

Brains, Beliefs, and Existentialism:  
Philosophies and Treatments Pertaining to Three Approaches to Social Anxiety  
Disorder, and the Prospect of a New Mental Health Paradigm

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## **Introduction**

This paper provides a brief overview of social anxiety disorder, and outlines three approaches discernible in the scientific literature to understanding the etiology and maintenance of social anxiety disorder. The connection of each of these approaches to a certain type of treatment is discussed, as well the philosophical assumptions supporting each of these approaches. These three approaches are then comparatively assessed in terms of their suitability for explaining the etiology and maintenance of social anxiety disorder, with an emphasis on the relationship between dynamics at distinct levels of abstraction. The shortcomings of the current DSM paradigm of mental illness are explored, and how improvements thereof may be related to the development of a more robust understanding of the mechanisms of mindfulness-based interventions. This paper concludes with a brief discussion of the potential value of existential philosophy in grounding and guiding the project of developing a new conceptual framework for mental health and illness that is less susceptible to the criticisms of the current DSM framework, and which can satisfyingly account for the effectiveness of mindfulness-based interventions for a wide range of psychiatric disorders.

## **Social anxiety disorder**

Social anxiety disorder (SAD), alternatively referred to as social phobia, is an anxiety disorder that pertains specifically to social situations. SAD affects approximately seven percent of Americans annually (American Psychiatric Association, 2013), and has a lifetime prevalence of approximately twelve percent (Kessler et al., 2005). SAD is

characterized by excessive fear or anxiety related to certain social situations in which one may be observed or scrutinized by others (APA, 2013). These situations may consist in normal social interactions, engaging in activities when one may be observed by others, or in performance settings (APA, 2103). This fear or anxiety may result in avoidant behavior towards the anxiety-inducing situations, or else lead to intense discomfort and difficulty in enduring such situations (APA, 2013). The 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) includes ten diagnostic criteria for social anxiety disorder. These criteria further specify that the affected individual fears negative evaluation by others in the anxiety-provoking situations; that the anxiety or fear occurs consistently in response to such situations and is persistent, typically lasting for at least six months; that the fear or anxiety is disproportionate to the actual threat posed in such situations (as assessed by a trained clinician); that “the fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning;” that it is not better accounted for by another disorder; and that it is not attributable to the effects of a substance nor explainable as a normal response to a medical condition (APA, 2013).

### **Three approaches**

Researchers and theorists concerned with SAD seek to understand the etiology, maintenance, and possible treatments of this disorder in varying ways. This paper categorizes the different ways of engaging this project into three approaches, which we term the “neuroscientific approach,” the “psychological approach,” and the “relational

approach.” The different methodologies proper to these approaches are founded on distinct underlying assumptions about the nature of the human mind and conscious experience. Implicit in each approach are certain philosophical positions, in light of which the aims of that approach become coherent, and from which its techniques derive epistemic validity. Although these distinct approaches concern themselves with common issues, their varied philosophical foundations lead them to diverge from one another in what they conceive to be the appropriate way to engage these issues, and in the kinds of understanding that they seek to develop.

Before discussing these three approaches in detail, we should note that their respective philosophical foundations do not necessarily reflect the beliefs of any individual persons. For example, a researcher whose work falls into the category of the neuroscientific approach need not personally subscribe to the beliefs and assumptions that this paper will ascribe to the neuroscientific approach; she may instead hold beliefs in line with the psychological and/or relational approaches. Such a seeming contradiction is possible because the underlying assumptions of these approaches are implicit in the activity of scientific research and in the conceptual vocabularies of theoretical models themselves, and are not necessarily espoused by individuals.

We should also note that these approaches are not mutually exclusive. One may simultaneously take all of them to be valid, with each approach describing the truth on its own level, in its own appropriate terms. By way of analogy, we may consider the fields of physics, chemistry, and biology: each of these disciplines describes the same reality, but at different levels of abstraction and, consequently, using different

vocabularies. In a similar manner, the neuroscientific, psychological, and relational approaches reflect distinguishable layers of understanding that are more complementary than mutually exclusive. Although opinionated individuals may discount altogether a certain approach and its mode of understanding human experience and behavior as invalid, in this paper we will not take any such exclusionary epistemological stance. For our purposes, any conflict between these approaches consists in disagreement about their suitability and relative value for our given project: that of understanding, explaining, and treating SAD.

It is also worth noting that although this paper outlines the three approaches in relation to SAD, these approaches do not apply exclusively to SAD; they are relevant to any psychiatric disorders for which pharmacological treatments, psychotherapies, and mindfulness-based interventions have been effectively employed, including but not limited to generalized anxiety disorder (Roemer, Orsillo, & Salters-Pedneault, 2008), major depressive disorder (Barnhofer et al., 2009), obsessive compulsive disorder (Twohig et al., 2010), substance use disorders, (Chiesa & Serretti, 2014), bipolar disorder (Williams et al., 2008), and post-traumatic stress disorder (King et al., 2013). Although SAD serves as the primary focus of this paper, we may also understand it as representative of a larger set of disorders to which these arguments apply to some degree.

## **The neuroscientific approach**

The first approach that we will examine is the neuroscientific approach. This line of research and theory focuses on neural substrates and neurophysiological dynamics, the possible abnormal functioning of which may underlie the pathological patterns of cognition, emotion, and behavior that characterize SAD. Brain-based studies have suggested various neurological bases for SAD, or at least neurobiological areas of interest that appear to be implicated in SAD. Specifically, multiple studies have pointed to the role of the amygdala in the abnormal responses of individuals with SAD to anxiety-inducing situations. One study found significantly higher levels activation in the right amygdala of individuals with social phobia in response to “harsh” facial expressions (angry, disgusted, or fearful) relative to healthy controls, which was not the case for happy or neutral facial expressions (Phan et al., 2006). Another study using PET found greater increase in blood flow to the right amygdaloid complex for individuals with social anxiety relative to healthy controls during a public speaking task (Tillfors et al., 2001). A meta-analysis conducted in 2007 of neuroimaging studies on anxiety disorders found that, relative to controls, patients with SAD displayed increased activation in the amygdala, parahippocampal gyrus, fusiform gyrus, globus pallidus, insula, inferior frontal gyrus, and superior temporal gyrus during emotional processing (Etkin & Wager, 2007). Of these, increased activation in the amygdala and insula were also shared by patients with specific phobia and post-traumatic stress disorder. Noting that increased activation in these areas is also shown by healthy volunteers undergoing fear conditioning (as observed by meta-analysis of relevant studies), the authors of this

meta-analysis interpret their results as evidence of an exaggerated fear response, operating via the relevant underlying neural mechanisms, as comprising a crucial element of anxiety disorders (Etkin & Wager, 2007). In contrast to this meta-analysis, a 2006 study found that *decreased* activity in the right amygdala was associated with symptomatic states of individuals with SAD, induced by mental rehearsal of anxiety-inducing social situations (Kilts et al., 2006). Regardless of this discrepancy, neuroimaging research implicates the amygdala as a region of particular interest for SAD.

Altogether, this line of research, focused on the brain, its component structures, and neurophysiological dynamics, comprises a neurological approach to SAD, and by implicative extension other mental disorders. This approach seeks to understand and, in light of this understanding, develop treatments for SAD primarily through the discipline of neuroscience, hence the name that we have applied to it. The problem and solution are, according to this approach and its guiding attitude, first and foremost biological matters, best understood by attending to the central nervous system as a physical entity. By taking this tack, one may largely avoid the vagueness, uncertainty, imprecision, and unreliability involved in taking subjective states and experiences as one's research focus. This biological approach implies by its methodology and proper lexicon of concepts that mental disorders such as SAD may, with sufficient effort, be conceptually reduced to the abnormal patterns of brain activity that characterize them.

This neurally-oriented attitude is well-founded; it is generally taken as a given in scientific circles that all mental events are the product of underlying physical events in

the brain. Although the question of how physical events give rise to subjective states at all remains an intractable mystery, often referred to as “the hard problem of consciousness,” (Chalmers, 1995, p. 207) the entire field of neuroscience comprises a vast array of evidence that all our subjective experiences and agential actions depend upon the functioning of relevant neural structures. So, we may naturally infer that abnormalities in our subjective experiences and agential actions, such as those that characterize SAD and other patterns of cognition, emotion, and behavior considered to be pathological, are dependent upon abnormalities in the neural functioning that gives rise to them. A robust understanding of the neural underpinnings of SAD, and any other mental disorder, will also implicate an appropriate and effective medical treatment that functions simply to correct the neural abnormality in question (which is to say, it will become clear precisely what a hypothetical treatment needs to accomplish, regardless of whether or not possess the technical ability to realize it as yet). A wholehearted extolment of this approach may be observed, by way of example, in a 2008 paper by Murray Stein and Dan Stein, who optimistically survey the current state of a neurological understanding of SAD:

Studies on the neural underpinnings of social anxiety disorder implicate abnormalities of corticolimbic and, possibly, corticostriatal circuitry in the cause or maintenance, or both of social anxiety disorder.... Findings that brain imaging abnormalities, such as those in the amygdala and insula, might normalise with successful drug treatment or psychotherapy, variation in the serotonin transporter gene promoter region affects the



extent of activation in these regions, and serotonin depletion reverses the benefits of antidepressant treatment, all point to a role for serotonergic dysfunction.... Preliminary evidence that neuropeptides such as oxytocin can affect the neural circuitry of social fear might lead to development of new drug treatments (Stein & Stein, 2008).

We may note that Stein and Stein remain open to the efficacy of psychotherapy, but only insofar as it can be shown to correct the neural abnormalities that they take to be ultimately responsible for the disorder in question.

This neuroscientific approach is in line with a philosophy of materialism. “Materialism” refers to a general belief that “all aspects of the universe are composed of matter and energy and can be explained by physical laws” (Chaffee, 2005, p. 141). A conception of the self based in a philosophy of materialism posits that “in the final analysis mental states are identical with, reducible to, or explainable in terms of physical brain states” (Chaffee, 2005, p. 141). Paul Churchland, a proponent of a philosophy of materialism, extols the value and importance of the development of a more accurate, adequate, and useful conceptual framework for the field of psychology, constructed in a bottom-up fashion from the empirical knowledge yielded by the discipline of neuroscience (Churchland, 2013). Churchland argues that the development of such a framework would entail a paradigmatic shift in psychology, such that we would no longer appeal to fundamentally subjective concepts such as “belief, desire, fear, pain, joy, and so on,” but rather to “such things as our

neuropharmacological states, our high-dimensional prototype representations, and the activation-patterns across specialized brain areas” (Churchland, 2013, p. 76). In this way, our psychological vocabulary would become more precise and well-founded in empirically observable reality.

This neuroscientific approach lends itself to the development of pharmacological treatments, which may act directly upon the neurophysiological dynamics that are posited to underlie the development and/or maintenance of SAD. And, indeed, pharmacological treatments for SAD have met with success. In keeping with Stein’s and Stein’s emphasis on the implicated role of serotonergic dysfunction in SAD, selective serotonin reuptake inhibitors (SSRIs), which increase the amount of serotonin present in serotonergic synapses by inhibiting their reuptake into presynaptic axons, have been shown to be efficacious in treating SAD (in addition to wide range of other psychiatric disorders) (Van der Linden, Stein, & Van Balkom, 2000). Monoamine oxidase inhibitors (MAOIs), which increase the synaptic concentration of monoamine neurotransmitters such as serotonin, dopamine, norepinephrine, epinephrine, and melatonin, and benzodiazepines, which act on GABA receptors, have also been shown to be effective for treating SAD (Liebowitz et al., 1992; Fedoroff & Taylor, 2001).

### **The psychological approach**

Another methodology for understanding and treating SAD (and similar disorders), referred to in this paper as the psychological approach, focuses not on neural structures and dynamics per se, but on cognition, emotion, and behavior. The

conceptual plane at which this approach operates is abstracted from the realm of neural functioning. Although cognition, emotion, and behavior are, as previously discussed, fundamentally inseparable from the neural activity that enables and instantiates them, we may still discuss the elements of these three categories in their own right. The psychological approach understands SAD primarily as a psychological phenomenon (hence the name we have applied to it), driven by dynamics at the level of cognition. Similar to the symptomatology used to define SAD in the DSM, its proper vocabulary is more familiar to our everyday understanding. Models in the vein of this approach seek to define SAD as consisting in entrenched patterns of cognition, inextricably bound up with associated emotions and behaviors, which have become pathological due to the impairment and distress they cause the individual who enacts and experiences them. The psychological approach does not seek to reduce the disorder to underlying phenomena, but rather to clarify it on the level at which it originally presents itself.

The model of SAD developed by Clark and Wells, which they term a “cognitive model,” typifies this psychological approach (Clark & Wells, 1995). This model proposes that the “core” of SAD consists in “a strong desire to convey a particular favorable impression of oneself to others and marked insecurity about one’s ability to do so” (Clark & Wells, 1995, p. 69). SAD, according to this model, is maintained by *beliefs* that are habitually held by the individual in question. Specifically, Clark and Wells point to beliefs on the part of an individual with SAD that in the anxiety-inducing situation, she is “in danger of behaving in an inept and unacceptable fashion,” and that “such behavior will have disastrous consequences, in terms of loss of status, loss of worth, and

rejection” (Clark & Wells, 1995, p. 69-70). Clark and Wells propose that such beliefs lead the “social phobic” to engage the social situation in a particular, set, and maladaptive manner. This involves the triggering of an “anxiety program,” which they describe as “a complex constellation of cognitive, somatic, affective, and behavioral changes” (Clark & Wells, 1995, p. 70). Although we will not delve into the minutiae of Clark’s and Wells’ anxiety program here, we may readily observe that this model is not concerned with patterns of neural activity or neurophysiological responses, but rather with thoughts and behaviors, which may span a spectrum from voluntary to involuntary.

Stefan Hofmann (2007) draws upon the scientific literature on SAD (including Clark’s and Wells’ model) to propose a psychological model for the maintenance of SAD, which utilizes a similar vocabulary and conceptual framework as that of Clark and Wells (Hofmann, 2007). Hoffman posits that individuals with SAD perceive social standards to be unrealistically high, and consequently become apprehensive about their ability to meet them. He proposes that, “When confronted with challenging social situations, individuals with SAD shift their attention toward their anxiety, view themselves negatively as a social object, overestimate the negative consequences of a social encounter, believe that they have little control over their emotional response, and view their social skills as inadequate to effectively cope with the social situation” (Hofmann, 2007, p. 203). As with Clark’s and Wells’ model, we may observe here a model of SAD maintenance that utilizes psychological concepts such as attentional habits, perceptions of self, and beliefs, without reference to any underlying neural substrates that instantiate these more abstract concepts.

The psychological approach calls attention to the central role of interpretation in structuring our understandings of and consequent emotional and behavioral responses to the situations in which we find ourselves. This crucially important *interpretation* may be understood as a cognitive activity in which people are constantly engaged. Events do not inherently contain emotional or cognitive significances; rather, these significances are assigned to them by the subject who experiences them. Furthermore, these significances are responsible for how events affect this subject and how she responds to them. We may observe the importance of such interpretive activity in Clark's and Wells' model as they describe a particular cognitive process involved in the maintenance of SAD. In this process, individuals with SAD interpret stimuli that become available to them by enhanced self-focused attention and self-observation such that they are imbued with negative valence and often distorted out of proportion (Clark & Wells, 1995). For example, an individual with SAD may "[equate] feeling humiliated with being humiliated, feeling out of control with being (observably) out of control, and feeling anxious with being noticeably anxious" (Clark & Wells, 1995, p. 71).

The attitude underlying the psychological approach involves an affirmation of the validity and importance of concepts and dynamics that exist at a level abstracted from neural activity, and are more readily available to immediate subjective experience. The psychological approach tacitly affirms that a robust understanding of the workings of the mind can give credence to things like beliefs, emotions, fears, and expectations, which we are originally familiar with by way of subjective experience (although these affective and cognitive concepts may be associated with neural and physiological states,

they are not defined according to these criteria; these biological responses have merely been correlated with pre-existing concepts, which are defined according to subjective experience). Furthermore, psychological models of SAD portray these cognitions as closely interrelated with outward behavior, in such a way that they give rise to behavior (both volitional and non-volitional) and are involved in scrutinizing and interpreting this same behavior (Clark and Wells, 1995; Hofmann, 2007).

The philosophical underpinnings of the psychological approach bear important similarities to the underlying assumptions of phenomenology. Phenomenology as a philosophical tradition is characterized by the endowment of human subjective experience with ontological primacy. This type of philosophy does “not assume that there are more ‘fundamental’ levels of reality beyond that of conscious human experience” (Chaffee, 2005, p. 137). Phenomenological work is guided by an underlying “belief that explanations for human behavior and experience are not to be sought by appeal to phenomena that are somehow behind, beneath, or beyond the phenomena of lived human experience, but instead are to be sought within the field of human experience itself, utilizing terminology and concepts appropriate to this field” (Chaffee, 2005, p. 137). The psychological approach’s implicit rejection of the tactic of reducing cognitive and emotional subjective experiences and observable behaviors to underlying neural activity, which is the guiding technique of the neuroscientific approach, mirrors the core distinction between the philosophical systems of materialism and phenomenology. Like phenomenological philosophy, the psychological approach does not seek to explain mental phenomena (in this case SAD) in terms of concepts at

different levels, but rather to clarify it at the level at which it originally presents itself, using concepts appropriate to this same level of description.

Psychological models of SAD go hand in hand with cognitive behavioral therapies (CBTs). Cognitive behavioral therapy, as a category of therapy, encompasses many varied treatments that target patients' thoughts, emotions, and behaviors. For our purposes, we may understand CBT as a type of treatment that seeks to alter the maladaptive ways in which a patient thinks about and interprets certain situations and events, and suggests new patterns of cognition in which the patient may seek to engage in response these situations (Beck, 2011). CBT addresses the pathological root of the disorders that it is used to treat (including SAD) as consisting first and foremost in the patient's interrelated beliefs, thoughts, and habits of interpretation; thus, by helping the patient to alter the content of these aspects of her mental life through conscious effort, a therapist may shift the patient away from her entrenched maladaptive mental habits, and in this way effectively remedy the issue in question (Beck, 2011). Grant and Wingate (2011) characterize CBT as a treatment that "focuses on specifically identifiable thought patterns, dysfunctional behaviors, and how these patterns interact to cause and maintain negative emotions" (Grant & Wingate, 2011, p. 234). Judith Beck states that the "cognitive model" underlying CBT "proposes that *dysfunctional thinking* (which influences the patient's mood and behavior) is common to all psychological disturbances," and that CBT targets "patients' basic beliefs about themselves, the world, and other people" (Beck, 2011, p. 3). The goal of CBT, then, is to correct whatever dysfunctional thinking about self, others, and the world is responsible for the issue in

question. In the form of CBT developed by Beck and Emery (1985) and Heimberg and Becker (2002), and described by Hofmann as “the most popular and best-researched treatment” for SAD, “patients practice identifying negative cognitions (automatic thoughts), observing the co-variation between anxious mood and automatic thoughts, examining the errors of logic, and formulating rational alternatives to their automatic thoughts” (Hofmann, 2007, p. 193). Collectively, these activities constitute “cognitive restructuring techniques” (Hofmann, 2007, p. 193). We may observe in all of these characterizations of CBT, though they may vary in their particulars, the close relation of this type of therapy to psychological models of SAD; CBT is concerned with cognitive habits, thoughts, emotions, and behaviors, and seeks to induce changes by manipulations on this level of abstraction.

### **The relational approach**

The third approach, the relational approach, differs only subtly from the psychological approach, and often in the literature appears intermixed with it. Nonetheless, this distinction reflects an appreciable difference in levels of abstraction with regard to mental life. The relational approach seeks to identify the root of disorders such as SAD in an individual’s modes of relating to self and world. It takes as its focus concepts that we will characterize as *attitudes* or *orientations*, which are more general and abstract than specific cognitions or habits of cognition.

This approach remains largely undeveloped in its own right, and robust relational models of SAD (and other disorders), which concern themselves with the individual’s



deep-seated attitudes towards herself and the world in which she participates, are still incipient in the scientific literature. This being the case, the vocabulary and types of concepts that such a project necessitates have only begun the process of carving out a space for themselves in the domain of mainstream psychological science and clinical theory. Examples of such concepts, proper to these inchoate relational models of SAD (and other disorders for which MBIs are employed), include self-compassion, cognitive (de)fusion, and psychological (in)flexibility.

Self-compassion is a construct that describes a way of relating to oneself, or, as pioneering self-compassion researcher Kristin Neff puts it, a “healthy attitude toward oneself” (Neff, 2003). Self-compassion, as characterized by Neff, encompasses three aspects:

“(a) self-kindness—being kind and understanding toward oneself in instances of pain or failure rather than being harshly self-critical, (b) common humanity—perceiving one’s experiences as part of the larger human experience rather than seeing them as separating and isolating, and (c) mindfulness—holding painful thoughts and feelings in balanced awareness rather than over-identifying with them” (Neff, 2003, p. 85).

We may observe that these three dimensions of self-compassion, and particularly the first and third, describe more abstract dynamics than the specific beliefs and cognitions addressed by the psychological approach. Whereas the psychological approach identifies self-critical cognitions and mental habits of self-scrutiny, the relational approach identifies an orientation towards oneself from which such cognitions and habits may

arise. For example, Hoffman, explaining negative self-perceptions in his psychological model of the maintenance of SAD, states that “patients with SAD...perceive their self attributes to fall short of the characteristics they believe others expect them to possess” (Hofmann, 2007, p. 199). Similarly, Clark and Wells state that social phobics hold “negative beliefs about their worth or value,” such as “I’m stupid,” or “I’m inadequate” (Clark & Wells, 1995, p. 76). All of these specific beliefs (that one is not meeting the social expectations of others, that one is stupid, or that one is inadequate) may be reflective of and grounded in a more general lack of self-compassion. And, in accordance with this notion, research has indicated that individuals with SAD demonstrate lower self-compassion than healthy controls (Werner et al., 2012). The underlying attitude or orientation towards oneself, such as self-compassion or lack thereof, engenders a tendency toward certain types of self-cognition and specific self-beliefs. In this sense, the distinction between the relational level, which is concerned with the more fundamental attitude, and the psychological level, which is concerned with the specific cognitions, becomes apparent.

To further clarify the distinction between the psychological and relational levels of abstraction, let us consider an illustrative example: a class field trip to a museum. As a student on this field trip, one may adopt a variety of different attitudes or orientations towards this experience. For example, one may relate to it as an exciting learning opportunity, or alternatively as a tiresome academic obligation. These more fundamental attitudes/orientations will then determine to a large degree the specific cognitions that one has with regard to the experience of the field trip. For example,

given the negative attitude, one might think, “this museum guide is so long-winded,” whereas given the positive attitude, one might think (in the same situation), “this museum guide is so knowledgeable.” When viewing a famous historical document, a student with a positive attitude would be inclined to feel excited, while a student with a negative attitude would be more inclined to feel bored or disappointed. In each case, the attitude is more fundamental than the thoughts and emotions, and determines in part the kinds of thoughts one is inclined to have and emotions that one is inclined to experience. One’s attitude will also determine to a large degree one’s behavior in response to the particular situations that arise. For example, the positive orientation will engender a tendency to ask questions, listen closely, and relate what one is seeing to what one has learned in lectures. The latter orientation, in contrast, may engender a tendency to look out the window and daydream, tap one’s foot impatiently, or devote one’s attention to socializing with classmates. We may see from this example that specific cognitions, emotions, and behaviors, and even patterns thereof, are to a certain extent the products of more abstract attitudes, or ways of relating to a given experience. Returning to the subject of SAD, the psychological approach concerns itself with the cognitions, emotions, behaviors, and patterns thereof, while the relational approach concerns itself with the underlying attitudes and orientations.

It bears noting that that these two approaches are cleanly distinguished in the extant scientific literature. Psychological models of SAD may often include relational elements, and vice versa. The distinction made here between the psychological and relational approaches, then, is both descriptive (insofar as this distinction is already

observable in the literature) and prescriptive (insofar as these approaches remain intermixed).

Another properly relational concept can be found in cognitive (de)fusion. Cognitive fusion describes a certain way of relating to one's cognitive-emotional experience. Specifically, it consists in a process by which one becomes attached to the contents of one's thoughts and feelings and responds to them as if they were literally true, rather than regarding them as transient mental phenomena arising from ongoing emotional and cognitive processes (Greco, Lambert, & Baer, 2008). When engaging in cognitive fusion, one identifies oneself to an excessive degree with the specific thoughts and emotions that one experiences. Cognitive defusion, then, describes a process of creating psychological distance between oneself and one's experiences such that one recognizes thoughts and feelings as such, rather than as literal realities (Hayes, 2006). Consequently, "the result of defusion is usually a decrease in believability of, or attachment to, private events rather than an immediate change in their frequency" (Hayes, 2006, p. 9). We may observe that cognitive (de)fusion does not describe any specific beliefs, habitual thoughts, or feelings, but rather a way of relating to these things; in this respect, it is a concept that belongs to the relational rather than the psychological approach.

Cognitive fusion in turn feeds into psychological inflexibility. Psychological inflexibility describes a dynamic in which an individual becomes entrenched in certain reactive cognitive and emotional habits, which may be maladaptive. Bond et al. (2011) characterize psychological inflexibility as "the rigid dominance of psychological

reactions over chosen values and contingencies in guiding action” (Bond et al., 2011, p. 678). They further note that “this often occurs when people fuse with evaluative and self-descriptive thoughts” (Bond et al., 2011, p. 678). When one is psychologically inflexible, one’s relation to one’s own cognitive-emotional reactions is such that one cannot exert flexible, conscious control over these reactions according to one’s values. By over-identifying with certain thoughts and emotions, one closes off the ability to alter one’s cognitive-emotional habits, and respond more adaptively to stressful situations. In contrast to inflexibility, Steven Hayes characterizes psychological flexibility as “the ability to contact the present moment more fully as a conscious human being, and to change or persist in behavior when doing so serves valued ends” (Hayes, 2006, p. 7). We may observe in psychological flexibility, and the lack thereof, a sense of agency that is at stake: when one is psychologically flexible, one relates to one’s cognitive-emotional reactions and behaviors as open to positive change according to one’s values, rather than as automatic responses over which one has little or no control.

This growing project of developing relational models of SAD (and other disorders), and the mandate for paradigmatic shifts that it entails, is founded in the demonstrated value of the treatments that implicate a relational approach to psychiatric disorders. Mindfulness-based interventions (MBIs) have in recent years attracted increasing levels of attention in the world of clinical psychology, and have met with considerable success in treating various mental disorders. Some examples of mindfulness-based interventions include mindfulness-based stress reduction (MBSR), mindfulness-based cognitive therapy (MBCT), and acceptance and commitment therapy

(ACT). Although differing in their specific methodologies, MBIs share some core principles, such as their foundation in the practice of mindfulness. Mindfulness (and the practice thereof) is characterized by Jon Kabat-Zinn, an influential pioneer and proponent of secularized mindfulness practices in the United States, as “the awareness that emerges through paying attention, on purpose, in the present moment, and non-judgmentally to the unfolding of experience moment by moment” (Kabat-Zinn, 2003, p. 145).

MBSR, a treatment program developed by Kabat-Zinn, focuses on intensive training in mindfulness meditation (Segal, Williams, & Teasdale, 2018). It is generally taught in eight weekly group classes that are between two and three hours in duration, plus an additional longer session (Irving, Dobkin, & Park, 2009). MBSR is centered around the practice of attending to the present moment (i.e. mindfulness), and makes use of a variety of meditation techniques (Irving, Dobkin, & Park, 2009). It has been shown to be effective in reducing symptoms of anxiety and depression in various populations (Serpa, Taylor, & Tillisch, 2014; Hofmann et al., 2010), including patients with SAD (Goldin & Gross, 2010).

MBCT is a treatment program derived from both MBSR and CBT (Kuyken et al., 2010). Its methodology largely resembles that of MBSR, consisting in an eight-week group intervention program (Baer, 2003). Originally designed to prevent relapse of major depressive episodes (Baer, 2003), MBCT has been shown to be effective for treating SAD (Koszycki et al., 2007) in addition to major depressive disorder (Kuyken et al., 2010). A study of MBCT compared to maintenance of pharmacotherapy for

depression found that “MBCT’s treatment effects were mediated by augmented self-compassion and mindfulness during treatment” (Kuyken et al., 2010, p. 1111); we may observe in these findings a concrete linkage between developing relational concepts and a MBI in line with the relational approach to treatment.

Acceptance and Commitment Therapy is another popular MBI that has been shown to be effective for treating SAD (Dalrymple & Herbert, 2007). ACT emphasizes acceptance of one’s thoughts and emotions (including negative ones) rather than avoidance or attempts at alteration, and committed action based on chosen values (Hayes et al., 2006). Bach and Hayes (2002) summarize ACT as a therapy that “teaches patients to accept unavoidable private events; to identify and focus on actions directed toward valued goals; and to defuse from odd cognition, just noticing thoughts rather than treating them as either true or false” (Bach & Hayes, 2002, p. 1129). Hayes writes that, “ACT attempts to change the way one *interacts with* or *relates to* thoughts (Hayes, 2006, p. 8). This characterization of the aims of ACT makes its relational nature plainly clear. He contrasts ACT, MBCT, and other relational approaches to traditional CBT, stating that, “Rather than focusing on changing psychological events directly these interventions seek to change the function of those events and the individual’s relationship to them through strategies such as mindfulness, acceptance, or cognitive defusion” (Hayes, 2006, p. 4).

In general terms, MBIs work by helping the patient to overcome unhealthy modes of relating to herself, the situations she finds herself in, and her cognitive-emotional experience--dynamics that relational models emphasize as crucial

factors in the etiology and/or maintenance of SAD. Through this shift, and particularly through defusion from negative thoughts and feelings, the patient may attain a greater sense independence from the pathological patterns of thought and emotion involved in SAD. This independence may reduce the distress and negative feedback loops that are characteristic of SAD, and furthermore foster a sense of agency to alter these patterns.

### **A comparative assessment of the three approaches**

We may examine the overall significance and value of the information regarding the etiology, maintenance, and potential treatment of SAD offered by these varied approaches, with their distinct levels of abstraction and corresponding conceptual vocabularies. It bears noting that each approach describes reality at a certain level, and uses the concepts and vocabulary appropriate to that level of description. None of these levels invalidate each other; rather, they complement one another, offering different ways of perceiving and understanding a particular subject. Nonetheless, certain concepts that are of central importance at one level may become wholly irrelevant at a more abstract level. Conversely, certain concepts which are entirely real at one level may become incoherent and impossible to grasp at a less abstract level. Neuroscientist Michael Gazzaniga provides a cogent discussion of different levels of description in his book *Who's In Charge?* Gazzaniga, employing the analogy of a ball, writes:

atoms come together and can generate the ball rolling across the floor, but the ball is still made up of atoms. We view the collective behavior of the atoms...at the higher organizational level of the ball...and we see it doing



ball behavior following Newton's laws, but the atoms are there at the core doing their own thing and following a different set of laws (Gazzaniga, 2011, p. 139).

Just because the ball is not a discernible entity when looking at atoms, and an atom is not a discernible entity when looking at the ball, this does not detract from the reality of either of these entities (or their validity as concepts). So, as we assess the strengths and weaknesses of each approach, we should bear in mind that we are not questioning how accurately an approach describes reality, but rather how helpful its mode of describing reality is to the project of understanding and treating SAD.

We may begin with an examination of the neuroscientific approach. This approach appears to derive the main of its force from the efficacy of pharmacological treatments, in combination with a general cultural confidence in concrete, physically-grounded science, rather than from any satisfying explanations it offers regarding the etiology or maintenance of pathological social anxiety. For example, from the several brain-imaging studies that we have discussed and Etkin's and Wager's 2007 meta-analysis, we learn that anxiety disorders, including SAD, involve increased activation during anxiety-related situations in brain regions that are generally associated with fear. We may be inclined to inquire, though, how much this adds to our understanding of SAD, which already included excessive anxiety and fear of certain social situations in its DSM definition. Over and above what is already given by the symptomatology of SAD, which is expressed primarily in terms of subjective experience

(emotional and cognitive) and some behavioral markers, this line of research points to some associated neural structures.

In their meta-analysis, Etkin and Wager state that, “these data support the hypothesis that shared symptoms [of anxiety disorders]—an exaggerated fear response—might be reflected in shared neurobiology” (Etkin & Wager, 2007, p. 1485). This hypothesis seems highly probable, if only because it comprises no more than an application of the principle that all subjective experiences are instantiated by neural activity in an organized, regular fashion. Does this hypothesis enhance our understanding of the etiology and maintenance of SAD, though? Are we to suppose that people develop SAD *because* their amygdalae have become over-active in certain situations, and continue to be affected by SAD *because* their amygdalae continue to be overactive in these scenarios? Or perhaps because of some form of serotonergic dysfunction? Explanations of this kind fail to provide satisfying answers to crucial questions regarding the nature of SAD. For example, they offer no account of why individuals with SAD are only pathologically fearful and anxious in response to particular types of situations, nor can they properly account for how specific cognitions, such as thoughts about how one’s self is being perceived by others, arise and have downstream effects on one’s behavior. Neuroscientific work strives to provide information on the neural level to match up with the more abstract concepts that are furnished to its purview from above, such as social fear or self-focused attention. But this process does not in itself clarify the origin of SAD and how it operates; it only adds brain-level information to the picture. Using the framework and language of the

neuroscientific approach, we may come to know in a robust way *what* is happening in the brain of an individual with SAD in a given situation, but not *why*; we thus achieve description without explanation.

One might object that there is good reason to believe that neurophysiological dynamics and neural activity do in fact cause the symptoms that collectively constitute social anxiety. Ample evidence highlights how brain-level changes can effect changes at the level of conscious experience and behavior. For example, a study by Argyropoulos et. al. (2004), which is cited by Etkin and Wager (2007), found that the depletion of tryptophan (an amino acid necessary for the production of serotonin) caused a significant increase in anxiety in SAD patients who had been successfully treated with SSRIs. And, most formidably, the demonstrated efficacy of pharmacological treatments for various psychiatric disorders (Abramowitz, 1997; Arroll et al., 2005; Marshall et al., 2001) demonstrates the upward causal efficacy of manipulations made directly at the neurophysiological level.

We find ourselves faced with a question, which we cannot hope to properly answer (in part because it is a poorly formulated question), but which we cannot avoid considering altogether: do neural events cause mental events, or do our mental events cause neural events? The neuroscientific approach, in seeking to develop neurobiologically based models for SAD, takes the former to reflect the truth of the matter. However, this position may rely on a conflation of instantiation and causality. The fact that all mental events are physically instantiated in the brain, and that subjective experience and neural activity are inseparable in this sense, does not

necessarily imply the primacy of neural events as the *cause*, per se, of mental events. The widely observable facts that psychoactive drugs affect our subjective experience, and that brain lesions can result in cognitive, perceptual, and/or behavioral impairments, do indeed confirm that changes on the physical level can and do cause changes on the level of subjective experience, cognition, and behavior. However, this domain of evidence does not preclude the possibility of a more nuanced relationship between these two levels, according to which they may be understood to be mutually efficacious.

Research findings that psychotherapies and MBIs, which are enacted in conscious agential activity, can produce observable changes on the level of neural activity and even neuroanatomy refute the notion of a simple unidirectional causality from the neural to the mental level. A study by Ochsner et al. (2002) used fMRI to examine the neural correlates of “cognitive transformation of emotional experience,” or “reappraisal” (Ochsner et al., 2002, p. 1215). To induce reappraisal, participants were instructed to interpret “negative photos” that they were presented with in such a way that “they no longer felt negative in response to them” (Ochsner et al., 2002, p. 1217). This study found that this activity of reappraisal, compared to merely attending to the photos, was correlated with increased activation in the lateral and medial prefrontal cortex and decreased activation in the amygdala and medial orbitofrontal cortex. We may note that the manipulation in this study took place on the level of conscious, agential mental activity, and induced observable changes in neural activity. This study thus presents an inverse dynamic to that of neurophysiologically induced changes in

conscious experience and mental activity (such as in the pharmacological treatment of SAD). Moreover, we may take this study only as one example of the vast literature of neuroimaging research in which manipulations of conscious, agential, mental activity induce observable changes in neural activity. Returning to the subject of treatments for psychiatric disorders, a paper reviewing the literature on the neurobiological effects of psychotherapies (for depression, panic disorder, phobia, and OCD) concludes that “empirical research indicates...that changes made at the mind level in a psychotherapeutic context produce changes at the brain level” (Kumari, 2006). Furthermore, multiple studies suggest that mindfulness practices can produce long-term structural changes in the brain (Hölzel et al., 2011; Pickut et al., 2013).

These studies show that deliberate activity at the level of consciousness, such as that involved in psychotherapies and MBIs, can “cause” neurobiological changes in the same way that pharmacological treatments can “cause” changes in our conscious experience--this sense of causality consists in manipulations at one level inducing observable changes at another. However, these effects are more aptly described in terms of concurrency than causality. Mind and brain are not two inseparable, intimately related entities, but two levels of description for the same entity. This radical unity means that changes at one level of description of mind/brain necessarily entail changes at all other levels (hence the aforementioned bidirectional efficacy).

Despite this radical unity, certain phenomena may arise and exist as such only at a certain level of description. As an illustrative example, let us consider planetary orbit. The phenomenon of planets revolving around a star operates at the level of astronomical

bodies. Stars and planets are composed entirely of atoms and molecules, and in theory may be reduced entirely to these lower-level concepts. However, planetary orbit, as a real observable phenomenon, is driven by dynamics at a level abstracted from these atoms and molecules. Specifically, this phenomenon is driven by the dynamics of gravitational force that astronomical bodies exert on one another. Examining how planets are made up of molecules and atoms may certainly *add* information to our understanding of astronomical phenomena; we may in this manner gain an understanding of how planets and stars are instantiated at lower levels of abstraction. But this examination will not explain how a planet began orbiting around a star or why it continues to do so. We may also note that planetary orbit entails changes in the positions and velocities of large quantities of molecules, precisely because the planet and its constituent molecules are the same entity described at different levels of abstraction. Dynamics being driven on one level will affect all the other levels accordingly. But the phenomenon in question here, planetary orbit as such, is driven by dynamics at the planetary--and not the molecular--level.

Taking the phenomenon of planetary orbit as an analogue for the phenomenon of SAD, we may distinguish a crucial question: at what level of abstraction do the dynamics driving SAD exist and operate? A comparative assessment of the treatment outcomes of pharmacotherapy, CBT, and MBIs, which enact manipulations on the neurobiological, psychological, and relational levels, respectively, may help us to address this question.

A meta-analysis of pharmacotherapies and cognitive behavioral therapies for SAD suggests that these two types of treatment do not differ significantly in their

efficacy (Gould, 1997). A study by Liebowitz et al. (1999) found that pharmacological therapy with phenelzine produced greater improvement than cognitive behavioral group therapy (CGBT) in the short term (twelve weeks) and after six months of treatment maintenance, but was associated with greater relapse rates after termination of treatment. Haug et al., (2003) found that CBT for SAD led to further improvement at a one-year follow-up, whereas the improvements of SSRI treatment had significantly deteriorated by this time. Another study found that cognitive therapy was significantly more effective than an SSRI treatment for SAD during and after treatment, as well as at a 12-month follow-up (Clark et al., 2003). This pattern of results suggests that cognitive behavioral therapies at least come closer to addressing the root of SAD rather than pharmacotherapies (this inference is premised on the notion that a treatment that addresses a disorder on the level upon which the dynamics driving it operate, rather than washing out symptoms by manipulations at lower levels of abstraction, will be more effective at reducing and eliminating pathological symptoms in the long-term, especially after treatment termination).

There is an unfortunate dearth of scientific literature directly comparing the long-term effectiveness of MBIs with pharmacological treatments and CBT for SAD. Nonetheless, we may derive some suggestive evidence from other studies. Miller, Fletcher, and Kabat-Zinn (1995) found that a mindfulness-based intervention called Stress Reduction and Relaxation Program (SR&RP) produced significant short-term improvements on multiple measures for patients with anxiety disorders, all of which were maintained at a three year follow up. Furthermore, MBIs have been shown to be

particularly effective at preventing relapse for other psychiatric disorders, such as major depressive disorder (MDD) and substance use disorders (SUDs). A meta-analysis by Piet and Hougaard (2011) found that MBCT (compared to treatment as usual and placebo) significantly decreased the risk of relapse for patients with recurrent MDD in remission, and was particularly effective at preventing relapse in individuals who had had three or more episodes of MDD. Another meta-analysis “found clear evidence that MBCT was associated with a significant reduction in the risk of depressive relapse/recurrence over 60 weeks compared with usual care,” and further determined that MBCT reduces the risk of depressive relapse/recurrence compared with...maintenance antidepressants” (Kuyken et al., 2016, p. 570-571). Evidence suggests that MBIs can effectively treat SUDs (Chiesa & Serretti, 2014), and that specially designed mindfulness therapies can be effective preventing SUD relapses (Bowen et al., 2009; 2014).

With this collection of evidence in mind (although it fails to directly address differential treatment outcomes for SAD), it appears worthwhile to at least consider that the third, most abstract level, that of one’s mode of self-relation, is the level at which SAD and related disorders take shape, and that psychological and neurophysiological symptoms are entailed by the unhealthy dynamics that arise and operate on this relational level. Indeed, the fact of MBIs’ effectiveness at all at treating SAD and other psychiatric disorders indicates that these disorders involve pathological dynamics on the relational level. This being the case, it seems highly likely that the relational approach has much to offer over and above a merely psychological approach precisely because specific beliefs, thoughts, and interpretations arise in a manner dependent upon more



fundamental attitudes and orientations towards oneself and the world (this is not to say that a relational approach should eclipse a psychological approach, but rather encompass it.) It seems likely that the *core* or *root* of a disorder characterized by dysfunctional thinking and negative beliefs (such as SAD) consists in unhealthy dynamics of the more fundamental attitudes that determine how we approach, interpret, and think about our experiences. Mindfulness-based interventions are characterized by their action upon these more fundamental attitudes, allowing for profound shifts in one's mode of relating to oneself and one's experience, which can ideally afford a related agency over how one engages challenging situations.

### **The need for a new paradigm for understanding mental health and illness**

But what exactly do these kinds of profound shifts consist in? What is shifting? From what and to what is it--whatever it may be--shifting? These are difficult questions to provide satisfying answers to. Relational concepts such as self-compassion, cognitive fusion, and psychological flexibility offer some insight into the dynamics that may be at play here, but it is evident that a larger and more refined vocabulary of relational concepts, as well as cogent frameworks in which to situate them, are necessitated by the goal of developing robust relational models of SAD and other disorders.

Even without regard for the pressing issue of understanding how and why MBIs work, many professionals in the field of psychiatric care acknowledge the necessity of developing new concepts and even conceptual frameworks for the purpose of understanding mental health and illness. For example, a report produced by a planning

committee for the most recent version of the DSM unflinchingly addresses many of the shortcomings of the prevailing manner in which psychiatric disorders and illnesses are understood, defined, and categorized (First, 2002). These include the high rates of comorbidities among discrete disorders defined in the DSM, the short-term diagnostic instability of many such disorders, and the lack of treatment specificity often observed among them (First, 2002). This report proceeds to declare in plain terms:

All these limitations in the current diagnostic paradigm suggest that research exclusively focused on refining the DSM-defined syndromes may never be successful in uncovering their underlying etiologies. For that to happen, an as yet unknown paradigm shift may need to occur. Therefore, another important goal of this volume is to transcend the limitations of the current DSM paradigm and to encourage a research agenda that goes beyond our current ways of thinking to attempt to integrate information from a wide variety of sources and technologies (First, 2002, p. xix).

We may see these two objectives, of developing an understanding of various mental disorders that enables us to make sense of the efficacy of MBIs, and of developing new conceptual frameworks that correct the blatant shortcomings of the current paradigm governing mental healthcare, as seeking a common goal. Both of these projects aim at a paradigm shift in our understanding of and approach to mental illness and healthcare, and a failure of either to address the issues driving the other would be to its own detriment. The development of a new conceptual framework, such as that which we perceive here to be doubly

warranted, would consist in redefining the underlying assumptions that structure our understanding of various psychiatric disorders. The conceptual framework so developed would derive validation from the very issues that threaten the validity of the current framework, such as high comorbidity rates, short-term diagnostic instability, and lack of treatment specificity--and, at the same time, it would readily accommodate relational models (in some form) of the myriad disorders for which MBIs are efficacious.

Buddhism provided the original conceptual framework in which mindfulness practices existed and were made sense of, and may continue to do so for the many religious adherents of Buddhism around the world. However, as mindfulness practices have been brought into mainstream practice in the West, even being adapted into clinical interventions for various psychiatric disorders, considerable effort has been made to secularize these practices, purging them of religious significances and relations (Braun, 2013). And, especially in light of their growing role as a class of clinical treatment for mental illnesses, researchers and theorists have taken up the project of developing scientifically viable constructs that can explain how MBIs work. Our exploration of the relational concepts of self-compassion, cognitive fusion, and psychological inflexibility outlined some such constructs. However, as researchers in this domain attest, this project is only in its early stages, and much theoretical and empirical work remains to be done in order to provide satisfying explanations for the mechanisms of MBIs (Kuyken et al., 2010; Hayes, 2006). Specifically, the expansion of our vocabulary of relational concepts, and the development of a robust and coherent

conceptual structure to clarify the relationships between these concepts would be of particular benefit.

### **Enter existentialism**

There exists a well-developed field of knowledge and inquiry that is of considerable relevance to this present project, whose vast stores of pertinent concepts and vocabulary has yet to be tapped for this purpose. This field is the philosophy of existentialism. Existential philosophy offers a vocabulary of concepts, many of which we may grasp as relational concepts, that are not based in religious belief or practice nor mired in the insufficient scientific paradigms that we presently work to overcome. Indeed, existential philosophy abounds in examination and discussion of individuals' ways of relating to themselves, the worlds in which they are immersed, and the situations and events of their everyday lives.

The relational approach to SAD and other disorders may implicitly derive justification from existential philosophy, in much the same way that the neuroscientific approach appeals to materialism and the psychological approach appeals to phenomenology. Existential philosophy, to use exceedingly broad terms, emphasizes the importance of one's modes of relating to the various aspects and elements of one's existence in determining the realities that one experiences (Heidegger, 1962; Camus, 2013; Sartre, 2012). In this respect, underlying assumptions of relational models, such as that "the relationship a person has with their thoughts and beliefs [is] potentially more relevant than belief content in predicting the emotional and behavioral

consequences of cognition” (Gillanders et al., 2014, p. 84) can be philosophically grounded in the tradition of existentialism.

Furthermore, existential philosophers present a wide range of relational concepts that can inform the development and refinement of a fuller, more robust, and coherent relational framework for understanding psychiatric disorders. The present discussion will focus on Jean-Paul Sartre’s concepts of freedom and bad faith, illustrating the relational nature of these ideas and their relevance to mental healthcare. These ideas, and the broader range of relational concepts in the domain of existential philosophy of which they comprise only two illustrative examples, have the potential to deepen, enrich, and shift our understanding of SAD and similar disorders in a beneficial manner. In this manner, the works of existential philosophers have the potential to be of considerable benefit in the project of developing a new paradigm for understanding mental health and illness.

Before proceeding, we may take note that the terminology and methodology of existential philosophy may appear quite foreign to what we recognize as the proper domain of theory for clinical psychology and psychiatry. Nonetheless, concepts coming from the existential tradition may be abbreviated and tailored to suit our present needs at no detriment to our purposes; we need not fret as to whether or not we are properly beholden to all the details of these thinkers’ original definitions, so long as the more general thrust of their ideas helps us along. Moreover, the initial disorientation entailed by bridging disciplines in this manner seems to be precisely in order given the goal of constructing a new paradigm that can “transcend the limitations of the current DSM

paradigm” and go “beyond our current ways of thinking to attempt to integrate information from a wide variety of sources” (First, 2002, p. xix).

The intention here is not to provide a comprehensive explication of Sartre’s philosophy, nor a fully formed account of how these ideas can be brought to bear on clinical psychology, but merely to provide a rough sketch of certain ideas and how they may connect to and inform relational understandings of SAD. Nonetheless, this brief suggestive exploration may help to illuminate the promise of the tactic of drawing upon existential philosophy to enrich and deepen relational understandings of SAD, and mental illness more generally.

### **Sartre, freedom, and bad faith**

Sartre’s conception of “bad faith” describes a mode of understanding and relating to oneself that is dishonest, failing to reflect the reality of one’s nature and situation as a human (or as the kind of being that humans are). To understand bad faith, we must first have a sense of Sartre’s notions of freedom. Sartre argues that people, as the kinds of beings that humans are, are fundamentally free. According to the Stanford Encyclopedia of Philosophy, freedom for Sartre “is the dislocation of consciousness from its object, the fundamental ‘nihilation’ or negation by means of which consciousness can grasp its object without losing itself in it: to be conscious of something is to be conscious of *not* being it, a ‘not’ that arises in the very structure of consciousness” (Crowell, 2017). For Sartre, the fact that consciousness can grasp its object implies its freedom with regard to that object; in its distinction from the object, consciousness has space to freely choose

its activity in relation to that object, rather than being totally determined by it or lost in it, which would be the case if it were identical to the object. Because consciousness is an object for itself (and not in itself), it is fundamentally free to choose and self-determine (Sartre, 2012).

We can detect in this conception of freedom resonances with ACT and Hayes' definitions of cognitive defusion and psychological flexibility. Hayes argues that a practice that calls attention to our non-identity with the objects of our mental/emotional experience (ACT) allows us to adopt a new relation to those experiences as transient, contingent psychological states rather than as constitutive of our identity (cognitive defusion), and we thus develop the ability to flexibly change these psychological responses in accordance to our values (psychological flexibility) (Hayes, 2006). This combination of cognitive defusion and psychological flexibility clearly bears a strong resemblance to Sartre's freedom, only portrayed as a healthy attitude or mode of self-relation rather than as an inherent attribute of human consciousness.

Sartre also emphasizes our fraught relationship with our own freedom. He characterizes the consciousness of our own freedom as "anguish" (Sartre, 2012). People are often inclined to flee this anguish, and in doing so to deny their own freedom. Sartre terms the attitude that this fleeing from anguish engenders "bad faith." Bad faith, for the most part, refers to a mode of relating to oneself as a determined object. In the attitude of bad faith, one projects the past onto the future, implicitly assuming that what has been will continue to be the case, and so deny one's own ability to transcend this factual past through choice. The Stanford Encyclopedia of Philosophy states:

The most familiar form of bad faith is acting as if one were a mere thing...and thereby denying one's own freedom to make oneself into something very different. Thus, the person who thinks she is a coward 'just as a matter of fact' is excluding from view the ability to transform her existence through changed ways of behaving. Such bad faith is a denial of transcendence or freedom (Varga & Guignon, 2017).

We may observe that bad faith describes a mode of relating to oneself, and an accompanying understanding of oneself, which engenders a belief in one's lack of control over one's own behavior. Thus, this attitude presents a relational concept, describing a fundamental attitude or orientation, that may underlie and give rise to the specific habit of interpreting one's cognitive-emotional reactions to stressful social situations as automatic responses mostly outside of one's control, which Hofmann's psychological model posits as a factor in the maintenance of SAD (Hofmann, 2007).

These examples of freedom and bad faith from Sartre's *Being and Nothingness*, sketched out only very roughly and devoid of their full context, readily exhibit the potential to enrich pre-existing relational concepts as well as inform the development of useful new relational concepts for understanding the dynamics at the core of SAD. In these ways, existential philosophy more broadly shows promise not only as a philosophical tradition that can implicitly serve as an epistemological foundation for the relational approach, but also as a rich field of relational concepts that may be tapped for and adapted to the project of developing a robust, relationally oriented framework for understanding mental health and illness.



## **Conclusion**

We have distinguished between three approaches--the neuroscientific approach, the psychological approach, and the relational approach--which seek to understand and address SAD (and by extension other psychiatric disorders) at three different levels of abstraction. Each of these levels is grounded in assumptions that are characteristic of a certain philosophical tradition, and is interrelated with a particular category of treatment. Of these, the relational approach, which addresses the deep-seated attitudes/orientations that underlie our beliefs and our cognitive-emotional responses to experiences, shows particular promise for explaining the dynamics driving SAD. A new paradigm (or conceptual framework) for understanding mental health and illness could simultaneously resolve many of the issues plaguing the current DSM paradigm and lend itself to the explanation of the broad efficacy of MBIs. This new framework would ideally involve a coherent and robust network of relational concepts, some of which are already cropping up carving out a space for themselves in the scientific literature. In developing such a framework, theorists and researchers may draw upon the rich conceptual lexicon of existential philosophy, which largely concerns itself with questions of how people relate to themselves and their experience of the world.

### Author contributions

Campbell developed the concept for this thesis based on discussions with Dr. Crowley. Campbell and Dr. Crowley continued to discuss the ideas included in this thesis as it further developed, with Dr. Crowley providing input and literature suggestions. Portions of this thesis were inspired by work done by Dr. Crowley at the Yale Child Study Center, in which Campbell was involved.

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