CHAPTER

7

Is Depressive Rumination Rational?

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> *…he who learns must suffer. And even in our sleep pain that cannot forget, falls drop by drop upon the heart, and in our own despite, against our will, comes wisdom to us…* **(*Hamilton, Trans., 1958*, *p. 170*)**

7.1 INTRODUCTION

Nearly half a century ago Hempel (1965, p. 150) opined that "classifications of mental disorders will increasingly reflect theoretical considerations." More than three decades later Murphy & Stich (2000), in addition to claiming that clinical practice is based upon false theory, lamented that little progress had been made along the lines that Hempel anticipated. They contended that evolutionary psychology has a natural and central role to play in the development of a new taxonomy that is grounded in natural science. One goal of such a project is the determination of just "what conditions count as disorders at all" (2000, p. 71).

Surveying candidate theoretical developments, Murphy and Stich cited several theories of depression, all of which concern problems pertaining to social relations (2000, pp. 74–84): (1) malfunction of a reciprocal altruism module, (2) social competition switching strategies, and (3)

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defection. As for the first among these, McGuire and Troisi (1998) argue that depression results from a tendency to overestimate one's own contributions to social relationships while underestimating the contributions of others. Because of their chronic misestimates they feel exploited and therefore choose to avoid social interaction. As for the second, Nesse (2000) hypothesizes that depression is an evolved response to the loss of status, an introspective marker that indicates a need to switch social strategies. And, as for the third, Watson and Andrews (2002) argue that depression is a means by which persons can derive more investment from their social network, as in the case of postpartum depression when mothers feel unable to nurture their children unless conspecifics lend more assistance.

Even those who voice skepticism of "social theories of depression" (Raison & Miller, 2013) accord some recognition to the possibility that social stressors may have played a significant evolutionary role. These authors argue that risk alleles for depression have been retained in the human genome because they encode for an "integrated suite" of immunological and behavioral responses that promote defense against infection, especially during infancy when the immune system is not fully operational and when selection pressures from infection are strongest. The idea is that depression is associated with elevated immune inflammatory responses, and these elevated responses are critical to fighting infection.[1] This seems to sit well with the social theories, because psychosocial stress puts people at risk for developing depression, even while that same stress serves as a potent activator of immune defense by increasing inflammation. But the authors argue that social concerns, per se, are secondary: what matters most is that "in ancestral environments, the association between stress perception and risk of subsequent wounding was reliable enough that evolution operated by…(favoring) organisms that prepotently activated inflammatory systems in response to a wide array of environmental threats and challenges" (Raison & Miller, 2013, p. 22). These stressors would have, incidentally, included psychosocial stressors.

But many problems append to these "evolutionary psychology" approaches to explaining depression. Not only can they be found wanting on conceptual grounds (Woodward & Cowie, 2004), the standard for an ideally complete adaptation explanation is extremely difficult to satisfy (Brandon, 1990). Yet more problematically, there is a dearth of experimental evidence showing that humans possess the psychological mechanisms posited by theories based upon evolutionary psychology (Buller, 2005).

Very recently, some among those who presuppose an adaptive advantage for dealing with social pressures that might be derived from depression have turned their attention to this problem, the dearth of experimental

evidence concerning the posited psychological mechanisms. They have argued that work on the typical animal models, mice and rats, can mislead, because they do not have the right kind of social organization (Hendrie & Pickles, 2009). Next, turning their attention to humans, they hypothesize that a specific brain region, the third ventricle,[2] which appears to mediate many behaviors associated with depression—sleep-wake cycles, appetite for food and sex, social affiliations, and fear or defensive behaviors—can help focus experimental work, especially with regard to development of more drug-based therapies (Hendrie & Pickles, 2010).

Whether experimental work focused on the third ventricle will succeed remains to be seen. But what does seem clear is that one can acknowledge the utility of negative emotional states when responding to stressors, without proclaiming that those states necessarily confer an adaptive advantage. Such negative states might be more like height: for men reproductive fitness increases steeply with increasing height, up to the point at which musculoskeletal and other health problems begin to outweigh the social and mating advantages of being tall (Nettle, 2004). In other words, there is only a thin adaptive peak between being too tall or too short, and in every generation there is a normal distribution of statures around that peak. On this view, one could argue that increasing height—or a disposition to respond to certain stressors with depression—is selected for because of the beneficial effects limned earlier, until that is the negative effects begin to outweigh the positive.[3] Depression then would arise "because of the tendency of the affect system, in extreme deviations from center, to go into a self-reinforcing cycle, at both the neurobiological and psychological level, which traps it at pathological negativity" (Nettle, 2004, p. 99). In a word, depression might not be an adaptation so much as it is dysregulation of mechanisms underlying normal variation.

Still, certain nagging facts about depression continue to suggest that it is best thought of as an adaptation. Especially noteworthy is that depression is unique among mental health problems in being so commonplace (Hagen, 2011, p. 720). Lifetime prevalence of disorders like schizophrenia or autism is about 1% or less, while it exceeds 20% for major depressive disorder (MDD). What makes this difference even more striking is that, when epidemiological estimates are based on longitudinal data, lifetime risks for succumbing to depression approach 50% (Blanco et al., 2008). It is this extremely high incidence rate, inter alia, which suggests to some theorists that some distinctive feature of depression—for example, rumination—is the manifestation of a properly functioning stress response mechanism, not a biological malfunction. Therefore, in the next section we present this most recent attempt at formulating an evolutionary explanation, a formulation that emphasizes depression's ruminative cognitive style.

7.2 THE ANALYTICAL RUMINATION HYPOTHESIS

Andrews and Thomson (2009) argue that unipolar depression's ruminations should be thought of as analogous to fever: that is, both are mechanisms that have evolved so as to produce effective responses to stressors.[4] On this view, organisms evolve stress response mechanisms that are triggered by specific stressors. Because resources cannot be devoted to all problems at once, stress response mechanisms prioritize fitness-related goals, coordinate trade-offs among body and mental functions—physiology, immune functions, attention and cognition, etc.—and allocate resources in such a way as to reflect priorities and trade-offs.

According to the Andrews and Thomson "Analytical Rumination Hypothesis" (ARH), the intrusive, persistent ruminating over social problems so characteristic of depression is not, per se, a good thing.[5] Nevertheless, it is an evolutionary adaptation. For adaptations, as can be seen from the example of fever, trade-offs are commonplace. Fever has costs: it is metabolically expensive and it can have deleterious effects upon work, sexual function, social relations, and so forth. But fever also enables organisms to coordinate aspects of the immune system in response to a stressor, infection (Kluger, 1986). Impairments associated with fever, thus understood, are not the result of a disorder; rather, they are the outcome of an adaptive trade-off, a trade-off that is necessary in order to produce an effective response to the stress of infection.

As for depression, consistent with the evolutionary theories discussed in the previous section, here the salient stressor is usually a social problem. Depression's costs are similar to those of fever: correlating with the sad mood and anhedonia are a host of deleterious effects on sexual functioning, work, sleeping, eating and social relations. But like fever, according to the Andrews and Thomson "design analysis" argument, there is an upside as well. In developing their argument, they presuppose that if a trait's features "proficiently promote" a specific effect, this very fact can be taken to support the claim that the effect is an evolved function of the trait, because of the unlikelihood that the trait's features could be wholly attributable to chance.

The effects they believe to be proficiently promoted by sadness or depression are four: (1) an analytical reasoning style, (2) accompanied by coordination of body systems to promote ruminative analysis of the triggering problem, (3) that aids development and evaluation of potential solutions, and (4) that diminishes resources available for other cognitive tasks, thereby resulting in the decrements often exhibited when depressed patients perform problem solving or cognitive tasks in the laboratory. The positive effect of the trade-off then is enhanced likelihood of being able to solve a serious social problem, at the cost of diminished performance in other domains. What is unique about the intrusive thoughts

associated with rumination is that they involve analysis (Andrews & Thomson, 2009, p. 629). And, as is the case with fever, depressed moods are not pleasant, but they are claimed to proficiently promote a gene-propagating effect.

Why does this matter? Among other things, it goes to the heart of whether people suffering from depression should be treated with medication. ARH suggests that psychotherapies that can assist people to identify and solve problems should be favored, for example, having patients write about the thoughts and feelings associated with depressed episodes (Andrews & Thomson, 2009, p. 635). Andrews and Thomson argue (2009, p. 645) that people should "stop trying to quickly resolve their pain with simple solutions, transition to a slower, analytical approach to problem solving, and *learn how to endure the pain until the problem is solved*."[6] ARH proposes that it is the emotional pain and extended nature of depressive rumination that should be valued: were it not for these characteristics, people would not be motivated to devote the long-term effort essential to solving complex problems.[7] What is needed—what should not be avoided or medicated away—is a slow, problem-solving approach that includes learning how to endure pain. Learning to endure and make use of the pain associated with depression might be part of depression's evolutionary heritage, a heritage that explains the "venerable philosophical traditions that view emotional pain as the impetus for growth and insight into oneself and the problems of life" (Andrews & Thomson, 2009, p. 645)

7.3 RUMINATION

The ARH emphasizes the importance of rumination, but "rumination" is a multi-dimensional concept, admitting of distinct modes. According to the "response style theory" (RST), the type of rumination that accompanies depression is "a mode of responding to distress that involves repetitively and passively focusing on symptoms of distress and on the possible causes and consequences of these symptoms" (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008, p. 400; cf. Nejad, Fossati, & Lemogne, 2013). RST holds that such passive, repetitive rumination can neither salve feelings nor solve problems. On the contrary, it has multiple deleterious consequences: it aggravates depressed moods by activating negative thoughts and memories, it interferes with problem solving by making thought more pessimistic and fatalistic, it interferes with instrumental behavior, and it causes loss of social support which further fuels depression.[8]

More generally, rumination may be thought of as a disposition to dwell on negative stimuli or memories and inhibit processing of or accessing of positive stimuli (Nolen-Hoeksema et al., 2008, pp. 411–412). For example, when rumination is induced in subjects—commonly by asking that they

think about a recent, stressful event, like a fight or the death of a loved one—these negative dispositions become especially evident. After 10 min of thinking about unpleasant interpersonal exchanges or loss, subjects exhibit negative biases in retrieving autobiographical information, predicting the future, and distributing attentional resources. Dot probe tasks of attention, whether auditory or visual,[9] reveal dispositions among ruminators to attend to the negative more than the positive, whether the stimuli are task relevant or task irrelevant (Foland-Ross et al., 2013).

Rumination is assessed by a 22-item scale that describes responses to depressed mood that are self-focused, symptom-focused, and focused on potential consequences of one's mood. Examples include the following: "I think, 'Why do I react this way?'; "I think about how hard it is to concentrate"; and, "I think I won't be able to do my job if I don't snap out of this' (Nolen-Hoeksema et al., 2008, p. 401). Tendencies measured by this scale tend to be relatively stable, even for persons whose depressive symptoms change significantly.

If rumination is indeed so stable though, how is it related to depression, especially MDD? One possibility is that rumination contributes to a person's descent from dysphoria into MDD, "but once an individual is in an episode, other autonomous self-perpetuating processes emerge that determine the duration of episodes" (Nolen-Hoeksema et al., 2008, p. 404). Among these processes are elevated peripheral levels of norepinephrine metabolites, increased phasic REM sleep, poor sleep maintenance, hypercortisolism, decreased cerebral blood flow and glucose metabolism within anterior cortical structures accompanied by increased blood flow, and glucose metabolism in paralimbic regions.[10] Even if these processes themselves did not trigger MDD symptoms, they may help to maintain and extend those symptoms.

But is it ruminations of any type that trigger, extend, or maintain depression? As mentioned previously, "rumination" is multidimensional, admitting of constructive and nonconstructive types.[11] The former, variously referred to as "pondering," "self-reflective," or "adaptive," is concrete and process-focused; the latter, variously referred to as "brooding," "passive," or "maladaptive," is abstract and associated with a strong negative bias (Nejad et al., 2013, pp. 1–2; Nolen-Hoeksema et al., 2008, pp. 413–414; Trapnell & Campbell, 1999; Treynor, Gonzalez, & Nolen-Hoeksema, 2003; Watkins, & Moulds, 2005). Clinical-scale items that target brooding reflect abstract forms of self-focus that emphasize obstacles to overcoming problems: for example, "I think, 'what am I doing to deserve this?" or "I think, 'why can't I handle problems better?" It is this—brooding—that has been found to positively correlate with depression, both concurrently and longitudinally.

The profile for pondering is different. For pondering, typical clinical-scale items include, for example, "I go someplace alone to think about my feelings" or "I analyze recent events to try to understand why I am

depressed." Unlike brooding, pondering is positively correlated only with concurrent depression; it is negatively correlated with depression longitudinally (Treynor et al., 2003). Brooding, therefore is a trait, while pondering is not.

In a review of recent studies attempting to flesh out the distinction between these two, Nolen-Hoeksema et al. (2008, p. 414) conclude that pondering is "a form of self-reflection that may be emotionally distressing in the short-run, but *adaptive in the long run* because it leads to successful problem solving."[12] For those who do become clinically depressed pondering is a constructive response. Brooding, on the other hand, appears to be a trait that can trigger, maintain, or aggravate depressive moods. In sum, it appears that pondering—but not brooding—is that which can play a role of the sort envisioned by advocates of the ARH.

7.4 RUMINATION AND THE RESTING STATE HYPOTHESIS OF MDD

The relevance of this distinction between the different types of ruminative cognition to MDD's neural substrate has recently been established by Hamilton et al. (2011). The authors discovered that dominance by the brain's "default mode network" (DMN) positively correlates with elevated levels of brooding, but with only low levels of pondering (cf., Hamilton, Chen, & Gotlib, 2013). It seems, thereby, that the neuronal activity in virtue of which people are caused to suffer the symptoms of depression is not related to the type of rumination that the ARH requires. And this link between brooding and the DMN can serve as a point of departure for showing why it is not likely that depressive rumination is an adaptation, and why it does not afford a rational path "for growth and insight into oneself and the problems of life."

The concept, DMN, was introduced by Raichle et al. (2001) to describe a set of dispersed brain regions[13] that exhibit a stable pattern of resting state metabolic activity and blood flow. This resting state "connectivity" pattern—as manifest by signal fluctuations that covary—is identifiable when subjects undergo functional magnetic resonance imaging (fMRI). High levels of DMN activity occur when people are daydreaming, mind wandering, or otherwise not engaged in tasks that involve attending to or responding to specific external stimuli.[14] Hence it is thought of as a default mode (Raichle, 2010).

This resting state or default mode is particularly intriguing because the expenditure of energy when the brain reacts to external stimuli is only slightly more than what is required when it is "at rest" or, say, daydreaming. The brain's energy budget is about 20% of the body's total, a surprisingly large amount.[15] And of this 20%, between 60% and 80% supports

communication among neurons and their supporting cells (Raichle, 2006a,b; Raichle and Mintun, 2006; pp. 467–468). Yet more intriguingly, when the brain responds to the external environment only a small increment in energy consumption—less than 5% more than the brain's resting blood flow—is required. Therefore, it seems that when the brain is not responding to the world, its spontaneous, intrinsic, "resting," activity is not without purpose. It seems less like a resting and more like a preparatory or anticipatory state. In fact, with respect to overall brain function, the intrinsic activity may be far more important than evoked activity.

When people engage in goal-directed, externally oriented cognition, on the other hand, these DMN regions exhibit a distinctive pattern of deactivation. That is, when persons engage in stimulus- or task-induced activity, the DMN exhibits a decrease in metabolic activity and blood flow. The DMN then is inversely related to what is often referred to as the task-positive network (TPN), a network that includes regions like the dorsolateral prefrontal cortex (DLPFC), a network that becomes more active when attention must be distributed to tasks involving external stimuli (Northoff, 2013a, pp. 73–118).[16] In effect what Hamilton et al. (2011) discovered is that in depressed patients—but not in healthy subjects—greater DMN activity relative to TPN activity correlated with depressive rumination, but not with pondering. This discovery is important because it dovetails neatly with two converging lines of research that might be able to contribute to a theoretically motivated explanation of depression of the sort envisioned by Hempel 50 years ago.

The first of these, cognitive science research extending over three decades, has been exploring the relationship between self-focused attention and negative affect (Ingram, 1990; Mor & Winquist, 2002; Pyszczynski & Greenberg, 1987). Echoing the brooding-pondering distinction adumbrated earlier, these findings indicate that self-focus is not a unitary concept: ruminative self-focus—viz, brooding—differs from other types of self-focus in that it tends to be repetitive, unproductive, and inclined to dwell on private, negative aspects of self, thereby intensifying negative moods (Mor & Winquist, 2002). Although positive self-focus does occur, to date most research suggests that the brooding, ruminative aspects of self-focus predominate.[17]

The second line of research, that which will be considered in some detail here, concerns imaging studies of the brain's resting state that have helped illuminate how DMN dominance might be related to ruminative self-focus in the etiology of depression as well as in its resistance to therapeutic treatments.[18] Northoff (2013b, 251–327; also see Northoff & Bermpohl, 2004; Northoff et al., 2006) has argued that certain core regions of the DMN, in particular anterior cortical midline structures (aCMS) like the perigenual anterior cingulate cortex (PACC), are uniquely involved in the processing of self-related stimuli. The focus, despite being motivated by philosophical

concerns (Northoff, 2012; Lane, 2012), is not on self as conventionally understood by philosophers (Lane, 2012, Lane & Liang, 2010, 2011).[19] Instead, it is on what Northoff (2013a, p. 253) refers to as "organization in relation to the organism itself," or what Lane (2014, 2015) refers to as the "subpersonal self." In short, "self"-focus so understood refers to the neuronal mechanisms in virtue of which stimuli are perceived as or judged to be related to "self" (or, "this organism"). Despite this difference in how "self" is conceived, the mental and the neuronal, the personal and the subpersonal, levels can be shown to merge in many experimental or clinical settings. Of most direct relevance to the subject at hand, ruminative self-focus and resting state activity in the PACC correlate positively with one another in patients suffering from depression (Grimm et al., 2009).

Metaphorically, the subpersonal self can be thought of as "a neuronal grid or structure" onto which stimuli are mapped (Northoff, 2013a, p. 275). When a stimulus with specific content and function, say, a baseball, is perceived, for one of the authors (Lane), it is likely to be perceived and subsequently judged as highly self-relevant; for the other author (Northoff), not. Accordingly, by hypothesis, Lane's neuronal activity should exhibit strong "overlap" with the resting state activity typically exhibited by aCMS regions (Northoff, 2013b, pp. 257–258). For Northoff, the degree of "overlap" should be considerably less.[20]

How then might the resting state's "neuronal grid" be related to the kind of self-focus that typifies depression? In order to better explain this relationship, first it should be noted that resting state activity is not confined to the DMN or the CMS (Northoff, Qin, & Nakao, 2010). Electrophysiological studies show that resting activity is prevalent throughout the entire brain: spontaneous neuronal oscillations and synchronizations have been identified in various parts of the brain, including the thalamus, the hypothalamus, the ventral tegmental area, the hippocampus, the visual cortex, and so forth. Because resting state activity is so widespread, it can influence all manner of neuronal activity that is induced by external stimuli; indeed, patterns of resting state activity in different brain regions can also directly influence one another.

Second, it should be noted that the neuroanatomy specified by DMN or CMS may have failed to identify the neural substrate of self with sufficient precision. Recent findings suggest that, although involved in self-processing, neither the DMN nor the CMS can be claimed to be uniquely involved in self-processing (Qin & Northoff, 2011). In other words, they engage in self-related, but not necessarily self-specific processing.

In order to hone in on the neural underpinnings of that which is self-specific, instead of merely emphasizing the distinction between medial and lateral regions as is done by CMS and DMN, a threefold distinction among paralimbic, medial heteromodal, and exterosensorimoter/lateral regions provides a more appropriate framework (Northoff, Wiebking,

Feinberg, & Panksepp, 2011). Not only is this distinction compatible with distinctions based upon cytoarchitecture, neurochemistry, and connectional features (Feinberg, 2009, 2011), it also links the PACC to the insula within the anterior paralimbic region as specific for the mediation of self (Northoff, 2013a, pp. 255–256), a finding that converges with important work on the neural basis of self being carried out by Craig (2009).[21] Of special relevance to our concerns here, the paralimbic regions are anatomically linked to ancient emotional and motivational networks. Given the relationship between paralimbic regions and self, along with their relationship to affect or motivation, it should not surprise that excessive self-focus can have significant consequences for mental health.

And, third, it should be noted that the resting state does not just passively respond to stimuli. Instead, it actively contributes to the constitution of mental states, which is one reason why the term "resting" state might be a misnomer. The state is more usefully regarded as the "brain's intrinsic activity."[22] Consider, for example, a study published by our group:[23] participants were asked to indicate whether emotional photos were self-related. Many of their choices struck us as odd, in that they did not comport with our intuitive assessments of participants' personalities. But we discovered that the degree of low-frequency alpha power (8–9 Hz)—even before the photos were presented—could predict the degree of self-relatedness. That is, a higher degree of alpha power disposes participants to experience pictures as more self-related; a lower degree, as less. Moreover, during the resting state, elevated levels of glutamate, which is typically associated with excitatory functions, were observed in the PACC. These findings suggest that PACC glutamate can predispose subjects to spontaneous fluctuations in frequencies, like low alpha, which in turn predisposes those subjects to perceiving stimuli as self-specific.

The "neuronal grid" idea helps explain the behavioral data, the self-relevant choices rendered by participants in the experiment. Even before the photos were shown, EEG waves in a frequency (8–9 Hz) that has previously been associated with self-relatedness were observed.[24] Moreover, during the resting state, elevated levels of glutamate in the PACC seem to predispose participants to having those spontaneous fluctuations in the 8–9 Hz frequency range. The implication seems to be that the resting and prestimulus "neuronal grid" disposes the person to perceive and judge certain stimuli to be highly self-related;[25] the specific content of a stimulus can even be well nigh irrelevant to the determination that a given photo is reported by the subject to be self-related. Inference to the best explanation suggests that elevated levels of an excitatory neurotransmitter in the PACC predispose subjects to "self-specific" frequencies, which in turn result in self-specific judgments. Succinctly, the subject is focused on self and when presented with a forced choice determination concerning an external stimulus is disposed to treat it as self-specific.

But how might these findings relate to depression? The resting state hypothesis (RSH) provides many insights. It aspires to explain MDD by bridging multiple levels, including brain networks, psychological symptoms, biochemical activity, and genetic-molecular mechanisms. The RSH aims to establish a framework that can both explain all existing data and motivate new research. As for the existing data, RSH points up that elevated resting state activity in anterior paralimbic regions—like the PACC and the anterior insula—is one among the most consistent findings in MDD research (Northoff et al., 2011; Northoff, 2013b, pp. 398–407). For this reason, and because of our intent to assess plausibility of the analytical rumination hypothesis, our discussion here is confined to brain networks (the TPN and DMN) and psychological symptoms (self-focus cum negative affect).

In addition to noting that elevated resting state in anterior paralimbic regions is characteristic of MDD, RSH further calls attention to the fact that these same regions are intimately related to basic subcortical regions that mediate processing of ancient or fundamental emotions, including physical distress, disgust, anger, fear, and sadness (Feinberg, 2009, p. 55).[26] RSH also notes that lateral regions which mediate the TPN tend to exhibit lowered resting state activity. Indeed, this contrast between medial and lateral is indicative of a perfectly general pattern of brain activity, the inverse relationship between TPN and DMN described earlier. The two networks tend to interact in an oscillatory, give-and-take, or seesaw manner: when medial regions that underlie self-related, or intereoceptive and emotional processing, undergo excitation, lateral regions that underlie the TPN tend to be inhibited. By contrast, when lateral regions are aroused, medial regions tend to be inhibited.

Concerning the medial-lateral "see-saw" one of the most important studies that sheds light on MDD was a meta-analysis conducted by Alcaro, Panksepp, Witczak, Hayes, & Northoff (2010), an analysis that included all imaging studies of human MDD focused on resting state activity. The authors found that medial regions like the PACC exhibit resting state hyperactivity; they also found that lateral regions like the DLPFC exhibit resting state hypoactivity. The relevant medial regions, in addition to exhibiting hyperactive resting states, also show structural abnormalities: reduced gray matter volume and reduced cell count markers of cellular function. Furthermore, investigations of MDD resting state in animal models reveal a similar pattern of hyperactivity.

What the findings assessed by Alcaro et al. (2010) and others (Northoff et al., 2011, pp. 1935–1945) suggest is that a neural correlate of depressive rumination, brooding, or other psychological symptoms of MDD is an imbalance between paralimbic and lateral activity. Hyperactive resting state activity in critical paralimbic regions might be that in virtue of which self-focus laden with negative affect is precipitated and sustained. This

hyperactivity, accompanied by hypoactivity in lateral regions, may help explain why positive stimuli cannot easily alter an MDD patient's mood: the former, correlating with internal focus, might effectively "block" external stimuli, while the latter seems not disposed to respond to external stimuli. A diminished disposition to respond to external stimuli seems to correlate with an abnormal pattern of rest-stimulus interaction (Northoff et al., 2010), as is exhibited in MDD. In a word, when the resting state is excessively active, it blocks entry of stimuli; when excessively inactive, it is unable to respond to those stimuli.

Assuming that this view of MDD is true, why then is the self-focused rumination so intertwined with the negative affect characteristic of brooding? Why, in short, does depression "hurt?" Panksepp and Watt (2011) suggest that primary-process emotional systems, especially the separation-distress PANIC/GRIEF systems, are the major contributor to this "hurt." What seems to happen is that when PANIC/GRIEF (perhaps as well FEAR and RAGE) occur, if the paralimbic resting state hyperactivity that accompanies intense self-focus insulates the person from positive stimuli, negative emotions can "highjack" the person's overall affective and cognitive states, even the conative states. We include "conative" here because the negative affect can be consolidated and intensified by diminished SEEKING urges: that is, the person is disinclined to break out of these brooding states and seek rewarding stimuli in the external environment. In sum, when PANIC/GRIEF occur in people who exhibit the resting state imbalance described earlier, mental life can be flooded by negative affect in such a way that is aggravated by diminished inclination to go in search of rewarding stimuli.

To help understand how this "flood" of negative affect can have such an extensive impact, it is useful to distinguish between nested and control hierarchies (Feinberg, 2009, pp. 159–185). In nested hierarchies, like the brain, any given level of organization is entirely composed of its constituent parts: higher level cortical regions are not independent of the rest of the brain. Those higher levels regions physically comprise paralimbic, limbic, and other regions. Control hierarchies, on the other hand, have pyramidal structures, like an army; a general is not physically comprised by lower ranking officers and enlisted men. Accordingly, in a control hierarchy constraints can be centralized and emanate from the top; in nested hierarchies there is no centralized control and system constraints are embodied within the hierarchy itself. Although in healthy subjects the brain's nested hierarchy exhibits a pattern of mutual, more-or-less balanced modulation, for those suffering from MDD, top-down modulation is significantly diminished, thereby allowing negative affect to "flood" higher level cognition, notably brooding rumination.

On the biochemical level, what seems to be happening is that the usual excitatory-inhibitory balance that obtains between glutamate and GABA

is upset. Elevated glutamate levels in critical paralimbic regions reflect decreased neural inhibition which is crucial to constraining the excessive self-focus characteristic of rumination. Excessively high levels of resting state activity in the PACC, for example, appear to be mediated by the neural excitation caused by glutamate. On the psychological level this is manifest as extreme self-focus and hopelessness. It are these findings concerning glutamate-ergic excitation in MDD that might explain why GABA-ergic drugs like ketamine can act so quickly (within 24 h) to bring relief to depressed patients who are suicidal (Niciu et al., 2014). GABA-ergic drugs can help dampen the self-focus, thereby making it possible that externally introduced positive stimuli can effectively reduce negative affect.

We regard the RSH as consistent with recent work on the neurobiology of resilience (Kalisch, Muller, & Tuscher, 2015). "Resilience" refers to the empirically observable phenomenon that not all people who are exposed to the same stressors, whether physical or social, succumb to mental health problems like MDD. Accordingly, researchers in this area do not focus on pathology per se; instead, they investigate mechanisms that prevent illness. One such mechanism, we propose, is a balanced give-and-take relationship between TPN and DMN networks. What seems to occur in MDD is that negative emotional responses to stress—say, PANIC/GRIEF or FEAR—cannot be properly adjusted because the imbalance and consequent abnormal self-focus prevents a more positive or commensurate appraisal of stressors.

The RSH hypothesis is as well compatible with work in computational neuroscience that regards the brain as an inference machine. According to the "free-energy principle" (Friston, 2009; Hohwy, 2013), for example, free energy is a quantifiable measure of surprise that can be used to model neuronal simulations of perception and action. This framework presupposes that brains employ hierarchical models dedicated to predicting sensory input with the aim of minimizing free energy, viz, surprise or predictive error. As is the case with our view of the seesaw relationship between networks, here too the idea of hierarchy is crucial: the free energy principle holds that the brain constructs sets of top-down prior expectations about sensory samples from the world. What appears to occur in MDD is "a loss of top-down control over limbic activity" (Carhart-Harris & Friston, 2010, p. 1267).[27] The flood of negative affect and the heightened self-focus as reflected in abnormally elevated paralimbic resting state activity seems to prevent the possibility of reappraising one's situation vis-à-vis stressors in a more positive light, not only because external stimuli are blocked, but also because top-down modulation of negative affect is inhibited. Baldly, the hopelessness that is symptomatic of depression might be explainable as the result of a diminution of surprise, but at the cost of depriving self of new information as well as of the capability to modulate limbic activity.

7.4.1 Other Forms of Self-Focus and Negative Affect

Still it might strike some readers as odd that self-focus as characterized here should be so strongly associated with negative affect and depression, for after all narcissists and those inclined to mind wander in the manner of James Thurber's Walter Mitty seem to be no less turned inward, yet their affective states might be largely positive. Minds insulated from the world can wander into worlds wherein self reigns narcissistically or performs heroically. And when our minds are wholly isolated from the world, as is the case when we dream, some among those dreams are infused with positive affect.

To begin with the last among these three, when the mind is severed from the external world during REM dream sleep, although dream emotion is common[28] and positive emotions are reported, negative emotions predominate (Merritt, Sickgold, Pace-Schott, Williams, & Hobson, 1994). Positive emotions (eg, joy, elation, or eroticism) account for less than one-third of emotion reports; negative emotions (eg, anxiety, fear, or sadness), more than two-thirds (Merritt et al., 1994, p. 50). Some studies even indicate that reports of negative emotions are as high as 80% of the total, and that "misfortune" is the norm for the "dream self" (Revonsuo, 2006, pp. 404–413). Moreover, although during the first half of an REM dream, the positive–negative imbalance is somewhat less, during the final half 76% of emotions are negative (Merritt et al., 1994, p. 56). Consistent with the RSH, these findings suggest that the longer self-focus persists, the more extended the period of insulation from external stimuli, the worse the mood.

Second, perhaps though the case with mind wandering or daydreaming is different? Cannot we emulate Walter Mitty? The data suggest that Walter Mitty is the exception not the rule. Carciofo, Du, Song, & Zhang (2014) report that many studies have shown there to be a link between frequent mind wandering and negative affect: Giambra and Traynor (1978) discovered correlations between frequency of mind wandering and three questionnaire measures of depression (cf., Mar, Mason, & Litvack, 2012), and Smallwood, O'Connor, Sudbery, & Obonsawin (2007) discovered that mind wandering is associated with dysphoria. Furthermore, mind wandering can predict subsequent negative affect (Killingsworth & Gilbert, 2010), and induced negative affect increases the frequency of mind wandering (Smallwood, Fitzgerald, Miles, & Phillips, 2009). It seems then that the type of self-focus associated with mind wandering resembles that found in sleep and depression.

And, third, even for narcissism there seems to be a significant relationship with depression. Kernberg and Yeomans (2013, pp. 14–15) observe that those who suffer from narcissistic personality disorder (NPD) appear to be masking "the fragmentation and weakness of their identity under a brittle and fragile grandiose self," and that they often present with

"severe feelings of inferiority and failure…corresponding to depressive reactions." These observations are consistent with a case study recently reported by Saito, Kobayashi, & Kato (2013). The case concerns a man in his late twenties who exhibited a variety of narcissistic symptoms. Although the patient was treated for NPD, the authors suggest that amelioration of his symptoms was due in large part to the use of antidepressant medications. These medications, in conjunction with supportive psychotherapy, seemed simultaneously to reduce both NPD and depressive symptoms, thereby suggesting a common etiology.

But recall that not all forms of self-focus are indicative of pernicious, depressive rumination. Pondering, as opposed to brooding, can be constructive, possibly in a way that is consistent with the analytic rumination hypothesis. What might help explain the difference between these two modes of self-focused thought?

We propose that the answer is to be found in the difference between self-relatedness and self-specificity. Earlier we argued that although early discussions of the DMN and the CMS emphasized their role in mediating self or self-reference, both failed to adequately distinguish what is merely related to self, as opposed to that which is specific to self. Indeed, that concern along with a more general concern about the principal psychological roles played by DMN and CMS regions have been raised previously (Legrand & Ruby, 2009; cf., Lane, 2012). Here we conjecture that pondering of the sort conducive to dealing with social stressors implicated in the etiology of depression involves those regions of the DMN or the CMS that have not only been implicated in self-reference, they have also been implicated in the social understanding of others.

In view of the need to more clearly distinguish between that which is related to and that which is specific for self, as well as the distinction between pondering and brooding, we think it worth pointing out that one region within the DMN, the medial prefrontal cortex (mPFC), has been found to play an important role in the social understanding of others (Li, Mai, & Liu, 2014). Experimental findings concerning social tasks involving representation of the cognitive and affective states of others, attribution of mental states to others, and predicting the behaviors of others show that there is striking overlap between parts of the mPFC and other regions involved in social cognition that lie outside of the DMN or the CMS. Whereas, for example, the ventral mPFC seems more responsive to self, the dorsal mPFC seems to be involved in both self- and other- referential processing.

In sum, our prediction is that the self-focused, brooding ruminations characteristic of depression are likely to be associated with elevated levels of resting state activity in the PACC and the anterior insula, but not in the dorsal mPFC. The dorsal mPFC may, however, play a significant role in self-focused pondering. Irrespective of whether this specific hypothesis is

confirmed, evidence from prior investigations of depression, REM sleep, mind wandering, and narcissism suggest a strong relationship between self-focus insulated from external stimuli and negative affect. It appears to be the case that the resting state's effectiveness at blocking external stimuli and maintaining an inward focus on self might be sufficient to strongly dispose one to experiencing negative affect.

7.5 THE RESTING STATE, DEPRESSIVE RUMINATION, AND RATIONALITY

Recall that according to the ARH, intrusive, persistent rumination is an evolutionary adaptation. It results from an evolutionary trade-off, much as is the case with fever or pain. These are not pleasant things. No one would claim that. But they are averred to be adaptations because they "proficiently promote" special effects that enhance our ability to deal with social stressors. One of the effects promoted is a coordination of body systems to facilitate rumination.

Advocates of the ARH counsel avoiding resort to medication as a way of relieving the negative affect associated with depressive ruminations. According to the ARH, it is better "to learn how to endure the pain until the problem is solved." They even suggest that their view is consistent with a "venerable philosophical tradition" which holds that pain or suffering motivates "growth and insight into oneself." The coordination of body systems that enables a closing in on self-focused, ruminative thoughts and the accompanying negative affect is something to be embraced.

The ARH and the RSH are not compatible. According to the RSH, a key to coordination of body systems is an abnormal resting state imbalance between medial and lateral regions. Lateral hypoactivity inhibits receptiveness to external stimuli and medial hyperactivity blocks the introduction of positive stimuli or top-down modulation. One probable biochemical cause for this abnormal state is an elevated level of glutamate in the PACC that aggravates the intensity of self-focus. If this view is correct, GABA-ergic drugs like ketamine can promote recovery from depression because they accomplish what the ARH advocates admonish people not to do: seek pharmaceutical relief from the ruminative thoughts and their accompanying negative affect.

The suggestion here is that the ARH conflates distinct types of self-focused thoughts and, by implication, their neural substrates. Pondering may indeed be constructive. But it is brooding, not pondering, that is associated with the self-focus and negative affect of depression. And this type of self-focus seems unlikely to promote the type of "growth and insight into oneself and the problems of life" that ARH alleges to be the functional equivalent of fever's coordination of immune responses to infection. The

problem is that the hypo- and hyperresting state imbalance makes rational consideration of social stressors difficult, if not impossible, because it focuses attention exclusively onto the negative. This precipitates the hopelessness so characteristic of depression and the inability to discover positive, constructive responses while engaged in persistent, intrusive rumination.

Nozick has written (1993, p. 120) that "reasons and reasoning all would be useful to an organism facing new situations and trying to avoid future difficulties. Such a capacity for rationality…might well serve an organism in its life tasks and increase its inclusive fitness." Were the ARH to direct attention merely to the likelihood that a general-purpose capacity for rationality has been selected for, along the lines suggested by Nozick, we could endorse it.[29] The problem is that the ARH is arguing for a special purpose capacity designed to deal not with "life tasks," generally considered, but with a specific subset of life tasks, social stressors that incline persons toward depressive rumination. It is this special purpose capacity that we believe unlikely to increase the inclusive fitness of humans.

Concerning what is often called epistemic or evidential rationality, Nozick further opined that beliefs are changeable and when changes "are based upon reasons and upon reasoning to new conclusions, *on a balancing of reasons for and against*, they can be attuned to match new or changing situations and then usefully affect the behavior of an organism facing such a situation" (Nozick, 1993, p. 94).[30] In other words, epistemic rationality is concerned with holding or formulating beliefs that get things right. And this is the problem with the ARH. Depressive rumination is unlikely to contribute to the holding or formulation of belief that get things right, because it does not allow for appropriate "balancing of reasons for and against."

To see why depressive rumination is not likely to yield appropriately "balanced" reasoning, it helps to observe that epistemic rationality is consistent with a broad consensus within analytic philosophy concerning the nature of belief. The majority of analytic philosophers who investigate belief advocate some version of the idea that beliefs "aim at the truth" (Williams, 1973, pp. 137–138). Davidson (2003, pp. 366–367) emphasizes their "veridical nature"; Searle (2001, pp. 37–38) claims it is their "job to represent how things are"; Crane (2001, p. 103) says that "holding true" is a synonym for belief; Wedgwood (2002, p. 273) observes that "for every proposition p that one consciously considers, the best outcome is to believe p when p is true"; Shah and Velleman (2005, pp. 498–500; cf., Velleman, 2000, pp. 182–188) contend that beliefs are "truth-regulated acceptance"; and, Railton (2003, p. 297) holds that a belief "not only represents its propositional content as true," it "cannot represent itself as unresponsive to…truth." Although with regard to a small subset of beliefs, we think these views may be somewhat problematic (Lane, 2010; Lane &

Flanagan, 2016; Churchland, 2013, p. 81); generally speaking, we endorse the view that rational deliberations should aim at formulating beliefs that are responsive to and that aim at the truth.

Depressive rumination is not rational, and is not likely to have been rational in ancestral environments, because it lacks the type of balance that reasoning requires. Because depressive rumination is laden with negative affect, it is unable to see the good along with the bad, a state of affairs which inhibits responsiveness to the truth, and implies failure to aim at the truth. If the RSH approximates the truth, if it is indeed an accurate account of depressive rumination, then depression lacks the ability to proficiently promote responses to social stress that enhance survivability. It is highly unlikely that natural selection would design organisms with the type of self-focus characteristic of depression, even if as a trade-off, because the lack of "balance" and responsiveness to the truth is not conducive to discovery of constructive responses to social stressors.

A motivation for formulating the ARH is depression's uniqueness among mental health problems: it is so widespread. If the views expressed here are correct, however, the proper place to search for an explanation of MDD's incidence rate does not lie in our evolutionary past. Perhaps the frequency of social stressors has increased or perhaps our resilience to those stressors has been weakened.[31] Perhaps as well there might be other environmental factors that contribute to imbalances between GABA and glutamate in specific brain regions. What seems clear though is that while rationality may well be an evolved trait, the inability to access a balance of reasons when formulating or changing beliefs is not.

7.6 CONCLUSIONS

If the resting state hypothesis approximates the truth, then the analytic rumination hypothesis could not be true, because the hypo- and hyperresting state imbalance would make it impossible for the "analytic ruminations" to form beliefs that would allow for sufficiently flexible response. Turning inward, intense self-focus tends to flood the cognitive system with negative affect, while simultaneously blocking the introduction of potentially positive stimuli. Reasons and reasoning do contribute to survival, but not when the accessible reasons are so narrowly focused, and when those that are accessible are cloaked in despondence.

Jennifer Corns, writing on "hedonic rationality," has suggested that there are many instances when intervening to eliminate suffering is inappropriate, more instances than we may once have thought (Corns, 2016).[32] Simply, there are times when it is rational to suffer. Generally speaking, we are inclined to agree with Corns. But we do not think MDD is one of

those instances. When patients suffer from MDD, abnormal resting state activity prevents patients from being properly responsive to reasons.

We began this essay with an epigraph penned by Aeschylus for his play *Agamemnon*. The gist of these words spoken by the Chorus is consistent with the ARH: wisdom is derived from suffering. Similar expressions of this idea appear recurrently in the writings of Aeschylus and other Greek poets and philosophers. The insight is not novel to 21st-century philosophers or scientists. Indeed, we think there are occasions when to suffer is rational and when to interfere with suffering prevents attainment of wisdom. But MDD is not one of those occasions. Medications—perhaps GABA-ergic—that adjust the resting state medial-lateral imbalance and reduce self-focus are necessary and appropriate.[33] They do not block wisdom; they make its achievement more likely.

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References

Alcaro, A., Panksepp, J., Witczak, J., Hayes, D. J., & Northoff, G. (2010). Is subcortical-cortical midline activity in depression mediated by glutamate and GABA? A cross-species translational approach. *Neuroscience and Biobehavioral Reviews*, *34*, 592–605.

Andrews, P. W., & Thomson, J. A., Jr. (2009). The bright side of being blue: Depression as an adaptation for analyzing complex problems. *Psychological Review*, *116*(3), 620–654.

Bai, Y., Nakao, T., Xu, J., Qin, P., Chaves, P., Heinzel, A., Duncan, N., Lane, T., Yen, N., Tsai, S., & Northoff, G. (2015). Resting state glutamate predicts pre-stimulus alpha. Increase during self-relatedness—A combined EEG-MRS study on rest-self overlap. *Social Neuroscience*, *11*(3), 249–263.

Bar-On, E., Weigl, D., Katz, K., Weitz, R., Steinberg, T., & Parvari, R. (2002). Congenital insensitivity to pain: Orthopaedic manifestations. *The Journal of Bone and Joint Surgery*, *84-B*, 252–257.

Blanco, C., Okuda, M., Wright, C., Hasin, D. S., Grant, B. F., & Liu, S. M. (2008). Mental health of college students and their non-college-attending peers: Results from the National Epidemiologic Study on alcohol and related conditions. *Archives of General Psychiatry*, *65*, 1429–1437.

Brandon, R. (1990). *Adaptation and environment*. Princeton, NJ: Princeton University Press.

Buller, D. J. (2005). *Adapting minds: Evolutionary psychology and the persistent quest for human nature*. Cambridge, MA: The MIT Press.

Carciofo, R., Du, F., Song, N., & Zhang, K. (2014). Mind wandering, sleep quality, affect and chronotype : An exploratory study. *PLoS ONE*, *9*(3), e91285.

Carhart-Harris, R. L., & Friston, K. J. (2010). The default-mode, ego-functions and free energy: A neurobiological account of Freudian ideas. *Brain*, *133*, 1256–1283.

Churchland, P. S. (2013). *Touching a nerve: The self as brain*. New York: W. W. Norton & Company.

Corns, J. (2016). Hedonic rationality. Unpublished manuscript.

Craig, A. D. (2009). How do you feel—now? The anterior insula and human awareness. *Nature Reviews Neuroscience*, *7*, 189–195.

Crane, T. (2001). *Elements of mind: An introduction to the philosophy of mind*. New York: Oxford University Press.

Davidson, D. (2003). Thought and talk. In T. O'Connor, & D. Robb (Eds.), *Philosophy of mind: Contemporary readings* (pp. 353–369). London: Routledge.

Demertzi, A., Soddu, A., Vanhaudenhuyse, A., Schabus, M., Noirhomme, Q., Bredart, S., Boly, M., Phillips, C., Luxen, A., Moonen, G., & Laureys, S. (2011). Two distinct neuronal networks mediate the awareness of environment and of self. *Journal of Cognitive Neuroscience*, *23*, 570–578.

Diaz, B. A., Sluis, S., Moens, S., Benjamins, J. S., Migliorati, F., Stoffers, D., Braber, A., Poil, S., Hardstone, R., Van't Ent, D., Boomsma, D., De Geus, E., Mansvelder, H., Van Someren, E., & Linkenkaer-Hansen, K. (2013). The Amsterdam Resting State Questionnaire reveals multiple phenotypes of resting-state cognition. *Frontiers in Human Neuroscience*, *7*, 446.

Feinberg, T. (2009). *From axons to identity: Neurobiological explorations of the nature of self*. New York: W. W. Norton & Company.

Feinberg, T. (2011). The nested neural hierarchy and the self. *Consciousness and Cognition*, *20*, 4–15.

Foland-Ross, L. C., Hamilton, J. P., Joormann, J., Berman, M. G., Jonides, J., & Gotlib, H. (2013). The neural basis of difficulties disengaging from negative irrelevant material in major depression. *Psychological Science*, *24*(3), 334–344.

Friston, K. (2009). The free-energy principle: A rough guide to the brain? *Trends in Cognitive Science*, *13*, 293–301.

Giambra, L. M., & Traynor, T. D. (1978). Depression and daydreaming: An analysis based upon self-ratings. *Journal of Clinical Psychology*, *34*(1), 14–25.

Grimm, S., Ernst, J., Boesiger, P., Schuepback, D., Hell, D., Boeker, H., & Northoff, G. (2009). Increased self-focus in major depressive disorder is related to neural abnormalities in subcortical-cortical midline structures. *Human Brain Mapping*, *30*, 2617–2627.

Hagen, E. H. (2011). Evolutionary theories of depression. *Canadian Journal of Psychiatry*, *56*(12), 716–726.

Hamilton, E. (Trans.) 1958. *Three Greek plays: Prometheus Bound, Agamemnon, and The Trojan Woman*. New York: W. W. Norton & Company

Hamilton, J. P., Furman, D. J., Chang, C., Thomason, M. E., Dennis, E., & Gotlib, I. H. (2011). Default-mode and task positive network activity in major depressive disorder: Implications for adaptive and maladaptive rumination. *Biological Psychiatry*, *70*(4), 327–333.

Hamilton, J. P., Chen, M. C., & Gotlib, I. H. (2013). Neural systems approaches to understanding major depressive disorder: An intrinsic functional organization perspective. *Neurobiology of Diseases*, *52*, 4–11.

Hempel, C. G. (1965). *Aspects of scientific explanation*. New York: The Free Press.

Hendrie, C. A., & Pickles, A. R. (2009). Depression as an evolutionary adaptation: Implications for the development of pre-clinial models. *Medical Hypotheses*, *72*, 342–347.

Hendrie, C. A., & Pickles, A. R. (2010). Depression as an evolutionary adaptation: Anatomical organization around the third ventricle. *Medical Hypotheses*, *74*(4), 736–740.

Hohwy, J. (2013). *The predictive mind*. New York: Oxford University Press.

Ingram, R. E. (1990). Self-focused attention in clinical disorders: Review and a conceptual model. *Psychological Bulletin*, *107*, 156–176.

Kalisch, R., Muller, M. B., & Tuscher, O. (2015). A conceptual framework for the neurobiological study of resilience. *Behavioral and Brain Sciences*, *38*, e92.

Kernberg, O. F., & Yeomans, F. E. (2013). Borderline personality disorder, bipolar disorder, depression, attention deficit/hyperactivity disorder, and narcissistic personality disorder: Practical differential diagnosis. *Bulletin of the Menninger Clinic*, *77*(1), 1–23.

Killingsworth, M. A., & Gilbert, D. T. (2010). A wandering mind is an unhappy mind. *Science*, *330*, 932.

Kluger, M. J. (1986). Is fever beneficial? *The Yale Journal of Biology and Medicine*, *59*, 89–95.

Lane, T. (2010). The ethics of false belief. *EurAmerica*, *40*(3), 591–633.

Lane, T. (2012). Toward an explanatory framework for mental ownership. *Phenomenology and the Cognitive Sciences*, *11*(2), 251–286.

Lane, T. (2014). When actions feel alien—An explanatory model. In T. W. Hung (Ed.), *Communicative action* (pp. 53–74). Springer Science + Business Media.

Lane, T. (2015). Self, belonging, and conscious experience: A critique of subjectivity theories of consciousness. In Rocco Gennaro (Ed.), *Disturbed consciousness: New essays on psychopathology and theories of consciousness* (pp. 103–140). Cambridge, MA: The MIT Press.

Lane, T., & Flanagan, O. (2016). Neuroexistentialism, eudaimonics, and positive illusions. In B. Kaldis (Ed.), *Mind and society: Cognitive science meets the philosophy of social sciences, Synthese Library Series of Studies in Epistemology, Logic, Methodology, and Philosophy of Science*. New York: Springer Science + Business Media.

Lane, T., & Liang, C. (2010). Mental ownership and higher-order thought. *Analysis*, *70*(3), 496–501.

Lane, T., & Liang, C. (2011). Self-consciousness and immunity. *The Journal of Philosophy*, *108*(2), 78–99.

Legrand, D., & Ruby, P. (2009). What is self specific? Theoretical investigation and critical review of neuroimaging results. *Psychological Review*, *116*(1), 252–282.

Li, W., Mai, X., & Liu, C. (2014). The default mode network and social understanding of others: What do brain connectivity studies tell us. *Frontiers in Human Neuroscience*, *8*(74), .

Mar, R. A., Mason, M. F., & Litvack, A. (2012). How daydreaming relates to life satisfaction, loneliness, and social support: The importance of gender and daydream content. *Consciousness and Cognition*, *21*, 401–407.

McGuire, M., & Troisi, A. (1998). *Darwinian psychiatry*. New York: Oxford University Press.

Merritt, J. M., Sickgold, R., Pace-Schott, E., Williams, J., & Hobson, J. A. (1994). Emotion profiles in the dreams of men and women. *Consciousness and Cognition*, *3*, 46–60.

Mor, N., & Winquist, J. (2002). Self-focused attention and negative affect: A meta-analysis. *Psychological Bulletin*, *128*(4), 638–662.

Murphy, D., & Stich, S. (2000). Darwin in the madhouse: Evolutionary psychology and the classification of mental disorders. In P. Carruthers, & A. Chamberlain (Eds.), *Evolution and the human mind: Modularity, language and meta-cognition* (pp. 62–92). Cambridge, UK: Cambridge University Press.

Nejad, A. B., Fossati, P., & Lemogne, C. (2013). Self-referential processing, rumination, and cortical midline structures in major depression. *Frontiers in human neuroscience*, *7*, 666.

Nesse, R. M. (2000). Is depression an adaptation? *Archives of General Psychiatry*, *57*, 14–20.

Nettle, D. (2004). Evolutionary origins of depression: a review and reformulation. *Journal of Affective Disorders*, *81*, 91–102.

Niciu, M. J., Luckenbaugh, D. A., Ionescu, D. F., Guevara, S., Machado-Vieiva, R., Richards, E. M., Brutsche, N. E., Nolan, N. M., & Zarate, C. A. (2014). Clinical predictors of ketamine response in treatment-resistant major depression. *The Journal of Clinical Psychiatry*, *75*(5), e417–e423.

Nolen-Hoeksema, S., Morrow, J., & Fredrickson, B. L. (1993). Response styles and the duration of episodes of depressed mood. *Journal of Abnormal Psychology*, *102*, 20–28.

Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. *Perspectives on Psychological Science*, *3*(5), 400–424.

Northoff, G. (2012). Immanuel Kant's mind and the brain's resting state. *Trends in Cognitive Sciences*, *16*(7), 356–359.

Northoff, G. (2013a). *Unlocking the brain. Volume I: Coding*. New York: Oxford University Press.
Northoff, G. (2013b). *Unlocking the brain. Volume II: Consciousness*. New York: Oxford University Press.
Northoff, G., & Bermpohl, F. (2004). Cortical midline structures and the self. *Trends in Cognitive Science*, *8*(3), 102–107.
Northoff, G., Heinzel, A., de Greck, M., Bermpohl, F., Dobrowolny, H., & Panksepp, J. (2006). Self-referential processing in our brain—A meta-analysis of imaging studies on the self. *Neuroimage*, *15*(31a), 440–457.
Northoff, G., Qin, P., & Nakao, T. (2010). Rest-stimulus interaction in the bra a review. *Trends in Neuroscience*, *33*(6), 277–284.
Northoff, G., Wiebking, C., Feinberg, T., & Panksepp, J. (2011). The resting state hypothesis of major depressive disorder—A translational subcortical-cortical framework for a system disorder. *Neuroscience and Biobehavioral Reviews*, *35*(9), 1929–1945.
Nozick, R. (1993). *The nature of rationality*. Princeton, NJ: Princeton University Press.
Panksepp, J., & Watt, D. F. (2011). Why does depression hurt? Ancestral primary-process separation-distress (PANIC) and diminished brain reward (SEEKING) processes in the genesis of depressive affect. *Psychiatry*, *74*(1), 5–13.
Pyszczynski, T., & Greenberg, J. (1987). Self-regulatory perseveration and the depressive self-focusing style: A self-awareness theory of reactive depression. *Psychological Bulletin*, *102*, 122–138.
Qin, P., & Northoff, G. (2011). How is our self related to midline regions and the default mode network. *NeuroImage*, *57*, 1221–1233.
Raichle, M. E., & Mintun, M. A. (2006). Brain work and brain imaging. *Annual Review of Neuroscience*, *29*, 449–476.
Raichle, M. E. (2006a). The brain's dark energy. *Science*, *314*, 1249–1250.
Raichle, M. E. (2006b). Brain work and brain imaging. In S. E. Hyman, & T. J. et al. Jessell (Eds.), *Annual review of neuroscience* (pp. 449–476). Palo Alto, CA: Annual Reviews.
Raichle, M. E. (2010). Two views of brain function. *Trends in Cognitive Sciences*, *14*(4), 180–190.
Raichle, M. E., & Gusnard, D. A. (2002). Appraising the brain's energy budget. *Proceedings of the National Academy of Sciences*, *99*(16), 10237–10239.
Raichle, M. E., MacLeod, M. A., Snyder, A. Z., Powers, W. J., Gusnard, D. A., & Shulman, G. A. (2001). A default mode of brain function. *Proceedings of the National Academy of Sciences*, *98*(2), 676–682.
Railton, P. (2003). *Facts, values, and norms: Essays toward a morality of consequence*. Cambridge, UK: Cambridge University Press.
Raison, C. L., & Miller, A. H. (2013). The evolutionary significance of depression in pathogen host defense (Pathos-D). *Molecular Psychiatry*, *18*, 15–37.
Revonsuo, A. (2006). *Inner presence: Consciousness as a biological phenomenon*. Cambridge, MA: The MIT Press.
Saito, S., Kobayashi, T., & Kato, S. (2013). A case of major depressive disorder barely distinguishable from narcissistic personality disorder. *Seishin Shinkelgaku Zasshi*, *115*(4), 363–371.
Searle, J. (2001). *The rediscovery of mind*. Cambridge, MA: The MIT Press.
Seligman, M., & Yellen, A. (1987). What is a dream? *Behavior Research and Therapy*, *25*, 1–24.
Shah, N., & Velleman, J. (2005). Doxastic deliberation. *The Philosophical Review*, *114*, 497–534.
Smallwood, J., Fitzgerald, A., Miles, L. K., & Phillips, L. H. (2009). Shifting moods, wandering minds: Negative moods lead the mind to wander. *Emotion*, *9*(2), 271–276.
Smallwood, J., O'Connor, R. C., Sudbery, M. V., & Obonsawin, M. (2007). Mind-wandering and dysphoria. *Cognition and Emotion*, *21*(4), 816–842.
Sterelny, K. (2003). *Thought in a hostile world: The evolution of human cognition*. Malden, MA: Blackwell Publishing.
Trapnell, P. D., & Campbell, J. D. (1999). Private self-consciousness and the five-factor model of personality: Distinguishing rumination from reflection. *Journal of Personality and Social Psychology*, *76*, 284–304.

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research*, *27*, 247–259.
Velleman, J. D. (2000). *The possibility of practical reason*. New York: Oxford University Press.
Watkins, E. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin*, *134*(2), 163–206.
Watkins, E., & Moulds, M. (2005). Distinct modes of ruminative self-focus: Impact of abstract versus concrete rumination on problem solving in depression. *Emotion*, *5*, 319–329.
Watson, P. J., & Andrews, P. W. (2002). Towards a revised evolutionary adaptationist analysis of depression: The social navigation hypothesis. *Journal of Affective Disorders*, *72*, 1–14.
Wedgwood, R. (2002). The aim of belief. *Philosophical Perspectives*, *16*, 267–297.
Williams, B. (1973). *Problems of the self*. Cambridge, UK: Cambridge University Press.
Woodward, J., & Cowie, F. (2004). The mind is not [just] a system of modules shaped [just] by natural selection. In C. Hitchcock (Ed.), *Contemporary debates in philosophy of science* (pp. 312–334). Malden, MA: Blackwell.

Endnotes

1. According to this PATHOS-D hypothesis, it is not that the alleles for depression co-evolved with immunological alleles that support pathogen defense; instead, "the alleles for depression… are in fact one in the same as…" those immunological alleles (Raison & Miller, 2013, p. 16).
2. The third ventricle includes the pineal gland, the hypothalamus, and the amygdala.
3. Of course the adaptive landscape in this vicinity is likely more complex than the single peak model described here.
4. The hypothesis proposed by Andrews and Thomson concerns only unipolar depression; they do not challenge the view that bipolar differs qualitatively from unipolar depression.
5. The point is that although severe pain is aversive and disabling, it is nevertheless beneficial; so too is severe emotional response. For those who might think pain not to be beneficial, consider the suffering endured by those who are congenitally insensitive to pain (Bar-On et al., 2002).
6. Italics not contained in original.
7. Consider that people contemplating divorce might lose children, money, and home by leaving. Alternatively, by not leaving, they risk continued marital conflict. Determining the optimal solution requires extended analysis and the capacity to endure the emotional pain that supplies the motivation.
8. Functional neuroimaging during the performance of emotional working memory tasks can also be used to show that negative, irrelevant stimuli can be especially difficult for persons suffering from depression to disregard.
9. Dichotic listening probes can, eg, be used to simultaneously present positive and negative stimuli.
10. Cerebral blood flow and metabolic changes will be a focus of concern in the next section, where we discuss the resting state hypothesis.
11. Investigators using the ruminative responses scale and correlational along with principal component analysis have identified at least three distinct types of items: depressive, brooding, and self-reflective (Nolen-Hoeksema et al., 1993).
12. Italics not contained in original.
13. Among the core regions are the posterior cingulate cortex, a medial prefrontal area, and the inferior parietal lobule.
14. During functional magnetic resonance imaging (fMRI), the resting state is typically measured by asking subjects to close their eyes or fixate on a cross while lying quietly.

15. This is 10 times higher than what would be expected were calculations based upon weight alone (Raichle & Gusnard, 2002).
16. There are several different ways of drawing this distinction: some, for example, distinguish between the "internal and external awareness network" (Demertzi et al. 2011).
17. Whether this emphasis more nearly reflects a common characteristic of self-focus itself, or whether it is an artifact of researchers' selective focus remains to be seen (Watkins, 2008). We return to discussion of the link between self-focus and negative affect in the next section.
18. A recently developed resting state questionnaire that is based upon data gathered from 813 subjects indicates that "self" is one of seven distinctive dimensions of resting state cognition (Diaz et al., 2013).
19. The philosophical sense of "self" tends to emphasize self-as-subject, or the subject of experience.
20. The neuronal activity that is manifest during both the resting state and self-related processing is spatial and temporal: both functional connectivity among critical regions and strong low-frequency fluctuations are exhibited (Northoff, 2013a, pp. 299–301).
21. Paralimbic regions include lower portions of the orbitofrontal cortex, perigenual and supragenual anterior cingulate cortex, the posterior cingulate cortex, the retrosplenial cortex, the temporal pole, and the insula.
22. There are, however, important conceptual distinctions in this vicinity—intrinsic activity, resting state, and baseline—that should be preserved. For explication, see Northoff (2013a, pp. 74–76).
23. Bai et al., 2015.
24. Exaggerated emphasis on the search for neural correlates of consciousness may have been an obstacle to discovery the way in which the "neuronal grid" can structure experience. A more comprehensive understanding of conscious experience, including the pathological experiences under consideration here, will require that attention be given to the "neural predispositions of consciousness" (Northoff, 2013, pp. 541–542).
25. "Grid" should not be interpreted as something that is in any literal sense inflexible; prior interaction between stimuli and resting state activity can modulate the resting state (the "grid") such that it "prepares itself" for subsequent processing of the same or similar stimuli (Northoff, 2013a, p. 246).
26. Because primary emotions emerge by the age of one, and because they are expressed in cross-culturally stereotypical fashion, it seems they are "hardwired" into the developing nervous system.
27. The view we articulate here is in many—but not all—respects consistent with the view presented by Carhart-Harris & Friston (2010).
28. Seligman and Yellen (1987) say of dream emotion that it is a "limbic bath" that persists throughout a dream's entirety. According to the study discussed in the text (Merritt et al., 1994, p. 47, 50), 95% of dream reports were associated with emotion; only 11 of 200 indicated no emotion.
29. "General" here does not imply that mechanisms are adapted to some "general" feature of the environment; instead, what matters is that there be mechanisms of "phenotypic plasticity" which are not committed to producing any specific response before interacting with the environment. The beliefs that supervene on such a mechanism are "functionally decoupled from any specific actions, while being potentially relevant to many" (Sterelny, 2003, pp. 30–40).
30. Italics not contained in original.
31. Although we do not develop our argument here, we speculate that adequate explanation of the incidence rate will give special emphasis to a diminution of resilience. The social

stressors are not new; rather, what has changed is our capacity for dealing with those stressors.

32. In this cogent manuscript Corns argues that agents can be found to be rational, or not, simply in virtue of what they feel, the pleasantness or unpleasantness of their emotions.
33. It is not our intent to dismiss the value of cognitive therapies that can promote pondering social stressors.

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PART IV

IRRATIONALITY

Is rationality a characteristic shared by all human beings? Or can human populations from diverse social backgrounds develop ways of thinking that are ontologically dissimilar from one another? While coherence is a key element in the Western notion of rationality, textual inconsistency seems much more prevalent in ancient Chinese philosophy. "Irrational" thus seems to be the first impression that ancient Chinese philosophy gives to many Western scholars. Some argue that these phenomena reveal the cultural diversity of rationality. Others hold that the alleged cultural differences have been exaggerated. Part IV considers whether ancient Chinese philosophy really does diverge substantially from Western ideas of rationality, or whether the appearance of irrationality in various classics of Chinese philosophy can be explained in some other way.

In Chapter 8, Yiu-ming Fung challenges the perceived view that ancient Chinese thinkers are nonanalytical, and that their ways of reasoning are incommensurate with thinkers in the Western philosophical tradition. He locates this view in the works of a number of scholars, including Marcel Granet, Joseph Needham, A. C. Graham, David Hall, and Roger Ames. Fung argues that this view should be rejected, not just because it contradicts ancient texts, but also because it is self-defeating.

In Chapter 9, Wai Chun Leong reviews the widespread claim that ancient Chinese philosophy features a kind of "logic" or "rationality" that is distinct from Western notions. For example, some have alleged that Chinese philosophy involves an acceptance of contradictions as if they were rational, whereas Western philosophy does not. In that case, attempting to understand Chinese philosophy in terms of current, Western notions of rationality might lead to a distortion of ancient traditions. Leong argues that the usual evidence for this claim either attributes no real contradiction to Chinese philosophy, or simply fails to make ancient Chinese philosophical texts intelligible.

In Chapter 10, Ting-mien Lee considers the cultural relativity of rationality from the viewpoint of Sinology. According to Lee, early Chinese philosophical texts are notorious for contradictions and a lack of apparent coherence. She argues that these apparently incoherent texts do not support the claim that ancient Chinese philosophy features a notion of rationality different from that operative in the Western tradition. She also provides an alternative explanation for textual incoherence, one that does not invoke a separate notion of rationality.

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