

Psychosis as a Mechanism for Coping with Existential Distress

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Abstract

This paper attempts to explain occurrences of psychosis through a new understanding: as a coping mechanism. This hypothesis seems to have broad explanatory power. Some qualitative evidence also supports this understanding. This paper concludes that it is possible to understand psychotic episodes as mechanisms for coping with existential distress.

Keywords: Existential Psychology; Psychosis; Psychotic Disorders; Coping

1. Introduction

The most puzzling part of psychotic disorders is their etiology. While a variety of hypotheses offer potential explanations, most individuals today accept the theory of psychosis known as the dopamine hypothesis (Howes & Kapur, 2009). This hypothesis posits that a biological malfunction—a dysregulation of dopaminergic activity—leads to psychosis. Precisely why this biological malfunction occurs is unknown—though most agree that genetic and environmental influences play a role—and exactly how this dopaminergic dysregulation leads to psychosis is not agreed upon (Howes & Kapur, 2009). However, while pragmatically useful, purely biological definitions and treatments are not entirely satisfactory. No clear objective, biological marker has emerged with which to identify psychosis (*DSM-IV-TR*). In addition, biological treatments of psychosis are somewhat ineffective: the efficacy of antipsychotic drugs to reduce psychotic symptoms and prevent relapse is only 41% (Leucht, Arbter et al., 2009; Leucht, Corves et al., 2009). The failure of these treatments leaves open a question: are psychotic disorders more than purely biological?

Existential psychology may provide an answer to this question. Existential psychology holds that mortality, responsibility, isolation, uncertainty or groundlessness, and a need for meaning are integral to being human, and as humans, we must face and accept these realities (Yalom, 1980). Indeed, recent research suggests that these existential issues are so profoundly important that we anchor our entire lives around them (Hirsh, 2010). Nevertheless, facing and dealing with these realities can be a painful and overwhelming experience in itself. Many individuals, therefore, avoid dealing with these realities until confronted with them in a boundary situation—that is, any abrupt or intrusive event that immediately confronts an individual with his or her existential issues (Yalom, 1980). Because these issues are so important, existential

psychotherapists see the avoidance of these issues as the cause of psychopathology (Yalom, 1980). If this were true, what would happen when an individual could no longer avoid these issues, but the extreme, overwhelming emotional distress caused by facing these issues prompted that individual to continue to attempt to avoid them? What would happen if someone avoided dealing with these issues when they were unavoidable? At this point, a break from reality would necessarily occur. This paper proposes that, in this light, one sees psychotic episodes for what they may be: a mechanism for coping with existential distress—a way of being that allows an individual to escape existential realities when that individual cannot avoid these things otherwise.

A note on typical coping mechanisms for existential distress is in order before proceeding further. Most individuals mollify existential distress in two ways: through a worldview that provides hope of literal immortality, or through the symbolic immortality of self-esteem garnered from exceeding societal expectations (Burke, Martens, & Faucher, 2010). Terror management theory has shown that these methods of coping have a robust and ubiquitous effect on easing the severity of existential distress. Of note for this paper's hypothesis, research has repeatedly shown that broad measures of religiosity and prayer have significant negative correlations with psychoticism—with small to moderate correlation coefficients (Francis & Wilcox, 1996; Lewis, Francis, & Enger, 2004; Roman & Lester, 1999). However, not all individuals can find relief through these ways of coping. For instance, some individuals may find no comfort from religion or religious beliefs. Additionally, an unresolved feeling of doubt and uncertainty about one's beliefs, which accompanies many religious believers and some consider an integral part of faith itself (Tillich, 1957), can have profoundly negative effects on a person's health and ability to cope with stress (Krause & Wulff, 2004). Moreover, self-esteem is not necessarily stable, and even high self-esteem can, when threatened, increase rates of psychopathological behaviors

(Borton, Crimmins, Ashby, & Ruddiman, 2012). Therefore, both conventional ways of coping with existential distress are not always sufficient, and extreme situations may render both of these methods impotent, necessitating an extreme way to cope.

It is important to note that the hypothesis outlined in this paper is biopsychosocial. While most acknowledge that existential crises can occur for psychological or social reasons, this paper is careful to note that certain brain regions play a crucial role in mediating existential distress. As this paper will later explain, deficits in these brain regions entail a diminished ability to mollify existential distress. As such, individuals with these deficits may suffer a psychotic break when individuals without these deficits would not. However, this understanding of psychosis is not purely biological: these deficits are not necessary for a psychotic episode to occur, as severity of existential distress can overwhelm any individual.

In the next section, this paper offers a brief historical perspective on this hypothesis to better illustrate how this hypothesis is similar and how it is different from other hypotheses. Following that, this paper then explores the explanatory power of this new hypothesis. Next, this paper addresses the traditional, biological model of psychosis. Included in this section is a novel way to understand the efficacy of antipsychotics and a discussion of the predispositions to psychosis. In the following section, this paper examines the supporting evidence for the hypothesis that psychosis often is a mechanism for coping with existential distress—such as studies of individuals who have fully recovered from psychosis. After the evidence for this hypothesis, this paper discusses some limitations of this theoretical perspective. Finally, this paper summarizes the implications of the research discussed herein and offers suggestions for further research.

2. Review

2.1 Historical Perspectives

Bateson, Jackson, Haley, and Weakland (1956) formulated what is perhaps the most famous psychological hypothesis of schizophrenia—the double-bind hypothesis. This hypothesis proposed that psychotic disorders arose out of a history of receiving constant conflicting messages from persons in one's family. The researchers formulated this hypothesis deductively from what most researchers thought about psychotic disorders at that time (Bateson, Jackson, Haley, & Weakland, 1963). However, this hypothesis failed to find empirical support, and after only a decade of research, most acknowledged this hypothesis was inconsistent with the literature (Schuham, 1967). It was around the time of this recognized failure that the one researcher made the first formal articulation of the dopamine hypothesis of schizophrenia (Rossum, 1966). Contrary to the double-bind hypothesis, the dopamine hypothesis rested on a foundation of empirical evidence. Various lines of research, such as the mechanism of action of neuroleptic drugs and amphetamine-induced psychosis, indicated that dopaminergic neurotransmission played a crucial role in producing schizophrenic symptoms. These strong data ultimately solidified this hypothesis as the foundation for an entire generation of researchers investigating the etiology of schizophrenia (Baumeister & Francis, 2002).

In the wake of the success of the dopamine hypothesis of schizophrenia, speculative hypotheses failing to proffer empirical support received little attention in research. The existential psychotherapists Irvin Yalom (1980) and Rollo May (1996) each put forward a hypothesis speculating on the etiology of psychosis from a phenomenological or psychological level. Yalom thought that psychosis might arise when death anxiety overwhelms an individual. Similarly, May proposed that psychotic episodes could occur to cope with anxiety when it is unmanageable by any other method. The hypothesis proposed herein is similar to that of both

Yalom and May, but it has nuanced differences. The proposed hypothesis is similar to Yalom's in that it views existential issues as the underlying prompt of psychotic episodes. It is different, however, because it specifies how this overwhelming distress might prompt a psychotic break—namely, if existential distress becomes unavoidable but unmanageable and a psychotic episode can function as a dissociative mechanism for avoiding that distress. The proposed hypothesis is also similar to May's because it views psychosis as a dissociative coping mechanism that arises in response to distress, but it differs in that it does not assert that a general type of distress could prompt these breaks—instead, it specifies existential distress in particular. The reason for this difference is that we do not anchor our lives around anxiety in general, whereas—as discussed in Section 1—we do for existential issues, and overwhelming generalized anxiety therefore does not cause the same fragmentation of the self that existential distress does. The proposed hypothesis can thus be seen as a synthesis of elements from each of these historical perspectives. However, this hypothesis does not view these psychological processes as operating independently of biological factors; these psychological and existential factors work closely with the structural or functional integrity of the brain in producing psychotic episodes.

2.2 The Explanatory Power of the Hypothesis

2.2.1 The symptomatic manifestation of psychosis. Psychosis has a variety of previously unexplained peculiarities, and this paper will now explore the application of the hypothesis proposed herein to these peculiarities. To begin, positive symptoms and negative symptoms occur together frequently in psychotic individuals (*DSM-IV-TR*). Despite their commonality of occurrence, the reason these seemingly opposite symptoms manifest in tandem is currently unexplained. Viewing psychosis as a coping mechanism allows one to see positive symptoms as part of the coping process: positive symptoms are the construction of an alternate

reality that allows the individual to escape from reality as it actually is. Similarly, negative symptoms are reflective of a withdrawal from reality. Positive and negative symptoms—rather than being dichotomous—work in tandem to allow the psychotic individual to avoid the issues that reality brings when these issues are otherwise unavoidable. Additionally, the reason for the occurrence of one particular type of positive symptom—grandiose delusions—continues to elude researchers (McKay & Kinsbourne, 2010), but this hypothesis seems able to explain its presence. This hypothesis explains the occurrence of grandiose delusions as being an artificial instillation of meaning. These delusions involve being a part of, or used by, something greater or more meaningful than one's self, and they instill meaning to a person's life that otherwise is meaningless. Consistent with this view of grandiose delusions, researchers have found that psychotic individuals experiencing grandiose delusions have a higher self-esteem and mood than psychotic individuals without grandiose delusions (Smith et al., 2006). We imbue everyday events with meaning in response to existential threats (Landau, Kosloff, & Schmeichel, 2011), and grandiose delusions may be an extreme example of this. Additionally, the symptomatic manifestation of psychosis does not relegate itself to psychotic individuals. Instead, psychotic symptoms exist on a continuum even in healthy individuals (Stefanis et al., 2002). This, too, seems to be explicable if psychosis is a way to cope with existential distress—as psychosis would be quantitatively, rather than qualitatively, different from normal.

One final aspect of the manifestation of psychosis that the proposed hypothesis can explain is the fact that most psychotic episodes occur in late adolescence or the early college years. There have been proposed explanations for this fact relating to an apparent psychosis-prone psychological state of adolescents (Harrop & Trower, 2001). The hypothesis proposed herein appears to be able to add dimension to the explanation of this fact. A recent meta-analysis

of data from terror management theory showed that the fear of death has its strongest effect during the college years (Burke, Martens, & Faucher, 2010). This could be because individuals during this time-period are now capable of understanding their own mortality but do not yet have a solidified worldview to buffer existential distress, leading to an increased fear of death. If individuals at this stage of life experience a heightened state of existential distress when confronted with distressing stimuli, this appears to explain why adolescents or young adults exhibit traits in common with psychosis-prone or psychotic individuals. The hypothesis that psychosis is a mechanism for coping with existential distress thus appears to be able to explain a variety of aspects of the symptomatic manifestation of psychosis.

2.2.2 Aspects of delusions. This hypothesis can explain another aspect of psychosis: psychotic individuals do not choose realistic explanations for their experiences (Freeman et al., 2004). If the explanations chosen by psychotic individuals were more realistic, then the problem of reality would continue to intrude on an individual that cannot deal with reality. Because of this, psychotic individuals may choose unrealistic explanations for their experiences to avoid the otherwise unavoidable implications of their experiences. This hypothesis potentially explains an additional aspect of delusions. Psychotic delusions oftentimes relate to the psychotic individual's life problems or goals (Jakes, Rhodes, & Issa, 2004). This fact is presumably consistent with the hypothesis proposed herein. These delusions may be confabulations that allow an individual to handle any aspect of reality that necessarily intrudes into their experience (McKay & Kinsbourne, 2010). If an individual's psychotic state alone is not enough to bear the weight of all issues pressing down on them, distorting the truth of the realities that continue to press down would help alleviate their suffering. If one is able to confabulate to the degree that the delusion fulfills a

given goal or relieves a given problem, then that delusion thereby completely alleviates that part of their suffering.

Delusions may thus serve the dual purpose of generally allowing an individual to avoid reality as well as potentially helping an individual cope with a particularly stressful issue not otherwise avoided. Consistent with this conceptualization, a variety of traumatic existential events prior to the onset of first-episode psychosis predict the content of delusions and hallucinations for that psychotic break (Raune, Bebbington, Dunn, & Kuipers, 2006). Furthermore, this hypothesis can explain the fact that psychotic individuals hold to their delusions with a greater degree of certainty than they do their regular beliefs (Freeman et al., 2004). Delusions play a key role in allowing these individuals to cope, and since these beliefs must hold if an individual's psychosis is to allow an avoidance of reality, psychotic individuals might hold these beliefs with great certainty. The hypothesis that psychosis is an existential coping mechanism thus appears able to account for a variety of facts about delusions.

2.2.3 The dispositions of psychotic individuals. This hypothesis therefore may explain the symptomatic manifestation and content of psychosis. However, one might extend the explanatory power of this hypothesis further still: to the dispositions held by psychotic individuals towards their psychosis. Those suffering from psychosis exhibit one of two dispositions toward their experience: wanting to rid themselves of reality and their experience at all costs, or having an absolute certainty of their experience as veridical (Spinelli, 2001). This hypothesis proposes that these two dispositions are reflective of the ability of a given psychotic episode to alleviate the weight of the issues facing an individual. Psychotic breaks that do not entirely allow psychotic individuals to avoid dealing with the issues that prompted their psychosis would leave these individuals no way to avoid reality—though they desire to.

Therefore, individuals in this state would want to rid themselves of reality and their experience however necessary—if their psychosis is, indeed, an attempted escape from an unbearable reality. Alternatively, for psychotic breaks that do truly allow individuals to avoid dealing with the issues that prompted their psychosis, these individuals would cling to their psychosis with certainty, as their psychosis is what permits them to cope with the crushing distress that prompted their episode. This hypothesis may therefore account for the dispositions of psychotic individuals towards their experience.

The explanatory power of this new hypothesis in explaining the psychological aspects of psychotic disorders thus seems broad and forceful. However, broad explanatory power is not sufficient to establish the truth of a hypothesis—none can call a scientific hypothesis such without proffering evidence in support of it or addressing data that seem to support other hypotheses. It is to the dominant hypothesis of psychosis that this paper now turns.

2.3 Addressing the Dominant Hypothesis

2.3.1 The biological model of psychosis. The biological model of psychosis proposes that a dysregulation in neurotransmission involving dopamine, due to biological factors, somehow induces a psychotic break (Howes & Kapur, 2009). Though a variety of hypotheses exist that purport to explain how this dopaminergic dysregulation causes psychosis, none has emerged as satisfactory. Nevertheless, the biological model is highly successful, and evidence for it is not shortcoming. Therefore, any model of psychosis that purports to explain its occurrence must address the evidence supporting the biological model of psychosis.

2.3.2 Biological predispositions to psychosis. In addressing the evidence supporting the biological model of psychosis, this paper will first discuss biological risk factors for developing a psychotic disorder. One classic and commonly discussed risk factor for psychosis is a head

injury (Symonds, 1937). Although there are conflicting data (cf. David & Prince, 2005), there is good reason to believe that a traumatic brain injury does indeed predispose individuals to psychosis (Molloy, Conroy, Cotter, & Cannon, 2011). However, why and how traumatic brain injuries bring about the occurrence of psychosis is unknown. This paper proposes that this predisposition is due to damage of the lateral prefrontal cortex (IPFC) or connectivity to it. The IPFC is responsible for suppressing unwanted thoughts and memories (Anderson et al., 2004). A consequence of this is that impairing the IPFC entails a diminished ability to avoid dealing with unwanted thoughts and memories. Therefore, an individual who has incurred a head injury that damaged or impaired his or her IPFC cannot avoid dealing with unwanted thoughts or issues to the same degree that a healthy individual can. Because of that, when faced with existentially distressing issues, an individual with an injured IPFC who chooses to try to avoid these issues would suffer psychotic breaks when a healthy individual faced with the same issues would not—as the weight of these issues would not press down as hard on healthy individuals who can repress them. Concurrent with this idea, reduced functioning in this area of the brain seems to increase rates of psychopathology (Anderson & Levy, 2009). Indeed, one difference commonly observed in psychotic individuals is a functional reduction in IPFC activity (e.g., Andreasen et al., 1997; Dolan et al., 1993). Data therefore support the notion that head injuries may predispose individuals to psychosis because they hinder an individual from repressing unwanted thoughts.

One can thus possibly explain the predisposition to developing psychotic disorders from head injuries without reference to the purely biological model, but this is not the only predisposition that a new hypothesis needs to address. Other predispositions exist as well, including genetic abnormalities (Hall et al., 2006) and prenatal infections (Brown, 2006). However, the reason for the association between genetic abnormalities or prenatal infections and

psychosis is unclear (Brown, 2006). It should be noted that these predispositions do not predetermine psychosis; many individuals who develop psychotic disorders did not have these predispositions. Perhaps, rather than these predispositions affecting the developing brain in a way that directly produces psychosis, these genetic or prenatal influences affect the developing brain in a way that alters cognition. These predispositions, then, would not be seen as predispositions towards psychosis directly, but instead as predispositions to aberrant cognitive styles—thereby altering how and individual processes existentially distress. Support for this view comes from a study that examined the link between psychosis and giftedness (Karlsson, 1970). This study found that close relatives of psychotic individuals had significantly increased probabilities for high achievement in scholastic and artistic areas. More forcefully, though, research has discovered that the same genetic abnormalities that predispose individuals to psychosis also predispose individuals with a high intellectual ability to creative achievement (Kéri, 2009). Alternatively, these genetic predispositions produce structural diminutions in the IPFC (Harms et al., 2010). Because these genetic abnormalities—and perhaps prenatal infections—decrease the functioning of the IPFC, these genetic abnormalities thereby partially prohibit affected individuals from repressing unwanted issues and thus increase the severity of any existential distress that they may have. It is therefore reasonable to suggest that these predispositions affect the brain in ways that do not directly provoke psychosis but instead alter cognitive styles, which then intensify existential distress.

While one can account for the aforementioned predispositions by noting the role of the IPFC in repressing unwanted thoughts, one cannot account for another predisposition to psychosis in this way. Drug use—particularly cannabis use—is associated with an increased risk of developing psychosis (Moore et al., 2007). This hypothesis, however, notes that drug use if

often an attempt by an individual to escape from reality. Rather than drug use being the cause of psychosis, then, drug use would be a symptom of an inability to deal with reality as it is—which is a clear precursor to psychosis on this view. Therefore, while there are a variety of predispositions that seem to provide evidence for the purely biological model of psychosis, the hypothesis proposed in this paper may be able to explain these predispositions as well.

2.3.3 The efficacy of antipsychotic drugs. The purely biological model of psychosis does not receive the majority of its strength from citing predispositions to psychosis; instead, the efficacy of antipsychotic drugs is the driving force behind the power of this model. Antipsychotic drugs work by modulating dopaminergic neurotransmission, and by doing so, they provide powerful evidence for the dopamine hypothesis of psychosis (Howes & Kapur, 2009). It is worth noting, again, that the efficacy of antipsychotic drugs is very limited—around 41% (Leucht, Arbter, Engel, Kissling, & Davis, 2009). Most individuals who experience symptom relief eventually relapse, and the difference in efficacy of antipsychotic drugs and placebo disintegrates over time (Leucht et al., 2009). Additionally, overall recovery rates are much better for those suffering from psychotic disorders who elect not to take antipsychotic medication than they are for those who do take antipsychotic medication (Harrow & Jobe, 2007). Furthermore, one randomized trial found that individuals with psychosocial support, but not antipsychotic medication, had better outcomes than individuals provided with both psychosocial support and antipsychotic medication (Carpenter, McGlashan, & Strauss, 1977; cf. Bola & Mosher, 2003). Therefore, it is not obvious that antipsychotic drugs represent the best treatment for psychotic individuals; indeed, they may even do more harm than good (Whitaker, 2004). However, the fact that antipsychotic drugs are efficacious at all in treating psychosis demands an explanation.

This paper's hypothesis—that psychosis might function as a mechanism for coping with existential distress—explains the efficacy of antipsychotic drugs by their ability to help a patient avoid existentially distressing issues. Antipsychotic drugs contribute to a dose-related overall cognitive deficit (Elie et al., 2010). Antipsychotic drugs also cause an overall reduction in brain volume (Ho, Andreasen, Ziebell, Pierson, & Magnotta, 2011), which reflects this overall cognitive deficit. After a lengthy review of the evidence for and against the dopamine hypothesis, one psychiatrist proposed that antipsychotics primarily work not by modifying dopamine but instead by inducing neurocognitive suppression, which diminishes the severity of psychotic symptoms (Moncrieff, 2009).

If these were all the data around the efficacy of antipsychotics, one could reasonably conclude that they work by cognitively reducing the severity of existential distress; however, there are more data. Recently, a neuroimaging study examined the neurological correlates of existential distress (Quirin et al., 2011). This study found that existential distress exceptionally increased activity in the amygdala, right caudate nucleus, and left anterior cingulate cortex. If antipsychotic drugs ameliorated existential distress, one would expect to see either marked antagonism or reduced activity in each of these areas following antipsychotic administration, and, indeed, research confirms this expectation (Blasi et al., 2009; Chakos et al., 1994; Holcomb et al., 1996). Additionally, some antipsychotic drugs increase activity in the LPFC (Blasi et al., 2009). Therefore, not only do antipsychotic drugs reduce the severity of existential distress, some actually endow an individual with an increased ability to repress unwanted thoughts. Antipsychotic drugs also affect the brain in a variety of other ways, but they also have a variety of side effects not related to their efficacy. However, if the hypothesis outlined herein is true, the nature of the antipsychotic cure is temporary rather than permanent; the individual still has not

dealt with the issues that prompted their psychotic break. This temporary nature may explain why antipsychotics increase the chronicity of psychosis: antipsychotics are simply another measure in avoiding the same issues that need resolved. To summarize, this paper explains the efficacy of antipsychotic drugs by their ability to reduce the neurocognitive severity of existential distress.

2.4 Supporting Evidence for the New Hypothesis

While there has not been research performed directly on the hypothesis proposed herein, there is still some justifying evidence. For instance, a qualitative study of those with psychotic disorders in Brazil revealed that existential needs ranked highest out of all their needs (Wagner & King, 2005). The needs for integrity of the self, autonomy, acceptance, love, spirituality, and especially meaning were more important to these individuals than their basic needs—the needs of food, water, and other basic necessities. This peculiar reversal of Maslow's hierarchy of needs is evidence for the hypothesis presented in this paper. Meeting these existential needs would be one of the most fundamental necessities in an individual suffering from psychosis; once these needs are met, a psychotic individual no longer needs to escape reality and can properly engage reality once again. Another piece of evidence for this hypothesis comes from qualitative studies on those who have fully recovered from psychosis, which found that creating a new self-narrative was integral to recovery (Roe & Davidson, 2005). The new narrative created by these recovered individuals allowed them the opportunity to regain a sense of self, to gain a degree of autonomy, and to create meaning from their experience. It therefore appears that addressing the existential is an integral aspect of recovery from psychotic disorders. Lastly, one researcher interviewed six individuals who suffered psychotic disorders and eventually attained full remission in a qualitative study (Williams, 2012). In this study, each of the individuals who

suffered from psychosis incurred an actual or existential threat to the self just before their psychotic break. Out of these individuals, two individuals experienced an actual threat to their lives, two experienced a profound sense of isolation, one experienced a feeling of living in a void, and one felt as if she was losing her sense of self. In each of their cases, these individuals all emerged, personally transformed, out of their psychotic state, which allowed them to overcome the distress of their existential crises. This process and personal transformation seemed to be what brought about the resolution of their psychotic disorders. These qualitative studies seem to lend support to the hypothesis that psychosis might be a way of coping with extreme existential distress, though confirmation of this hypothesis requires more research.

3. Limitations

While this hypothesis appears to have a fair amount of persuasiveness, it does suffer from some apparent limitations. The first of these limitations is that the relationship of the hypothesis proposed herein to amphetamine psychosis, psychotomimetics, psychosis resulting from sleep deprivation, and psychosis resulting from neurodegeneration to the hypothesis proposed herein is not currently delineated. However, these forms of psychosis are explainable given this theoretical perspective; this hypothesis sees biological factors working in tandem with psychological factors, and forcibly altering neurotransmission similar to match what occurs under existential distress may result in psychotic episodes. The second limitation of this hypothesis is that many individuals undergo existential crises without having a psychotic break. However, it may be that individuals who do not have a psychotic break do not attempt to avoid the existential issues they are facing; instead, they may accept these issues or deal with them. Again, though, this is speculation, and it does not easily lead to predictions for further research. A final possible limitation of this hypothesis is that it implicitly assumes that psychosis is a unitary phenomenon,

potentially manifest in differing ways, but some argue that a single concept of psychosis ought to be abandoned for a pentagonal model (Os & Kapur, 2009; White, Harvey, Opler, & Lindenmayer, 1997). However, there is a lack of experimental evidence for a pentagonal model of psychosis (Gaag et al., 2006), and good evidence indicating that a unitary psychotic dimension underlies a pentagonal manifestation (Reininghaus, Priebe, & Bentall, 2012). This supposed limitation therefore does not actually appear to damage the credulity of this hypothesis.

4. Discussion and Conclusion

To recapitulate, the impact of existential issues on one's quality of life cannot be understated. Meaning, mortality, and relationships form the core of who we are as individuals. Because of this, when a crisis in these areas is provoked and apparently irreconcilable, some might wish to try to avoid these issues, though they cannot be avoided. The hypothesis that psychosis is a way of being that allows an individual to avoid existential distress when it is otherwise unavoidable appears to have considerable explanatory power, few limitations, and a fair amount of supporting evidence. In addition, the purely biological model of psychosis does not seem to have enough evidence to command allegiance. As any scientific hypothesis, the hypothesis proposed in this paper has proposed experimental verifications. In a randomized trial of treatment for psychotic individuals, those treated with existential psychotherapy—especially a type of existential psychotherapy that provides an ontological ground for the resolution of existential issues, such as the one provided by Bretherton (2006)—should have significantly better outcomes than those treated with biological methods. Additionally, this hypothesis would predict that individuals prone to psychosis might exhibit a greater level of distress when presented with an existential threat. Research is currently being conducted on this prediction. Furthermore, this hypothesis also predicts that a group of individuals presented with existential

threats, given an antipsychotic, and presented with another existential threat would show a greater decrease in fMRI reactivity to that second existential threat than would a group given either a placebo or an anxiolytic. This hypothesis seems strong enough to merit research, but until this hypothesis is tested, one cannot firmly hold this hypothesis as true. In all, though, the hypothesis proposed herein—that psychosis is a way for an individual to cope with otherwise intolerable existential distress—seems to be a coherent and cogent hypothesis, given the current data.

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