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PTSD and Rilkean Memory

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Abstract: This is a paper on the philosophical clinical psychology of PTSD. How best to improve our treatment plans for the disorder is the primary imperative in the clinical literature. Our failure to properly treat those suffering from PTSD up until now could be either the result of merely a problem in practice or, more seriously, a problem in principle. In this essay, I explore three possible accounts consistent with the supposition that what we have here is a problem in principle. I call the first the "Somatic" hypothesis; the second, the "Memorial"; and the third, a "Rilkean" hypothesis of PTSD, which is inspired by the recent work of M. Rowlands (2015, 2017). The first two have been more-or-less articulated previously; the third is introduced here for the first time. The primary argument of this essay is abductive: I argue in favor of a Rilkean Memory hypothesis of PTSD by comparing it to the other two accounts and showing that it possesses greater theoretical virtue than they do. I then conclude the essay by noting certain limitations with the account here sketched, nonetheless suggesting that this hypothesis can open up new avenues of clinical research and treatment options for the disorder.

Key Words: Posttraumatic Stress Disorder (PTSD); Rilkean Memory; Trauma; Memory; Psychiatry and Psychotherapy

Introduction

Posttraumatic stress disorder (PTSD) is one of the most devastating psychological disorders currently recognized by the American Psychological Association's *Diagnostic and Statistic Manual* of psychological disorders. By "psychological disorder" I mean to refer to a psychological dysfunction "associated with distress or impairment that is not a typical or culturally expected response" (Durand & Barlow, 2016). PTSD is a psychological disorder that follows the stress experienced in the face of some perceived potentially traumatic event (PTE), which occurs at least one month after that experience of the event (DSM-5). Statistics on the disorder are difficult to precisely ascertain, but it would seem that, on average, about 20-30% of people who experience a severe PTE will go on to develop symptoms consistent with a diagnosis of PTSD (Desmedt, Marighetto, & Piazza, 2015; de Vries & Olff, 2009). Somewhere between 6-10% of the general US population will experience symptoms consistent with a diagnosis of PTSD at some point during their lives (Kesler, Berglund, et al., 2005; Hidalgo & Davidson, 2000; Yule 2001). The disorder has about a 3.6% yearly incidence rate (Kessler, Chiu, et al., 2005; Breslau, 2012). The most frequent PTEs that precede the onset of PTSD include injury, threat of injury, and sexual assault, though it is not uncommon to develop PTSD vicariously, as a result of learning of a traumatic experience that another person has had (Devilly & Cotton, 2003; Shaw, 2016). Consequently, those most at risk for developing PTSD are armed forces veterans and sexual assault survivors. It is estimated that approximately 18.7% of Vietnam veterans developed PTSD (Dohrenwend, Turner, & Turse, 2006). By contrast, only about 4.3% of veterans from the wars in Iraq and Afghanistan have developed PTSD; among those who directly experienced combat during those wars, the rates increase to about 7.6% (Smith et al., 2008). Those who have been diagnosed with PTSD are at a greater risk for suicide than the average population. Durkheim (1951) estimated that European armed forces veterans were 10 times more likely to commit suicide than those among the general population. Bullman & Kang (1996), more conservatively, estimate that veterans with PTSD are about 4 times more likely, a still highly significant correlation. If left untreated (and, oftentimes, even when treated), PTSD may run a chronic course.

Frequent presenting symptoms of PTSD cut across several psychological dimensions. Most markedly, those with PTSD show unusually high and sustained hyper-arousal and hyper-vigilance, sometimes resulting in over-aggression. They experience affective symptoms, such as those resembling depression and anxiety-disorders. PTSD is classified as a type of anxiety disorder. However, as Wastell (2005) notes, "[t]here are, however, critical divergences between PTSD and the anxiety disorders. The most important of these is the recognition of an event or situation that triggers the symptoms" (p. 19). See also de Loos (1990) for a discussion between the symptoms of PTSD and anxiety, on one hand, and PTSD and depression, on the other. Sufferers of PTSD experience apparently memorial symptoms, such as intrusive and recurring images and episodes of the PTE, both in wake (flashbacks) and sleep (nightmares); and, paradoxically, those with PTSD often show signs of retrograde (and/or functional) amnesia for significant details of the PTE. This frequently occurring symptom is noted by the DSM and corroborated by Krikorian and Layton (1998), Yovell, Bannett, and Shalev (2003), and Granja, Gomes, Amaro, et al. (2008). (But, given the importance of the symptom in what follows, see also Berntsen and Rubin (2013) for dissenting opinion.) Additionally, those with PTSD often show signs of broad cognitive dysfunction(s) (e.g., erroneously blaming themselves for the occurrence of the event and over-estimating the likelihood of re-experiencing similar events), as well as maladaptive behavioral dysfunctions (such as avoiding all stimuli, however remote, associated with the memory of the PTE) (see the DSM-5, as well as Durand & Barlow, 2016). Moving forward, I will take all of these symptoms as important for understanding the disorder, such that an adequate hypothesis of PTSD and have something to say about them all.

Neurological research on PTSD has made some progress since the disorder was first standardized by the DSM in 1980. We now know that, by and large, two neurological systems are implicated in the disorder's etiology and maintenance: the amygdala and the hippocampus. This is not to say that these neurological systems are exclusively responsible for the disorder, but only that these two systems have been most consistently observed and determined especially important. Other neurological systems, less consistently observed, include Broca's area, which can explain why

PTSD flashbacks are often experienced as cases of "speechless terror." The amygdala is largely responsible for psychological affective states, especially fear, and is thought to assign emotional valence to experience given to it by the thalamus. As van der Kolk (2005) writes, "A large body of animal research, mostly in rodents, has established the importance of the amygdala for emotional processes (Cahill and McGaugh, 1998; LeDous, 1996). The amygdala establishes the initial interpretation of the nature of the particular stress and initiates the process of activating neurochemical and neuroanatomic fear circuitires (LeDoux, 1998)" (see also van der Kolk, 1994, p. 259). And the hippocampus is largely responsible for contextual memory formation and memory retrieval.

> The septohippocampal system, which is abject to the amygdala, is thought to record in memory the spatial and temporal dimensions of experience and to play an important role in the categorization and storage of incoming stimuli in memory. Proper functioning of the hippocampus is necessary for explicit or declarative memory. The hippocampus is believed to be involved in the evaluation of spatially and temporally unrelated events, comparing them with previously stored information and determining whether and how they are associated with each other and reward, punishment, novelty, or nonreward (van der Kolk, 1994).

Additionally, "[t]he hippocampus plays a significant role in the capacity to consciously recall a previous life event, that is, in declarative memory" (van der Kolk, 2004, p. 332; see also Gray 1982). Generally speaking, increased amygdalar activity in the face of an attended-to event increases hippocampal memorial formation of the event (see, e.g., Shaw, 2016, pp. 165-169, on "flashbulb memories; see also Desmedt, Marighetto, & Piazza, 2015, p. 292). Moreover, under-active amygdalar activity is correlated with a decrease in memorial formation and retrieval for that event. Extreme over-active amygdalar activity, however, can short-circuit the hippocampus, and in those suffering from PTSD, it is common to observe continued over-active amygdalar activity, as well as lower hippocampal volume relative to the average person (Kitayama et al., 2005; Bremner, 1997; Gurvits et al., 1998). Etiologically, then, the amygdala is a prime player in the development of PTSD, and the prolonged symptoms associated with the disorder are the result of these two systems — though the exact causal story of the

disorder's maintaining conditions is much disputed. I won't go into much further detail of the neurobiology of PTSD throughout the rest of the essay, but see Vasterling and Brewin (2005) for further study.

While great progress has made been in improving the diagnostic validity of the disorder and uncovering the neurological systems implicated in it, treatment for PTSD has staggered; progress is slow (Wolfe & Keane, 1990). It is estimated that only about one in every three persons suffering from PTSD who undergo our best evidence-based treatments for the disorder are subsequently cured, leaving about two-thirds posteriorly morbid (Cukor et al., 2010; Bradley et al., 2005). At present, Trauma-Focused Cognitive-Behavioral Therapy (TF-CBT) is the front-line response to PTSD. However, some meta-analyses suggest that Brief Psychodynamic Therapy is, perhaps, just as effective (Bradley et al., 2005; Shedler, 2010). See Hendin (2017) and Krupnick (2002) for more information on the latter psychotherapeutic technique. For this reason, finding better methods for treating those suffering from PTSD is the prime imperative in the clinical literature.

Answering this imperative, however, is difficult. Why are our best treatments for PTSD largely ineffective? Note the phrase "best treatments." As Cukor et al. (2010) note, many clinicians either do not use these methods or are unaware of them, compounding the problem substantially. There are at least two ways we might diagnose the problem. First, it might be thought that our failure to treat those suffering from PTSD is merely a problem *in practice*: we are correctly understanding what the disorder is, but we have just yet to create the best methods for interfering with the disorder's maintaining conditions. This is one possibility. However, second, it might be thought that we are failing to properly treat those with PTSD because of a problem *in principle*: notwithstanding our limited clinical success, we have yet to properly understand what the disorder actually is and how it maintains itself. Consequently, we are mostly targeting the symptoms of the disorder while failing to address the disorder itself. In an attempt to answer this imperative, I would like to explore this second option in this paper; we are failing to treat those suffering from PTSD because we do not yet have a clear understanding of the disorder's ontology.

There is some reason to suspect that this second possibility is the correct

diagnosis. To demonstrate that this is the case, I would like to begin by reviewing two of the most promising hypotheses of PTSD to date. I'll call the first hypothesis the Somatic Hypothesis. B. A. van der Kolk is largely accredited with this view, though he himself attributes it to the pioneering work of (especially) Janet (1889), Freud, and Kardiner. And I'll call the second, the Memorial Hypothesis. This hypothesis is discussed most frequently within the literature on propranolol and its potential use as a treatment for PTSD. (More on this later.) Each hypothesis is prima facie plausible, largely because each takes seriously the neurology implicated in the disorder. This feature, of course, is not enough to make a theory worthy of acceptance, but it is nonetheless a *conditio sine non qua* on any theory's immediate plausibility. However, both cannot be true, and both have certain problems making sense of the presenting symptoms of the disorder. I'll begin by detailing both theories and showing that this is the case. Then, I would like to introduce a third hypothesis — what I will call a Rilkean Memory Hypothesis of PTSD, which is inspired by some of the recent work of M. Rowlands (2015, 2017). This hypothesis, I will argue, has all of the merits of the other two and yet is able to avoid the unique problems associated with each. After introducing this hypothesis and demonstrating that this is the case, I will then consider certain objections one might have to the proposal and end the paper by discussing its implications for treatment options for those suffering from PTSD. While there are certain limitations present, there is some reason to believe that this hypothesis could be the correct one. If so, it could thereby open up exciting new avenues of practical clinical research.

Somatic Hypothesis of PTSD

The Somatic Hypothesis of PTSD presumes, like all good theories of PTSD, that in the face of the PTE, the amygdala becomes over-active, and that this over-activity causes broad hippocampal dysfunction. However, this hypothesis interprets this hippocampal dysfunction in a distinct way. It is postulated that, as a result of the hippocampal dysfunction, the emotional significance attached to the experience fails to get fully integrated into memory. Instead, the experience remains and gets transformed into a series of "sub-cortically mediated responses." In other words, instead of being directed

to memory, the stress gets directly transcribed into the body. “Although memory is ordinarily an active and constructive process, in PTSD failure of declarative memory may lead to organization of the trauma on a somatosensory level (as visual images or physical sensations) that is relatively impervious to change” (van der Kolk, 1994, p. 253). “Various external and internal stimuli... decrease hippocampal activity. However, even when stress interferes with hippocampally mediated memory storage and categorization, some mental representation of the experience is probably laid down by means of a system that records affective experience but has no capacity for symbolic processing or placement in space and time” (van der Kolk, 1994, p. 261). The slogan of this hypothesis, then, is, with respect to trauma “the body keeps the score.” These sub-cortically mediated states then continue to stimulate the amygdala, which continues to cause hippocampal dysfunction.

The Somatic Hypothesis does an excellent job at explaining several of the symptoms associated with the disorder. The amnesiac symptoms follow from it straight-forwardly: having failed to get integrated into the person's memorial system, it is to be expected that he / she would experience memorial recall failure. This theory also directly explains the persistent hyper-arousal seen in those suffering from the disorder — the hyperarousal is just the direct manifestation of those sub-cortically mediated states. Additionally, this hypothesis can explain the flashbacks and nightmares experienced by those suffering from the disorder — since those experiences failed to get integrated and contextualized by the hippocampus, when the amygdala is notably over-active, those unprocessed experiences reoccur as if they were being experienced in the present, as fragmentary and distorted current perceptions. In this way, this hypothesis can not only explain some of the most significant presenting symptoms of PTSD, but it is also consistent with the neurology implicated in the disorder.

Additionally, it has been found that those who have experienced a PTE and will go on to develop PTSD are often unable to recount many of the details of the PTE immediately following the event, suggesting that those experiences have failed to be integrated into contextual memory (van der Kolk, 2005, p. 330). Moreover, it is not uncommon for persons with PTSD to visit their physicians first complaining of

unspecified somatic symptoms, well before experiencing a flashback or nightmare of the PTE, indicating the actual etiology of the symptoms.

> Usually, when survivors of trauma apply for help, either to their family doctors, to emergency rooms, or to different physician specialists, they accentuate their physical complaints. Survivors rarely mention a traumatic war history in their initial medical contacts unless they have already been identified as victims... The histories of survivors of all kind of severe war experience have demonstrated that many of these individuals did not understand the underlying cause of their problems until a syndrome of nightmares and intrusive recollections provided thematic material necessary for recognition... In retrospect, clinical recognition [is] often preceded by more subtle signs of impending decompensation. These are usually described as 'nonspecific' when the underlying cause is either not recognized or investigated by an emphatic clinical. Such nonspecific complaints often prove to be of a physical character and may persist after the manifestation of frank posttraumatic stress disorder (de Loos, 1990, p. 95).

Finally, if this hypothesis is correct, it could also explain why many of our best treatment plans for the disorder fail: the central nervous system (CNS) has limited ability to interfere with sub-cortically mediated states; so, since most of our best evidence-based therapies (e.g., cognitive restructuring therapy) attempt to interfere with the disorder via this and similar channels, we can make sense of why the disorder has remained so recalcitrant to intervention. For more information on CRT, see Stahl & Grady (2010), Grady (2010), and Cukor et al. (2010). All of this strengthens the case that this hypothesis is correct.

However, there is certain evidence that would suggest that, while this hypothesis is partially correct, it is not likely fully correct. It has been found that, despite sufferers of PTSD not being able to immediately recount details of the PTE following the event, autobiographical memory is nonetheless strongly implicated in the disorder. First, over time, many persons with PTSD are able to recount additional details of the PTE. If these experiences were not actually integrated by hippocampal memorial systems, it is difficult to explain how this could be the case. Moreover, it has been found that persons with PTSD are more likely to assign great autobiographical significance to those details of the event that they do remember. It certain studies,

when prompted with word-cues (of both positive and negative valence) and asked to recall a time in their lives involving the concepts, persons with PTSD are much more likely to report experiences directly pertaining the PTE (Sutherland & Bryant, 2008). So, while immediate recall of the PTE may be impaired, these observations indicate that autobiographical memory for the PTE has, in fact, occurred. If the emotional significance assigned to the experience by the amygdala has bypassed hippocampal integration, it is, again, difficult to make sense of how this could be the case.

Finally, this hypothesis offers an unintuitive explanation for how delayed onset PTSD works. As indicated, PTSD is a psychological disorder that occurs at least one month after the experience of the PTE. Some who experience a PTE develop trauma-like symptoms immediately following the event. Those who experience PTSD-like symptoms before one month after the PTE are generally diagnosed with acute stress disorder (ASD) (see DSM-5). On average, about 50% of those who suffer from ASD will later qualify for a diagnosis of PTSD; 50% of people, happily, will not (Bryant, 2010). However, oddly enough, only about 50% of people who develop PTSD previously met criteria for a diagnosis of ASD (Bryant et al., 2011). And even more oddly, some people do not develop PTSD until much, much later after the experience of a PTE. Consistent with the Somatic Hypothesis of PTSD, we should expect a higher percent of those suffering from PTSD to have met criteria for ASD. Additionally, it is difficult to make sense of what is happening in cases of very delayed PTSD onset. This hypothesis would propose that there must be active and inactive sub-cortically mediated states; some activate immediately, and some, for whatever reason, do not. This hypothesis would need to offer some additional causal story for how and why this happens. Such a story does not appear forthcoming.

Memorial Hypothesis of PTSD

So, despite a great deal of intuitive appeal and evidence in its favor, the Somatic Hypothesis of PTSD is likely not the full explanation of how PTSD maintains itself. Another point is worth stressing: the Somatic Hypothesis postulates that the low hippocampal volume observed in those suffering from PTSD is likely correlated with under-active hippocampal memorial functioning. This is one possibility, but another

possibility is that it may be associated with hyper-active hippocampal activity. After all, higher amygdalar activity in the face of an event is usually correlated with greater memorial formation for that event (e.g., "flashbulb memories"). Consequently, lower hippocampal volume may not reliably indicate lower hippocampal activity levels.

The Memorial Hypothesis of PTSD postulates an alternative explanation. Theories of this sort are suggested by, for example, McCleery and Harvey (2004) and are also commonly suggested in literatures surrounding memorial-focused treatment plans for PTSD, such as the use of propranolol. I'll present a reconstructed precisification of one such proposal here. Like the alternative hypothesis, the Memorial Hypothesis presumes that over-active amygdalar activity causes broad hippocampal dysfunction — but, in this case, over-active hippocampal functioning. According to this theory, as a result of the significant emotional valence assigned to the experience of the PTE, the memory of the PTE gets over-consolidated in memory. Proponents of this hypothesis point out that over-active amygdalar activity generally also causes the person to narrow his / her attention to environmental cues; the salient features of the context are remembered very vividly, while peripheral details are largely ignored and so fail to make their way into long-term memory (McCleery & Harvey, 2004). Accordingly, this hypothesis postulates that PTSD maintains itself as a result of over-consolidated memories. Once over-consolidated, pathological remembering of those salient details takes place. Schacter (1999) has proposed that there are "seven sins of memory," one of which is memorial over-persistence. Hui and Fisher (2015), likewise, describe it as a kind of "pathological" form of memory occurrence. This could be what we see here.

In this way, the Memorial Hypothesis of PTSD is, likewise, able to offer an account of how several of the symptoms of PTSD occur and persist. The flashbacks and nightmares are a direct result of over-consolidated memories pathologically intruding upon the person. As indicated, the amygdala is able to directly influence the hippocampus; however, the hippocampus is also able to stimulate the amygdala (Soeter & Kindt, 2010; Phelps, 2004). When one recalls an emotional salient experience, the amygdala is activated in ways similar to the way it was activated in the face of the event – i.e., processes of long-term potentiation. So, the over-active

amygdalar activity (and the corresponding somatic and affective symptoms consequently produced) can be explained as indirect causes of over-active hippocampal activity. Whenever the experience is relived and the person further experiences terror in the face of it, the memory continually gets over-consolidated and re-consolidated, creating a vicious cycle (see, e.g., McCleery & Harvey, 2004, p. 490). In this way, the disorder maintains itself primarily as a result of memorial dysfunction. Whatever the details exactly, this hypothesis is clearly correct that memory is an important variable in PTSD and its treatment outcomes (see Tyron & McKay, 2009).

Like the Somatic Hypothesis of PTSD, the Memorial Hypothesis is also able to explain how the symptoms arise from the dynamic relationship between the amygdala and the hippocampus. There is additional evidence is support of this hypothesis. Recently, many clinicians have found some success at treating and preventing PTSD using the drug propranolol. Propranolol is a beta-blocker that is thought to interfere with amygdalar protein synthetization, which can indirectly slow down hyper-active amygdalar activity and interfere with processes of re-consolidation. Though note that some dispute this and see propranolol instead as enhancing extinction learning for conditioned stimuli (see Giustino, Fitzgerald, & Marren, 2016). When persons who have recently experienced some PTE are given propranolol within six hours of experiencing the event, they are less likely to later meet criteria for PTSD (Pitman et al., 2002). Additionally, some have found that, even much later, processes of reconsolidation can be interrupted. See Kredlow & Otto (2015) for a study that attempted to target processes of re-consolidation psychotherapeutically, rather than psychopharmacologically. In these cases, the person is told to recall as many details of the event as they are able, and his / her memories are confounded. Later, when asked to recall the event, they experience much less distress in the face of it. It is thought that, in the first case, propranolol works to strip the experience of much of its negative affective valence, leading to weaker consolidation. And in the second case, it is thought that when memories are recalled, they enter a “labile” state, in which concurrent experience, affect, and information is able to directly interfere with the memory's re-consolidation.

Additionally, it is possible that Exposure Therapy (ET) — perhaps our best

evidence based treatment plan for PTSD — works in a similar fashion (see, again, Stahl & Grady (2010) for more on this therapeutic technique). The person is made to confront stimuli directly related to, or reminding the person of, the PTE and to form new emotional attachments to it / them. For example, a person who has developed PTSD as a result of experiencing a horrific car crash may be asked to visualize driving; then to come into contact with a car; then to sit in a car; and, finally, to drive a car. At each stage, the person is rewarded for their behavior, creating more positive emotional valences associated with the conditioned stimulus. After repeated exposure and de-conditioning, the memories of the PTE, it is thought, can be reconsolidated in a much weaker fashion, eventually extinguishing the pathological remembering associated with the PTE. The (albeit limited) efficacy of ET adds additional support to the Memorial Hypothesis.

However, like the Somatic Hypothesis, the Memorial Hypothesis of PTSD suffers from certain setbacks. First, this hypothesis doesn't account well for the retrograde amnesia observed in many persons suffering from PTSD. As indicated, over-active amygdalar activity is known to cause narrower attentional focus. In this way, the Memorial Hypothesis presumes that it is not so much the case that forgetting is involved as it is a failure of integration of peripheral details. However, in many cases of PTSD, especially cases involving childhood trauma, it is not uncommon to observe total amnesia — the person suffering from the disorder apparently has no memories of the PTE whatsoever. In such cases, there is a great risk, when talking with the person, of inadvertently helping them to create false memories of the event, or, at the very least, of helping them to amplify whatever fragmentary memories of the event that remain (see Shaw, 2016; Oulton et al., 2018, for more on memory amplification and elaborate cognition in relation to PTSD). It is difficult to say that the problem here is one of over-consolidation, since apparently no factual memory of the event remains at all.

Indeed, some theorists have proposed, alternatively, that retrograde amnesia is at the heart of the disorder and that propranolol, even if it seems like it works, likely does more harm than good, except in severe cases, since it may, in fact, mask the direct symptoms of the disorder and destroys the person's opportunity to work through and

integrate the experiences the person does have of the PTE (Desmedt, Marighetto, & Piazza, 2015). While I myself remain neutral with respect to this proposal, I believe this alternative proposal is at least correct to point out the importance of forgetting in the maintaining conditions of PTSD — a fact that is not properly accounted for in the Memorial Hypothesis of the disorder.

Rilkean Memory

To summarize the argument so far, I claim that the Somatic Hypothesis is right to emphasize the role of the affective and somatic symptoms of PTSD but is flawed insofar as it fails to properly account for traumatic remembering. And the Memorial Hypothesis is right to emphasize the autobiographical memorial significance of the trauma for the person suffering from PTSD but flawed insofar as it fails to properly account for traumatic forgetting. I would now like to introduce a third account. It is in many ways a hybrid of the two and attempts to synthesize the insights from both while avoiding the unique pitfalls of each. The hypothesis I propose relies centrally on a concept recently engineered by M. Rowlands (2015, 2017), called "Rilkean Memory," which is also further developed here. I'll begin by elucidating the concept, and then I'll detail my proposal involving it.

Rilkean memory may be defined as a form of non-declarative, embodied, autobiographical memory. Rowlands (2015) himself defines Rilkean memory as "a type of involuntary autobiographical memory that is not Freudian, neither implicit nor explicit, neither procedural nor declarative, and neither episodic nor semantic" (p. 148). I have offered an alternative definition since this definition is unnecessarily apophatic. However, interestingly, depending on one's ontology of memory, the two definitions may amount to the same thing. Rowlands (2015, 2017) reports that he was inspired to develop the concept while reading through the writings of the German poet, Rainer Maria Rilke. In his novel, *The Notebooks of Malte Laurid Brigge*, Rilke's narrator writes:

> And yet it is not enough to have memories. You must be able to forget them when they are many, and you must have the immense patience to wait until they return. For the memories themselves are not

> important. *Only when they have changed into our very blood, into glance and gesture, and are nameless, no longer to be distinguished from ourselves* — only then can it happen that in some very rare hour the first word of a poem arises in their midst and goes forth from them (Rilke, 1985, p. 14).

The passage I have italicized is the significant one in this context; a Rilkean memory is a memory that has lost its content but nonetheless been "changed into [the] very blood, into glance and gesture, and [is] nameless, no longer to be distinguished [from the person]."

The concept of Rilkean memory is controversial. This is so because, if such memories exist, then our traditional taxonomy of (long-term) memory is in need of revision (see Michaelian & Sutton, 2017, for a set of popular ontologies, some alternative to the one that I presume). Generally speaking, all memories are usually cataloged into one of two broad genera: declarative and non-declarative memory. Declarative memory has two dominant species: episodic memory (the kind of perspectival memory one has when one remembers an actual experience one has formerly had) and semantic memory (roughly, memory of facts, either about oneself or otherwise); autobiographical memory may be one or both of these kinds. Non-declarative memory, by contrast, is generally said to be content-less, or, at the very least, consciously inaccessible; consequently, it is generally said that there are no autobiographical non-declarative memories. Examples of non-declarative memory include procedural memory (memories of how to perform some tasks) and implicit memory (memories of stimuli that one has, or so it is thought, even if inaccessible, since, by virtue of having them, one performs better on some task that could only be explained by supposing that one has them). Therefore, if Rilkean memories exist, it would follow that this ontology of memory is not jointly exhaustive, not mutually exclusive, or neither jointly exhaustive nor mutually exclusive. Though nondeclarative and nonintensional, they are nonetheless, in a significant sense, autobiographical.

Rowlands hypothesizes that there may be at least two kinds of Rilkean memory: affective and behavioral. Rowlands (2015, 2017) also sometimes refers to this second kind of memory as just "embodied Rilkean memory." I have chosen the alternative

designation because, with Prinz (2004), I suppose that affect is somatosensory and thus embodied, as well. Here's an example of the former (compare with Rowlands, 2015, p. 148): Suppose you dated a person named Beau back when you were a freshman in high school. Beau used to wear a distinct perfume. You're now thirty-five; you haven't thought about Beau in at least fifteen years, at least. You're walking through a department store, and suddenly you get a whiff of that scent. Sensing it, you're likely to stop and prick up your nose, taking it in. In such a situation, memories of Beau may come flooding back. On the other hand, they may not — after all, it has been a long time and it is not unreasonable to suppose that your memories of Beau have gone. In either case, you still experience an affective state — the same kind(s) of states you experienced long ago, when dating Beau. According to Rowlands, we have here an example of affective Rilkean memory. Your affective state, regardless of whether it is accompanied by declarative memory, just is your autobiographical memories of dating Beau. Rowlands clarifies that he does not just mean that your memories are the *cause* of your affective state(s); rather, he means that your memories are now *constituted* by your affective state(s). Your affective state is the memorial acts you once had that were accompanied by declarative memory that has been transformed into an affective state. Here we have a memory, though forgotten, that has changed into your "very blood," no longer to be separated from yourself.

That would be the kind of thing that would count as an affective Rilkean memory. An affective Rilkean memory is a kind of non-declarative, embodied, autobiographical memory that just is an affective state. A behavioral Rilkean memory, but contrast, would be a Rilkean memory that just is a behavioral disposition. A brief example of this kind of memory would be the sloped shoulders that accompanies a person who has experienced a lifetime of disappointment (Rowlands, 2017, p. 58). The specific episodes of disappointment, happily, may be forgotten (but maybe not); nonetheless, the sloped shoulders remain. This unique posture and behavioral disposition just is what the memories of those experiences have become — it is the result of those memories changing "into glance and gesture," no longer to be distinguished from oneself.

Both kinds of memories are similar in terms of their causal origin and common

processes each has undergone. The thought is, one comes to feel a certain way or act in a certain way on the basis of some autobiographical memory (or memories). Those feelings and behavioral dispositions come to partially *constitute* the memorial acts themselves. Usually, for every memorial act, there is a memorial content. But, in certain cases, the memorial content gets lost (i.e. undergoes "intentional breakdown"), nonetheless leaving the affect or behavioral disposition behind (see Rowlands, 2015, 2017). Despite being contentless, there's a sense in which they nonetheless still count as types of memories; as Rowlands says, they link us in very concrete ways to our personal pasts. Rowlands (2015): "Rilkean memories are like the dark matter of memory. They hold everything together."

Rilkean Memory Hypothesis of PTSD

I'll now propose that the concept of Rilkean memory — especially affective Rilkean memory — can be used to preserve the insights of both the Somatic and Memorial Hypotheses of PTSD, while making for a novel hypothesis of what PTSD is and how the disorder maintains itself. I'll focus on affective Rilkean memory, though, given the neurobiology of PTSD, behavioral Rilkean memories could be useful for explaining some of the symptoms of PTSD, as well.

If affective Rilkean memories exist, then it is plausible to suppose that Rilkean memory would implicate two neurological systems: the hippocampus and that / those neurological systems implicated in affective states — if the affective state is fear, then it will implicate the amygdala. It would implicate the hippocampus because Rilkean memories would have begun as memorial acts (likely) with episodic memorial contents. According to Rowlands (2017), Rilkean memories would most likely be transformed episodic memories. Those affective states would have been such to stimulate the amygdala, since fearful episodic memories all do so. Then, after undergoing intentional breakdown, those fearful memories would have just been transcribed into the body as fearful affective states (sub-cortically mediated amygdalar activity). (In conversation, a colleague has suggested the intriguing possibility that such a process might be measured via allostatic load count.) This is not to say that the process of intentional breakdown must be complete, nor that the person has entirely lost the

corresponding ability to consciously recall the episode of those memorial acts, but only that it would likely imply there being sub-cortically mediated affective states. (Note that Rowlands does not make room for the possibility that intentional breakdown of an episodic memory may be partial. So let this be understood as a development of the concept.) Importantly, these are, of course, just those observations made with respect to the neurology of PTSD.

A Rilkean memory hypothesis of PTSD, then, may be externally consistent with the neurology of PTSD, making it a candidate competing explanation. How, then, might a Rilkean memory hypothesis of PTSD go? Here's one explanation — When the person experiences the PTE, the amygdala becomes over-active; it then short-circuits the hippocampus, leading to reduced hippocampal volume. Just as in the Memorial Hypothesis, I presume this leads to hippocampal over-activity and processes of increased memorial functioning. However, this increased memorial activity is only partially constituted by processes of over-consolidation and reconsolidation; it is also constituted by increased processes of intentional breakdown (i.e. Rilkean memory production), which produces a surplus of affective states. It is known that the hippocampus is partly responsible, additionally, for emotional control — i.e., monitoring the activity of the amygdala. “The high density of glucocorticoid receptors in this structure supports the idea that the hippocampus may play an important role in emotion regulation” (van der Kolk, 2004), p. 332). Because the hippocampus also shrinks as a result of over-active amygdalar activity, I presume it loses some functionality, especially functionality pertaining to regulated amgydalar-mediated states. This not only causes an increase in the production of episodic memories undergoing intentional breakdown and being written “in the very blood,” but it also causes those affective Rilkean memories to arise pathologically and over-frequently, without the hippocampus being able to prevent it. This is experienced, primarily, as a bodily disorder — a constant state of over-arousal and fear (reminiscent of that fear experienced in the face of the PTE).

A hypothesis of this sort can also explain the symptoms related to the disorder. Like in the Somatic account, the affective and somatic symptoms of the disorder may be thought of as the direct manifestation of the primary causal player in the disorder's

maintenance. However, like the Memorial account, it can also explain the memorial symptoms of the account, since it treats both hippocampal and amygdalar activity (those processes implicated by affective Rilkean memory) as equally central. The flashbacks occur as the result of the memory pathologically recurring and the person's attention being constantly stimulated to process every cue as a present threat. This goes also for the nightmares. And the amnesia occurs as the result of the hippocampus simultaneously over-consolidating episodic memory and over-intentionally-analyzing them. More memorial acts lose their content than on average, and those that remain are particularly fearful. Finally, this hypothesis can account for observed pathological misremembering associated with the disorder too. In such cases, much of the actual memory of the PTE has undergone intentional breakdown. Hence, when the person suffering from the disorder does attempt to remember the full event, they are unable to do so and inadvertently end up fabricating new details to complete the record incompletely stored in declarative memory. (Similar behavior, incidentally, is observed in people who have undergone hypnosis and are later discovered to have false memories of why they have acted as they did while under the influence of it. Loftus (1997) suggests that such misremembering occurs as the result of "imagination inflation," which plausibly is also what occurs in cases of posttraumatic misremembering too.)

In this way, this hypothesis relies on insights from both the Somatic Hypothesis and the Memorial Hypothesis; PTSD is centrally a disorder involving both over-consolidated memory and an over-production of sub-cortically mediated states (rogue affective states resembling those experienced in the face of the PTE). Moreover, this hypothesis also avoids the pitfalls of both. The Somatic Hypothesis was found lacking because it could not explain how autobiographical memory could be implicated in the disorder. On this account, autobiographical memory is necessary, since Rilkean memories are formed from autobiographical episodic memories, and those alone. It should be noted that Desmedt et al. (2015); Brewin, Dalgleish, & Joseph (1996); and others also put forward a dual memory hypothesis of PTSD. However, on these accounts, it is generally presumed that both memorial systems are able to form their respective memories independently of the other. If this is so, then it would likewise be

difficult to account for how the stimuli is thought to have strong autobiographical significance for the individual, since, on the standard taxonomies, non-declarative memory is necessarily non-autobiographical memory. On my hypothesis, on the other hand, this can be accounted for by considering that the affective memorial states once *just were* autobiographical episodic memories and were once associated with declarative content. The Somatic Hypothesis also had difficulty explaining delayed onset PTSD and had to presume that some sub-cortically mediated fear-states exist as inactive within the body. This hypothesis makes no such supposition: in delayed onset PTSD, those experiences exist in memory, as per usual.

The Memorial Hypothesis, likewise, had some difficulty with its external consistency. In particular, it has difficulty explaining cases of PTSD with total amnesia. On this account, however, total amnesia presents no problem. This hypothesis supposes that PTSD is essentially disorderly Rilkean memory — therefore, one could have PTSD comprised entirely of disorderly Rilkean memory without any corresponding autobiographical episodic memory remaining of those events implicated in their formation. This case of PTSD would be entirely bodily, and it would be appropriate to describe the situation as "the body keep[ing] the score." Moreover, since intentional breakdown is a type of amnesiatic process (phenomenologically, it might also appear as a disassociate process), this hypothesis also preserves the clinical insight that genuine retrograde amnesia occurs in cases of PTSD, a fact that the Memorial Hypothesis indicates is only apparent.

This hypothesis, then, not only can overcome the pitfalls observed with respect to its competing hypotheses, but it would also predict their occurrences, given how it understands what constitutes PTSD. I'll say, finally, that this hypothesis has several additional virtues. First, it is simpler than the two hypotheses presented. Both the Somatic Hypothesis and the Memorial Hypothesis suppose that some of the symptoms of PTSD are direct, and others are indirect. This hypothesis, by contrast, supposes that all of the symptoms are direct: traumatic Rilkean memory implies both fear and memory, the dominant attributes of PTSD. Additionally, to the extent that its competitors could explain why our current best treatments have been effective (or ineffective, as the case may be), it borrows its explanation from them and predicts,

likewise, that they would only be partially effective for addressing the disorder.

Objections and Replies

A Rilkean Memory Hypothesis, however, may be objectionable. Before discussing its practical implications, I would first like to respond to two potential objections. The first objection is that a Rilkean Memory Hypothesis of PTSD is too ontologically radical; it requires us to accept a kind of memory that would require that we drastically alter our metaphysics of memory. In this way, it lacks the virtue of theoretical conservationism (see Keas, 2018, for more on theoretical virtues and vices).

I have two replies to this first worry. First, there is only a possible case to be made against this proposal that it lacks global theoretical conservativism, not local conservativism. By global theoretical conservativism, I mean conservative with respect to the broader domain of psychology in general, as well as the various core concepts comprising the discipline. And by local conservativism, I mean with respect to the clinical research literature on PTSD, specifically. It is certainly locally conservative. I have already demonstrated that it is conservative with respect to the Somatic Hypothesis and the Memorial Hypothesis. I'll say briefly that it is also conservative with respect to other popular theories of PTSD: Psychoanalytic, Behavioral, and Affective Hypotheses of the disorder.

Psychoanalytic Hypotheses of PTSD generally suppose that processes of repression are involved in the etiology and maintenance of the disorder (for more information on psychoanalytic theories of PTSD, see Freud, 1917; Ferenczi et al., 1921; Wastell, 2006, pp. 5-9). As Rowlands (2015, p. 149) notes, on its surface, intentional breakdown (described above) resembles processes of repression. However, whereas the latter are necessarily defensive mechanisms, intentional breakdown need not be (as in my examples above). Psychoanalytic theories also generally presume that the person suffering from PTSD needs to "work through" the memories of the disorder and experience an affective "catharsis" in order to reach full recovery (Durand & Barlow, 2016, p. 149). A Rilkean memory hypothesis, by contrast, makes no such assumption, since, as indicated, in certain cases of PTSD, there may be no declarative memory remaining of the trauma at all towards which one can cathect. According to some

behavioral analysts, PTSD is primarily a disorder of extinction learning; that is, the person has over-generalized his / her fear response to innocuous stimuli, and a failure of extinction has taken place, and continues to take place, probably as a result of dysfunctional hippocampal activity (VanElzakker et al., 2014). Like this hypothesis, a Rilkean Memory Hypothesis supposes that there is an over-active fear response. In my examples above, I described the affective Rilkean memory triggered by the smell of perfume. As a general rule, Rilkean Memories are responsive to the environment – at least as much as any non-intentional affective state can be. Importantly, however, disorderly Rilkean memory need not always be response-dependent. As a form of memory (or so, it is contented), it may be susceptible to the same kind of memory error mentioned earlier — a kind of overly-frequent occurring. Moreover, processes of generalization are known to depend on affective amygdalar activity, which corresponds, roughly, to the neurological systems implicated in disordered Rilkean memory. Those with PTSD show an unusual startle response and sensitivity to pain (analgesia), which may be difficult to explain on a behavioral explanation on its own (van der Kolk, 2004; Pitman et al., 1990). Finally, Affective Hypotheses of PTSD presume that PTSD is primarily the disorder of an over-active fear response. See Brown et al. (2018) for one such negative affect hypothesis. See also Wastell (2006) for a fairly sophisticated emotion-based theory of PTSD. It is related intimately to anxiety and depressive-like states. My proposal, likewise, supposes that many cases of PTSD involve an abundance of negative affective states; but these affective states are special, insofar as they were once associated with autobiographically significant memorial content.

My proposal, therefore, is very locally conservative. The objection, on the other hand, that it is not globally conservative has greater merit. The concept of Rilkean memory is new — at present, it is has generated discussion for a short time. However, despite being new, some theorists from other fields have given compelling arguments to the effect that it may be useful. Recently, A. Layva (2018) has argued that Rilkean memories may be constitutive of our best explanation for how expert sport-specific knowledge may work. He relies in his account especially on behavioral Rilkean memory. Additionally, L. Barrett (2017) has recently suggested that the concept of

Rilkean memory may be useful as a fourth term in behavior analysis. Generally, three central terms are used in behavior analyses: stimulus, response, and consequence. Barrett (2017) suggests that this model might be improved by making room for Rilkean memories into the equation. Barrett (2017):

> The concept of Rilkean memories seems particularly useful in... [the] new [suggested] quadruple because there is no danger of [the behaviorist] sliding into cognitivism. Rilkean memories are not memories of facts or episodes. They are caused by such things, but there are not about them. Indeed, they are not about anything at all; they have no content. Rilkean memories are thus inherently resistant to cognitivist reinterpretation: they lack all characteristics that would allow them to be transformed into intentional propositions. They remain entirely historical and... are inscribed on the organism's body itself (p. 98).

The point of mentioning these two applications is to say that, while, at present, the proposal conflicts with our standard scientific ontolog(ies), very soon, it might not, as Rilkean memory continues to gain attention and appreciation in diverse fields. (For further application of Rilkean memory to philosophical topics (epistemic injustice), see Piñeiro, 2023, and Woolwine, 2023).

The second objection to the Rilkean Memory Hypothesis might be issued by a reader of Rowlands. I have taken the concept of Rilkean memory and applied it within a theory of psychopathology. But one might think that Rowlands would wish to resist this application. (Thanks to an anonymous reviewer for suggesting this objection.) He says explicitly on several occasions that he does not take Rilkean memories or intentional breakdown to be at all pathological. Instead, they are a normal part of healthy psychological life. His apparent resistance to Rilkean memory as being pathological is most evident in his discussion contrasting Rilkean memories, on the one hand, and Freudian (or Neo-Freudian) repressed memories, on the other. For example:

> First, repressed memories derive from unpleasant experiences. Rilkean memories can derive from experiences that are pleasant, unpleasant or neutral. Second, Freudian repressed memories result from the operations of a psychic defense mechanism, whose function is to transform memories of traumatic experiences into another form,

> in which they will do less immediate harm (although the long-term consequences, of course, may not be so good). Rilkean memories are not, in general, the product of the operations of a psychic defense mechanism. Third, the 'vertical' imagery embodied in standard Freudian theory does not really fit very well with the more 'horizontal' – embodied and embedded – conception implicated in the idea of Rilkean memory. In standard Freudian accounts, repressed memories bubble away beneath the surface of the conscious mind. Rilkean memories don't bubble beneath the surface of anything. They are spread out, often incorporating the entirety of a person's body and embedded in her environment (Rowlands, 2015, p. 149).

On another occasion, he contrasts Rilkean memory with Casey's (1980) concept of "traumatic bodily memory." Casey writes:

> Each time my tongue passes over my right lower molar tooth these days, distinct memories of being in a dentist's chair and, somewhat less frequently, of chewing on a hard kernel of popcorn somewhat earlier, are elicited. In particular, I recall biting down on the kernel and feeling immediately afterward parts of something very hard lying loose in my mouth: at first I wasn't sure whether they were bits of kernel or bits of tooth. I also remember, from a period of about a month later, being in the dentist's chair and experiencing acute pain as my dentists drilled deeply into the broken tooth as part of the procedure of crowning it (p. 154).

And Rowlands (2015) replies, "This is a perfectly legitimate sense of embodied remembering, but it is not my sense" (p. 6). Hence, a critic may say, even if my proposal may be an interesting hypothesis, it is not a genuine "Rilkean memory" explanation of PTSD, as I have advertised it.

But this objection requires only a brief reply. First, Rowlands clarifies his position with respect to Rilkean memories contrasted with repressed memories in his later work, writing:

> Repressed memories are postulated to explain certain types of pathology. The explanatory work of Rilkean memories is, I shall argue, *far broader*: Rilkean memories are postulated, and enlisted, in the attempt to explain what makes us the persons we are—*whether pathological or healthy*, whether good or evil" (2017, p. 68 – italics added for emphasis).

In other words, he does not protest against Rilkean memory being put to work in an

explanation of pathology; he simply insists that psychopathology is not the *only* area where they may be fruitfully applied. Rilkean memories are not essentially pathological, but they may still be accidentally so. Second, it is not because Casey has applied "bodily memory" to trauma that Rowlands has distanced Rilkean memory from it. Rather, it is because Casey's notion of bodily memory details merely a causal relation between memory and affect. The experience of pain triggers the memory of the dentist's chair. Rowlands, on the other hand, seeks to develop a more integral concept, wherein the affective state bears a constitutive relation to the memorial act. The experience of pain just is the memorial act of pain on the dentist's chair, now stripped of its content and transformed into affect. The fact that Casey's example involves trauma is simply irrelevant in this context. Consequently, the critic's objection would seem on both counts to be based on a superficial reading of Rowlands. Even so, I must stress again that I have not only applied Rowlands's concept here but have also further developed it by allowing that intentional breakdown involved in the formation of Rilkean memory may be more or less complete. Episodic memory of an event is after all complex; hence, some parts of such a memory may retain their content whilst other parts become Rilkean.

Rilkean Memory Treatment Plans

I argue, then, that a Rilkean Memory Hypothesis of PTSD is plausible, despite asking of us to accept a radical new concept — Rilkean memory. If this hypothesis is plausible, it is therefore plausible to suppose that our failure to treat PTSD has been, at least in part, a problem in principle, and only consequently also a problem in practice. I would like to conclude this essay by addressing directly what I have called "the prime imperative in the clinical literature on PTSD": how best to help those suffering from it.

My hypothesis proposes that both over-active hippocampal activity and over-active amygdalar activity are implicated in the maintaining conditions of the disorder. For this reason, our methods that attempt to interfere with over-active hippocampal activity — such as over-active processes of consolidation and reconsolidation — ought to be conserved and continued to be employed in treatment plans for PTSD. Anyway

we can interfere with hippocampal activity via processes of re-conditioning and cognitive intervention are useful to this end.

However, additionally, we ought in whatever way reasonable also attempt to employ somatic therapies in our treatment plans of PTSD. After all, according to my proposal, PTSD is partly a condition of an over-abundance of sub-cortically mediated states changed into "blood" (i.e. rogue affective Rilkean memories). B. A. van der Kolk (2006) has suggested a similar point. He argues that "interoceptive, body-oriented therapies," such as "sensory awareness, Feldenkrais, Rolfing, the F. M. Alexander Technique, body-mind entering, somatic experiencing, Pesso-Boyden psychotherapy, Rubenfeld synergy, Hokomi," yoga, and somatic massage therapy

> … can directly confront a core clinical issue in PTSD... If past experience is embodied in current physiological states and action tendencies and the trauma is reenacted in breath, gestures, sensory perceptions, movement, emotion and thought, therapy may be most effective if it facilitates self-awareness and self-regulation. Once patients become aware of their sensations and action tendencies they can set about discovering new ways of orienting themselves in their surroundings and exploring novel ways of engaging with potential sources of mastery and pleasure (pp. 282 & 289).

There is some evidence to suggest that this may be correct. In a study of body-oriented therapies as a treatment plan for sexual abuse recovery, C. Price (2007) has found some effect in reducing dissociation (a common element in PTSD and in autobiographical memory generally) (see McCleery & Harvey (2004) and Spiegel & Cardena (1990) for more on the relation between dissociation and PTSD). Additionally, Price et al. (2007) have found some effect in using mindfulness therapies to combat the disorder. Another reason worth further researching this type of therapy is because those with PTSD also often develop alexithymia — literally "the absence of words for emotion" (Wastell, 2005, pp. 36-38). As indicated, this could be associated with unusually underactive neuronal activity in Broca's area. So far, I have said nothing about the relation between intentional breakdown and Broca's area, but it does not seem unreasonable to suggest that with the deterioration of symbolic memorial content, Broca's area may be adversely affected.

Given all that I have said, I believe it is imperative that somatic forms of

psychotherapy for PTSD be further explored and used alongside trauma focused cognitive behavioral therapy, cognitive restructuring therapy, and / or brief psychodynamic therapy, where applicable. This is, of course, on the supposition that our failure to properly treat those suffering from PTSD thus far has been a problem in principle and not merely a problem in practice. But, if all that I have said is reasonable, then so too is it reasonable that these methods are worth further study.

References

Barrett, L. (2017). Reinforcing Rilkean memories. *The Behavior Analyst, 40*, 95-99.

Berntsen, D., & Rubin, D. C. (2013). Involuntary memories and dissociative amnesia: Assessing key assumptions in posttraumatic stress disorder research. *Clinical Psychological Science, 2*(2), 174-186.

Bradley, R., Greene, J., Russ, E., et al. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *American Journal of Psychiatry, 162*, 214-227.

Bremner, J. D. (1997). Magnetic resonance imaging based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse: A preliminary report. *Biological Psychiatry, 41*, 23-32.

Breslau, N. (2012). Epidemiology of posttraumatic stress disorder in adults. In J. G. Beck & D. M. Sloan (Eds.), *The Oxford handbook of traumatic stress disorder* (pp. 84-97).

Brewin, C. R., Dalgleish, T., & Joseph, S. (1996). A dual representation theory of posttraumatic stress disorder. *Psychological Review, 103*, 670-690.

Brown, W. J., Dewey, D., Bunnell, B. E., et al. (2018). A critical review of negative affect and the application of CBT for PTSD. *Trauma, Violence, & Abuse, 19*(2), 176-194.

Bryant, R. A. (2010). The complexity of complex PTSD. *American Journal of Psychiatry, 167*, 879-881.

Bryant, R. A., Friedman, M. J., Spiegel, D., Ursano, R., & Strain, J. (2011). A review of acute stress disorder in DSM-5. *Depression and Anxiety, 28*(9), 802-817.

Casey, E. (1980). Remembering: A phenomenological study. University of Indiana Press.

Cukor, J., Olden, M., Lee, F., & Difede, J. (2010). Evidence-based treatments for PTSD: New directions and special challenges. *Annals of the New York Academy of Sciences, 1208*, 82-89.

de Loos, W. S. (1990). Psychosomatic manifestations of chronic posttraumatic stress disorder. In M. E. Wolf & A. D. Mosnaim (Eds.), *Posttraumatic stress disorder:*

Etiology, phenomenology, and treatment (pp. 95-104).

de Vries, G. J., & Olff, M. (2009). The lifetime prevalence of posttraumatic events and posttraumatic stress disorder in the Netherlands. *Journal of Traumatic Stress, 22*(4), 259-267.

Desmedt, A., Marighetto, A., & Piazza, P. V. (2015). Abnormal fear memory as a model for posttraumatic stress disorder. *Biological Psychiatry, 78*, 290-297.

Devilly, G. J., & Cotton. P. (2003). Psychological debriefing and the workplace: Defining a concept, controversies, and guidelines for intervention. *Australian Psychologist, 38*(2), 144-150.

Dohrenwend, B. P., Turner, J. B., & Turse, N. A. (2006). The psychological risks of Vietnam for U.S. veterans: A revisit with new data and methods. *Science, 313*, 979-982.

Durand, V. M., & Barlow, D. H. (2016). *Essentials of abnormal psychology*. Cengage Learning.

Ferenciz, S., Abraham, K., & Freud, S. (1921). *Psychoanalysis and the war neuroses*. Psychoanalytic Press.

Freud, S. (1917). *Mourning and melancholia*. J. Strachey (Ed.). Hogarth Press and Institute for Psychoanalysis.

Giustino, T. F., Fitzgerald, P. J., & Maren, S. (2016). Revisiting propranolol and PTSD: Memory erasure or extinction enhancement? *Neurobiology of Learning and Memory, 130*, 26-33.

Grady, M. M. (2010). *Second messenger — Cognitive behavior therapy (CBT) for PTSD: An excerpt from* Stahl's illustrated anxiety, stress, and PTSD. Neuroscience Education Institute.

Granja, C., Gomes, E., Amaro, A., Ribeiro, O., Jones, C., Carneiro, A., & Costa-Pereira, A. (2008). Understanding posttraumatic stress disorder-related symptims after critical care: The early illness amnesia hypothesis. *Critical Care Medicine, 36*(10), 2801-2809.

Gray, J. (1982). *The neurophysiology of anxiety: An inquiry into the functions of the septo-hippocampal system*. Oxford University Press.

Gurvits, T., Shenton, M., Hokama, H., et al. (1998). Magnetic resonance imaging (MRI)

study of hippocampal volume in chronic combat related posttraumatic stress disorder. *Biological Psychiatry, 40*, 1091-1099.

Hendin, H. (2017). Psychodynamic treatment for combat veterans with PTSD at risk for suicide. *Psychodynamic Psychiatry, 45*(2), 217-236.

Hidalgo, R. B., & Davidson, J. R. (2000). Posttraumatic stress disorder: Epidemiology and health-related considerations. *Journal of Clinical Psychology, 61*(7), 5-13.

Hui, K., & Fisher, C. E. (2015). The ethics of memory modification. *Journal of Medical Ethics, 41*, 515-520.

Janet, P. (1889). *L'automatise psychologique*. Alcan Publishing.

Keas, M. N. (2018). Systematizing the theoretical virtues. *Synthese, 195*(6), 2761-2793.

Kessler, R. C., Berglund, P., WatTaim, C., et al. (2008). The national comorbidity survey replication (NCS-R): Cornerstone in improving mental health and mental health care in the United States. *The WHO World Health Surveys: Global Perspectives on Epidemiology of Mental Disorders*, 165-210.

Kessler, R. C., Chiu, W. T., Jin, R., et al. (2006). The epidemiology of panic attacks, panic disorder, and agoraphobia in the national comorbidity survey replication. *Archives of General Psychiatry, 63*(4), 415-424.

Kitayama, N., Vaccarino, V., Kutner, M., Weiss, P, & Bremner, J. D. (2005). Magnetic resonance imaging (MRI) measurement of hippocampal volume in posttraumatic stress disorder: A meta-analysis. *Journal of Affect Disorders, 88*, 79-86.

Kredlow, M. A., & Otto, M. W. (2014). Interference with the reconsolidation of trauma-related memories in adults. *Depression and Anxiety, 32*, 32-37.

Krikorian, R., & Layton, B. S. (1998). Implicit memory in posttraumatic stress disorder with amnesia for the traumatic event. *Journal of Neuropsychiatry and Clinical Neurosciences, 10*(3), 359-362.

Krupnick, J. L. (2002). Brief psychodynamic treatment of PTSD. *Journal of Clinical Psychology, 58*(8), 919-932.

Leyva, A. (2018). Embodied Rilkean sport-specific knowledge. *Journal of Philosophy of Sport, 45*(2), 128-143.

Loftus, E. F. (1997). Creating false memories. *Scientific American, 277*(3), 70-75.

McCleery, J. M., & Harvey, A. G. (2004). Integration of psychological and biological approaches to trauma memory: Implications for pharmacological prevention of PTSD. *Journal of Traumatic Stress, 17*(6), 485-496.

Michaelian, K., & Sutton, J. (2017). Memory. In E. N. Zalta (Ed.), *The Stanford Encyclopedia of Philosophy*.

Oulton, J. M., Strange, D., Nixon, R. D. V., & Takarangi, M. K. T. (2018). Imagining trauma: Memory amplification and the role of elaborative cognitions. *Journal of Therapy and Experimental Psychiatry, 60*, 78-86.

Phelps, E. A. (2004). Human emotion and memory: Interactions of the amygdala and hippocampal complex. *Current Opinions in Neurobiology, 14*, 198-202.

Piñeiro, J. M. (2023). Rilkean memory, epistemic injustice, and epistemic violence. *Southwest Philosophy Review, 39*(1), 269-279.

Pitman, R. K., Orr, S. P, van der Kolk, B. A., et al. (1990). Analgesia: A new dependent variable for the biological study of PTSD. In M. E. Wolf & A. D. Mosnaim (Eds.), *Posttraumatic stress disorder: Etiology, phenomenology, and treatment* (pp. 140-148).

Pitman, R. K., Sanders, K. M., Zusman, R. M., et al. (2002). Pilot study of secondary prevention of posttraumatic stress disorder with propranolol. *Biological Psychiatry, 51*(2), 189-192.

Price, C. (2007). Dissociation reduction in body therapy during sexual abuse recovery. *Contemporary Therapies in Clinical Practice, 13*(2), 116-128.

Price, C. J., McBride, B., Hylere, L., & Kivlahan, D. R. (2007). Mindful awareness in body-oriented therapy for female veterans with posttraumatic stress disorder taking prescription analgesics for chronic pain: A feasibility study. *Alternative Therapies in Health and Medicine, 13*(6), 32-40.

Prinz, J. (2004). *Gut reactions: A perceptual theory of emotions*. Oxford University Press.

Rilke, R. M. (1985). *The notebooks of Malte Laurids Brigge*. S. Mitchell (Trans.). Vintage Books.

Rowlands, M. (2015). Rilkean memory. *The Southern Journal of Philosophy, 53*, 141-154.

Rowlands, M. (2017). *Memory and the self: Phenomenology, science and autobiographical memory*. Oxford University Press.

Schacter, D. L. (1999). The seven sins of memory: Insights from psychology and cognitive neuroscience. *American Psychologist, 54*, 182-203.

Shaw, J. (2016). *The memory illusion: Remembering, forgetting, and the science of false memory*. Random House.

Shedler, J. (2010). The efficacy of psychodynamic therapy. *American Psychological Association, 65*(2), 98-109.

Smith, T. C., Ryan, M. A. K., Wingard, D. L., et al. (2008). New onset and persistent symptoms of post-traumatic stress disorder self reported after deployment and combat exposures: Prospective population based U.S. military cohort study. *British Medical Journal, 336*(7640), 366-371.

Soeter, M., & Kindt, M. (2010). Dissociating response systems: Erasing fear from memory. *Neurobiology of Learning and Memory, 94*, 30-41.

Spiegel, D., & Cardena, E. (1990). Dissociative mechanisms in posttraumatic stress disorder. In M. E. Wolf & A. D. Mosnaim (Eds.)*, Posttraumatic stress disorder: Etiology, phenomenology, and treatment* (pp. 22-35).

Stahl, S. M., & Grady, M. M. (2010). *Stahl's illustrated anxiety, stress, and PTSD*. Cambridge University Press.

Sutherland, K., & Bryant, R. A. (2008). Autobiographical memory and the self-memory system in posttraumatic stress disorder. *Journal of Anxiety Disorders, 22*, 555-560.

Tyron, W. W., & McKay, D. (2009). Memory modification as an outcome variable in anxiety disorder treatment. *Journal of Anxiety Disorders, 23*, 546-556.

van der Kolk, B. A. (1994). The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress. *Harvard Review of Psychiatry, 1*(5), 253-265.

van der Kolk, B. A. (2004). Psychobiology of posttraumatic stress disorder. In J. Panksepp (Ed), *Textbook of biological psychiatry*.

van der Kolk, B. A. (2006). Clinical implications of neuroscience research in PTSD. *Annals of the New York Academy of Sciences, 1071*, 277-293.

VanElzakker, M. B., Dahlgren, M. K., Davis, F. C., et al. (2014). From Pavlov to PTSD: The extinction of conditioned fear in rodents, humans, and in anxiety disorders. *Neurology of Learning and Memory, 133*, 3-18.

Vasterling, J. J., & Brewin, C. R. (Eds.). (2005). Neuropsychology of PTSD: Biological, cognitive, and clinical perspectives. Guilford Press.

Walsh, K., Danielson, C. K., McCauley, J. L., et al. (2012). National prevalence of posttraumatic stress disorder among sexually revictimized adolescent, college, and adult household-residing women. *Archives of General Psychiatry, 69*(9), 935-942.

Wastell, C. (2005). *Understanding trauma and emotion: Dealing with trauma using an emotion-focused approach*. Open University Press.

Wolfe, J., & Keane, T. M. (1990). Diagnostic validity of posttraumatic stress disorder. In M. E. Wolf & A. D. Mosnaim (Eds.), *Posttraumatic stress disorder: Etiology, phenomenology, and treatment* (pp. 48-63).

Woolwine, S. (2023). Comments on Josué Piñeiro "Rilkean memory and testimonial injustice." *Southwest Philosophy Review, 39*(2), 109-111.

Yovell, Y., Bannett, Y., Shalev, A. Y. (2003). Amnesia for traumatic events among recent survivors: A pilot study. *CNS Perspectives, 8*(9), 676-685.

Yule, W. (2001). Posttraumatic stress disorder in the general population and in children. *Journal of Clinical Psychology, 62*(17), 23-28.