DMN was first described, it was characterized as a network of regions that consistently show decreased activation from baseline during a wide range of "goal-directed" behaviors (Raichle et al., 2001). Numerous studies have since demonstrated an "anti-correlated" relationship between the DMN and the executive network supporting goal pursuit, including causal evidence that the executive network can negatively regulate the DMN (A. C. Chen et al., 2013; Uddin, Kelly, Biswal, Xavier Castellanos, & Milham, 2009). Thus, at a basic level, pursuing a goal or a purpose by itself can result in reduced DMN activity. Religiousness and spirituality are also linked with reduced DMN function. For example, the typical MPFC activation observed during self-referential thinking is not observed in Tibetan Buddhists, who follow the doctrine *anatta* ("no-self"), denying oneself (Wu et al., 2010). Similarly, when DMN function is disrupted, for example by surgical selective lesion (Urgesi, Aglioti, Skrap, & Fabbro, 2010) or magnetically

induced temporary "lesion" (Crescentini, Aglioti, Fabbro, & Urgesi, 2014) of a component of DMN called the temporoparietal junction, self-reported self-transcendence and implicit religiousness/spirituality increase. Conversely, when this region is temporarily excited by magnetic stimulation, implicit religiousness/spirituality decreases (Crescentini, Di Bucchianico, Fabbro, & Urgesi, 2015). Importantly, when a personally meaningful spiritual experience or a sense of religious ecstasy is experimentally elicited by an individualized guided-imagery task (L. Miller et al., 2019) or by psychedelic drugs, such as psilocybin (Carhart-Harris et al., 2012), ayahuasca (Palhano-Fontes et al., 2015), or lysergic acid diethylamide (LSD) (Carhart-Harris et al., 2016), activity in and functional connectivity within DMN are reduced.

6.3. Exposure to nature

Another emerging contributor to resilience is exposure to nature, including such practices as "forest bathing" and garden therapy that have gained increased academic and popular attention in the last two decades (Annerstedt & Wahrborg, 2011; Hartig, Mitchell, de Vries, & Frumkin, 2014). Exposure to nature can improve cognitive function (Keniger, Gaston, Irvine, & Fuller, 2013) and have health benefits (for review see Hartig et al., 2014), such as lower blood pressure and cortisol (Haluza, Schonbauer, & Cervinka, 2014; Keniger et al., 2013), lower inflammatory cytokines and enhanced immune function (Haluza et al., 2014; Kuo, 2015), improved autonomic stress recovery (D. K. Brown, Barton, & Gladwell, 2013), and

improved circadian function (Stothard et al., 2017). Although the path from nature exposure to psychological and physiological health is complex, it includes respite from life stressors and urban pollutants (Hartig et al., 2014; Tost, Champagne, & Meyer-Lindenberg, 2015), as well as transcendence of self (Williams & Harvey, 2001). While urban pollutants can have numerous effects on the brain and well-being (van den Bosch & Meyer-Lindenberg, 2019), here I review the effect of nature on reducing activation in the DMN. In general, even brief exposure to nature, such as 15 minutes of sitting in a forest, can decrease activity in PFC and PCC, and reduce subjective negative affect and amygdala activation (reviewed in Norwood et al., 2019). In a correlational study, rural versus urban upbringing was specifically associated with lower stress reactivity in MPFC only, while current city living was specifically associated with greater stress reactivity in amygdala only (Lederbogen et al., 2011). Importantly, a randomly-assigned 90-minute walk in a natural, but not urban, setting reduced self-reported rumination and MPFC activity (Bratman, Hamilton, Hahn, Daily, & Gross, 2015).

6.4. Flow

Another way to lose oneself is through the experience of flow, a state of deep engagement in a challenging activity that requires high level of skill, such as playing a sport, a musical instrument, or chess (Csikszentmihalyi, 1999). The state of flow requires concentration, perseverance, and skill that matches the challenge; if the task is too challenging, it can lead to anxiety, and if the task is too easy, it can lead to

boredom. When perceived challenges and skills are both high and in balance, a state of optimal experience arises and subjective well-being increases (Csikszentmihalyi & LeFevre, 1989). Consistent with this framework, flow can reduce negative affect and boost positive affect (Rankin, Walsh, & Sweeny, 2018), as well as increase the relaxing activity of the parasympathetic nervous system (Knierim, Rissler, Dorner, Maedche, & Weinhardt, 2018). Similarly, individuals who experience flow more frequently report lower depressive symptoms and emotional exhaustion, even after controlling for genetic and familial factors (Mosing, Butkovic, & Ullen, 2018). There is also evidence of high subjective well-being among individuals who regularly experience flow, including advanced musicians (Ascenso, Williamon, & Perkins, 2016; Fritz & Avsec, 2007), elite athletes (Gould, Dieffenbach, & Moffett, 2010; Jackson, Ford, Kimiecik, & Marsh, 1998), and full-time employees who experience flow in their jobs (Ilies et al., 2017). In fact, the field of occupational therapy may have been born out of observations that being "occupied" by activities like the arts reduces stress and enhances well-being, and may be viewed as an early form of "flow therapy" (Sadlo, 2016).

A nascent and growing literature on the neural basis of flow suggests that being in a state of flow can reduce activation in the DMN and the amygdala and possibly activate the reward pathway. Consistent with the idea of experts experiencing flow and self-transcendence during engagement in the expert task, grandmaster and master chess players showed lower DMN activity during a chess problem-solving task than novice players (Duan et al., 2012). Similarly, across two separate fMRI studies, when difficulty of an arithmetic task was continuously

adjusted to each participant's skill level, being in a state of flow compared to boredom and overload, as confirmed by self-report, decreased activation in the amygdala and DMN, including MPFC and PCC, along with increased activation in the attention network (Ulrich, Keller, & Gron, 2016; Ulrich, Keller, Hoenig, Waller, & Gron, 2014) and striatum (Ulrich et al., 2014). In another fMRI study, using a naturalistic video game, balance between game difficulty and the player's ability resulted in greater self-reported intrinsic reward and activation in the striatum, compared to when game difficulty was too high or too low, and in lower DMN activation compared to when game difficulty was too low (Huskey, Craighead, Miller, & Weber, 2018). Consistent with the potential role of the reward pathway in flow, self-reported flow-proneness is correlated with dopamine D2 receptor availability in the striatum (de Manzano et al., 2013) and with a dopamine D2 receptor gene polymorphism (Gyurkovics et al., 2016).

6.5. Summary of strategies that promote transcending the self

The four strategies reviewed in this section all reduce activity in the DMN. They each also impact some aspect of the distress networks. Two of the strategies, specifically purpose in life and flow, are also associated with activation in the striatum, a reward-related region (Table 1).

7. Future Directions: Predictions and Implications of the Tripartite Model

The tripartite model makes several predictions and is therefore testable. First, the model predicts that manipulating distress, reward, or default mode networks will impact resilience. As reviewed below, predictions of this kind are testable with experimental studies, which are important for understanding the causal mechanisms of building resilience and for testing efficacy of new interventions. The tripartite model also hypothesizes that individual differences in baseline activation of these networks may predict stress resilience and intervention outcomes. These types of hypotheses are addressed with prediction (e.g., prospective longitudinal) studies, which can help identify processes underlying resilience and potentially lead to individualized intervention. Here, I review each of these types of investigations, including their potential implications. Finally, I suggest additional future directions that can help test and expand the model.

7.1. Experimental Approaches

If the tripartite model is valid, then any intervention that (moderately) stimulates the reward network or reduces over-activation in the default mode or distress networks should boost resilience. This framework of resilience-building can help lead to novel or largely ignored strategies. In particular, the tripartite model makes two unique types of predictions that could lead to novel and potentially more effective approaches to resilience-building. First, the model formally recognizes and predicts efficacy of strategies that can transiently reduce DMN activity and induce a

sense of self-transcendence. Specifically, as outlined below, if the tripartite model is correct in that reducing DMN activity boosts resilience, then awe and hypnotic state, experiences that reduce DMN activity, should each improve health outcomes. Second, the model predicts that interventions that activate more than one pathway to resilience would be particularly effective, likely in an additive fashion (Tabibnia & Radecki, 2018). Here I introduce some strategies that can test these predictions.

7.1.1. Awe

A subjective state that has received recent attention is awe, the feeling of wonder experienced when facing something greater than the self and beyond current understanding, such as witnessing a panoramic view of nature, childbirth, or a magnificent work of art (Keltner & Haidt, 2003). Consistent with the notion that awe can help dissolve awareness of the self, viewing photographs or videos of nature that are rated as most "sublime" or awe-inducing reduces activation in DMN regions including PCC and MPFC (Ishizu & Zeki, 2014; van Elk, Arciniegas Gomez, van der Zwaag, van Schie, & Sauter, 2019). Further supporting a role of DMN in awe, self-reported dispositional awe is inversely correlated with gray matter in three cortical regions, all within the DMN, namely MPFC, PCC, and temporo-parietal junction (Guan, Xiang, Chen, Wang, & Chen, 2018). There is some preliminary evidence in support of the resilience-boosting potential of awe. Experimentally inducing awe, for example by asking participants to imagine or experience an inspiring scene (e.g., the view of Paris from above or the view of towering trees from below), increases subjective well-being and prosocial behavior (Anderson, Monroy,

& Keltner, 2018; Piff, Dietze, Feinberg, Stancato, & Keltner, 2015; Rudd, Vohs, & Aaker, 2012). Additionally, dispositional awe has been associated with lower pro-inflammatory cytokines (Stellar et al., 2015). However, randomized controlled studies, particularly those that control for nature exposure, are needed to confirm the causal effect of awe on long-term mental or physical health outcomes.

7.1.2. Hypnotic state

Another mental state associated with decreased DMN activity is the hypnotic state. Hypnosis has been defined as "a state of consciousness involving focused attention and … enhanced capacity for response to suggestion" (Elkins, Barabasz, Council, & Spiegel, 2015). Hypnotic induction typically involves instructions to relax and focus attention on the present moment, resulting in mental absorption, reduced mind-wandering, and an altered sense of self (Kihlstrom, 2018), states that can be likened to mindfulness and flow. As such, despite inconsistencies in methodology and results of neuroimaging studies of hypnosis, a recent narrative review (Landry, Lifshitz, & Raz, 2017) and empirical study (Y. Liu et al., 2018) indicate mounting evidence associating the hypnotic state with decreased DMN activation. Numerous randomized controlled studies have demonstrated high efficacy of hypnotherapy in treatment of specific disorders, including functional gastrointestinal disorders (reviewed in Vasant & Whorwell, 2019), physical pain (reviewed in Thompson et al., 2019), and nicotine dependence (reviewed in Barnes, McRobbie, Dong, Walker, & Hartmann‐Boyce, 2019). However, according to the tripartite model, experiencing "neutral hypnosis" alone (i.e., simply being in a state of hypnosis, even without

specific suggestions to alter experience or behavior) should itself boost resilience in a wide range of contexts. While the notion that hypnotic induction alone may have much impact on intervention outcome has been questioned (e.g., Kihlstrom, 2018), improved subjective affect can in fact follow hypnotic induction (Laborde, Heuer, & Mosley, 2018). Nonetheless, well-controlled experiments are needed to test the hypothesis that neutral hypnosis can be leveraged to help boost mental or physical health outcomes.

7.1.3. Adding self-transcendence to other strategies

Another prediction of the tripartite model is that incorporating self-transcendence into another strategy would render that strategy more effective. Self-distancing and self-affirmation are two examples that can support this prediction. Part of the reason self-distancing is a particularly effective reappraisal strategy (Webb et al., 2012) may be that the use of third-person and second-person appraisals not only help change the interpretation of a negative experience but that they do so by normalizing the experience beyond the self and facilitating meaning-making (Orvell, Kross, & Gelman, 2017). Consistent with this self-transcendent effect of distancing, third-person self-talk during distancing reduces activity in a part of the DMN associated with self-reflection, namely MPFC (Moser et al., 2017). Self-transcendence can also be incorporated into self-affirmation to boost its effect. For example, affirming self-transcendent values (e.g., "contributing to something larger than oneself ") is more effective in buffering the negative consequences of social exclusion than affirming self-enhancing values (e.g., "appearing

intelligent/competent") (Burson, Crocker, & Mischkowski, 2012). Consistent with this finding, neural response to prosocial relative to selfish rewards is more predictive of longitudinal mental health outcomes (Telzer, Fuligni, Lieberman, & Galvan, 2014). Future studies can investigate the added benefit of incorporating self-transcendence into other strategies, such as recalling positive memories (i.e., of self-transcendent experiences) and physical exercise (that is done outdoors or that induces flow).

7.1.4. Recruiting multiple pathways

Just as risk factors tend to contribute to vulnerability in an additive manner, so too resilience factors likely contribute to resilience in an additive manner (Southwick & Charney, 2012; Tabibnia & Radecki, 2018). Accordingly, a strategy that recruits more than one pathway would be expected to be more effective than a comparable strategy that recruits only one. For example, the tripartite model predicts that a particularly effective strategy would be participating in team sports, because it would activate not only the reward pathway through exercise, social connectedness, and self-efficacy, but also the distress-reduction pathway through stress inoculation, as well as the DMN pathway through the experience of "flow". Consistent with this additive prediction, in cross-sectional studies team-sport athletes report lower rates of poor mental health than individual-sport athletes (Chekroud et al., 2018; Pluhar et al., 2019). Some high-intensity outdoor sports like mountaineering and surfing could have the added benefit of further reducing DMN activity via nature exposure and potentially awe. While some correlational reports

link high-intensity sports with affective psychopathology, such as bipolarity (Siwek et al., 2015), others link them to well-being (Houge Mackenzie & Brymer, 2018). Given that emotion regulation (Barlow, Woodman, & Hardy, 2013) and emotional catharsis (Zhou, Chlebosz, Tower, & Morris, 2019) may be important motivations for participating in some of these sports, it is possible that these activities are sought as a form of mood repair. Consistent with the idea that participation in such sports may be therapeutic, sports-oriented occupational therapy, such as "surf-therapy", is being investigated as a potential intervention for mental health problems in military service members (Walter et al., 2019) and vulnerable youth (Godfrey, Devine-Wright, & Taylor, 2015). Thus, the proposition that participation in team and/or high-intensity sports can enhance resilience is an empirical question that warrants examination. Similarly, being in a musical band is predicted to be particularly effective, because it would not only activate the reward pathway through sensory pleasure, social connectedness, and self-efficacy, but it would likely also affect the DMN pathway through the experience of "flow" and potentially awe.

To test the tripartite model and practical usefulness of these interventions, randomized trials can investigate the effect of each intervention on resilience outcomes and comparative studies can assess the relative efficacy of single- vs. multiple-route strategies. Particularly useful would be studies with longitudinal designs that can assess long-term effects of the intervention.

7.2. Prediction Approaches

Longitudinal designs are also important for prediction studies. Specifically, if

the tripartite model is valid, then to the extent that there are stable individual differences in neural activation in the distress, reward, and default mode networks, baseline activation in each network should predict not only differences in subsequent stress resilience but potentially also in treatment response. As reviewed in this section, prospective longitudinal designs in which individuals are followed before, during, and after exposure to stressors, can best test such hypotheses. Beyond providing tests of the tripartite model, prediction studies may also identify new targets for intervention and contribute to individualized intervention.

7.2.1 Predicting Resilience

As urged by other resilience researchers, there is currently a pressing need for prospective longitudinal studies of resilience (Kalisch et al., 2017). In particular, there is a relative dearth of human studies on whether functioning of the distress, reward, or default mode networks can predict health outcome after subsequent exposure to stressors. Nonetheless, some findings compatible with the tripartite model do exist. For example, prospective studies have identified HPA markers of subsequent PTSD symptoms in male soldiers undergoing warzone deployment (reviewed in Kalisch et al., 2017) and ventral striatum markers of subsequent depressive symptoms in adolescents (Telzer et al., 2014). Prospective studies of this type can test the involvement of the distress, reward, and default networks in resilience and identify other targets for intervention. These studies may also help determine the predictive value of neural resilience markers, for example for identifying the most resilient individuals to confront an upcoming stressor (e.g., war

deployment) versus the less resilient individuals who may benefit from proactive resilience-training first. Studies that measure neural activity at multiple time-points – before, during, and after a stressor – can also help elucidate the mechanisms of adaptive coping.

7.2.2 Predicting Treatment Response: Tailored Medicine

Baseline activation in brain networks may not only predict resilience outcomes but may also offer a tool for individualized or tailored intervention. For example, individuals low on reward network activation might benefit more from one type of intervention, while individuals high on distress or default mode network activation might benefit more from another. Similarly, given that many psychopathologies are associated with LPFC deficits (e.g., Tabibnia et al., 2011; Zilverstand et al., 2017), these individuals may not benefit as much from cognitive coping strategies, such as cognitive reappraisal, that recruit this region (e.g., Morawetz, Bode, Derntl, et al., 2017; Tabibnia et al., 2014) and instead may respond better to alternative, more bottom-up strategies, such as those in the positivity boosting or self-transcendence routes (e.g., Westbrook et al., 2013). Prospective longitudinal intervention studies are needed to evaluate the predictive value of neural markers for tailoring intervention.

7.3. Other future directions

To help test and further develop the tripartite model, future studies can not

only test the predictions outlined above, but also conduct more formal, quantitative, and bias-corrected reviews of the literature to ascertain reliable involvement of each neural pathway in a given strategy. Future work can also elucidate underlying mechanisms by investigating neural-level interactions among the 3 pathways. For example, findings that stimulation of the reward network can down-regulate amygdala/HPA activity (Dutcher & Creswell, 2018) offer a potential explanation for how strategies like social connectedness can not only be rewarding but also down-regulate negative affect. Similarly, reduction of DMN activity, such as through mindfulness, may impact amygdala/HPA response, perhaps by reducing ruminative thinking that could otherwise stimulate distress-related activity in these regions (Nejad, Fossati, & Lemogne, 2013; Sheline et al., 2009). In addition to individual differences in baseline activation that may moderate resilience and intervention outcomes, other moderating factors, such as personality (Segerstrom & Smith, 2019) and culture (Tsai, 2017), would also be important to investigate.

8. Conclusions

8.1. Summary and significance

Taking an affective neuroscience approach, I proposed a simple three-route model of boosting resilience in adulthood that aims to unify the large body of literature on resilience. Specifically, according to the tripartite model of resilience-

building, the three distinct general routes to resilience are 1) down-regulating the negative (reducing distress-related responses of the amygdala and hypothalamic-pituitary-adrenal (HPA) axis), 2) up-regulating the positive (activating mesostriatal reward pathways), and 3) transcending the self (reducing activation in the default mode network). To build the theory, I first described the mechanism underlying each neural pathway to resilience. Then, to support the theory, I systematically reviewed over two-dozen behavioral and psychosocial strategies that boost resilience and demonstrated that each strategy recruits at least one of these pathways.

To date, no single model of resilience can account for the dozens of behavioral and psychosocial resilience-building strategies that dominate the literature. Prior models, although important and similar in many ways, differ in significant ways from the tripartite model. Like Kalisch et al. (2015)'s PASTOR model, the tripartite model assumes there are far fewer distinct psychobiological mechanisms than factors to resilience and recognizes the importance of identifying these few shared mechanisms. While both models account for a wide range of cognitive-based strategies to resilience, the tripartite model also accounts for a host of additional strategies, including social and physical health strategies. In contrast, Waugh and Koster (2015)'s resilience framework, a model of resilience interventions for remission from depression, includes both a distress downregulation and a positivity upregulation route, in addition to a third route they call "flexibility", which they suggest may be enhanced through meditation. The tripartite model formalizes these 3 routes in terms of their plausible functional

neuroanatomical mechanisms, which helps account for a far greater variety of strategies than either of these models.

An implication of the tripartite model is that merely eliminating stressors and perceptions of threat are not sufficient for optimal resilience-building. As espoused by "positive psychology" approaches, successful intervention should also include strategies from the positivity-boosting route, and likely from the self-transcending route as well. This idea is consistent with recent neurobiological and evolutionary models of the stress response and disease (Brosschot, Verkuil, & Thayer, 2017) positing that the absence of threat is not the same as the presence of safety. Both distress-reduction and positivity-boosting strategies are needed to more successfully minimize chronic stress and its downstream consequences.

Despite the general recognition in health and clinical psychology that both positive and negative affect should be targeted in resilience-building (Johnson & Wood, 2017; Southwick & Charney, 2012; Waugh & Koster, 2015), there is still some resistance in incorporating "positive interventions" into psychological therapy (Johnson & Wood, 2017), and some ambiguity remains as to whether positive and negative human functioning (e.g., happiness and depression) are opposite ends of the same continuum (Johnson & Wood, 2017; Wood & Tarrier, 2010) or two distinct, at least partly orthogonal, continua (B. D. Dunn, 2017; Ryff et al., 2006; Westerhof & Keyes, 2010). The affective neuroscience approach of the tripartite model helps clarify the important and unique contributions of each of these two routes to resilience and helps resolve the theoretical ambiguity, as it points to distinct (albeit interacting) networks in the brain for negative and positive affective function.

8.2. Limitations & Conclusion

While resilience in the face of trauma is not uncommon (Bonanno, 2004), there are limits to the prevalence or ease of resilience, such as in chronic mass-trauma or politically violent regions, where prevalence of PTSD and depression are higher and resilience trajectories are poorer than in other populations (Hobfoll, Mancini, Hall, Canetti, & Bonanno, 2011). Even in peaceful regions, prevalence of resilience, in terms of subjective well-being and self-reported physical health, in the long-term aftermath of such adversities as spousal loss and unemployment, can be lower than 50% (Infurna & Luthar, 2016). Also limited is the effectiveness of specific resilience strategies. While engaging in activities that increase positive affect tends to boost resilience, positive affect is not always beneficial (Pressman & Cross, 2018). For example, excessive happiness or mania can lead to neglect of threat, and inappropriate expressions of happiness, such as when in need of help or during confrontation or negotiation, can solicit unwanted social responses (reviewed in Gruber, Mauss, & Tamir, 2011).

There are limits to any theory of resilience as well. Three facets of the tripartite model that highlight its boundaries are: 1) it is an *affective neuroscience* model that focuses on brain networks; 2) it is a model of *behavioral and psychosocial* strategies*;* and 3) it is a model of resilience-building in *adulthood*. Thus, the model does not exhaustively incorporate neurochemical or other biological pathways that are important for resilience. In its current form, it does not model the important

roles of individual or sociological factors (Dunkel Schetter & Dolbier, 2011), pharmaceutical interventions, or developmental factors (Casey, Glatt, & Lee, 2015), that can influence resilience outcomes. That is not to say that these factors are not important or that they cannot be integrated into future iterations of the model. The current version of the tripartite model is intended to be a simple framework for identifying common mechanisms underlying behavioral and psychosocial (i.e., non-invasive and relatively easy to implement) strategies that can help boost resilience in adults.

In conclusion, the tripartite model is a unifying theory of resilience-building in adulthood, a model that makes predictions and opens the door to discovery of potential new or largely ignored cognitive and/or psychosocial strategies and approaches to resilience-building. Although the model does not formally incorporate individual differences factors, it paves the way for an individualized- or tailored-medicine approach to resilience-building. Given the limitations of current resilience-building approaches and the prevalence of stress-related mental and physical health problems, novel approaches, such as those proposed in this paper, are needed for conceptualizing resilience-building and generating novel interventions.

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Figure Captions

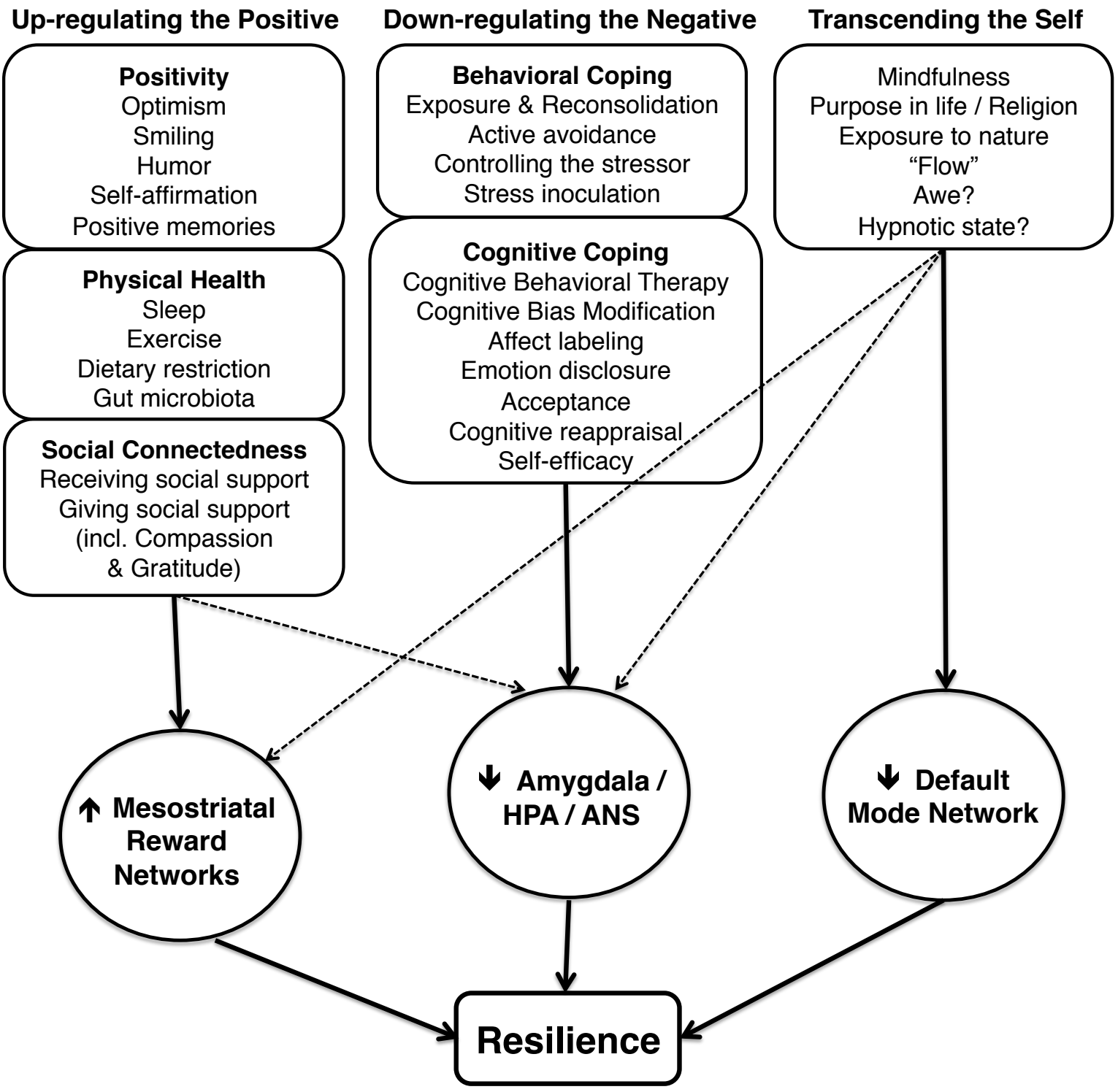
Fig. 1A. The tripartite model of resilience-building. Taking an affective neuroscience approach, three distinct general routes, and hence three distinct categories of strategies, to resilience are proposed: 1) down-regulating the negative, 2) up-regulating the positive, and 3) transcending the self. First, at the center of the model, are strategies that directly down-regulate negative affect and reduce distress-related responses of the amygdala, hypothalamic-pituitary-adrenal (HPA) axis, and autonomic nervous system (ANS). The second route to resilience encompasses strategies that up-regulate positive affect, including psychological, social, and physical well-being, activating mesostriatal pathways of reward and motivation. The third route to resilience encompasses strategies that promote an experience of self-transcendence and reduce activation in the default mode network (DMN), a network of brain regions associated with self-reflection, mind-wandering, and rumination. Solid arrow from a category of strategies to a brain network indicates that there is evidence for every one of the strategies within the category to activate the brain network. Dashed arrow indicates that there is evidence for only some of the strategies in the category to impact the brain network. For example, all strategies that up-regulate the positive have been shown to activate the reward network. However, only some strategies that up-regulate the positive, including social support and sleep, have been shown to also reduce distress-related responses of the amygdala and HPA. Question mark (?) indicates emerging strategy that the model

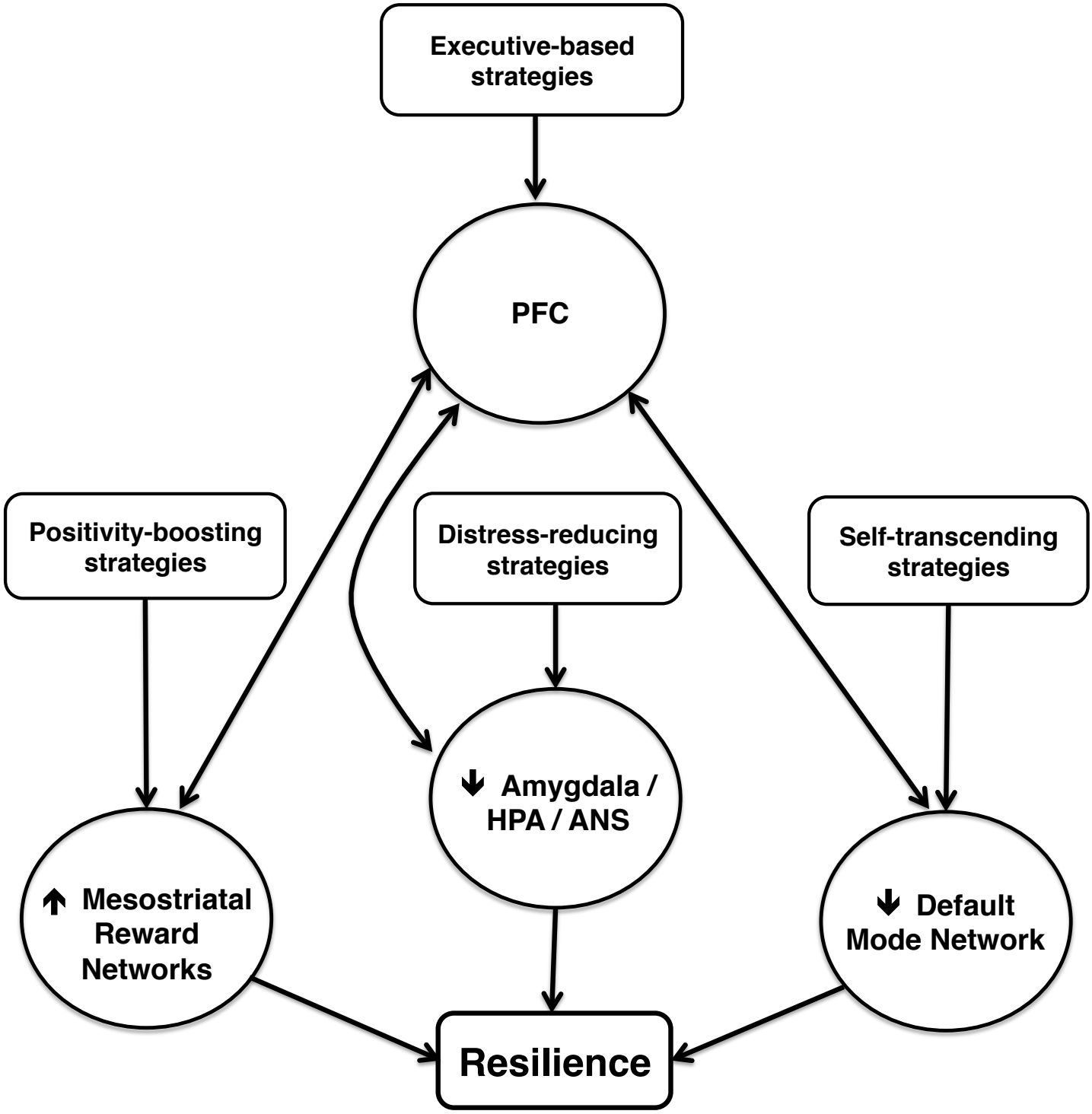
would predict as beneficial to resilience via the depicted neural pathway but that needs further investigation.

Fig. 1B. Tripartite model, zoomed out. Top-down voluntary or implicit control over each of the three pathways to resilience can be exerted via prefrontal (PFC) regions that support decision-making, self-regulation, and other executive function. Strategies that recruit or improve PFC executive function, such as psychological flexibility and cognitive training, may impact resilience via the mesostriatal, amygdala/distress, and/or default mode pathways.

Table 1. Strategies to Resilience and Their Neural Pathways

Table Notes. ACC = Anterior Cingulate Cortex; DMN = Default Mode Network; HPA = Hypothalamic Pituitary Adrenal; LPFC = Lateral Prefrontal Cortex; MPFC = Medial Prefrontal Cortex; PTSD = Post-Traumatic Stress Disorder; VTA = Ventral Tegmental Area


Up-regulating the Positive
Down-regulating the Negative
Transcending the Self
Positivity
Optimism
Smiling
Humor
Self-affirmation
Positive memories
Physical Health
Sleep
Exercise
Dietary restriction
Gut microbiota
Social Connectedness
Receiving social support
Giving social support
(incl. Compassion
& Gratitude)
Behavioral Coping
Exposure & Reconsolidation
Active avoidance
Controlling the stressor
Stress inoculation
Cognitive Coping
Cognitive Behavioral Therapy
Cognitive Bias Modification
Affect labeling
Emotion disclosure
Acceptance
Cognitive reappraisal
Self-efficacy
Mindfulness
Purpose in life / Religion
Exposure to nature
"Flow"
Awe?
Hypnotic state?
↑ Mesostriatal Reward Networks
↓ Amygdala / HPA / ANS
↓ Default Mode Network
Resilience


Executive-based strategies
PFC
Positivity-boosting strategies
Distress-reducing strategies
Self-transcending strategies
↓ Amygdala / HPA / ANS
↑ Mesostriatal Reward Networks
↓ Default Mode Network
Resilience

Strategy	Link to Resilience	Sample References (Resilience)	Neural Pathway(s)	Sample References (Pathways)
1. Downregulating the negative				
1a. Behavioral coping				
Exposure	Common treatment for anxiety disorders, including phobias and PTSD (Reduces fear over time, but fear may return)	reviewed in Craske, Treanor, Conway, Zbozinek, & Vervliet (2014) and in Foa & McLean (2016)	New safety memory formed in amygdala, consolidated in MPFC, inhibits old amygdala-based fear memory	reviewed in Furini, Myskiw, & Izquierdo (2014), in Milad & Quirk (2012), in Sotres-Bayon, Cain, & LeDoux (2006), in Fullana et al. (2018), in Hartley & Phelps (2010,) and in Sehlmeyer et al. (2009); Goossens, Sunaert, Peeters, Griez, & Schruers (2007); Herrmann et al. (2017); Raij et al. (2017); Hauner, Mineka, Voss, & Paller (2012)
Reconsolidation	Emerging treatment approach for anxiety disorders, including phobias and PTSD (Intended to reduce fear long term)	reviewed in Kredlow, Unger, & Otto (2016) and in Craske, Treanor, Conway, Zbozinek, & Vervliet (2014)	May weaken exisiting amygdala-based fear memory	reviewed in Beckers & Kindt (2017) and in Baldi & Bucherelli (2015); Argen et al. (2012); Bjorkstrand et al. (2017); Schiller, Kanen, LeDoux, Monfils, & Phelps (2013)
Active coping (active avoidance, controlling the stressor)	Better mental and physical health; better pain management	reviewed in Koolhaas et al. (1999) & Russo et al. (2012); Emmert et al. (2017); Bowen et al. (2014); Y. Ono et al. (2012); Y. Ono et al. (2012); Snow-Turek, Norris, & Tan (1996)	Plasticity in amygdala --> corticostriatal circuitry; Plasticity in MPFC--> brainstem/limbic circuitry; Reduces autonomic reactivity	reviewed in LeDoux & Gorman (2001); LeDoux, Moscarello, Sears, & Campese (2017); Cain & LeDoux (2007); Moscarello & LeDoux (2013); Delgado et al. (2009); Maier (2015); Hartley et al. (2014), Kerr et al. (2012)
Stress inoculation	Better subjective affect; successfully used to treat mood and anxiety disorders	reviewed in R. T. Liu (2015), in Seery (2011), in Levine (1957), in Rutter (1981), in Dienstbier (1989), in Maier, (2015), in Parker & Maestripieri (2011), in Russo et al. (2012), in Meichenbaum (2017), and in Saunders, Driskell, Johnston, & Salas (1996); Shapero et al. (2015)	Plasticity in MPFC (e.g., increase in volume); modulation of HPA axis & amygdala	reviewed in Levine & Mody (2003), in Russo et al. (2012), and in Tottenham (2015); Brockhurst, Cheleuitte-Nieves, Buckmaster, Schatzberg, & Lyons (2015); Amat, Paul, Zarza, Watkins, & Maier (2006); Parker et al. (2004); Katz et al. (2009); Lee, Buckmaster, Yi, Schatzberg, & Lyons (2014); Sinha, Lacadie, Constable, & Seo (2016)
1b. Cognitive coping (incl. emotion regulation)				
Cognitive behavioral therapy (CBT)	Common treatment for mood and anxiety disorders	reviewed in Clark & Beck (2010), in Cuijpers et al. (2013), in Hofmann & Smits (2008), in Craske et al. (2017), in Tolin (2010), and in DeRubeis, Siegle, & Hollon (2008)	Reduces amygdala activation and enhances prefrontal function	reviewed in Porto et al. (2009); Furmark et al. (2002); Lipka et al. (2014); reviewed in Clark & Beck (2010); DeRubeis, Siegle, & Hollon (2008); de Lange et al. (2008); Shou et al. (2017)
Affect labeling	Reduces subjective distress; can improve outcome of exposure therapy	Lieberman, Inagaki, Tabibnia, & Crockett (2011); Constantinou, Van Den Houte, Bogaerts, Van Diest, & Van den Bergh (2014); Kircanski, Lieberman, & Craske (2012); Niles, Craske, Lieberman, & Hur (2015)	Reduces autonomic reactivity in short- and long-terms; reduces amygdala activation, likely via LPFC	Hariri et al. (2003); Lieberman et al. (2007); Lieberman et al. (2011); Tabibnia et al. (2008); Torrisi et al. (2013); Brooks et al. (2017)
Emotion disclosure	Improves mental and physical health outcomes	reviewed in Hemenover (2003), in Pennebaker (1997), in Frattaroli (2006), and in Riddle, Smith, & Jones (2016); Krapan et al. (2013)	Likely re-encodes amygdala-based trauma memory in neocortex	Brewin (2001); Careaga, Girardi, & Suchecki (2016); Wang et al. (2014); Memarian et al. (2017); Bourassa et al. (2017)
Emotion acceptance	Reduces subjective distress ; successfully used to treat mood and anxiety disorders; improves pain tolerance	reviewed in Hayes (2004), in Lindsay & Creswell (2017), andin Kohl, Rief, & Glombiewski (2012); Dan-Glauser & Gross, (2015); Arch et al. (2012); Forman, Herbert, Moitra, Yeomans, & Geller (2007)	Likely stregnthens LPFC-amygdala functional connectivity	Smoski et al. (2015); Ellard et al. (2017); Young et al. (2017); Young et al. (2019)
Cognitive reappraisal (incl. distancing)	Reduces subjective distress; important component of CBT	reviewed in Powers & LaBar (2019), in Augustine & Hemenover (2009), in Webb et al. (2012), in John & Gross (2004), in Southwick & Charney (2012), and in Troy & Mauss (2011)	Reduces autonomic arousal; reduces amygdala activation, likely via LPFC	reviewed in Buhle et al. (2014) and in Powers & LaBar (2019); Klumpp et al. (2018); Morawetz, Bode, Baudewig, & Heekeren (2017); d'Arbeloff et al. (2018); Feeser et al. (2014); Marques et al. (2018); Denny et al. (2015)
Self-efficacy (incl. perception of control)	Better mental health, esp. following trauma; facilitates mental health interventions; reduces pain perception	reviewed in Benight & Bandura (2004) and in Schwarzer & Warner (2013); Blackburn & Owens (2015); Schnoll et al. (2011); Schuck, Otten, Kleinjan, Bricker, & Engels (2014); Goldin et al. (2012); Zlomuzica, Preusser, Schneider, & Margraf, 2015; Morina et al. (2018); Bowers (1968); Mackie, Coda, & Hill (1991)	Reduces activation in pain network and amygdala, likely via LPFC; alters MPFC connectivity, including with amygdala	Salomons et al. (2004); Brascher et al. (2016); Wiech et al. (2006); Titcombe-Parekh et al. (2018); M. Ono et al. (2017)

Cognitive Bias Modification (CBM)	Reduces depression and anxiety symptoms	reviewed in Koster & Hoorelbeke (2015), in Jones & Sharpe (2017), and in Mogg, Waters, & Bradley (2017)	Decreases amygdala reactivity; increases LPFC activation; LPFC stimulation improves outcome	Hiland et al. (2019); Taylor et al. (2014); Browning, Holmes, Murphy, Goodwin, & Harmer (2010); Clarke, Browning, Hammond, Notebaert, & MacLeod (2014); Heeren et al. (2017); Ironside, O'Shea, Cowen, & Harmer (2016)
2. Upregulating the positive				
2a. Psychologically (Positivity)				
Optimism	Improves mental and physical health, esp. following stress; improves psychotherapy outcome; decreases pain perception	reviewed in Scheier & Carver (2018); L. O. Lee et al. (2019); Seligman et al. (1988); Reivich, Gillham, Chaplin, & Seligman (2013); Seligman, Steen, Park, & Peterson (2005); L. A. King (2001); Meevissen, Peters, & Alberts (2011); Hanssen, Peters, Vlaeyen, Meevissen, & Vancleef (2013)	Increases dopaminergic neural firing; activates reward network, including MPFC and ventral striatum; greater striatal gray matter	Schultz, Dayan, & Montague (1997); Knutson, Adams, Fong, & Hommer (2001); Sharot et al. (2012); Kuzmanovic, Jefferson, & Vogeley (2016); Yamada et al. (2013); Flagan & Beer (2013); D'Argembeau et al. (2008); Benoit et al. (2014); Gerlach et al. (2014); Lai, Wang, Zhao, Qiu, & Gong (2019)
Smiling	Improves subjective affect; greater longevity	reviewed in Fernández-Dols & Crivelli (2013), in Reisenzein, Studtmann, & Horstmann (2013), and in Coles, Larsen, & Lench (2019); Seder & Oishi (2011); Abel & Kruger (2010); Kraft & Pressman (2012)	Reduces autonomic arousal during stress recovery; activates the striatum, MPFC, and amygdala; activates the same patterns of regional brain activity as spontaneous smiling	Kraft & Pressman (2012); Ekman & Davidson (1993); Hennenlotter et al. (2005); Hsu, Sims, & Chakrabarti (2018); T. W. Lee, Josephs, Dolan, & Critchley (2006)
Humor	Lower mortality; improves mental health outcomes, incl. following stress	reviewed in Kuiper (2012), in Bonanno (2004), and in Southwick et al. (2005); Sliter, Kale, & Yuan (2014); Romundstad, Svebak, Holen, & Holmen (2016); Ford, Lappi, O'Connor, & Banos (2017); Maiolino & Kuiper (2016)	Activates reward network, incl. VTA, nucleus accumbens, amygdala, and medial orbitofrontal cortex	Chan et al. (2018); Goel & Dolan (2001); Mobbs, Greicius, Abdel-Azim, Menon, & Reiss (2003); Shibata, Terasawa, & Umeda (2014); Bartolo, Benuzzi, Nocetti, Baraldi, & Nichelle (2006); Mobbs, Hagan, Azim, Menon, & Reiss (2005); Amir & Biederman (2016)
Self-affirmation	Improves subjective affect, esp. following stress; facilitates executive function	reviewed in G. L. Cohen & Sherman (2014) and in Steele (1988); Hales, Wesselmann, & Williams (2016); Creswell, Dutcher, Klein, Harris, & Levine (2013); Churchill, Jessop, Green, & Harris (2018); P. S. Harris, Harris, & Miles (2017); Schmeichel & Vohs (2009); Logel, Kathmandu, & Cohen (2018); Brady et al. (2016)	Reduces autonomic response to stress; activates reward network, incl. ventral striatum and MPFC	Sherman, Bunyan, Creswell, & Jaremka (2009); Cascio et al. (2016); Dutcher et al. (2016)
Positive memories	Improves subjective affect, incl. in clinical samples	reviewed in Westermann, Spies, Stahl, & Hesse (1996); Johnson, Gooding, Wood, Fair, & Tarrier (2013); Panagioti, Gooding, & Tarrier (2012); Arditte Hall, De Raedt, Timpano, & Joormann (2018)	Reduces HPA stress response; activates reward network, incl. nucleus accumbens and MPFC	Ramirez et al. (2015); Speer & Delgado (2017); Speer, Bhanji, & Delgado (2014); Suardi et al. (2016)
2b. Physically				
Sleep	Improves mental and physical health; may also facilitate stress recovery	reviewed in McEwen (2006), in Irwin, Olmstead, & Carroll (2016), in Rumble, White, & Benca (2015), in Vandekerckhove & Cluydts (2010), and in A. N. Goldstein & Walker (2014); N. K. Tang, Fiecas, Afolalu, & Wolke (2017)	Reduces autonomic, HPA, and amygdala distresss response; affects amygdala-MPFC functional connectivity; activates reward network; affects MPFC-to-accumbens signaling and integrity of ventral striatum	reviewed in Krause et al. (2017), in Meerlo, Sgoifo, & Suchecki (2008), in van Dalfsen & Markus (2018), in Goldstein-Piekarski, Greer, Saletin, & Walker (2015), and in Volkow et al. (2012); Motomura et al. (2013); Yoo, Gujar, Hu, Jolesz, & Walker (2007); Perogamvros & Schwartz (2012); Z. Liu et al. (2016); Whitman et al. (2017)
Exercise	Improves physical and mental health; improves executive function	reviewed in Ashdown-Franks et al. (2020), in Kramer & Erickson (2007), in Penedo & Dahn (2005), and in Warburton & Bredin (2017)	Neuroplasticity in LPFC and MPFC; activates reward network and affects its integrity	Basso & Morrell (2015); Greenwood et al. (2011); Meeusen & Fontenelle (2012); Herrera et al. (2016); Marais, Stein, & Daniels (2009); Mul et al. (2018); Batouli & Saba (2017); Dang et al. (2017); Robertson et al. (2016); C. Chen et al. (2017); Byun et al. (2014).
Food restriction	Increases healthy lifespan; can improve physical and mental health	reviewed in Longo & Mattson (2014), in C. Lee & Longo (2016), and in Horne, Muhlestein, & Anderson (2015); Martin et al. (2016); Ravussin et al. (2015)	Facilitates amygdalar fear extinction; has neuroprotective effects on striatal and midbrain dopamine neurons; may activate reward network via orexin	Verma et al. (2016); Maalouf, Rho, & Mattson (2009); Maswood et al. (2004); Bruce-Keller, Umberger, McFall, & Mattson (1999); Marie, Bralet, Gueldry, & Bralet (1990); G. C. Harris & Aston-Jones (2006); Lutter et al. (2008); Manchishi, Cui, Zou, Cheng, & Li (2018); Zhang et al. (2015)

Gut microbiome	Increase healthy lifespan; can improve physical and mental health	reviewed in Long-Smith et al. (2020), in Pereira et al. (2020), in Spielman, Gibson, & Klegeris (2018), in Mayer, Knight, Mazmanian, Cryan, & Tillisch (2014), in Foster, Rinaman, & Cryan (2017), in Sarkar et al. (2016), and in Cryan & Dinan (2012); Buffington et al. (2016); Gacias et al. (2016); Jiang et al. (2015); Luna & Foster (2015); Sharon, Sampson, Geschwind, & Mazmanian (2016); Patterson et al. (2014); Dinan, Stanton, & Cryan (2013); Butler, Cryan, & Dinan (2019); R. T. Liu, Walsh, & Sheehan (2019)	Regulates amygdala fear-related function & morphology; modulates HPA stress-response; can affect microglia growth in VTA; can affect prefrontal and striatal dopamine; associated with reward anticipation response in ventral striatum and with connectivity of the network, incl. amygdala and nucleus accumbens	reviewed in Cryan & Dinan (2012); Luczynski, McVey Neufeld, et al. (2016); Sarkar et al. (2018); Sharon et al. (2016); reviewed in de Weerth (2017); Foster et al. (2017); Hoban et al. (2018); Luczynski, Whelan, et al. (2016); Patterson et al. (2014); Aarts et al. (2017); K. Lee et al. (2018); Osadchiy et al. (2018); Wei et al. (2019); Huang et al. (2018)
2c. Socially				
Receiving social validation and support	Improves mental and physical health; lower morbidity and mortality	reviewed in House, Landis, & Umberson (1988), in Buettner (2012), in Holt-Lunstad, Smith, & Layton (2010), in Thoits (2011), in Uchino (2006), and in Southwick et al. (2005); Fowler & Christakis (2008); Chopik & O'Brien (2017)	Reduces distress response (e.g., HPA, pain network); activates reward network, incl. ventral striatum; possibly via oxytocin-reward pathways that promote social attachment and attenuate HPA & ACC stress responses	Gunaydin et al. (2014); reviewed in Kiyokawa & Hennessy (2018) and in Eisenberger (2013); Morelli, Torre, & Eisenberger (2014); Tabibnia, Satpute, & Lieberman (2008); reviewed in Tabibnia & Lieberman (2007); Heinrichs, Baumgartner, Kirschbaum, & Ehlers (2003); Shamay-Tsoory & Abu-Akel (2016); Hung et al. (2017); Morhenn, Beavin, & Zak (2012); Coan, Schaefer, & Davidson (2006)
Giving social validation and support	Better mental and physical health; greater longevity	reviewed in Eisenberger (2013), in Southwick et al. (2005), in Curry et al. (2018), in Post (2005), and in Rachman (1979); Inagaki et al. (2016); S. L. Brown, Brown, House, & Smith (2008)	Reduces distress response (e.g., amygdala, autonomic); activates reward network, incl. ventral striatum	reviewed in Eisenberger (2013), in Cutler & Campbell-Meiklejohn (2019), and in Hubbard, Harbaugh, Srivastava, Degras, & Mayr (2016); Inagaki et al. (2016); Inagaki & Eisenberger (2012; 2016); Zaki & Mitchell (2011); Decety, Jackson, Sommerville, Chaminade, & Meltzoff (2004)
Giving: Compassion	Improves mental and physical health; improves subjective affect; improves psychotherapy outcome	Fredrickson, Cohn, Coffey, Pek, & Finkel (2008); reviewed in Hofmann, Grossman, & Hinton (2011); Cosley, McCoy, Saslow, & Epel (2010); Engert, Kok, Papassotiriou, Chrousos, & Singer (2017); Klimecki, Leiberg, Ricard, & Singer (2014)	Reduces autonomic and HPA stress response; activates reward network , incl. ventral striatum & MPFC	Cosley, McCoy, Saslow, & Epel (2010); Engert, Kok, Papassotiriou, Chrousos, & Singer (2017); reviewed in Hofmann et al. (2011); Klimecki et al. (2014); Preckel, Kanske, & Singer (2018); Weng et al. (2013); Engen & Singer, (2015); J. W. Kim et al. (2009); Mercadillo, Diaz, Pasaye, & Barrios (2011)
Giving: Gratitude	Improves mental and physical health; improves psychotherapy outcome	Hill, Allemand, & Roberts (2013); reviewed in Seligman et al. (2005); Kaczmarek et al. (2015); S. L. Kerr, O'Donovan, & Pepping (2015); Wong et al. (2018)	Reduces autonomic arousal; associated with connectivity of and activation in reward network, incl. ventral striatum & MPFC; has lasting effect on MPFC activation	Kyeong, Kim, Kim, Kim, & Kim (2017); Yu, Gao, Zhou, & Zhou (2018); Karns, Moore, & Mayr (2017); Kini, Wong, McInnis, Gabana, & Brown (2016); Fox, Kaplan, Damasio, & Damasio (2015); Zahn et al. (2009)
3. Self-Transcendence				
Mindfulness	Can improve mental and physical health, as well as executive function; effective treatment tool for depression and anxiety	reviewed in Holzel et al., 2011), in Lin, Callahan, & Moser, 2018), in Vago & Silbersweig, 2012), in Creswell & Lindsay, 2014), in Creswell (2017), and in Goldberg et al. (2018); Basso, McHale, Ende, Oberlin, & Suzuki (2019); Wielgosz, Goldberg, Kral, Dunne, & Davidson (2019)	Changes amygdala and PFC structure and function associated with self-regulation and attention; reduces activity in DMN	reviewed in Holzel et al. (2011), in Tang et al. (2015), in Brewer et al. (2011), in Fox et al. (2016), and in Guendelman et al. (2017); Kral et al. (2018)
Purpose in life (incl. religion and spirituality)	Can improve mental and physical health	Reviewed in Reker et al. (1987), in Zika & Chamberlain (1992), in Ryan & Deci (2001), in Southwick et al. (2005), in Cheadle & Dunkel Schetter (2017), in Bouso, Dos Santos, Alcazar-Corcoles, & Hallak (2018), in Nunes et al. (2016), and in Myers (2000); Alim et al. (2008); Griffiths et al. (2016; Ross et al. (2016)	Associated with reduced activity in distress networks; associated with sustained activity in reward network; reduces activity in and functional connectivity within DMN	Inzlicht et al. (2011); Heller et al. (2013); Van Reekum et al. (2007); Kang et al. (2017); Raichle et al. (2001); A. C. Chen et al. (2013); Uddin, Kelly, Biswal, Xavier Castellanos, & Milham (2009); Wu et al. (2010); Carhart-Harris et al. (2012); Urgesi, Aglioti, Skrap, & Fabbro (2010); Crescentini, Aglioti, Fabbro, & Urgesi (2014); Crescentini, Di Bucchianico, Fabbro, & Urgesi (2015); Palhano-Fontes et al. (2015); Carhart-Harris et al. (2016)

Exposure to nature	Improves mental and physical health, as well as executive function	reviewed in Annerstedt & Wahrborg (2011), in Hartig, Mitchell, de Vries, & Frumkin (2014), in Keniger, Gaston, Irvine, & Fuller (2013), in Haluza, Schonbauer, & Cervinka (2014), in Kuo (2015), and in Tost, Champagne, & Meyer-Lindenberg (2015); Stothard et al. (2017)	Reduces autonomic and HPA activity; reduces activity in DMN; correlated with gray matter in DMN	reviewed in Norwood et al. (2019); Haluza, Schonbauer, & Cervinka (2014); Keniger et al. (2013); D. K. Brown, Barton, & Gladwell (2013); Bratman, Hamilton, Hahn, Daily, & Gross (2015)
Flow	Improves subjective affect; better mental health	Csikszentmihalyi & LeFevre (1989); Rankin, Walsh, & Sweeny (2018); Mosing, Butkovic, & Ullen (2018); Ascenso, Williamon, & Perkins (2016); Fritz & Avsec (2007); Gould, Dieffenbach, & Moffett (2010); Jackson, Ford, Kimiecik, & Marsh (1998); Ilies et al. (2017); Sadlo (2016)	Reduces autonomic and amygdala arousal; activates striatum; correlated with dopamine receptor availability and gene polymorphism; reduces activity in DMN	Duan et al. (2012); Knierim, Rissler, Dorner, Maedche, & Weinhardt, 2018); Ulrich, Keller, & Gron (2016); Ulrich et al. (2014); Huskey et al. (2018); de Manzano et al. (2013); Gyurkovics et al. (2016)